The effects of social support, inclusion in social networks and alcohol consumption on depressive symptoms in the adult urban population of the UK and of Central and Eastern Europe.

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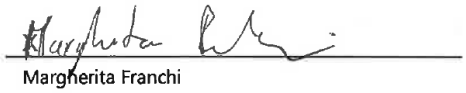
I, Margherita Franchi, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.



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Abstract

Background

Depression is the most common mental disorder and the leading cause of disability worldwide. Three main risk factors for depression are inclusion in social networks, social support and alcohol consumption. Although research has focused on the association between each risk factor and depression, virtually no study has yet attempted to investigate how they interact in affecting the risk of depression. This thesis aims to fill this gap in our understanding of risk factors for depression.

Methods

The association between social support, inclusion in social networks, alcohol consumption and depressive symptoms was investigated in three ways. First cross-sectionally, using data from the Whitehall II and HAPIEE cohorts to compare the patterns of association observed in the UK with those observed in Central and Eastern Europe. Then longitudinally, using data from four phases of the Whitehall II cohort to assess the magnitude and duration of the association. Finally, through growth curve models aimed at modelling patterns of covariation between social support, inclusion in social networks, alcohol consumption and depressive symptoms through time.

Results

In all four countries odds of depressive symptoms were higher among individuals who did not have or never saw their friends and relatives. In Central and Eastern Europe high odds of depressive symptoms were found among both abstainers and heavy drinkers. In the UK, negative social support, social isolation, daily drinking and abstention were predictive of high odds of depressive symptoms for as long as nineteen years. Participants who suffered from intense depressive symptoms, were socially isolated and received inadequate support were more likely to engage in frequent drinking than their more socially connected counterparts. This suggests that interventions aimed at improving social connections could prove particularly effective in preventing depressive symptoms but also at containing hazardous alcohol consumption.

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Contents

Tables and Figures

1. Introduction

1

There had been as many plagues in the world as there had been wars, yet plagues and wars always find people equally unprepared.

-Albert Camus, The Plague-

Violent outbursts of infectious diseases feature in all written records since the beginning of history; and before writing was invented, traces of tuberculosis and pox have been found on the bones of the first farmers. With the beginning of agriculture and the introduction of stocks living in close proximity with people, many animal diseases evolved a human strand and took their toll on farmers. These zoonoses were often described in ancient texts as divine punishments, from the plague with which God struck down every first born in Egypt in the Old Testament, to the outburst of bubonic plague that gods sent on to Athens to determine its loss of the Peloponnesian war. By medieval times, epidemics were a part of everyday life and still caught people completely unprepared. In the Middle-ages, thanks the enlargements of cities, a widespread road system across Europe and increased movements of goods and people, epidemics could spread faster, travel further and kill more and more people in shorter periods of time. Thus the Black Death, the terrible bubonic plague outburst of 1348-1350, managed to kill between 45% and 50% of the European population in merely two years (Dobson 2008).

Those who survived the Black Death were then immune to it, but their grand-grandchildren did not seem to be immune to syphilis when it was brought into Europe from the newly discovered Americas a century and a half later. The first outburst of syphilis forced the French army to abandon their siege of Naples in 1498 and meant that disease was then called by all but the French 'the French pox'. Being a venereal disease syphilis did not spread as fast as bubonic plague, but humanity could not find a cure for it until the discovery of penicillin in 1928.

Penicillin was indeed the cure to many diseases including tuberculosis, which had been decimating the working classes for a century causing 25% of all deaths in Europe. Penicillin was also the first medicine which directly targeted a bacterium with the intent of killing it, marking the beginning of modern medicine. Since the discovery of penicillin, modern medicine has managed to defeat most infectious diseases, containing their spread, curing their symptoms and in some cases eradicating them entirely. And thus, with the exception of HIV/AIDS which remains the number one killer in the world, in the past fifty years or so, more people die of non-communicable diseases than of communicable diseases, with heart disease and cancer topping the list of non-communicable killers (Murray and Lopez 1997).

So far I have been talking about how deadly diseases are, and for years that was the main focus of attention of international public health community as well. Diseases were deemed more or less dangerous and accordingly targeted, according to their rates of mortality alone, until in 1993 a paper from the World Bank drew attention to the relative

burden associated with disease morbidity as well as mortality (Berkley, Bobadilla et al. 1993). Since then, burden is the primary measure of a disease's impact on the population and it is quantified through three main measures: years of life lost (YLL) due to premature mortality, years lost due to disability (YLD) for people living with the health condition or its consequences and disability-adjusted life years (DALY) which are the sum of the previous two measures (WHO, website) (Murray and Lopez 1997). Although technically DALYs encompass YLDs it is not infrequent in the literature to find the two measures used alternatively. This shift of attention from mortality alone to burden of morbidity allowed to highlight the importance of targeting some diseases which do not have a high mortality but do create disability. Among these diseases with low mortality but high burden, are mental and substance abuse disorders.

Mental and substance abuse disorders are a group of disorders that affect the brain such as depressive disorders, schizophrenia, anxiety disorders, bipolar disorder, eating disorders, childhood behavioural disorders, pervasive developmental disorders, idiopathic intellectual disability, alcohol use disorder, drug use disorder and other mental disorders. All of which had been largely neglected until 1993 as their mortality is really low, and perhaps most importantly because they are associated with great stigma (Witheford, Dagenhardt et al. 2013). However, mental and substance use disorders are not to be underestimated as they exert a great burden of disability worldwide (Witheford, Dagenhardt et al. 2013). In fact, in their recent measurement of the global burden of disease attributable to mental and substance use disorders Whiteford and colleagues (2013) found that mental and substance use disorders accounted for 7.4% of

all DALYs, and 22.9% of all YLDs worldwide, representing the leading cause of YLDs worldwide.

Among mental and substance use disorders the most common and most burdensome are depressive disorders, which account for 40.5% of DALYs caused by mental and substance use disorders (Whiteford, Degenhardt et al. 2013). Depressive disorders are known under a variety of names including: unipolar major depression and major depressive disorder, and are a major public health concern as they represent the leading cause of disability worldwide accounting for 13.0% of YLDs in women and 8.3% of YLDs in men worldwide (Ustun, Ayuso-Mateos et al. 2004). The burden of depressive disorders is a concern also because it has been steadily growing from accounting for 3.7% of all DALYs in 1990, to accounting to 4.3% of DALYs worldwide in 2000 (Ferrari, Somerville et al. 2013).

However, despite the great public health concern they pose the epidemiology of depressive disorders is still confusing as there is no single measure of prevalence of depressive disorders and data is missing or patchy from many areas of the world. Two main global projects have been collecting data on prevalence and incidence of depressive disorders, as well as many literature reviews being conducted (Ferrari, Somerville et al. 2013). The two global projects are the Global Burden of Disease (GBD) 2010 project which aims at collecting data on the burden of a number of diseases; and the World Mental Health Survey, launched by the WHO which has been collecting data

specifically on mental disorders through population surveys in 28 countries using the WHO Composite International Diagnostic Interview (Bromet, Andrade et al. 2011).

The GBD 2010 required epidemiological data on occurrence and course of illness of major depressive disorders in order to quantify their morbidity. The study reported that although there is a vast literature on the different epidemiological parameters of depressive disorders this still needs to be systematically summarised at a global level. If such a systematic summarisation would have both clinical and public health applications as well as informing the GBD 2010 study, problems were encountered due to heterogeneity both in the epidemiological estimates resulting from true differences in the epidemiology of major depressive disorders, and in the methodology used to capture data (Skapinakis and Lewis 2001; Weich and Araya 2004; Bromet, Andrade et al. 2011; Ferrari, Somerville et al. 2013). The same heterogeneity has been reported by a number of literature reviews on prevalence and incidence of major depressive disorders which concluded that it is indeed difficult to explain regional variation in prevalence of depressive disorders given the variation in methodologies used (Weich and Araya 2004). In this regard the efforts of the World Mental Health Survey offers systematic data collected with the same methodology if not for the world, at least for 28 countries.

Using data from the Mental Health Survey, Bromet et al (2011) report both lifetime and 12-months prevalence of depressive disorders in 28 countries, divided between low and high income. 12-months prevalence varied from 2.2% in Japan to 10.4% in Sau Paulo, Brazil; while lifetime prevalence varied from 6.5% in Schenzen, China to 21.0% in France

(Bromet, Andrade et al. 2011). In Europe alone, Bromet and colleagues (2011) found a great oscillation from 9.9% life time prevalence in Italy and Germany, to 10.4% in Spain, to 21.0% in France. Similarly, 12-months prevalence of depression varied from 3.0% in Italy and Germany, to 4.0% in Spain, to 5.9% in France.

Country specific variation in incidence and prevalence of depression in Europe was also reported in a review of existing studies by Paykel and colleagues (2005). In their review, Paykel and colleagues found that 12-month prevalence of depression in countries of Western Europe averaged around 5%, with higher prevalence among women, middle aged individuals and more disadvantaged social groups; they also found a two-fold variation in 12-months prevalence of depression across countries which was attributed to methodological variation in the studies reviewed (Paykel, Brugha et al. 2005). Paykel and colleagues (2005) concluded highlighting a lack of data on lifetime prevalence of depression in Europe and on depression in general from countries of Central and Eastern Europe.

Coincidentally, Europe is also the region of the world spending the most on medicines for mental health (Saxena 2011) that women are more affected by depression than men, and that low social economic circumstances are a risk strong factor for depression (Lorant, Eaton et al. 2003; Marmot and Brunner 2005; Nicholson, Pikhart et al. 2008). For this reason, in Europe, mental health concerns regarding depression have been raising since the economic crisis that started in 2007. As the economy regressed, people economic circumstances worsened and unemployment raised, putting more and more

people at a higher risk of depression (Wahlbeck, Anderson et al. 2011) . This was worsened by the fact that most European governments reacted to the crisis with cuts in public expenditure which affected most areas of the public sector, including public health (Wahlbeck, Anderson et al. 2011). This has the potential to create a vicious cycle in which unemployment and impoverishment contribute to increase the incidence of depression in Europe, and depression contribute to diminish the productivity of individuals (Wahlbeck, Anderson et al. 2011), as depression has been shown to be the second most common disorder on the work place with incidence as high as 4% to 7% in Europe (Wahlbeck and Makinen 2008).

The European Union has responded to this risk by implementing a vast number of policies and legislations targeting mental health in the member countries. According to the WHO Mental Health Atlas 2011, in Europe the median proportion of health budget, allocated to mental health is 5%, which is relatively high compared to the 3.75% allocated to mental health in the Eastern Mediterranean and 1.53% in the Americas (Saxena 2011). The WHO Mental Health Atlas 2011 also highlighted how Europe is consistently the region of the world with better access and more facilities for mental health patients (Saxena 2011).

The European Union has also been calling for more research and attention on the matter of public health, through a number of reports highlighting the pressing need for prevention of mental disorders. The most recent consensus paper on mental health published by the Directorate-General for Health & Consumers started by reporting that the annual cost of depression in Europe in 2004 was 118 billion euros, or 250 euros per inhabitant, largely amenable to early retirement, days of sickness absence from work and suicide (Wahlbeck and Makinen 2008). For this reason, in 2008 the Slovenian Presidency of the European Union called for a meeting devoted to design a plan of action regarding mental health and wellbeing, the result was the European Pact on Mental Health.

The European Pact on Mental Health is based on several papers researching the cost and burden of mental health in countries of the European Union, which reported that mental health issues have a great impact on the economy as mental health problem account for 25% of all new disability benefit cases in Europe (McCollam, O'Sullivan et al. 2008). And in particular a paper by Sobocki and colleagues (2006) highlighted the enormous cost of depression in Europe. According to Sobocki and colleagues (2006) the direct annual cost of depression amounts to 41 billion euros, of which 22 billion are spent on outpatient care, 9 billion on drugs and 10 billion on hospitalization. In addition to this, there are the 76 billion euros estimated to be the cost of morbidity and mortality of depression. These estimates make depression the most expensive brain disorder in Europe, accounting for 33% of the total cost; they also mean that the cost of depression corresponds to 1% of the total European economy (Sobocki, Jonosson et al. 2006). For these reasons the

European Pact on Mental Health stated prevention of depression and suicide as the number one priority for action in Europe (Vassiliou 2008).

In fact, if preventing the onset of any disease is preferable to having to cure it, prevention of depression in particular is of paramount importance for three main reasons. Firstly, depression is a very debilitating disorder, being the leading cause of years lost to disability worldwide (Witheford, Dagenhardt et al. 2013), and its prevalence keeps growing, affecting more and more people every year (Murray and Lopez 1997; Mathers and Loncar 2006). This leads to the exorbitant and ever growing costs of depression, as high as a staggering annual 117 billion euros in Europe alone (Sobocki, Jonosson et al. 2006), most of which are due to sickness absence from work, early retirement and suicide (Wahlbeck and Makinen 2008). And finally, there is evidence to support the fact that the available drugs for curing depression not only are not effective but could cause further damage in the brain and reduce patients' productivity and impair their recovery (Whitaker 2010).

Therefore, the European Pact on Mental Health (2008) identified prevention of depression and suicide as the first priority for future policies and interventions in mental health. The Pact identified five main areas of intervention: (1) improve the training of professionals and key actors within the social sector of mental health; (2) restrict access to potential means for suicide; (3) take measures to raise mental health awareness in the general public, among health professionals and other relevant sectors; (4) take measures to reduce risk factors for suicide such as excessive drinking, drug abuse and

social exclusion, depression and stress; (5) provide support mechanisms after suicide attempts and for those bereaved by suicide, such as emotional support helplines (Vassiliou 2008).

The effectiveness of interventions for the prevention of depression depends on a thorough understanding of factors affecting individual risk of the disorder. These factors could be either beneficial acting as a protection against depression, or deleterious, increasing the risk of depression. Research has identified a few main factors that negatively influence the risk of depression. Two of these are age and gender, as middle-aged people are known to be at higher risk than both younger and older age groups, and women are known to be at almost double the risk of depression than men (Piccinelli and Wilkinson 2000; Kawachi and Berkman 2001; Bellman, Forster et al. 2003; Kuehener 2003; Goodwin and Gotlib 2004; Zunzunegui, Minicuci et al. 2007; Nicholson, Pikhart et al. 2008; Michel 2009). Other risk factors are stress, loneliness or social isolation, adverse socio-economic circumstances and excessive alcohol consumption (Rodgers, Korten et al. 2000; Rodgers, Korten et al. 2000; Caldwell, Rodgers et al. 2002; Paykel, Brugha et al. 2005; Skogen, Harvey et al. 2009). On the other hand, factors that help protecting from depression are social inclusion and support, physical activity, positive socio-economic circumstances and moderate alcohol consumption (Berkman and Glass 2000; Berkman, Glass et al. 2000; Cohen 2004; Thoits 2011).

Therefore, to help develop effective interventions aimed at preventing depression in Europe, the European Union has commissioned papers that would draft guidelines for tackling all the main risk factors of depression. McCollam et al (2008) have identified three broad groups of actions affecting different sectors, - from the household, to the work place and community environments - aimed at strengthening factors that enhance mental health and at reducing those factors which are detrimental for mental health. These three groups are: first, actions aimed at strengthening individuals and families by increasing emotional resilience through intervention designed at increasing self-esteem and coping skills (McCollam, O'Sullivan et al. 2008); secondly, actions aimed at developing and maintaining strong and safe communities by increasing social support, social inclusion and participation, improving community safety, neighbourhood environments, promoting child care and self-help networks, developing health and social services which support mental health, improving mental health within schools and workplaces (McCollam, O'Sullivan et al. 2008); finally, there are actions aimed at reducing structural barriers to mental health through initiatives to reduce discrimination and inequalities and to promote access to education, meaningful employment, housing, services and support for those who are vulnerable (WHO 2004; McCollam, O'Sullivan et al. 2008).

Specific prevention interventions following these guidelines should effectively tackle most of the main risk factors for depression, with the exclusion of alcohol consumption which is not mentioned in the McCollam et al (2008) report. Furthermore, there is a fourth form of action that is fundamental for developing effective interventions and that

is research and improved communication between researchers and policy makers (Vassiliou 2008). Further research on the risk factors for depression is really important as the associations between depression and its risk factors are often complex and multifaceted. For example, individuals who are socially isolated and lonely are known to be at a higher risk of contracting depression, but at the same time individuals who are already affected by depression are known to isolate themselves (Segrin, Powell et al. 2003; Lasgaard, Goossens et al. 2011), and very little research has been devoted to determining the direction of the association. Similarly, high levels of positive social support are known to protect from depression and improving social support is at the heart of all policy interventions, however negative support or inability to reciprocate support can be a source of stress and a factor increasing the risk of depression (Deelstra, Peeters et al. 2003; Gleason, Mausmi et al. 2008; Thoits 2011).

In the same fashion, alcohol affects the risk of depression differently according to amount consumed, as moderate consumption is known to be beneficial for mental health while excessive consumption is associated with increased risk of depression as well as being a drug use disorder per se in extreme cases (Lipton 1994; Rodgers, Parslow et al. 2007; Boden and Fergusson 2011). In addition, as in the case of social inclusion, individuals who are affected by depression are more likely to drink in excess as a form of self-medication (Boden and Fergusson 2011). Furthermore, alcohol consumption can interact with social inclusion and support affecting depression differently according to the levels of social inclusion of an individual (Rimal and Real 2005).

The intricate nature of the associations between depression and two of its most common risk factors calls for more research aimed at unravelling the pathways linking social inclusion and support, alcohol consumption and depression. Such research could then be the basis for informed policy decisions aimed at preventing depression and suicide in Europe. This thesis sets out precisely to unravel the patterns of association between alcohol consumption, social inclusion and support, and depression through the longitudinal analysis of data coming from the UK. The longitudinal nature of the data and analysis will help establishing temporality and potentially causality of the associations under investigation. In addition, this thesis will present the comparison of patterns of association between alcohol consumption, social inclusion and support, and depression observed in the UK and in Russia, Poland and the Czech Republic. This will be comparison of cross-sectional data, but it is of importance as information on depression from Central and Eastern Europe is somewhat lacking (Paykel, Brugha et al. 2005). Further, this cross-sectional comparison could form the basis for identifying possible culture-specific patterns of association which in turn could be of importance in developing prevention policies aimed at the whole European Union, or specifically designed for particular countries.

2. Background and Literature Review

2

2.1 Introduction

In view of the exorbitant burden and cost of depression on the European population, the European Union has recently called for research to focus primarily on providing evidence for a better understanding of factors that influence this mental disorder in order to develop policies targeted at prevention. Hence this thesis aspires to provide new insights into the relationship between depression and three of its main risk factors: social support, inclusion in social networks and alcohol consumption. These three factors were chosen among the many determinants of depression because the literature regarding their association with the disorder presents gaps and questions still in want of an answer. These questions, as well as the existing evidence focusing on the ties linking social support, inclusion in social networks, alcohol consumption and depression will be reviewed in this chapter.

The first section of this chapter will present depression as a disorder, outlining its symptoms and the process of diagnosis, as well as briefly touching upon the problem of definition of the disorder and introducing the issue of the stigma that still surrounds mental health and the possible repercussions that this stigma has on diagnosis. This section will also touch upon some of the treatments for depression, to then introduce two of the main the epidemiological tools used to detect depressive symptoms in population surveys: the

Centre for Epidemiological Studies depression scale (CES-D) and the General Health Questionnaire (GHQ) depression subscale (section 2.2.iv).

The following section will present the concepts of social support and inclusion in social networks and some of their many definitions, highlighting the different theories that have been put forward over the years to explain how these two factors affect health in general and depressive symptoms in particular. The literature providing the evidence on which these theories are based will then be reviewed, with a focus both on evidence supporting an effect of social support and inclusion in social networks on depressive symptoms and on the evidence suggesting that depression could play a role on the levels of inclusion and support experienced by individuals who suffer from the disorder.

Section 2.4 will then present alcohol consumption, exposing the deleterious effects of excessive consumption on health and introducing the methods of measurement of excessive consumption in the UK. The literature linking alcohol consumption and depressive symptoms will then be reviewed, highlighting the peculiar J or U shaped association between the two. Attention will also be given to the possible role that depression could play in inducing individuals who suffer from the disorder to drink excessively as a form of self-medication.

Section 2.5 will review the literature on the links between depression and all three factors here investigated, with a special focus on the literature suggesting how inclusion in social networks could affect alcohol consumption and, conversely, on how alcohol consumption could affect inclusion in social networks, to finish with a review of the scarce literature investigating the association between social support, inclusion in social networks and alcohol consumption and depressive symptoms.

As one of the aims of this thesis is to compare the patterns of association between social support, inclusion in social networks and alcohol consumption and depressive symptoms observed in different European countries, through analysis of data coming from the Whitehall II cohort in the UK and the Health Alcohol and Psychosocial factors In Eastern Europe (HAPIEE) cohort based in the Czech Republic, Russia and Poland, section 2.6 will review the literature stemming from these two studies, with particular focus on the questions that still have to be answered in these particular populations.

Finally, section 2.7 will summarise the evidence presented in this chapter trying to clearly identify the areas that need further research or clarification, both in the general literature and with special regards to the population of the UK and Russia, Poland and the Czech Republic which are here under study.

2.2 Depression

2.2.1 Historical context

In 1948 the Danish philosopher Soeren Kierkegaard published an essay titled *Sickness Onto Death* in which he stated that: "*when death is the greatest danger, one hopes for life; but when one becomes acquainted with an even more dreadful danger, one hopes for death. So, when the danger is so great that death has become one's hope, despair is the disconsolateness of not being able to die"*(Kierkergaard 1849 [1983]). This essay is the Nineteen Century Christian expression of a literary tradition that has spanned the course of history from ancient Greece to the modern day, centuries of philosophical works aimed at explaining what, from period to period, has been called Melancholy, Despair, or more recently, Depression.

In the Fifth Century BC, Greek natural philosophers following the teaching of Pythagoras thought that Melancholy was caused by an excess of black bile, which was responsible for inducing anger, ill-temper and sad thoughts. A century later Plato suggested that a melancholic mood was the necessary condition for literary genius. Following Plato's work all major Greek Tragedians depicted their heroes as dramatically sad, longing for death and tormented by outbursts of anger (Klibansky, Panofsky et al. 1964). During the Middle Ages, the common understanding was that melancholia and ill-temper were governed by the influence of Saturn. The planed was considered to be cold, dark and violent and therefore associated with all earthly things characterised by the same features, including human moods and tempers (Klibansky, Panofsky et al. 1964). Following the Middle Ages, the Platonic notion of melancholy as being instrumental for poetic achievement continued to

underpin a great body of literature, finding its peak in the Romantic Movement. In fact, both the Romantic artists and their heroes were pictured as restless, mournful individuals who could not find their place in society and found comfort only in long journeys into the wildest landscapes. Today, scientists and doctors consider patients showing the symptoms of melancholia as affected by clinical depression.

2.2.2 Symptoms and Diagnosis

Clinical depression is the most common mental disorder and the leading cause of disability worldwide. However, there is still considerable variation in the use of technical names used to define the disorder in the existing literature, with unipolar major depression and major depressive disorder being but two of the numerous labels encountered in the literature (Ferrari, Somerville et al. 2013; Witheford, Dagenhardt et al. 2013). The reason behind this variation in nomenclature stands in two main factors: first, depression affects each patient differently as its symptoms are very subjective; and secondly in epidemiological surveys of the general population, depression is measured with a number of different tools which yield slightly different results as they focus on different aspects of the disease (Paykel, Brugha et al. 2005). Generally speaking, as stated by the American Psychiatric Association in the latest Diagnostic and Statistical Manual of Mental Disorder (DSM-IV) depression is characterised by a deep and unwavering sadness and a loss of interest in nearly all activities which can severely impair a person's ability to function in social situations or at the workplace (APA 2000).

Further, despite taking as many forms as there are people affected by it, depression is characterised by eight main symptoms whose presence is fundamental to identify and diagnose the disorder. Two of these eight symptoms had already been identified by Sigmund Freud in 1917 and are: (1) a deeply depressed mood and (2) a markedly diminished interest in all activities. The remaining six symptoms are: (3) a significant variation in appetite and weight, more often than not resulting in diminished appetite and subsequent weight loss (Carney and Freedland 2000); (4) a significant variation in sleeping patterns, often resulting in insomnia, as patients find themselves going to bed relatively early only to sleep restlessly and wake up in the early hours of the morning, which often reported to be the worst; (5) a significant variation in energy levels, as patients often report feeling either restless and agitated, or inactive, indolent and experiencing great fatigue; (6) an increase inability to concentrate, think, or make decisions; (7) recurrent feelings of worthlessness and/or inappropriate guilt; and (8) recurrent feelings of death and suicide (APA 2000; Carney and Freedland 2000). With the regards to the last symptoms, it is believed that as many as 15% to 20% of patients affected by depression actually commit suicide (Goodwin and Jamison 1990).

Diagnosis of depression is usually formulated on the basis of the severity and duration of symptoms. According to the Diagnostic Statistical Manual of Mental Disorder (DSM-IV) a diagnosis of depression is justified when five or more of the symptoms described above "have been present during the same two-weeks period and represent a change from previous functioning" (Mental Health Matters Website, 2000). For a clinical diagnosis to be justified, the five symptoms present should include depressed mood and loss of interest or

pleasure in most activities (APA 2000). Regardless of these diagnostic guidelines, a vast proportion of cases remain undiagnosed and untreated because of the variation of symptoms and because an observed tendency of patients to complain about physical pains rather than their psychological distress, thus making it difficult to recognise the presence of mental disorders (Mulrow, Williams et al. 1995). In addition, depression is an episodic disorder, with each episode typically lasting between a few months and a few years interspersed by periods of normality of at least two months occurring between episodes (APA 2000), and with an estimated 85% of patients bound to suffer from a second episode after recovery from the first one (APA 2000); this second episode has been estimated to occur within two years from the first one in 35% of patients, and within twelve years in 60% of patients (WHO 2001).

2.2.3 Reporting and Treatment

One of the main problems concerned with diagnosing depression is the strong stigma that still surrounds mental health issues. Because individuals suffering from mental disorders are often still referred to as 'crazy' in popular culture and informal slang, many people find it difficult to admit to a mental disorder and to seek help for fear of stigma and subsequent social isolation (APA 2000). This is particularly true among men, and in cultures – such as Central and Eastern Europe - where the stereotypical concept of masculinity translates into a widespread reluctance among men to admit to mental issues and seek help; for example it has been shown that Russian men are very unlikely to report depressive symptoms to the point that the gender difference in prevalence of depression in Russia is much bigger than anywhere else in the world (Nicholson, Pikhart et al. 2008).

However, when patients do chose to report their symptoms and seek help, depression can be treated. Albeit conflicting views on the effectiveness of antidepressant it seems that they are the fastest and most effective way of bringing relief to patients suffering from severe depression, as 50-65% of patients treated with antidepressants see an improvement. However, antidepressants are not recommended in cases of mild depression where cognitive behavioural therapy (CBT) is now thought to be more effective than drug treatment; regular exercise is also recommended in cases of mild depression (NHS 2013). Antidepressants work by increasing the levels of a group of chemicals in the brain called neurotransmitters; these include serotonin and noradrenaline which are thought to improve mood and emotion although the way in which this process happens is not fully

understood, which is why prescription of antidepressants is usually accompanied by therapy aimed at treating emotional distress (NHS 2013).

When prescription of antidepressants is justified by the intensity of the symptoms, patients are prescribed drugs which fall into four broad groups, differentiated by the neurotransmitters whose levels they are designed to alter. These drugs can be prescribed one by one, or sometimes in a cocktail of two or more drugs together. The two oldest types of antidepressants, Tricyclic Antidepressants (TCAs) and Monoamine Oxidase Inhibitors (MAOIs) are now prescribed only in case other antidepressants have failed because they have strong side effects and a high risk of overdose. The newest drugs are now preferred and selective serotonin reuptake inhibitors (SSRIs) are the most widely prescribed as they have fewer side effects than other drugs. SSRIs include fluoxetine (commonly sold as Prozac), citalopram (sold as Cipramil), paroxetine (sold as Seroxat) and sertraline (sold as Lustral). The second type of new age antidepressants is serotonin-adrenaline reuptake inhibitors (SNRIs) which include duloxetine (sold as Cymbalta or Yentreve) and venlafaxine (sold as Efexor) (NHS 2013).

All of these antidepressants have strong side effects which include feeling sick, dry mouth, slightly blurred vision, constipation, dizziness, drowsiness, problems in sleeping (insomnia), sexual dysfunction, and in some extreme cases bladder blockage (NHS 2013). In addition, in his book 'Anatomy of an Epidemic', Robert Whitaker has recently argued that antidepressants might be deleterious for the brain in the long run (Whitaker 2010). Without denying that antidepressants do bring relief to patients, Whitaker argues that there is no

real scientific evidence to prove that depression is caused by an imbalance of neurotransmitters in the brain, but antidepressants are designed to cause one. Hence, it could be that long term exposure to antidepressants might lead to a permanent inability of the brain to restore the natural balance of neurotransmitters (Whitaker 2010). Which is why it is of paramount importance to research and invest in preventing depression: not only because of its high burden on society, but also because its treatments could cause more damage to the brain.

2.2.4 Epidemiological Tools

In order to implement effective prevention policies it is important to identify which groups of people are more at risk of depression. This has been done through population surveys that have been run in different countries as part of either independent national studies or international research projects such as the World Mental Health Survey. These population surveys use epidemiological tools designed to measure presence and frequency of depressive symptoms more than clinical depression per se, hence in this thesis the term depressive symptoms will be used instead of depression. A number of epidemiological tools have been developed, including: the Self-reported Depression Scale (SDS), a 20-items scale designed in 1965 to detect depressive symptoms in patients of all ages but whose validity is controversial (Zung, Richards et al. 1965; Croezen, Peasey et al. 2011); the Geriatric Depression Scale (GDS), designed to detect depressive symptoms in the elderly population, originally it contained 30 items but the authors subsequently designed a shorter 15-items version (Yesavage, Brink et al. 1983; Croezen, Peasey et al. 2011); the Composite International Diagnostic Interview Short Form (CIDI-SF) a self-reported scale currently used by the WHO in the World Mental Health Survey project, designed to detect symptoms of eight different syndromes: generalised anxiety disorder, agoraphobia, panic attack, drug dependence, social phobia, simple phobia, major depressive episode and alcohol dependence (Kessler, Andrews et al. 2006; Croezen, Peasey et al. 2011).

Two further such tools are the Centre for Epidemiologic Studies Depression Scale (CES-D), and the General Health Questionnaire (GHQ), which will be used in this thesis and described in more detail in Chapter 4. The CES-D scale is a short self-report scale designed to measure depressive symptomatology in the general population by Lenore Radloff (Radloff 1977), and one of the most widely used instrument to measure depressive symptoms. The CES-D is composed of twenty items investigating the presence of depressive symptoms, all items having been tailored as to detect clinical symptoms of depression in the general as well as the clinical population (Radloff 1977). The General health Questionnaire was also developed in the 1970s (Goldberg 1972), but it is a less specific instrument as it is designed to quantify the risk of developing psychiatric disorders in general rather than depression specifically. The GHQ assesses well-being in individuals by targeting two main areas: the ability, or inability, to carry out normal tasks and the appearance of distress (Goldberg 1972; Goldberg and Hiller 1979). The GHQ and the CES-D scales have been used to measure depressive symptoms in the Whitehall II and the Health Alcohol, and Psychosocial factors In central and Eastern Europe (HAPIEE) cohort studies respectively, and hence will be presented in more details in Chapter 4, where the methods of the analysis here presented will described.

2.2.5 Prevalence and Risk Factors

The epidemiological tools described above have allowed researchers to identify what groups of people are more affected by depressive symptoms in the general population. Hence, it is now known that, even though depression can occur at any age its incidence is higher among individuals aged 40-50 (WHO 2001). In addition, a wide body of research has confirmed that women tend to have rates of depression between two and three times higher than men (APA 2000; Kuehener 2003). The Global Burden of Disease (GBD) study estimated that in the year 2000, point prevalence for depression was 1.9% in men and 3.2% in women, and that 5.8% of men and 9.5% of women were to develop the disorder in the following twelve months (WHO 2001). This sex ration was found to be even more dramatic in Central and Eastern Europe, where Bobak and colleagues (2006) found that the prevalence of the disease was almost twice as high among women than among men (Bobak, Pikhart et al. 2006).

Marital status also seems to affect the risk of depression, as prevalence of depression is lower among married couples compared to single, divorced or widowed individuals. For example, Bobak et al (2006) found that single, divorced or widowed people in Russia, Poland and the Czech Republic were between twice and four times more likely to be depressed than their married counterparts. Finally, socio-favourable economic circumstances appear to play a role in protecting against depression, as prevalence of depression has been repeatedly recorded as higher among more disadvantaged individuals than among their better off counterparts (Lorant, Eaton et al. 2003; Paykel, Brugha et al. 2005). In their meta-analysis of the existing evidence Lorant and colleagues (2003) found

that individuals of low socio-economic status were almost twice as likely to be depressed than individuals of higher status; even though, the odds of being affected for the first time were lower than the odds of persisting depression in the more disadvantaged groups. In the same fashion, Nicholson and colleagues found that lower status individuals were up to five times more likely to report depressive symptoms (Nicholson, Pikhart et al. 2008). Further, physical activity is known to influence depressive symptoms (Lee, Lee et al. 2014; Rosenbaum, Tiedemann et al. 2014), to the point that the NHS recommends increasing levels of physical activity as a treatment for mild depression (NHS 2013). Finally, smoking has been linked to increased severity of depressive symptoms and to slow the remission from the disorder (Dierker, Avenenoli et al. 2002; Jamal, Van der Does et al. 2012).

2.3 Inclusion in Social Networks and Social Support

2.3.1 Historical Context

The idea that social relations, or lack of thereof, play a fundamental role in the onset of melancholia and tragic *furor* in literary heroes is a constant theme in literature, from the ancient Greek tragedies to modern fiction. In particular there seem to be two aspects to this theme: one is physical loneliness, as in the case of Odysseus who travels home for ten years without any companions and encounters many adventures made even greater by his being alone; the other is loneliness derived by the sudden loss of dear ones, or more importantly, the loss of faith and trust in other human beings (Klibansky, Panofsky et al. 1964). This was the case for Achilles, probably the most famous of heroes, who entered his state of melancholic rage in response to the death of his lover Patroclus (Klibansky, Panofsky et al. 1964). Less known is the case of Medea, who, according to the poet Euripidis, succumbed to rage and despair so strong as to lead her to murder her own children in revenge for being abandoned by her husband (Klibansky, Panofsky et al. 1964).

This theme of the hero assuming their heroic and melancholic status in response to a loss in social relations is found in subsequent literature as well. William Shakespeare's Hamlet, for example, was driven into madness by the death of his father, but more than anything by the feeling that he could no longer trust those closest to him; and the same fate attended Ophelia who was driven to madness and ultimately suicide by the loss of her father and the knowledge that her beloved Hamlet was mad (Shakespeare 1599 [2011]). At the beginning of the last century, in his novel *Nostromo*, Joseph Conrad comments on the death of one of his characters with: "*but the truth was that he died from solitude, the enemy known but to*

few on this Earth, and whom only the simplest of us are fit to withstand. The brilliant Costaguanaro of the boulevards had died from solitude and want of faith in himself and others" (Conrad 1904), clearly indicating both physical loneliness and lack of faith and trust in others as a possible direct cause of death.

This idea that loss of loved ones or of faith in them could lead to mental and even physical decay, was later conceptualised by Sigmund Freud who, in his essay Mourning and Melancholia (1917 [1964]) argues that mourning and melancholy have the same cause: the loss of a beloved object or person. The only difference is that, in the case of melancholy, the loss is not real. According to Freud, both psychological conditions are characterised by depression, diminished interested for the outside world, inability to love and unwillingness to perform any activity. A melancholic patient would, however, present all these symptoms coupled with a markedly low self-esteem, an acute sense of guilt and deep self-hate that are not present in mourning (Freud 1917 [1964]). Freud also saw melancholy as likely to turn into mania and alternating phases of sadness and mania (Freud 1917 [1964]).

Indeed, the notion of social isolation or loss of dear ones as being deleterious for mental health might find its roots in the forces that drove the evolution of the human brain. Evolutionary psychologist Robin Dunbar has focused his research on understanding what drove the evolution of the human brain through the observation of primate behaviour and of the analysis of the structure of the primate brain. The latter revealed that primates have brains considerably larger and more complex than mammal species of similar body size that do not live in groups. Hence, Dunbar speculated that the primate brain evolved to be larger and more complex as a response to the cognitive demands posed on individual by social

associations (Dunbar 1998; Dunbar and Schultz 2007). This speculation was driven by the observation that primates have a particularly large neocortex. The neocortex is the frontal part of the brain which in humans is responsible for higher functions such as sensory perceptions, generation of motor commands, spatial reasoning, conscious thought and language (Lui, Hansen et al. 2011). Dunbar attempted to prove his theory by plotting the size of the neocortex against the size of the social group in which different species of primates live. And indeed, when plotting the size of the neocortex against the size of social group in primates, Dunbar found that in many different species of primates the more complex the social life, the bigger the neocortex (Dunbar 1998). What is more, when looking at the behaviour of chimpanzees in the wild Dunbar noticed that, after foraging, the second most important activity in these primates daily life is social grooming. Chimpanzees groom to enhance relationships and seal alliances, they also resort to social grooming in times of great stress or fear, to find calm and peace again (Dunbar 1998). The higher the level of stress and uncertainty in a chimpanzee's life the more it will engage in grooming with closer allies (Dunbar 2003).

Dunbar tested his theory on different animal species to investigate whether a complex social life has been the trigger for an enhancement in the neocortex in all species or only in primates. Contrary to what was expected, Dunbar and his colleagues found an inverse correlation between group size and neocortex size. Interestingly enough among birds, ungulates and carnivores, monogamous species have a bigger neocortex. Dunbar argued that this is because pair bonding is more cognitively demanding than polygamy which does not involve co-operation and co-ordination between individuals (Dunbar and Schultz 2007).

Indeed, monogamous pair bonding, and social life not only seem to have been the trigger for brain evolution in small animals and primates respectively, but it has also been shown to affect mental health in humans. This was first noticed by the French sociologist Emile Durkheim who in 1897 published a study which was going to become one of the milestones of modern sociology and an inspiration for much future research, *Suicide: A Study in Sociology.* In this study Durkheim looked at patterns of suicide in France from a social rather than psychological perspective, suggesting that suicide might not be as intimate and individualistic an act as it is generally thought to be (Durkheim 1897 [1951]). Durkheim observed that suicide rates remained the same year after year despite the personal hardships or attitudes of single individuals, and argued that there must have been some overarching social forces acting to maintain the rates constant. He identified two such forces in marriage and social integration. Individuals might die, age or fall into misfortune but, Durkheim noticed, the rates of suicide among married individuals are always lower than among unmarried people. In the same fashion, more integrated societies have lower rates of suicide than less integrated ones (Durkheim 1897 [1951]). Durkheim postulated that what determines a society's level of integration is its religion. He argued that catholic societies are more integrated than protestant ones and therefore have lower rates of suicide. Durkheim highlighted two aspects of Catholicism that play a particular role in protecting from suicide. The first is its norms: Catholicism has stricter rules of behaviours than Protestantism and condemns suicide more vehemently, thus Catholics will be less likely to fall out of such strict regulation. The second important aspect of Catholicism is its enhanced social participation; in fact Catholics are required to attend mass every Sunday and to take part in several social occasions which both provide individuals with positive

experiences that increase their well-being, and create a community in which individuals will look after and support each other (Durkheim 1897 [1951]; Berkman, Glass et al. 2000)

Durkheim's pioneering work was rediscovered in the 1970s when a wave of studies started investigating the association between social relations and mental health. Sydney Cobb (1976), reviewed the evidence regarding whether supportive associations among people can be protective against the health consequences of life stresses. The review concluded that there is evidence that social support helps recovery from many illnesses including depression, and might also determine a reduction in the medication needed (Cobb 1976). The article was closely followed by the publication of a book by George Brown and Tirril Harris (1978) called *Social origins of depression: A study of psychiatric disorder in women.* In the book, Brown and Harris presented a study carried out on British women in London, in which they investigated possible social determinants of depression. They found that depression was more prevalent among women of lower social strata and that these social differences could be accounted for by the number of stressful life-events encountered in the previous year, and by the presence of vulnerability factors, first among which was the absence of a close confiding relationship (Brown and Harris 1978). Two years later, Scott Henderson (1980) investigated the role of deficiencies in social relationships in the onset of mental disorders in a community sample. Henderson found that among the half of this community sample, who were exposed to the higher level of life adversity, deficiencies in social relations explained 30 per cent of the variance in neurotic symptoms four months later. Interestingly he also found that it was the perceived inadequacy of relations in time

of adversity which was the stronger predictor of onset of mental disorders (Henderson 1980).

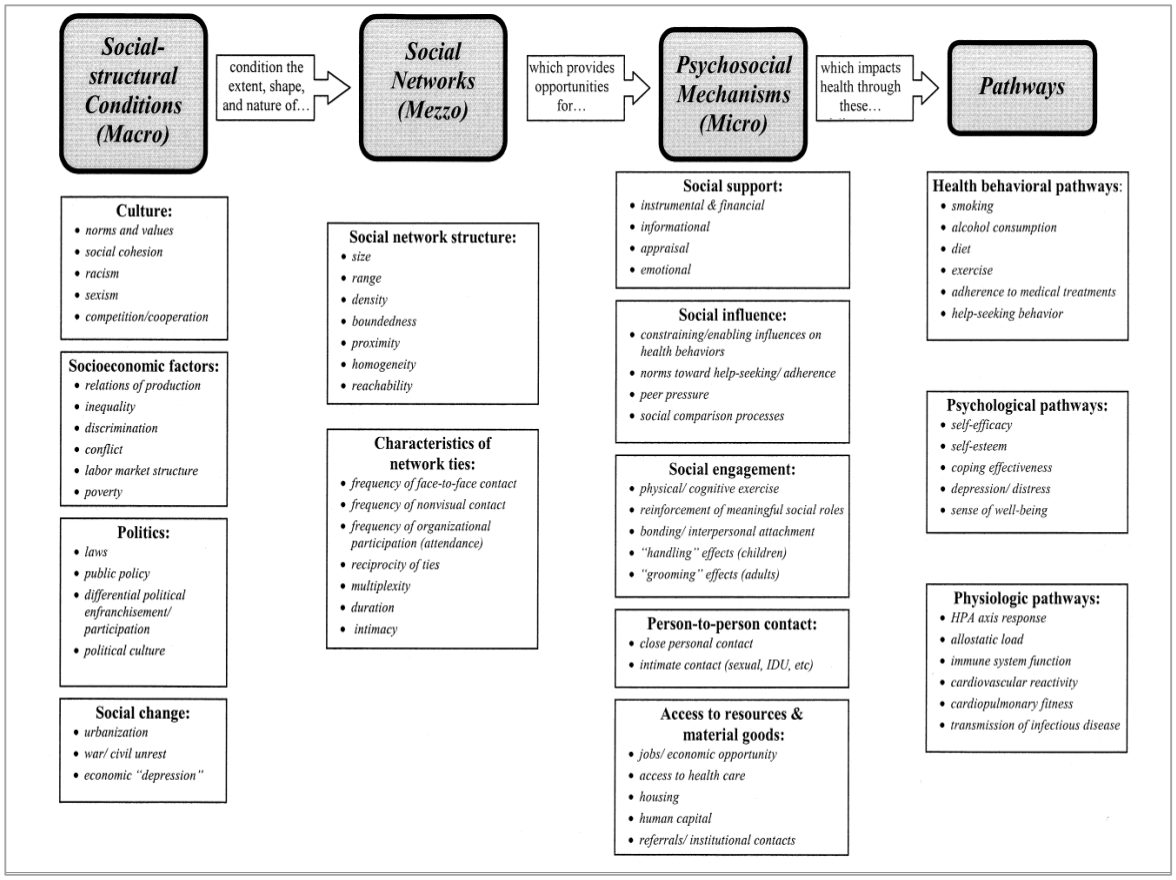
2.3.2 Modern Theory

To date, a number of hypotheses and conceptual frameworks have been put forward to explain the mechanisms through which social relations affect mental health (references). Two frameworks particularly well known and widely used in subsequent research are the ones by Cohen and Wills (1985) and Berkman and Glass (2000). Cohen and Wills (1985) proposed two models: the first, the so called main effect model, explained how the quantitative aspects of social relations affect mental health; the second, the stress-buffering model, explained how the qualitative aspects of social relations provide a protective buffer against stress (Cohen and Wills, 1985). On the other hand, the conceptual framework proposed by Berkman and Glass (2000), sees social relations as part of a wider chain of effects that start at the macro-level of culture, society and politics. These models are by no means mutually exclusive, and can be integrated to provide a comprehensive framework. In this thesis I will use Berkman and Glass (2000) cascade model as the underlying framework, and incorporate Cohen and Wills (1985) main effect and stress buffering models to explain the effects of social support on depressive symptoms.

The cascade model starts by considering how structural conditions at the macro level influence social networks. In fact, Berkman and Glass (2000) postulated that a society's cultural as well as socio-economic and political factors influence the extent, shape and nature of the social networks found within it. These cultural, socio-economic and political

factors include cultural norms and values, levels of social cohesion, as well as the structure of the labour market and the levels of poverty present, or the political culture and levels of political participation (Figure 2.3.1). The sum of these factors, constitutes what Robert Putnam (1995; 2000) and Pierre Bordieu (1986) have defined under the name social capital, which shall be discussed in more details in section 2.3.3. Social capital conditions the nature of social networks within a society. Social networks, or the mezzo level in the cascade model, vary in their structure as well as in the strength of the ties that constitute them. Structural characteristics of a social network are: size, range, density, boundedness, proximity, homogeneity and reachability. While characteristics of social ties within a network are: frequency of face-to-face contact, frequency of non-visual contact, frequency of organisational participation (attendance), reciprocity of ties, multiplexity , duration and intimacy (Figure 2.3.1 (Berkman and Glass 2000)). While details of all their characteristics and how they influence health will be given in section 2.3.4, it is important to explain here that social networks provide the opportunities for the psychosocial mechanisms that affect mental health to take place. In fact, when part of a social network, individuals experience social support, social influence, social engagement, person to person contact and access to resources and material goods, which they would not have the opportunity to experience were they alone (Berkman and Glass 2000). Sections 2.3.4 will illustrate the ways in which social influence, social engagement and person to person contact affect mental health, while the pathways through which social support affects mental health are explained in section 2.3.5.

Figure 2.3.1 *Berkman and Glass cascade model, from Berkman and Glass (2000) p 847.*



2.3.3 Social Capital

Social capital roughly refers to all those structural characteristics of society that represent the macro level in the cascade model described in section 2.3.2. The concept of social capital was first theorised in the mid-late-1980s by French sociologist Pierre Bordieu (1986) and American sociologist James Coleman (1988), and subsequently revisited and expanded by American sociologist Robert Putman (1993; 1995; 2000) who focused primarily on the social capital, or disappearance of thereof, of rural Italy and then, more extensively, of the United States.

Pierre Bordieu (1986) defined social capital as "the sum of resources, actual or virtual, that accrue to an individual or group by virtue of possessing a durable network of more or less institutionalised relationships of mutual acquaintance and recognition" (p. 249). Bordieu applied the economic concept of capital to social ties and conceptualised that social capital is product of all the effort that members of a group invest into social relations. In order for capital to be accumulated, members of a group not only need to be invested of a role within the group – such as brother, daughter, friend – but also need to invest considerable amount of time and energy in maintaining relations and rules within the group (Bordieu 1986). In this way the social capital created is so much greater than a single relationship, and it is inherited by new members as soon as they are invested of their identity in the group. Thus, the group is not only accumulating social capital but cultural and even economic capital which derive from the need of investing in material goods and define rules for life with the group (Bordieu 1986). Ultimately, according to Bordieu the group will decide to entrust all

this capital into the hands of a few selected members who will then manage the social capital of relations, and this is how societies are born (Bordieu 1986).

American sociologist Robert Putnam took Bordieu's concept of social capital and applied it to the civil society of the United States of America, trying to explain how civil society came to be and what forces drive it (Putnam 1993; Putnam 1995; Putnam 2000). In his book *Making Democracy Work: Civic Traditions in Modern Italy* (Putnam 1993) Putman argues that the quality of governance is determined by longstanding traditions of civil engagement, or lack of thereof. For Putnam the hallmarks of successful governance are to be found in voter turnout, newspaper readership, membership in choral societies and sport clubs, and all of these networks of organized reciprocity and civic solidarity are a precondition for socioeconomic modernisation (Putnam 1993).

In his subsequent work on the civil society in the United Sates, Putnam argues that the general participation in networks of civil engagement such as sport clubs or the Scouts, and even voter turnout and trust in neighbours have been drastically diminishing in the past two or three decades, thus undermining the principles of civil society (Putnam 1995; Putnam 2000). Putnam then calls for more civil engagement in the States, such as bonding with neighbours and joining clubs and societies, so as try and re-create those networks of organised reciprocity and solidarity which are at the heart of modern society (Putnam 1995; Putnam 2000).

Perhaps the most spectacular example of how the lack of an engaged civil society can affect health comes from countries of Central and Eastern Europe after the fall of the Soviet Union. In fact, even though Russia was the country that underwent the deepest social changes and suffered the greatest consequences, all countries of Central and Eastern Europe were hugely effected by the political and economic turmoil. The communist model of state was characterised by a high degree of centralisation at all levels of society and a distinct lack of all those non-political organisations that go by the name of civil society and contribute to create trust and social capital (Rose 1995; Rose 2000). When communism fell, the societal and economic organisation of the countries involved was disrupted almost entirely, creating what Durkheim (1857) had called "anomie", that is the absence of social values, norms and opportunities, which in turn poses individuals under acute stress (Cornia 2000). Indeed, the lack of institutions aimed at moderating the impact of political disruption played a role in exacerbating the stress that individuals were facing (Cornia 2000). The Italian economist Giovanni Andrea Cornia (2000) has argued that high levels of psychological stress caused by political turmoil are directly linked to increase in mortality (Cornia 2000). Sure enough, in the first year following the changes of 1989, countries of Central and Eastern Europe were swept by a peak in mortality rates and a sharp decline in life expectancy, particularly among men from a humble socio-economic background and with unstable family arrangements (Cornia 2000; Marmot 2004). Mortality rates in the area have been fluctuating throughout the Nineties following the economic ups and downs, and life expectancy slowly rose again almost to the levels of Western Europe in nearly all countries of the ex- URSS, with the exception of Russia, where life expectancy for men is still sixteen years lower than in the United Kingdom (Marmot and Bobak 2000; Marmot

and Brunner 2005). The immediate reasons behind such high mortality rates and low life expectancy in Central and Eastern Europe are to be found in an excessive consumption of alcohol which contributes to increasing the rates of cardiovascular heart diseases (Leon, Saburova et al. 2007). However, if Cornia and Putman are right, it is political changes coupled with lack of social trust and of informal social networks that underlie both the increase in alcohol consumption and in mortality.

2.3.4 Social Networks

Social networks are at the heart of the cascade model and of society as a whole. In fact not only they influence individual health both directly through social and behavioural pathways and through provision of support, but they also are instrumental in shaping the health of societies by increasing social capital which will then condition socio-economic and cultural development. Given the paramount importance of the role they play in conditioning the health of both individuals and societies, this section is devoted to illustrate the structural characteristics of networks as well as the pathways through which they affect health. A social network can be defined as the web of social relations that surround an individual coupled with the characteristics of its ties (Fischer 1982; Berkman and Glass 2000). This definition allows to identify the two different sets of structural characteristics used to define a networks; that is the structural characteristics of the network itself, and the structural characteristics of its ties (Figure 2.3.2).

2.3.4.1 Structural characteristics of social networks

A social network is usually defined on the basis of its size or range, density, boundedness, and homogeneity. Size or range, refers to the number of network members; density refers to the degree to which the members are connected to each other; boundedness is the extent to which network members are defined on the basis of traditional group roles such as parent, colleague, friend or neighbour; and finally homogeneity is the extent to which network members are similar to each other. These four characteristics define the type of network an individual is embedded in, although it is important to keep in mind that individuals are often surrounded by multiple social networks. In her review of existing

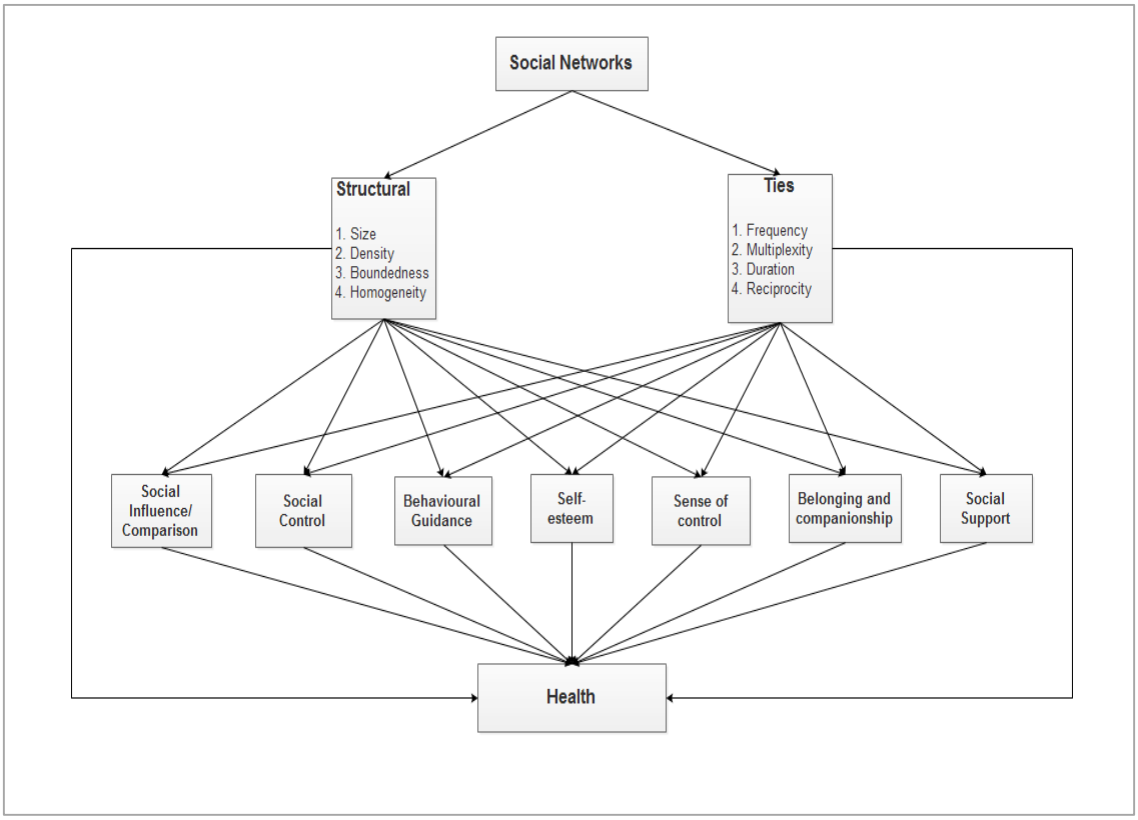
literature on the topic of how social networks influence health, Peggy Thoits (2011) identified two main types of networks: primary and secondary. A primary network is usually small in size, but highly dense and homogeneous, in that it is usually formed by a small group of very intimate individuals such as a significant other, closest of kin or very close friends. Members of the primary network are usually involved in the provision of confiding/emotional support, social control, sense of belonging and companionship, behavioural guidance and meaning and sense of control (Thoits 2011). The secondary network, on the other hand, is usually larger in size, less dense and less homogeneous as it is composed by less intimate friends, work colleagues or more removed family members, and it is usually involved in the provision of practical support and also social control (Thoits 2011).

2.3.4.2 Structural characteristics of social ties

Perhaps more important for individual health are the characteristics of the ties within the network. Berkman and Glass (2000) identified four main such characteristics: (1) frequency of contact, which refers to the number of face to face contacts or via phone, email, social media contacts between network members; (2) mutliplexity, refers to the number of types of transactions or support flowing through a set of social ties, or in other words, how many of the various pathways through which social networks affect health are regularly exchanged during normal network associations; (3) duration, refers to the amount of time members of the network know each other; and (4) reciprocity, or the degree to which social exchanges are even or reciprocated (Berkman and Glass 2000). Of these, frequency of contact, coupled with network size, is often used as a measure of how socially connected an individual might be, and many a study have reported a direct association with depressive

symptoms (Wildes, Harkness et al. 2002; Brugha, Weich et al. 2005; Chan and Lee 2006; Hedley and Young 2006; Haines, Beggs et al. 2011; van Beljouw, van Exel et al. 2014). For instance, in their study of adults aged 16-74 in British households, Brugha and colleagues (2005) suggested that individuals with smaller social networks at baseline were more at risk of mental disorder at the subsequent round of follow-up. Similarly, a study carried out in the elderly population of Beijing and Hong Kong found that participants with a larger network were happier than their more isolated counterparts (Chan and Lee 2006). Finally, Wildes, Harkness and Simons (2002) also found that number of social relationships was a strong predictor of depression in women aged 30 and above, even stronger than adverse life events.

Figure 2.3.2 *Structural characteristics of networks and their ties and pathways to health*



2.3.4.3 Pathways from social networks to health

If structural characteristics of social ties have been proven to affect mental health, it is probably because they are a proxy for the degree of transition of a number of pathways through which social relations affect health. Berkman and Glass (2000) identified six broad groups of pathways, but in her more recent paper social relations and mental health, Peggy Thoits (2011) reviewed the evidence supporting the action of seven distinct pathways leading from social networks to mental health, six of these are described below, the seventh, social support, is the topic of a separate section.

Social influence/ social comparison had already been identified as one of the properties of social ties affecting health by Berkman and Glass (2000) in their cascade model. It refers to the fact that individuals obtain both normative and behavioural guidance through comparison with members of their group who are similar to them. Individuals will modify their behaviour accordingly to the behaviour of others in their group by simply comparing themselves to others. An example of this could be found in smoking, as it has been shown that smoking prevalence among peers is the strongest predictors of taking up smoking in adolescents (Landrine, Richardson et al. 1994). Hence, social influence/ social comparison could be either beneficial or deleterious for health, depending on the type of behaviour individuals will decide to copy from other members of their group (Thoits 2011).

Social control roughly corresponds to what Berkman and Glass (2000) have called the active component of social influence, and refers to the direct attempts from other members of a group to influence, control, modify a person's behaviour (Uchino 2004; Umberson and

Montez 2010). Social control is usually practiced by member of the primary group, as they have the intimacy necessary to act directly. However, because of the direct nature of these interventions, social control could backfire if considered too invasive by individuals who are being actively controlled by other members of their group (Thoits 2011)

Behavioural guidance, purpose and meaning refers to the powerful effects of role relationships. In other words, within groups individuals have specific roles given by their social ties, for example husband, or mother, or friend; with each role comes responsibility and commitment as role obligations constrain behaviours (Thoits 2011). But role obligations also confer a sense of identity, purpose and meaning to one's life, coupled with the feeling of 'mattering' or in other words, of being important to somebody else (Thoits 2011), which plays a fundamental role in enhancing mental well-being and protecting from disorders such as depression (Berkman and Glass 2000; Thoits 2003; Uchino 2004; Umberson and Montez 2010; Thoits 2011).

Self-esteem is a by-product of role identities provided by social ties, as it stems from self-evaluation of performance in those roles. Individuals evaluate their role performances not only through comparison with socially similar others – other parents, friends, teachers – but also through imaginatively reviewing their performances from the point of view of those similar others or other members of audience. These self-evaluations affect self-esteem and self-worth by providing individuals with a sense of how good, worthy or competent they are in a role (Thoits 2011). Self-esteem and self-worth, in turn, are of

paramount importance in protecting from anxiety and depression (Baumeister, Campbell et al. 2003; Thoits 2003; Taylor and Stanton 2007).

Sense of control or mastery is another by-product of successful role performances, as it stems from the feeling of being "on top of one's game." Role performances require individuals to successfully carry out a vast amount of tasks on a daily basis, these tasks range from earning money, to doing the laundry, attending meeting, etc., and require a great investment of energy and time. Sense of control or mastery stems from being able to carry out all of these tasks on a regular basis, as well as from the belief of being particularly good in some areas of life (Thoits 2011). Sense of control or mastery contributes to generate the feeling that one is able to cope with most if not all stresses, which plays a role in protecting from anxiety and depression (Barrera 2000; Mirowsky and Ross 2003; Taylor and Stanton 2007).

Belonging and companionship. Aside from the positive effects of role performances, social associations per se contribute to creating a sense of belonging to a community. Sense of belonging stems from acceptance, which is not a given of a group, instead members of both the primary and the secondary group need to show signs of acceptance of an individual for them to feel they belong to a community (Thoits 2011). Belonging to a community provides the feeling that the group will look after an individual and help in times of crisis, both emotionally and materially (Hagerty, Lynch-Sauer et al. 1992; Hagerty, Williams et al. 1996; Hagerty and Williams 1999). This, in turns, proves protective against anxiety and depression as it provides a feeling of safeness in times of crisis (Hagerty, Lynch-Sauer et al. 1992; Hagerty, Williams et al. 1996). Companionship, on the other hand, stems from this sense of

belonging and corresponds to the feeling of having others with whom to share activities, thoughts and emotions. The lack of companionship results in loneliness which is deleterious for mental health (Nolen-Hoeksema and Harrell 2002; Heikkinen and Kauppinen 2004; Cacioppo, Hughes et al. 2006; Thoits 2011).

2.3.5 Social Support

The seventh pathway through which social networks affect health is the provision of social support. The effects of social support on health and especially mental health are so strong that for years it was considered to be the only pathway that led from social networks to health (Kahn and Antonucci 1980; House, Kahn et al. 1985; Sarason, Sarason et al. 1990; Berkman and Glass 2000). However, despite the great attention it has received over the decades, there still is little consensus on the definition of social support, to the point that in their review of the literature William and Barclay (2004) identified as many as twenty five different definitions. Among these definitions, one of the most widely used was formulated by Sydney Cobb (1976) who defined social support as "the perception or experience that one is loved and cared for by others, esteemed and valued, and (is) part of a social network of mutual assistance and obligations." This definition is widely used because it highlights two of the main aspects of social support. Namely, that support can be actually received or purely perceived and that it is transactional in nature as it stems from a system of mutual obligations (Cobb 1976; Kahn and Antonucci 1980; Berkman and Glass 2000; Berkman, Glass et al. 2000; Kawachi and Berkman 2001; Thoits 2011).

Since research on social support started in the 1970s many frameworks and hypotheses have been put forward to explain just how it affects health. Perhaps the best known of these models is the one put forward by Cohen and Wills (1985), who postulated that social support could either effect health directly, the main strain model, or by providing a buffer against stress, the stress buffering model (Cohen and Wills 1985). In later work, Sheldon Cohen (2004) suggested that the main effect model is more suitable to describe the direct

way in which inclusion in social networks affects health, while social support acts primarily as a buffer against stress, with different types of support proving more or less efficient in the task (Cohen 2004). However, other research has proven that support can in fact be beneficial even when not in a time of crisis, by creating a sense of security and love through day to day transactions (Vilhajalmsson 1993; Uchino 2004; Thoits 2011). To the point, that it has been argued that support can take two forms: day to day support, which contributes to form an underlying sense of importance to others and security; and emergency support which is provided in times of crisis and focuses on providing coping assistance against stress (Lin, Ye et al. 1999; Badr, Acitelli et al. 2001; Thoits 2011). Further, three main types of support are often identified in the literature: instrumental, informational, and emotional support (Cohen and Wills 1985; Berkman, Glass et al. 2000; Kawachi and Berkman 2001; Cohen 2004).

Instrumental support refers to help with tangible needs such as money lending or babysitting. Instrumental support can be provided in a time of crisis to infer a sense of security – for example being lent money in a situation of financial distress - but more often than not takes the form of all those small practical favour received by a number of different actors that help easing or speeding up our daily activities (Cohen 2004; Thoits 2011). As mentioned above, and depending on the situation instrumental support can be provided by members of both the primary and the secondary networks.

Informational support refers to the body of relevant information that can be provided by the social network to help a member cope with their current difficulties and hardships.

Typically it takes the form of advice or guidance in dealing with one's problems and it can be provided by both the primary or secondary network. Usually the primary networks will provide advice in important matters, while the members of the secondary networks might provide wisdom and information on a larger array of topics. Informational support can be provided in both times of crisis and on a day to day basis, in the form of informal conversations on topics such as children, work, being a parent etc (Thoits 1985; Taylor and Aspinwall 1996; Uchino 2004; Thoits 2011).

Emotional support refers to the expression of empathy, caring, reassurance and trust that is often provided by very close, intimate persons, and which offers opportunities for voicing emotions and venting about (Cohen 2004). As or the other two types, emotional support is provided both on a daily basis, allowing individuals to vent their emotions before issues escalate into a situation of crisis, allowing to de-escalate the appraisal of problems from future threats to manageable task; and in times of crisis, to assist in coping with stressors by providing emotional aid (Cohen and McKay 1984; Thoits 1985; Taylor and Aspinwall 1996; Uchino 2004; Thoits 2011).

If instrumental, informational and emotional support contribute to maintain or restore the psychological and physical health of individuals, there is a fourth type of support which has the exact opposite effect. *Negative support* refers to both the stressful aspects of social ties, such as argument, divorces, abusive relationships, or even just annoyance; and to the perceived inadequacy of the positive support received. Negative support has been shown to be particularly detrimental for mental health. For example, Croezen et al (2012) found

that in a cohort of Dutch adults, experienced negative support substantially increased the odds of prevalent and incident poor mental health. Similarly, Stansfeld and colleagues (1998) reported how among British Civil Servants, low emotional support and high negativity of social relations predicted increased risk of psychiatric morbidity.

This leads to the issue of received versus perceived support. Received support refers to support received in a particular stressful situation or during a delimited period of time; perceived support, on the other hand, is the feeling of availability of support that emerges from numerous real instances of help provided by different network's members at different stages of an individual's life (Hobfoll 2009; Thoits 2011). A number of studies have been looking at whether it is perceived or received support to be more beneficial for mental health. Bolger et al (2000) reported that while there is a documented association between perceived support and depressive symptoms, the same association is not present between received support and depression. They argued that often recipients of support fail to register acts of support at a conscious level, but never the less these invisible support transactions favour adjustment to major stressors by promoting a sense of belonging. Similarly, Bolger and Amarel (2007) reported that invisible support in practical tasks helped participants, while visible support increased their reactivity. It has been speculated that this is the case because visible support adds to the stressful situation the strain of reciprocating the favour, which individuals may not feel able to do (Deelstra, Peeters et al. 2003; Gleason, Mausmi et al. 2008).

2.3.6 Gender differences

When looking at prevalence of depression in the general population, a marked difference has been observed between men and women. To begin with, women have been repeatedly reported to be roughly twice as likely as men to be affected by depressive symptoms (Piccinelli and Wilkinson 2000; Kawachi and Berkman 2001; Bellman, Forster et al. 2003; Kuehener 2003; Goodwin and Gotlib 2004; Zunzunegui, Minicuci et al. 2007; Nicholson, Pikhart et al. 2008; Michel 2009) and to have higher rates of relapse and lower rates of complete remission from depression (Kuehener 2003). These marked gender differences could be due to artefactual, biological or social factors or, more likely, a combination of these (Kuehener 2003). Advocates of artefactual gender differences, claim that women are more likely to seek help and respond differently to depression measuring tools (Briscoe 1982). However, although women have found to report more symptoms than men and more likely to report certain symptoms (Angst and Dobler-Mikola 1984), this alone cannot account for the entirety of the gender differences observed (Kuehener 2003; Parker and Brotchie 2010).

For what concerns biological factors, if early studies on the genetic epidemiology of depression found similar heritability in men and women (Sullivan, Neale et al. 2000), recently Parker and Brotchie (2010) have suggested that women might have a greater biological predispositional vulnerability to depression and to social factors that can precipitate it, such as the stress of multiple social roles Kuehener 2003; Matud 2004; Panayiotou and Papageorgiou 2007). Women also differ from men in their coping

mechanisms, as they are more likely to indulge in negative thoughts and rumination, while men are more likely to externalize their feelings and find relief in physical activities (Wupperman and Neumann 2006).

However, very little research has focused on gender differences in the patterns of association between inclusion in social networks or social support and depressive symptoms. With the notable exception of Brown and Harris (Brown and Harris 1978) who observed that women are much more likely than men to rely on ventilation of their issues and on emotional support and argued that the gender gap in prevalence of depression might be much larger than what it already is if they did not. However, more recently Kendler and colleagues (2005) conducted a study on opposite-sex twins using levels of social support at wave one to predict risk of major depression at wave two and found that although levels of social support did not explain the gender difference in prevalence of depression, emotionally supportive social relationships were more protective against major depression than in men (Kendler, Meyers et al. 2005). Similarly, Dalgard and colleagues (2006) found that women enjoy more social support than men but this does not explain the gender differences in depression. However, women enjoyed no social support and were exposed to life events were more vulnerable to depression than men without support (Dalgard, Dowrick et al. 2006).

2.3.7 Depressive symptoms, inclusion in social networks and social support

If a vast body of literature including both cross-sectional and longitudinal studies has shown that social isolation is deleterious for mental health (Durkheim 1897 [1951]; Weiss 1973; Miller and Ingham 1976; Henderson 1977; Brown and Harris 1978; Henderson 1980; DiTommaso and Spinner 1997; Hagerty and Williams 1999; Nolen-Hoeksema and Harrell 2002; Chou and Chi 2004; Heikkinen and Kauppinen 2004; Cacioppo, Hughes et al. 2006)' and that social support also has an impact on mental health(Cohen and Wills 1985; Deelstra, Peeters et al. 2003; Cohen 2004; Bolger and Amarel 2007; Hobfoll 2009; Thoits 2011; Croezen, Picavet et al. 2012), considerably less research has been devoted to investigating the effects of existing depression on the levels of social inclusion and of social support that an individual enjoys. In other words, is it possible that individuals who suffer from depression would isolate themselves because of the disorder? And, is it possible that people affected by depression would perceive that they are not receiving any support, or that they are receiving so much they could never reciprocate, because of the disorder?

Indeed there is some evidence to suggest that depression may trigger increased social isolation or decreased quality of relations (Segrin, Powell et al. 2003; Maher, Mora et al. 2006; Lasgaard, Goossens et al. 2011). For example, Segrin and colleagues (2003) investigated the association between depressive symptoms, relational quality and potential emotional loneliness in 101 dating couples among university students and found that depressive symptoms were negatively associated with relational quality and that relational quality was negatively associated with loneliness (Segrin, Powell et al. 2003). Similarly, Maher et al (2006) investigated the effects of cognitive, mood, and somatic aspects of

depression on perception of social support and demands among older adults over a period of two years, and found that the cognitive component of depression predicted changes in perceived support and demand (Maher, Mora et al. 2006). Finally, Lasgaard and colleagues (2011) investigated the association between depressive symptoms, loneliness and suicide ideation among adolescents and reported how depressive symptoms predicted increased perceived loneliness over time but not the opposite. This calls for more research aimed at unravelling the association between social inclusion and depressive symptoms.

2.4 Alcohol consumption

2.4.1 Historical context

In literature and figurative art alcohol often accompanies the onset of melancholia among tormented fictional heroes and poets alike. In Greek mythology and poetry, melancholia was characterised by a series of behaviours including rage and closer contact with the gods and with one's own deepest emotions (Klibansky, Panofsky et al. 1964). In other words, melancholia freed the mind from rationality and connected man with the truth of the misery of human nature, which was considered the only way to poetic genius (Klibansky, Panofsky et al. 1964). This connection with the deepest feelings and emotions could have been obtained also through inebriation given by consumption of wine. So much so, that Dionysus, the god of wine, was also the god of inebriation, wild emotion and everything that is passionate rather than rational (Klibansky, Panofsky et al. 1964).

This was described perfectly in Euripides' tragedy Bacchae. The tragedy sees the king of Thebes, Pentheus, being visited by a handsome stranger who is nonetheless than Dionysus in disguise. Pentheus is unaware of being Dionysus cousin and is known for being a rational man who bases all his decision on rules, reason and tradition. When Dionysus visits Thebes, preceded by his cult, Pentheus opposes him claiming that inebriation will bring chaos to the city. However Dionysus talks his cousin into dismissing rationality, connecting to his deeper and darkest wishes and spying on the women who had joined the new cult. This ends in tragedy as the women literally rip Pentheus body apart (Euripides 2000).

The idea of alcohol induced inebriation as being both liberating and conductive to tragedy was rediscovered and reinterpreted by German philosopher Friedrich Nietzsche, who in his 1872 essay on Greek tragedy identified the source of art in the struggle between what he called the Dionysian and the Apollonian (Nietzsche 1872 [2000]). In this essay, Nietzsche introduced the intellectual dichotomy between Dionysian and Apollonian, two concepts that take their names from the Greek gods of wine and inebriation and light and rationality respectively (Nietzsche 1872 [2000]). Hence, the Dionysian represents all that is dark, wild, related to death, passionate, irrational, unordered, unshaped; while the Apollonian represents all that is light, orderly, rational, formed, related to life (Nietzsche 1872 [2000]). After introducing these concepts, Nietzsche claimed life and art are a constant struggle between Apollonian and Dionysian, and the Greek tragedy in its highest form is the perfect example of this existential struggle creating perfect art (Nietzsche 1872 [2000]).

Nietzsche's ideas were but one representation of the artistic mood of his time, when Romanticism was ruling the world of the arts and the conception of the artist had evolved to incorporate sadness and inebriation as some of the main characteristics of an artistic mind (Abbagnano and Fornero 2003). In fact, Romantic artists were, or liked to represents themselves as, tormented souls, living uneasily in their time and society, naturally excluded from social circles, always looking for more and prone to wander desolated landscapes as well as the desolation of their inner sadness (Abbagnano and Fornero 2003). Romantic artists would happily indulge in excessive drinking to mitigate their sorrows, finding in alcoholic inebriation the inspiration for many of their artistic works.

2.4.2 Defining alcohol consumption

After the end of romanticism and the advent of a more scientific framework of thought, a vast body of research has linked alcohol consumption to ill health in numerous ways (Rehm, Room et al. 2003; Wannamethee and Shaper 2003; Poschl and Seitz 2004; Boffetta and Hashibe 2006; Klatsky 2009; Wang, Lee et al. 2010). In fact, heavy alcohol consumption, both regular and occasional, has been associated with increased risk of all-causes mortality (Power, Rodgers et al. 1998; Rehm, Greenfield et al. 2001). In addition, in their study of the contribution of average volume of alcohol consumption and patterns of drinking to burden of disease, Lurgen Rehm and his colleagues (2003) reviewed the existing literature linking alcohol to a number of diseases and then run meta-analyses to assess the risk relationship between alcohol and disease (Rehm, Room et al. 2003). They included in the model only direct effects of alcohol on health, not including subsequent possible social exclusion or circumstances, and found that average volume of alcohol consumption increased the risk for a vast number of chronic diseases, including: mouth and oropharyngeal cancer; oesophageal cancer; liver cancer; breast cancer; epilepsy; hypertensive disease; hemorrhagic stroke; and cirrhosis of the liver (Rehm, Room et al. 2003).

However, there are two common chronic diseases on which the effects of alcohol are not quite so clear cut. These are coronary heart disease and depression. In fact, there is growing epidemiological evidence highlighting that regular light to moderate alcohol consumption protects against the risk of coronary heart disease through increasing the number of high-density lipoproteins and favourably affecting blood-clotting factors (Rehm, Room et al. 2003; Rehm, Sempos et al. 2003). This was first observed in Mediterranean countries such

as Italy and France, where it is customary to have a glass of wine with every meal and the rates of coronary heart disease are lower than expected (Rehm, Room et al. 2003; Marmot 2004). However, the Mediterranean style of consumption seems to be the only drinking pattern that plays a protective role against coronary heart disease, for any instance of heavy drinking as well as drinking outside of meals are associated with an increase in the risk of coronary heart disease (Rehm, Greenfield et al. 2001; Rehm, Room et al. 2003; Rehm, Sempos et al. 2003). The controversial association between alcohol consumption and depression will be addressed in detail in the next section.

Because of the established deleterious effects of alcohol, the NHS recommends to keep levels of consumption at a minimum. This minimum is considered to be 3 to 4 units per day for men and 2 to 3 units per day for women (NHS 2012). Alcohol units are a simple way of expressing the amount of pure alcohol in a drink and a single units of alcohol corresponds to 10ml or 8g of pure alcohol, which is the amount of alcohol that the average adult can process in an hour (NHS 2012). The amount of pure alcohol in a drink is usually indicated on bottles or cans near the abbreviation ABV which stands for alcohol by volume, or "sometimes vol", which is a measure of pure alcohol as a percentage of the total amount of liquid in a drink. For example, if a bottle of wine reads ABV 12% on the label it means that 12% of that bottle is pure alcohol. This would allow customers to calculate the units present in a drink simply by multiplying the total volume of a drink by its ABV and dividing by 1000. For example, a standard bottle of wine is 750ml, multiplied by 12%, divided by 1000, equals 9. Hence that bottle of wine contains 9 units (NHS 2012). However, given that not everybody wants to start doing math when having a drink, even if facilitated by technology, it is important to understand how many units are there in the most commonly

consumed alcoholic beverages. As a general rule of thumb, a small glass of wine contains 1.5 units, a medium one 2.1 and a large one 3 units. Three units are also found in a pint of higher strength beer, lager or cider, while 2 units are found in a pint of lower strength or a can of beer, lager or cider; and 1.7 units are found in a bottle of beer, lager or cider; an alcopop contains 1.5 units, and a single shot of spirit with a mixer contains 1 unit (Figure 2.4.1(NHS 2012).

Aside from the daily recommended dosage, there are situations in which it is deplorable to drink at all. For example the NHS strongly advises persons not to drink at all when driving or about to drive, and women are recommended not to drink when pregnant as absorption of alcohol through the placenta might damage the baby; if women do chose to drink during pregnancy, they are advised consume no more than two units once or twice a week (NHS 2012). People who consume alcohol according to these guidelines are considered at low risk, not safe, but at low risk from the deleterious effects of alcohol, such as cancer of the mouth, throat or breasts, liver cirrhosis and high blood pressure, and are advised to cut down their consumption. People who chose to drink more are considered at increasing or high risk (NHS 2012). Men who regularly consume between 5 and 7 units a day and women who consume between 4 and 6 units a day are considered by the NHS at increasing risk of the deleterious effects of alcohol and advised to reduce their consumption (NHS 2012). Men who regularly consume 8 or more units a day and women who consume 6 or more units a day are considered by the NHS of being at high risk of the deleterious effects of alcohol and advised to seek help immediately (NHS 2012). For, as well as increasing the risk

of other diseases, alcohol misuse is a mental disorder in its own right and as such should be treated (Rehm, Room et al. 2003).

Figure 2.4.1 *Alcohol units in the most common drinks*

From: http://www.nhs.uk/Livewell/alcohol/Pages/alcohol-units.aspx#t

There is general consensus supporting the existence of two types of alcohol use disorders: alcohol abuse and alcohol dependence. Alcohol abuse refers to a pattern of hazardous drinking which recurrently results in significant and adverse consequences and can lead individuals to fail to fulfil major work, school or family obligations. Alcohol abusers might also have recurrent legal or relationship problems induced by their drinking (APA website). Alcohol dependence, commonly known as alcoholism, on the other hand refers to the loss of reliable control over alcohol use. Alcohol dependent people are often unable to stop drinking once they have started and they face withdrawal symptoms if drinking is suddenly stopped, these symptoms included nausea, sweating, restlessness, irritability, tremors, hallucinations and convulsions (APA website).

2.4.3 Alcohol consumption and Depressive Symptoms

The relationship between alcohol consumption and depressive symptoms is multifaceted. In fact, alcohol use disorders and depressive symptoms have been shown to co-exist and to be comorbid but there is no definitive evidence supporting causality in either direction (Khantzian 1997; Rehm, Room et al. 2003; Boden and Fergusson 2011). However, the association between non-pathological patterns of alcohol use such as light, moderate or social consumption and depressive symptoms, has been repeatedly found to be shaped as a J or U (Lipton 1994; Fergusson, Boden et al. 2009; Boden and Fergusson 2011). This section will explore these issues, reviewing the existing literature on the topic.

Both alcohol abuse and alcohol dependency disorders have been repeatedly found to be highly comorbid with depressive symptoms (Rehm, Room et al. 2003; Boden and Fergusson 2011). For, several epidemiological studies carried out both on clinical and community samples have consistently reported that alcohol use disorders and depression co-occur to a degree that is higher than chance (Rehm, Room et al. 2003; Fergusson, Boden et al. 2009; Boden and Fergusson 2011), and that patients suffering from alcohol dependence have been shown to be at twice or even thrice higher risk of depressive symptoms than persons not affected by such disorder (Merikangas, Metha et al. 1998; Rehm, Room et al. 2003). Researchers have identified three main possible explanations for this comorbidity: (i) alcohol abuse increases the risk of depression by promoting inception, duration and recurrence of the disorder; (ii) depression leads to increased alcohol consumption as a form of self-medication, and to the persistence of alcohol dependence; and (iii) there are

common environmental or genetic determinants of both depression and alcohol abuse (Rodgers, Korten et al. 2000; Fergusson, Boden et al. 2009; Boden and Fergusson 2011). Indeed there is evidence to support all three mechanisms and the issue of direction of the association has not yet been definitively solved (Markou, Kosten et al. 1998; Merikangas, Metha et al. 1998; Gilman and Abraham 2001; Falk, Yi et al. 2008; Boden and Fergusson 2011).

If the issue is causality in the relationship between alcohol use disorders and depressive symptoms still remains unsolved, the pattern of association between non-pathological drinking patterns and depressive symptoms has consistently be found to be J or U shaped (Lipton 1994). Most people will indulge in a drinking pattern that lies somewhere between not drinking at all, having a drink with their meal, drinking only in social contexts, or even having a couple when stressed or tired, for after all alcohol has a sedative effect on the brain which momentarily helps relaxing and feeling calm (Khantzian 1997; Bolton, Robinson et al. 2009). In all these cases, people who drink moderately have been repeatedly found to be at a lower risk of depressive symptoms than both alcohol abusers and abstainers (Lipton 1994; Power, Rodgers et al. 1998; Rodgers, Korten et al. 2000; Rodgers, Korten et al. 2000; Caldwell, Rodgers et al. 2002; Skogen, Harvey et al. 2009).

Skogen and colleagues (2009) investigated the association between abstention versus low-alcohol consumption and depression and anxiety in population survey over a period of two weeks. They found a U shaped association between alcohol consumption and risk of anxiety and depression, as abstention was associated with increased odds of both disorders. In

addition, when differentiating self-reported abstainers from participants who reported drinking normally but not in those two weeks, abstainers were at a higher risk (Skogen, Harvey et al. 2009). The association was partly but not fully, accounted for by socio-economic status, social activity, somatic illness, age, gender and possible abandonment of alcohol consumption due to advent of disorders caused by excessive drinking (Skogen, Harvey et al. 2009). This U or J shaped association appears throughout the literature despite the different definition of heavy, moderate or light consumption (Boden and Fergusson 2011). This is all the more interesting because it reflects the shape of the association between alcohol consumption and mortality (Peele and Brodksy 2000), which could suggest that depression may be the link between alcohol consumption and mortality (Rodgers, Korten et al. 2000).

Figure 2.4.2 *Skogen et al (2009) odds ratio for depressive symptoms among abstainers and low level drinkers*

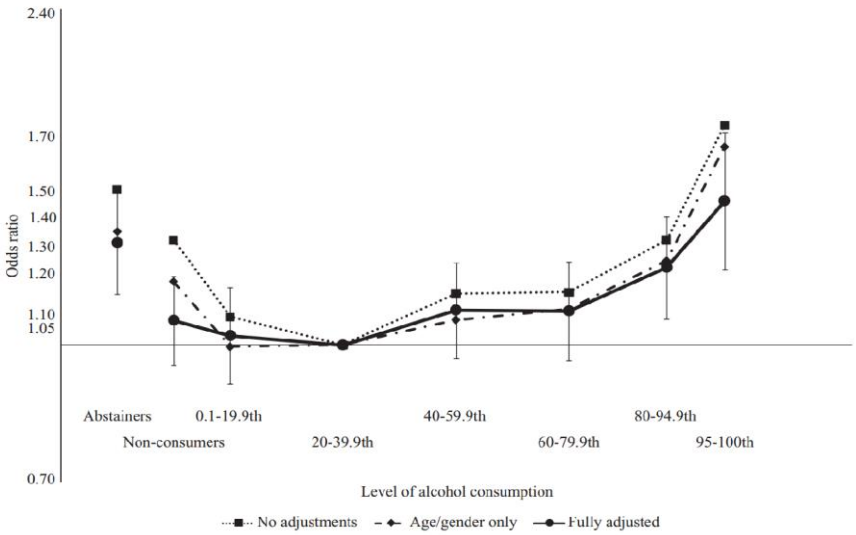


Figure 1 Unadjusted and adjusted odds ratio for case-level anxiety among different levels of alcohol consumption. Participants who deny alcohol abstinence were separated into groups based on gender specific percentiles of total alcohol consumption. The group with the lowest prevalence of case-level anxiety has been used as the reference group. Bars denote 95% confidence intervals for the fully adjusted model. The *y*-axis is log-scaled

Several explanations have been put forward for the shape of the association between alcohol consumption and depression. Lipton (1994) found that habitual moderate drinkers cope better with life events than abstainers or heavy drinkers and suggested a possible stress buffering role of moderate drinking. Another possible explanation, the so called "sick quitter" effect, sees the association to be skewed by the fact that abstainers are likely to be ex-heavy drinkers, thus having already been affected by alcohol and quit for health related reasons (Power, Rodgers et al. 1998; Rodgers, Korten et al. 2000). However, a number of studies showed that this hypothesis could not entirely account for the pattern observed (Alati, Lawlor et al. 2005; Rodgers, Parslow et al. 2007; Skogen, Harvey et al. 2009). For example, Rose Aalati and colleagues (2005) investigated the association between alcohol consumption and depressive symptoms among a cohort of women, with measurements taken at baseline, 5-years follow up and 14-years follow up. They found a J shaped association between alcohol consumption and depressive symptoms at the 5-years follow up, which became a positive linear association at the 14-years follow up with prevalence of depressive symptoms increasing with greater consumption (Alati, Lawlor et al. 2005). They also found that at the 5-years follow up prevalence of depressive symptoms was very similar among women who had been abstainers since baseline, and among women who had consumed alcohol and then quit. Similarly, at the 14-years follow up prevalence of depressive symptoms was the same among participants who had been abstainers all along, and women who used to consume alcohol and then stopped (Alati, Lawlor et al. 2005). Hence, the 'sick-quitter' hypothesis does not seem to be supported by data.

A third explanation proposes that abstainers and heavy drinkers are similar in other personal characteristics that favour drinking behaviours and at the same time are related to the onset of depression. These characteristics include education, economic circumstances - as both abstainers and heavy drinkers tend to be less educated and economically disadvantaged -, personality traits such as sociality and enthusiasm, and levels of social support (Peele and Brodksy 2000). Indeed, Pattenden and colleagues (2008) investigated the association between self-reported never drinking and living alone, and educational qualification among English adults, using data from the Health Survey for England. The results showed that never drinking was strongly associated with living with another adult and lower educational qualification (Skogen, Harvey et al. 2009). Lower levels of education were associated to increase risk of hazardous drinking as well. Tomkins and colleagues (2007), investigated hazardous drinking among Russian men aged 25-54 and reported that men with the lowest levels of education had the highest odds of hazardous drinking. They also reported low levels of education to be independently associated with unemployment (Tomkins, Saburova et al. 2007).

Finally, abstainers may be at increased risk of depressive symptoms because they have poorer social relationships than moderate drinkers (Peele and Brodksy 2000; Lucas, Windsor et al. 2010). Lucas and colleagues (2010) used data from an Australian national cross-sectional survey among men aged 20-22, 30-32 and 40-42 to investigate whether the higher distress experienced by abstainers compared to light and moderate drinkers was due to (i) the presence of many ex-drinkers among abstainers and (ii) abstainers having poorer social relationships than light/moderate drinkers (Lucas, Windsor et al. 2010).

Indeed, they found that among men aged 40-42 years abstainer were less socially integrated, less extroverted and have lower social support than light/moderate drinkers, and this partially explained their increased distress (Lucas, Windsor et al. 2010).

2.4.4 Gender differences

Patterns of alcohol consumption are predicted not only by education or socio-economic circumstances, but also by gender. In fact, a vast body of evidence has consistently being reporting how women tend to consume smaller quantities of alcohol compared to men and less frequently (Mumenthaler, Taylor et al. 1999; Wilsnack, Vogeltanz-Holm et al. 2000; Nolen-Hoeksema 2004; Wilsnack, Wilsnack et al. 2009). Two main theories have been put forward to explain this marked gender difference. The first sees women drinking less because they are physically unable to consume as much as alcohol as men do (Mumenthaler, Taylor et al. 1999). The second sees women drinking less because of social constrictions and rules that influence their behaviour (Nolen-Hoeksema and Harrell 2002; Makela, Gmel et al. 2006). In addition, gender differences have been highlighted also in the way in which patterns of alcohol consumption affect the risk of depressive symptoms (Makela and Mustonen 2000; Nolen-Hoeksema and Ahrens 2002; Nolen-Hoeksema 2004). All these gender related differences will be the topic of this section.

Gender differences in alcohol consumption could be due to biological differences in the way in which men and women assimilate and process alcohol (Mumenthaler, Taylor et al. 1999). In their review of the existing literature on the topic, Mumenthaler and colleagues (1999) identified two main biological mechanisms that could explain the gender differences in alcohol consumption and effects of alcohol on health. These mechanisms are: (i) gender differences in the physiological processing and elimination of alcohol (pharmacokinetics) and (ii) differential sensitivity of the nervous system to the effects of alcohol (Mumenthaler, Taylor et al. 1999). In regard to pharmacokinetics, women are known to have more body

fat and lower body water than men, and because alcohol is dispersed mainly in body water, women reach higher blood alcohol concentration (BAC) than men after consuming similar amounts of alcohol (Mumenthaler, Taylor et al. 1999). For what concerns differential neuro-sensitivity to alcohol, Mumenthaler et al (1999) reported that after consuming similar amounts of alcohol the ability to divide attention between two or more sources of visual information was more impaired in women than in men, however no gender differences were found in the alcohol induced impairment of psychomotor function or memory loss (Mumenthaler, Taylor et al. 1999).

In recent years a number of studies have been highlighting how biological differences in alcohol consumption might be magnified by gender roles and social sanctions (Wilsnack, Vogeltanz-Holm et al. 2000; Nolen-Hoeksema 2004; Holmila and Raitasalo 2005; Wilsnack, Wilsnack et al. 2009). As Wilsnack and colleagues (2000) pointed out, humans tend to interpret and codify minor biological differences into systematic and absolute categorisations of people and behaviours; in particular, all societies have codified presumed biological differences between the sexes into markedly different behaviours for men and women (Wilsnack, Vogeltanz-Holm et al. 2000). In particular, the uniquely female biological ability to bear children has been codified into a number of social behaviours and characteristics that shape women's life. For this reason, women are almost always considered to possess 'feminine' characteristics, which include a nurturing and caring nature, and an aversion to excesses, violence and risk taking (Wilsnack, Vogeltanz-Holm et al. 2000; Nolen-Hoeksema 2004; Holmila and Raitasalo 2005). Indeed, there is evidence that women are less likely to drink heavily than men, and when they do they are less likely

to show aggressive and antisocial behaviours, sensation-seeking and behavioural wildness (Nolen-Hoeksema 2004). In addition, when women do chose to drink, they encounter greater social sanctions and judgement (Nolen-Hoeksema 2004; Holmila and Raitasalo 2005). However, there is also evidence that in countries with a greater gender equality and women emancipation, the social sanctions against women who consume alcohol in excess are loosening and gender differences in patterns of alcohol consumptions are reducing (Bloomfield, Gmel et al. 2001; Makela, Gmel et al. 2006).

In addition, men and women also seem to differ in their motives for - and expectations about alcohol consumption. For example, in their study of the Finnish population, Makela and Mustonen (2000; Room and Makela 2000) highlighted how women reported turning to alcohol in order to able to better express their feelings, sort out interpersonal relationships at home or on the work place and feeling more optimistic about life when inebriated. Men, on the other hand, reported feeling funnier, wittier and able to get closer to the other sex as their main expectations from alcohol consumption (Makela and Mustonen 2000). Similarly, Mulligan Rauch and Becker Bryant (2000) found that young adult men drink more in the context of social facilitation, while young adult women consume more alcohol in the context of emotional pain (Mulligan Rauch and Becker Bryant 2000). However, there is also some evidence that when people consume alcohol as a coping mechanism, men experience stronger negative effects of alcohol than women. For example, in their study of college students Markman Geisner et al (2004) reported a stronger association between psychological distress and negative drinking consequences in men than in women.

2.4.5 Depressive symptoms and alcohol consumption

The NHS suggests that individuals who regularly consume more alcohol than the recommended doses of 3-4 daily units for men and 2-3 daily units for women, might feel depressed and are more at risk of depression (NHS 2012); and in their review of the existing literature on alcohol use disorder and depression, Boden and Fergusson (2011) revealed how most of the existing studies report alcohol use disorder to be a risk factor for depression. However, there is a substantial amount of evidence suggesting that individuals who are already suffering from depression are more likely to abuse of alcohol (Khantzian 1997; Markou, Kosten et al. 1998; Dixit and Crum 2000; Bolton, Robinson et al. 2009). This fact has been explained with the 'self-medication' hypothesis, which states that patients suffering from depression use alcohol as a way to alleviate their psychological distress (Khantzian 1997; Markou, Kosten et al. 1998).

The self-medication hypothesis was first introduced by Edward Khantzian (1997), who suggested that as alcohol creates the illusion of relief from psychological suffering because it temporarily softens mental defences and ameliorates states of isolation and emptiness that are characteristics of depression. Hence individuals who suffer from depression are likely to try and find refuge in the illusions that alcohol creates (Khantzian 1997). Athina Markou and colleagues (1998), tested this hypothesis through a study of the neurotransmitters affected by drug use disorders and by depression in the attempt to investigate the neurological mechanisms underlying both depression and drug use disorders. They suggested that depression and drug use disorders affect the same

neurotransmitters, and drugs such as alcohol have the power to enhance neurotransmission thus acting as antidepressants (Markou, Kosten et al. 1998).

The self-medication hypothesis has been supported by epidemiological studies as well as neurological evidence. For example, Bolton et al (2009) used data from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) to test the self-medication hypothesis. They reported how 24.1% of individuals with mood disorders used alcohol or drugs to relieve their symptoms, in particular 41.0% of self-medication was found among participants who suffered from depression (Bolton, Robinson et al. 2009). In addition, Bolton and colleagues (2009) found that men were more than twice as likely than women to engage in self-medication.

Similarly, Dixit and Crum (2000) investigated whether depression was associated with a greater risk of heavy alcohol consumption in women using data from the Baltimore cohort of the National Institute of Mental Health Epidemiologic Catchment Area project, and found that the risk of heavy alcohol consumption was 2.6 times higher among women with a history of depression than among women without such history. Further, Dixit and Crum found that a higher frequency in depressive symptoms was also associated with an increase in the risk of for heavy alcohol use (Dixit and Crum 2000). This evidence suggests that the association between alcohol consumption and depression is a complex and bi-directional one, and that more studies are required in order to better understand the mechanisms underlying the interplay between depression and alcohol.

2.5 Inclusion in Social Networks, Social Support, Alcohol Consumption and Depressive Symptoms

2.5.1 Introduction

This sub-chapter will look at the association between social capital, inclusion in social networks, social support and depressive symptoms. Indeed, in section 2.4.5., I have already reviewed literature supporting the fact that gender difference in alcohol consumption are defined by social roles and norms, but this is not the only case in which social norms affects alcohol consumption, as there is evidence to support the notion that social life affects alcohol consumption at various levels. In fact, we can apply Berkman and Glass (2000) cascade model introduced in section 2.2 to how social associations affect alcohol consumption.

At the macro, or country level, cultural norms and traditions are thought to affect the frequency and volume of individual alcohol consumption through social comparison and unspoken rules and permissions (Levine 1992; Room and Makela 2000; Bloomfield, Gmel et al. 2001; Bloomfield, Grittner et al. 2006; Rahav, Wilsnack et al. 2006; Joosten, Knibbe et al. 2009). Within this wider context, at the intermediate level, specific social networks have their own rules of what represents acceptable or unacceptable drinking patterns and provide behavioural guidance (Room and Makela 2000). But perhaps more importantly social groups influence individual alcohol consumption through social influence/social comparison and social control; or in other words the drinking pattern of a social group will be copied by its individual members and variations from the standard pattern will be actively controlled by the group (Monahan and Lannutti 2000; Borsari and Carey 2001;

Rimal and Real 2005; Makela, Gmel et al. 2006; Kuntsche, Knibbe et al. 2009). Moreover, there is evidence that individual patterns of alcohol consumption might affect the levels of social integration enjoyed by the drinker, as drinking too much or too little may result in exclusion from a social group (Rimal and Real 2005; Ahern, Galea et al. 2008; Ahern and Galea 2011). Finally I will review the literature regarding how inclusion in social networks and social support interact with alcohol consumption to affect the risk of depressive symptoms (Peirce, Frone et al. 2000; Allgower, Wardle et al. 2001; Buu, Wang et al. 2011).

2.5.2 Social Capital and Alcohol Consumption

All societies have their own, peculiar, relationship to alcohol partly determined by cultural norms, partly by daily associations, partly by structure of the society which provides the setting in which individual norms and attitudes towards alcohol are formed (Room and Makela 2000). To this date, several attempts have been made to categorise different patterns of alcohol consumption at the country level. Traditionally, studies on how national cultural norms influence alcohol consumption have used European countries as their settings, and divided them into 'wet' and 'dry' cultures (Room and Makela 2000; Joosten, Knibbe et al. 2009). 'Wet' cultures are usually identified with the Mediterranean countries in which wine is produced in vast amounts and regularly consumed with meals. Researchers have speculated that because wine is produced in Mediterranean countries it represents a source of income and hence is conceived as something precious. Hence, in these 'wet' societies, alcohol is consumed regularly but drunkenness if frowned upon because considered a waste (Room and Makela 2000; Joosten, Knibbe et al. 2009).

'Dry' cultures on the other hand, are usually identified with countries of Northern Europe and are characterised by a high proportion of abstainers but also by infrequent and very heavy drinking as the dominant pattern of heavy drinking and by higher violence and social disruption associated with drinking (Room and Makela 2000). Further, Harry Levine (1992) observed that 'dry' cultures also have in common the Protestant religion and postulated Protestantism may be conductive to heavier drinking because it traditionally emphasises on self-regulation and self-control, rather than on strict behavioural norms as Catholicism does (Levine 1992). An association between religion and prevalence of drinking was also

found by Rahav and colleagues (2006). Rahav and colleagues also found that prevalence of drinking was strongly associated with urbanization and economic development (Rahav, Wilsnack et al. 2006).

However, there is evidence that this dichotomous categorisation into 'wet' and 'dry' countries is somewhat reductive of the actual problem. In fact in recent years, national patterns of alcohol consumption have evolved, with Mediterranean countries increasingly drinking more and more spirits and beer and seeing considerably more social disruption due to alcohol, and per-capita levels of alcohol consumption converging in European countries (Room and Makela 2000). In addition, the traditional categorisation in 'wet' and 'dry' countries looked at male consumption, almost taking for granted that women would consume less and less often than men. This is of course partly true, but it is also true that a dramatic convergence in male and female patterns of consumption has been observed in countries where women enjoy higher levels of equality and social integration (Bloomfield, Gmel et al. 2001; Bloomfield, Grittner et al. 2006).

Therefore, more recent and flexible approaches to cultural differences in alcohol consumption focus on a number of different dimensions of drinking such as the cultural take on the drinker, the drinking group and the drinking occasion (Room and Makela 2000). In other words, researchers are taking into account the degree to which drinking is integrated with other aspects of social life with particular interested at which social groups, usually differentiated on the basis of age, gender and social status, are allowed to drink at all and how much intoxication is permitted (Room and Makela 2000). Another dimension

that is considered in new typological approaches is the role played by modes of social control on drinking, as different countries have adopted different tactics in their tackling of alcohol consumption and alcohol related problems such as alcohol induced diseases, injuries and disruptive or violent behaviours. The most famous example of how a country has tried to preventing its citizens to drink in excess, or at all what matters, is the Prohibition applied in the United States from 1920 to 1933 with catastrophic results as the rates of crime spiralled up as a consequence of the overarching ban on alcohol (Reference). More recently, Scandinavian countries have introduced strict regulations on the amount of alcohol purchasable by an individual per shopping occasion in the attempt to limit excessive drinking (Room and Makela 2000). However, while country level interventions on alcohol consumption can have mixed results, network levels norms and traditions regarding drinking are thought to be more consistently effective in regulating individual consumption (Rimal and Real 2005).

2.5.3 Inclusion in Social Networks and Alcohol Consumption

Social networks affect alcohol consumption in much the same ways in which they affect mental health. In fact, individual consumption is shaped by: *social influence and social comparison,* as persons will adapt their consumption to the patterns of drinking observed in their social group; *social control*, as the members of a social network can actively endeavour to ensure individuals follow the accepted patterns of consumption; and *behavioural guidance, purpose and meaning*, as the fulfilment of particular social roles will allow and justify different types of drinking patterns. This section will address how each of these mechanisms helps to shape individual alcohol consumption.

Social influence/social comparison, is the mechanisms through which individuals obtain normative and behavioural guidance by comparing themselves with the other members of their social groups (Thoits 2011). *Social influence/social comparison* is particularly powerful in the case of alcohol consumption as the different attitudes towards alcohol and codified patterns of consumption found in different social groups are often a statement of the identity of that group (Makela, Gmel et al. 2006). For instance, Keyes and Hasin (2008) investigated the relationship between alcohol abuse and income and found that of the indicators of alcohol only abuse hazardous drinking, was positively associated with income. Or in other words individuals with high personal income were more likely to engage in hazardous drinking patterns such as drinking before or during driving (Keyes and Hasin 2008). Moreover, in their recent cross-sectional study of patterns of alcohol consumption in Welsh neighbourhoods, Fone and colleagues (2013) found that neighbourhood deprivation was strongly associated with prevalence of binge drinking, regardless of

individual socio-economic status (Fone, Farewell et al. 2013). All these studies show how belonging to a specific social group affects patterns of alcohol consumption through comparison, for example being part of the high income group somehow allows people to believe that it is acceptable to drink and drive, or living in a neighbourhood in which binge drinking is tolerated and highly prevalent induce individuals to be more prone to binging (Bloomfield, Grittner et al. 2006; Keyes and Hasin 2008; Fone, Farewell et al. 2013).

Social control refers to active influence of individual behaviours from other network members (Thoits 2011). Social control can be preventive, when members of the group are actively encouraged to adopt a certain behaviour, or it can be a reaction to an unwanted behaviour (Thoits 2011). For what concerns alcohol consumption, there is evidence to support the idea that preventive social control can influence individual patterns of drinking. For example, in their review of the existing literature on peer pressure and drinking in university students Borsari and Carey (2001), showed how two of the most powerful weapons of peer pressure are offers of alcohol, both as polite gestures and as commands to imbibe, and perceived social norms which allow students to think that excessive drinking is not only acceptable but required of them (Borsari and Carey 2001). Similarly, Ahern and colleagues (2011) found that neighbourhood norms about drunkenness were strongly related to individual drinking behaviours. More specifically, even individuals who believed it acceptable to drink heavily were less likely to binge if living in a neighbourhood with strong norms against drunkenness, for the norms against drunkenness were enforced in the form of social disapproval and marginalisation (Ahern, Galea et al. 2008; Ahern and Galea 2011). Social networks rules on alcohol are so strong that Ahern and colleagues

(2008) found that the association between neighbourhood and individual drinking pattern were entirely accounted for by network and individual drinking rules (Ahern, Galea et al. 2008).

Behavioural guidance, purpose and meaning is the mechanism through which individuals who identify themselves in a specific social role will adopt the behaviours that are associated with that role (Thoits 2011). Evidence of how social roles influence alcohol consumption can be found in the universal gender differences in alcohol consumption. In fact, traditionally women in all society are identified with the social role of the mother and hence expected avoid intoxication and hazardous behaviours, favouring caring and loving life styles (Nolen-Hoeksema 2004). As a result, women who conform to this stereotype do in fact drink less than men (Wilsnack, Vogeltanz-Holm et al. 2000; Wilsnack, Wilsnack et al. 2009). However, in recent years women in many European countries have chosen not to conform to the traditional social role and adopted behaviours which are similar to those of men, including increased alcohol consumption (Makela and Mustonen 2000; Makela, Gmel et al. 2006; Bloomfield 2006). Another example of how social roles influence individual alcohol consumption are university students. Rimal and Real (2005) observed how upon entering university students drinking patterns changed accordingly to the drinking culture reigning on campus in general and in specific clubs and societies. Interestingly, Rimal and Real (2005) noted that individual patterns of consumption were affected only partially by peer pressure, and changed largely because students felt the need to conform to the surrounding drinking culture in order to be accepted in the new environment (Rimal and Real, 2005).

2.5.4 Alcohol Consumption and Inclusion in Social Networks

If social networks can influence individual drinking patterns through active or passive mechanisms, individual consumption has also been shown to hinder or enhance a person's inclusion in a specific network. This has been observed to occur through two main mechanisms: positive expectations, and social anxiety. In addition, different patterns of consumption are linked to varying levels of social inclusion. This section will explore how positive expectations and social anxiety operate and what drinking patterns influence social life and how.

Positive expectations, refer to the belief that alcohol would reduce social anxiety, induce self-confidence and sociability and ultimately promote social acceptance (Rimal and Real, 2005). In fact, a number of studies have reported that indeed this is the most common reason why people engage in moderate drinking (Monahan and Lannutti 2000; Kuntsche, Knibbe et al. 2005; Peters and Stringham 2006; Kuntsche, von Fischer et al. 2008). Positive expectations about the effects of alcohol on a person's performance in social settings means that people will engage in drinking in the hope it will help to overcome their social fears and inhibitions and facilitate social associations (Monahan and Lannutti 2000; Kuntsche, Knibbe et al. 2005; Peters and Stringham 2006; Kuntsche, von Fischer et al. 2008).

Excessive alcohol consumption on the other hand has been found to be associated with negative expectations about alcohol, when individuals imbibe for coping motives, hoping in the mood stabilising effects of alcohol (Lewis and O'Neill 2000; Loukas, Krull et al. 2000; Lyvers, Hasking et al. 2010). Excessive consumption also appears to be associated with

greater social anxiety and loneliness. In other words, not only individuals who drink in excess are more likely to be socially excluded, but it would seem they are already socially anxious and awkward (Bonin, McCreary et al. 2000; Koppes, Twisk et al. 2001; Korn and Maggs 2004; Clerkin and Barnett 2012). Conversely, Korn and Maggs (2004), looked at reasons for drinking or abstaining in a college setting and found that the most diffident students did not drink alcohol, despite believing it would have brought fun experiences. Similarly, Monahan and Lannutti (2000) found that young women with low social self-esteem were more likely to engage in flirtatious conversation with men when under the effects of alcohol. Further, Peters and Stringham (2006) looked at the association between drinking and higher earnings through increased social capital using data from the General Social Survey in the US. They observed how self-reported drinkers benefited from a 10-14 percent higher earning than abstainers; when looking further into the matter, men who reported drinking in social settings such as bars at least once a month had an additional 7 percent higher earnings. The authors concluded that drinking socially is linked to increased earnings through increasing social capital (Peters and Stringham 2006).

In their review of the literature regarding the psychological benefits of moderate alcohol consumption, Peele and Brodsky (2000) reviewed a number of studies providing mix evidence of how drinking in social setting affects consumption and effects of alcohol. Smith et al, (1975) reported how individuals drinking in social settings reported great effects of alcohol even when consuming minimal amounts. Pliner and Cappell (1974) found that subjects drinking in social contexts experienced greater euphoria than when drinking the same amount alone. Doty and de Wit (1995) suggested that when in social settings

individuals were more likely to choose alcoholic than non-alcoholic beverages, and had more positive reactions than those drinking in isolated contexts (Doty and de Wit 1995). Despite evidence that alcohol is more effective when consumed in social settings, there is also evidence that social drinking promotes heavy rather than moderate consumption, especially during celebrations (MacAndrew and Edgerton 1969; Peace 1992).

2.5.5 Inclusion in Social Networks, Social Support, Alcohol Consumption and Depressive Symptoms

The association between social inclusion, alcohol consumption and depressive symptoms has surprisingly received very little attention in the literature. Peirce and colleagues (2000) examined the longitudinal relations among contacts with social networks, perceived social support, depression and alcohol use. They found depression to be associated with increased alcohol consumption and alcohol consumption to be associated with decreased social contacts and perceived support; decreased social contacts and perceived support were in turn associated with depression. Allgower and colleagues (2001) also looked at the relationship between depressive symptoms, social support and a number of health behaviours including alcohol consumption among college students. They found habitual alcohol consumption to be more common among individuals with high rather than low social support, and depressive symptoms to be negatively correlated with social support. Further, they did not find any association between depressive symptoms and alcohol use specifically, but they found that depressive symptoms affected health behaviours independently of social support. More recently, Buu and colleagues (2011) looked at the effects of women's psychopathology history, social support, their husband's and children's symptomatology, family stress, and neighbourhood environment on their alcohol problems, antisocial behaviour and depression over 12 years. They observed how women's alcohol problems and antisocial behaviour decreased over time while their depressive symptoms increased. They also found a negative association between levels of support and depressive symptoms, and a positive association between neighbourhood residential instability and alcohol problems and depressive symptoms.

2.6 Evidence from the Whitehall II and HAPIEE cohort studies

2.6.1 Introduction

Life time prevalence of depression varies across countries. In western Europe alone it ranges from 9.9% in Italy and Germany, to 10.4% in Spain, to 21.0% in France (Bromet, Andrade et al. 2011). In previous sections I have addressed how social capital and importance of particular types of social networks also vary from country to country, according to traditions, economic circumstances and political participation (Rose 1995; Putnam 2000; Rose 2000). Perhaps the most famous example of this is the case of Russia and countries of the ex USSR, where the drastic political and economic change left a void where civil society should stand (Rose 1995; Rose 2000). Patterns of alcohol consumption also vary across countries according to production of alcoholic beverages, dietary culture and tolerance towards drunkenness (Wilsnack, Vogeltanz-Holm et al. 2000; Rahav, Wilsnack et al. 2006). Both social life, in the form of social capital, inclusion in social networks and social support, and alcohol consumption have been shown to affect the risk of depression (Brugha, Conroy et al. 1982; Lipton 1994; Cohen 2004; Brugha, Weich et al. 2005; Chan and Lee 2006; Bolger and Amarel 2007; Boden and Fergusson 2011; Croezen, Picavet et al. 2012).

Therefore, it could be that country variation in social capital and importance of different types of networks and in alcohol consumption influences the country variation in prevalence of depression. In order to test this hypothesis I will compare cross-sectional results from the UK and Russia, Poland and the Czech Republic using data from two cohorts: the Whitehall II cohort study involving British Civil Servants, and the HAPIEE cohort study

involving the adult urban population of Russia, Poland and the Czech Republic. Before analysing the data I will review the existing literature stemming from the two cohorts on the associations between inclusion in social networks and depressive symptoms, social support and depressive symptoms and alcohol consumption and depressive symptoms; trying to identify aspects of these associations that have not yet been addressed by previous research on the same data. The next few sections will review the existing literature coming from the Whitehall II and the HAPIEE cohort studies, starting with literature on the association between social support and depressive symptoms, followed by the literature on the association between inclusion in social networks and depressive symptoms, to finish with literature on the association between alcohol consumption and depressive symptoms.

2.6.2 Whitehall II

The Whitehall II cohort is a prospective cohort study involving British civil servants, which has been running since 1985. During the course of the twenty-nine years since the beginning of the cohort, participants have been regularly asked information about their participation in social networks and about the levels of social support they received. Social support has been measured through the close person questionnaire, a measure of support formally validated at the beginning of the study (Stansfeld and Marmot 1992). Studies on social support and psychiatric disorders in general, with attention to depression and anxiety in particular, have been carried out on Whitehall II data since the mid-late '90s and were largely the work of Stephen Stansfeld.

In the mid '90s Stansfeld and colleagues (1997) investigated the effects of support on the work place and outside of it on the risk of psychiatric sickness absence over a period of five year in a subsample of the Whitehall II cohort study. They reported how high levels of support from colleagues and work supervisors were associated with a lower risk of short spells of psychiatric sickness absence, while negative aspects of personal relationships with the closest person outside of work increased the risk of long spells of psychiatric sickness absence (Stansfeld, Rael et al. 1997).

The following year Stansfeld and colleagues published two articles, one was looking to explain the social gradient in depression and well-being (Stansfeld, Head et al. 1998), the other was further investigating the association between social support and psychiatric morbidity in general (Stansfeld, Fuhrer et al. 1998). In the first article, Stansfeld and

colleagues reported that a third of the social gradient in depressive symptoms could be explained by differences in support, as participants in the higher employment grades enjoyed more support and less stressful events than participants in the lower grades (Stansfeld, Head et al. 1998). In the second article, Stansfeld and colleagues (1998) investigated what types of social support were associated with increased risk of psychiatric morbidity, in a sub-sample of the cohort at baseline and second phase of follow up. They reported how low confiding/emotional support and high levels of negative aspects of relationships were associated with an increased risk of psychiatric morbidity, and how there was no evidence of a buffering effect of support (Stansfeld, Fuhrer et al. 1998).

Psychological distress was also the focus of attention in a subsequent paper by Fuhrer and colleagues (1999) which aimed at investigating gender differences on the relation between social support and relations and psychological distress. This paper highlighted how the effects of marital status, social support both within and outside the workplace and inclusion in networks on psychological distress were similar for men and women (Fuhrer, Stansfeld et al. 1999). Women were found to be under greater psychological distress than men, and to cope with this increased stress by relying on a higher number of close persons; while men enjoyed a greater immediate benefit from receiving emotional support from a close person such as a spouse and had in general larger social networks (Fuhrer, Stansfeld et al. 1999). Finally, Nabi and colleagues (2009) investigated whether hostile individuals were more likely to enjoy more interpersonal conflicts, less social support, more stressful life events and a greater likelihood of depressive symptoms. They reported how more hostile

individuals were indeed more likely to show depressive symptoms than their hostile counterparts (Nabi, Singh-Manoux et al. 2009).

For what concerns alcohol consumption, several studies carried out on Whitehall II data have investigated its effects on sickness absence (Marmot, North et al. 1993), cardiovascular disease (Britton and McKee 2000) and health in general (Britton 2002; Head, Martikainen et al. 2002; Britton 2006). Britton and colleagues (2006), focused on the association between alcohol consumption and cognitive function and reported how participants who admitted drinking in moderation were less likely to display poor cognitive function (Britton 2006). Finally Steven Bell (Bell et al, 2014) investigated the association between alcohol consumption and depressive symptoms and reported how within the Whitehall II cohort, individuals with better mental health were more likely to undertake greater reductions in their drinking pattern than participants who suffered or had suffered from depression or anxiety.

2.6.3 HAPIEE

The Health, Alcohol and Psychosocial factors In Eastern Europe study is a prospective cohort study started in 2002 which involves randomly selected adults from urban centres in Russia, Poland and the Czech Republic (Peasey, Bobak et al. 2006). During the twelve years of its life the study has focused primarily on determinants of all causes mortality and, in particular, of cardiovascular heart disease as the rates of the latter are particularly high in Central and Eastern Europe (Peasey, Bobak et al. 2006). Alcohol consumption has also been thoroughly investigated although almost exclusively in the light of its association with mortality or cardiovascular heart disease. However, a small number of studies investigated determinants of depression and I will review them here. Among these, Bell and colleagues (2014) investigated alcohol consumption as a risk factor for depressive symptoms in the HAPIEE cohort and found a twofold increase in odds of depressive symptoms in participants who engaged in hazardous drinking (Bell et al, 2014).

Martin Bobak and colleagues (2006) investigated the prevalence rates and distribution of depressive symptoms through cross-sectional analysis on data from the HAPIEE cohort study. They reported how depressive symptoms more likely to affect participants who found themselves in a situation of material depravation, and more importantly, participants who were not married (Bobak, Pikhart et al. 2006). Finally, Franchi et al (submitted) looked at the relationship between inclusion in social networks and depressive symptoms using cross-sectional data from the HAPIEE study, and showed how participants who reported not having any relatives outside their household or friends had higher odds of depressive symptoms than individuals who were more socially included.

2.7 Summary and gaps in the literature

This chapter was dedicated to introducing the theoretical models formulated to explain the effects of social life and alcohol consumption on health in general and depressive symptoms in particular, and to review the existing literature on the topic. In section 2.3.2 I introduced the modern theoretical model that sees social life affecting health at three different levels through social capital, inclusion in social networks and social support. Section 2.3.3 was devoted to define social capital and explain how it indirectly affects health by influencing individual socio-economic circumstances, social participation and inclusion in social networks. Section 2.3.4 introduced the seven mechanisms through which social networks directly affect health, briefly describing how social influence/ social comparison, social control, behavioural guidance, purpose and meaning, self-esteem, sense of control or mastery, and belonging and companionship affect health. Section 2.3.5 introduced the eighth mechanism through which social networks affect health: social support, describing the different types of support and the categories of people who can provide it, and mentioning that support can have a negative impact on health.

Section 2.3.6 reviewed the existing literature on how inclusion in social networks and social support affect the risk of depressive symptoms (Brugha, Conroy et al. 1982; Vilhajalmsson 1993; Hagerty and Williams 1999; Wildes, Harkness et al. 2002; Deelstra, Peeters et al. 2003; Brugha, Weich et al. 2005; Cacioppo, Hughes et al. 2006; Chan and Lee 2006; Bolger and Amarel 2007; Gleason, Mausmi et al. 2008; Hobfoll 2009; Croezen, Picavet et al. 2012) . Finally, section 3.3.viii reviewed the existing literature providing evidence that presence

of depressive symptoms can affect the levels of social inclusion of individuals, causing isolation (Segrin, Powell et al. 2003; Maher, Mora et al. 2006; Lasgaard, Goossens et al. 2011).

I then turned to defining alcohol consumption reporting the recommended daily amount to be consumed and the deleterious effects of excessive drinking on the body. Section 2.4.3 reviewed the existing literature on the association between alcohol consumption and depressive symptoms, which highlighted how particularly heavy alcohol consumption is a mental disorder in its own right and highly comorbid with depressive symptoms (Boden and Fergusson 2011). However, according to the existing literature among individuals who do not suffer from problem drinking, the association between alcohol consumption and depressive symptoms is J or U shaped, with moderate drinkers being at a lesser risk than both heavier drinkers and abstainers (Lipton 1994; Rodgers, Korten et al. 2000; Rodgers, Korten et al. 2000; Caldwell, Rodgers et al. 2002; Alati, Lawlor et al. 2005; Rodgers, Parslow et al. 2007; Skogen, Harvey et al. 2009; Lucas, Windsor et al. 2010). I then reviewed the existing literature providing evidence that depressive symptoms could trigger heavy alcohol consumption rather than heavy alcohol consumption leading to depressive symptoms (Khantzian 1997; Markou, Kosten et al. 1998; Dixit and Crum 2000; Bolton, Robinson et al. 2009).

Sub-chapter 2.5 looked at the association between social life and alcohol consumption. In section 2.5.2 I reviewed the evidence showing how cultural views of alcohol at the country level and norms on how much is it socially acceptable to drink influence individual

consumption (Levine 1992; Room and Makela 2000; Bloomfield, Stockwell et al. 2003; Bloomfield, Grittner et al. 2006; Rahav, Wilsnack et al. 2006). Section 2.5.3 reviewed the literature on how social networks actively influence their members alcohol consumption through social influence/social comparison and social control (Wilsnack, Vogeltanz-Holm et al. 2000; Borsari and Carey 2001; Nolen-Hoeksema 2004; Rimal and Real 2005; Bloomfield, Grittner et al. 2006; Makela, Gmel et al. 2006; Ahern, Galea et al. 2008; Keyes and Hasin 2008; Wilsnack, Wilsnack et al. 2009; Ahern and Galea 2011; Fone, Farewell et al. 2013).

Section 2.5.4 reviewed the literature on how alcohol consumption can determine individual inclusion in social networks, as people have been shown to consume alcohol in order to be more socially appealing and conversely individuals whose drinking patterns do not conform to the ones considered acceptable in their network can be isolated in response (Bonin, McCreary et al. 2000; Lewis and O'Neill 2000; Monahan and Lannutti 2000; Koppes, Twisk et al. 2001; Korn and Maggs 2004; Kuntsche, Knibbe et al. 2005; Peters and Stringham 2006; Kuntsche, von Fischer et al. 2008; Lyvers, Hasking et al. 2010; Clerkin and Barnett 2012). Finally, I reviewed the few existing studies which focused on the association between social support, alcohol consumption and depressive symptoms. Of the studies reviewed, one reported how depressive symptoms were associated with later increased alcohol consumption which in turn was associated with higher levels of social isolation (Peirce, Frone et al. 2000). A second study reported alcohol consumption to be higher among individuals who enjoyed high levels of social support while depressive symptoms were more frequent among individuals who received low levels of support (Allgower, Wardle et al. 2001).

I then reviewed the evidence on the topic stemming from the two cohort studies whose data I will be using, the Whitehall II cohort study and the HAPIEE cohort study. Several studies were carried out on Whitehall II data in the '90s, investigating the association between psychiatric disorders and social support at work (Stansfeld, North et al. 1995; Stansfeld, Rael et al. 1997; Stansfeld, Fuhrer et al. 1998; Fuhrer, Stansfeld et al. 1999), but only a few focused particularly on depressive symptoms (Stansfeld, Head et al. 1998; Stansfeld, Head et al. 2003; Nabi, Singh-Manoux et al. 2009), and very few considered social support outside the work place and virtually none considered inclusion in social networks. Similarly, several studies using Whitehall II data investigated the effects of alcohol consumption on health, sickness absence and cognitive function (Marmot, North et al. 1993; Head, Martikainen et al. 2002; Britton, Singh-Manoux et al. 2004), but only one focused on the association between alcohol consumption and depressive symptoms specifically (Bell et al, 2014).

The HAPIEE is a more recent study, encompassing three cohorts of adults in Central and Eastern Europe. The HAPIEE study was designed to investigate the effects of traditional and less conventional risk factors on cardiovascular and other non-communicable diseases in Central and Eastern Europe, with a particular focus on the role played by social and psychosocial factors, as the main hypotheses of the study involve alcohol, nutrition and psychosocial factors. Among the many publications on data from the study, depressive symptoms were the topic of only three studies, all of which looking at the association between socio-economic circumstances both present and during the life course, and

depressive symptoms. Even the association between alcohol consumption and depressive symptoms has yet to be investigated.

Therefore, after reviewing the literature on the association between inclusion in social networks, social support, alcohol consumption and depressive symptoms, it can be noticed that while a vast body of research has been devoted to understanding the associations between inclusion in social networks, social support and depressive symptoms and between alcohol consumption and depressive symptoms separately, the possible association between inclusion in networks, social support and alcohol consumption in the way the affect depressive symptoms has been largely overlooked. Further, the association between inclusion in social networks, social support, alcohol consumption and depressives symptoms has never been investigated in data from the Whitehall II or the HAPIEE cohort studies, and while some research has focused on the association between support and depressive symptoms and between alcohol and depressive symptoms in Whitehall II, the topic is yet to be investigated in data from the HAPIEE study.

3. Aims, Objectives and Hypotheses

3

3.1 Introduction and Research Aims

Depression is the most common mental disorder and the leading cause of disability worldwide, inflicting a great burden and cost on society. Hence, in order to tackle this growing public health concern in an efficient and cost-effective way the European Union has commissioned research onto determinants of depression so as to provide the foundation for informed policies aimed at preventing the disorder. Inclusion in social networks, social support received from these social networks and alcohol consumption have been identified as three of the main determinants of depression. However, the existing literature provides ambivalent evidence on the association between these three factors and depression. In fact, while there is evidence to support a direct effect of inclusion in social networks, social support and alcohol consumption on depressive symptoms, there is also evidence to show how individuals who suffer from depressive symptoms are more socially isolated, feel inadequately supported and drink more heavily as a form of self-medication. Further, there is evidence to support an association between social support, inclusion in social networks and alcohol consumption in their effects on depressive symptoms. And in particular, there is evidence illustrating how different social norms affect individual patterns of alcohol consumption, and conversely how individual patterns of consumption that differ from the norm may result in social isolation.

Therefore, this project sets out to provide answers to the questions of temporality in the associations between social support, inclusion in social networks and depressive symptoms

and between alcohol consumption and depressive symptoms. Particular attention will be given to the possible association between alcohol consumption, social support and inclusion in social networks in their effects on depressive symptoms, trying to establish whether these three factors influence each other as well as depressive symptoms through time. This longitudinal analysis will be carried out on data from the Whitehall II cohort study, accounting for the possible confounding effects of other known determinants of depressive symptoms. Secondly, this project aims to assess whether cultural differences in perception of alcohol affect consumption and its influence on depressive symptoms, and whether deviations from the most common pattern of drinking result in social exclusion. In order to do so I will use cross-sectional data from the Whitehall II cohort study and from the Health Alcohol and Psychosocial factors in Eastern Europe (HAPIEE) cohort study, based in Russia, Poland and the Czech Republic. And finally, this project aims at providing valuable results that could form the basis for relevant policy recommendations aimed at tackling the risk factors for depressive symptoms and thus reduce their burden.

3.2 Objectives and Hypotheses

3.2.1 Introduction

The aims outlined above will be addressed through formulation and investigation of three groups of research objectives and hypotheses. First, there will be objectives and hypotheses regarding the associations between inclusion in social networks and depressive symptoms, and between alcohol consumption and depressive symptoms at given point in time, and how these vary across four countries as different as the UK, the Czech Republic, Russia and Poland. Secondly, there will be objectives and hypotheses regarding the association between social support and depressive symptoms at a given point in time, but also regarding the direction and magnitude of the associations between social support, inclusion in social networks, alcohol consumption and depressive symptoms through time. And finally, there will be objective and hypotheses regarding the possible association between social support, inclusion in social networks and alcohol consumption in their effects on depressive symptoms. All these objectives and hypotheses are exposed below.

3.2.2 Cross-sectional Objectives and Hypotheses

O1: To investigate the association between measures of inclusion in social networks and depressive symptoms.

H1: *Individuals who do not have any or never meet their friends and relatives outside the household are more likely to suffer from depressive symptoms than their more socially connected counterparts.*

O2: To investigate the association between alcohol consumption and depressive symptoms

H2: *Both individuals who consume alcohol heavily and frequently and individuals who do not consume alcohol are more likely to suffer from depressive symptoms than moderate drinkers.*

O3: To compare patterns observed in the UK, the Czech Republic, Russia and Poland

H3: *The patterns of association between inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms would vary across countries.*

O4: To test whether the associations between inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are confounded by age, gender, marital status, occupation, alcohol consumption and inclusion in social networks.

H4: *The associations between inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are not confounded by age, gender, marital status, occupation, alcohol consumption and inclusion in social networks*.

3.2.3 Longitudinal Objectives and Hypotheses

O5: To investigate the association between measures of social support and depressive symptoms.

H5: *Individuals who receive low levels of confiding/emotional or practical support, and high levels of negative support are more likely to suffer from depressive symptoms.*

O6: To investigate the duration and magnitude of the association between inclusion in social networks, social support and depressive symptoms.

H6: *Individuals who are poorly connected to friends, relatives outside the household or other clubs and societies at Phase 1, will be more likely to be affected by depressive symptoms for many years.*

H7: *Individuals who receive low levels of confiding/emotional or practical support, and high levels of negative support at Phase 1, will be more likely to be affected by depressive symptoms for many years.*

O7: To investigate the magnitude and duration of the association between alcohol consumption and depressive symptoms.

H8: *Individuals who either consume alcohol heavily and frequently or who do not drink at all will be more likely to be affected by depressive symptoms for many years than moderate drinkers.*

O8: To test whether the associations social support, inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are confounded by the effects of age, gender, marital status, employment grade, smoking status, physical activity, and alcohol consumption or social support and inclusion in social networks respectively.

H9: *The associations social support, inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are not confounded by the effects of age, gender, marital status, employment grade, smoking status, physical activity, and alcohol consumption or social support and inclusion in social networks respectively*.

3.2.4 Temporality and Association

O9: To investigate the developmental trajectories of change in the association between social support, inclusion in social networks, alcohol consumption and depressive symptoms through time.

H10: *Individual variation in depressive symptoms through time is associated with individual variation in experienced levels of social support and inclusion in social networks.*

H11: *Individual variation in depressive symptoms through time is associated with individual variation in alcohol consumption – measured in terms of frequency of drinking sessions*

H12: *The effects of social support and inclusion in social networks on depressive symptoms vary when the effects of alcohol consumption are taken into consideration and vice versa.*

4. Methods

4

4.1 **Introduction**

In this chapter I will describe the data I will analyse in order to answer the research objectives and hypotheses outlined in Chapter 3. I will start by introducing the Whitehall II and the HAPIEE cohort studies, describing the time and mode of data collection for the two studies, their sample size and population included. I will then introduce the different measures of depressive symptoms used in the two cohorts, as in the HAPIEE cohort study depressive symptoms were measured through the Centre for Epidemiological Studies Depression scale (CES-D), while in the Whitehall II cohort a depression subscale of the general health questionnaire was used. I will describe the two instruments and discuss the differences they present in measuring depressive symptoms. After having introduced measures of depressive symptoms, I will introduce the measures of social support, inclusion in social networks and alcohol used in the two cohorts. I will then describe the measures of marital status, employment grade, smoking status and physical activity that will constitute possible confounders in the analysis. The issue of statistical power of the two data sets will also be addressed, both in terms of the overall power of the two cohorts and of the power of the sub samples here analysed. Finally I will introduce the ethical issues and approval obtained for the two cohorts. Details of the statistical analysis employed will be given in the following chapters, as the statistical techniques involved vary greatly from one set of the analysis to the other and could not be described in one common section.

4.2 Study population and sample

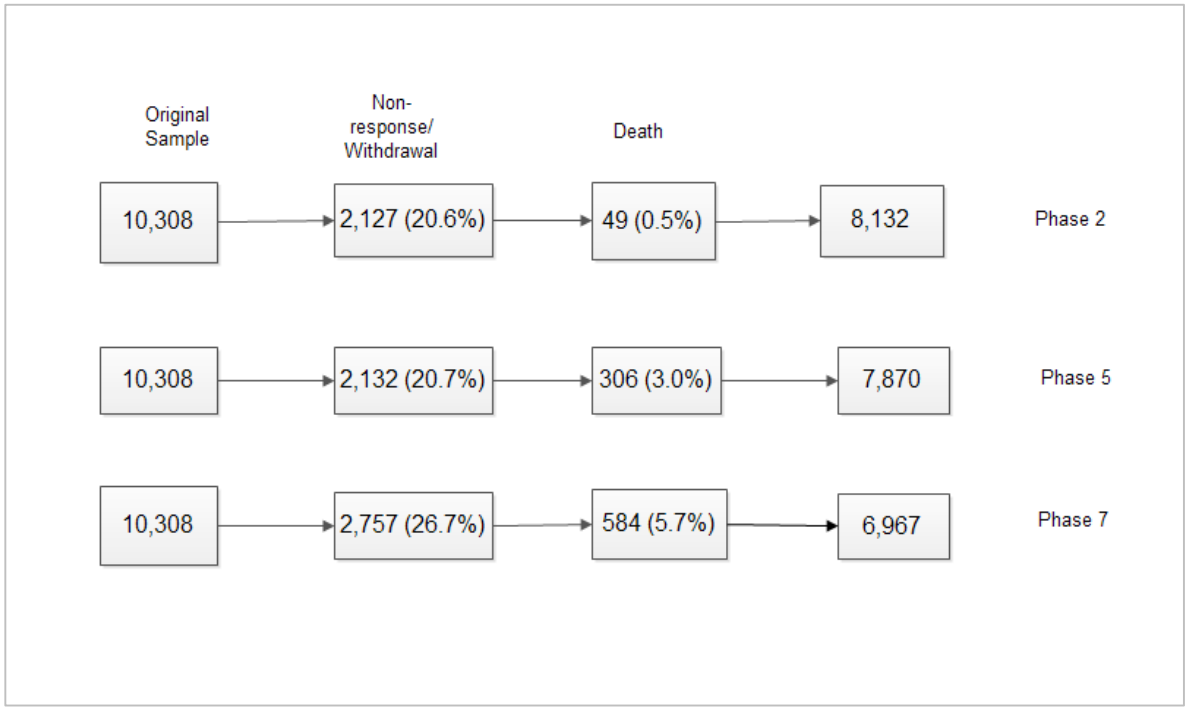
4.2.1 Whitehall II

The Whitehall II cohort study focuses on British civil servants who were working in the London offices of 20 Whitehall departments in 1985-1988, when baseline measurements took place. The original study population was composed of 6895 men and 3413 women (tot. 10308) aged 35-55 employed in clerical and office support grades, middle-ranking executive grades or senior administrative grades, with great differences in salaries. Baseline measurements took the form of a clinical screening and a structured questionnaire, and participants were subsequently invited back to the research clinic every five years, while a postal questionnaire has been sent out in between screenings (Marmot and Brunner 2005).

The analysis here presented will be based on data from Phase 1 and Phases 2, 5 and 7 which include information on social support as well as inclusion in networks, alcohol consumption and depressive symptoms. The data collection process for each Phase of the Whitehall II cohort took place at roughly regular intervals and each took between two and three years to complete. Thus, if Phase 1 measurements were collected between 1985 and 1988, Phase 2 measurements took the form of a questionnaire submitted to participants between 1989 and 1990; Phase 5 measurements included both a screening and a questionnaire and were carried out between 1997 and 1999; and the Phase 7 screening and questionnaire process was finalised between 2002 and 2004. In the course of the nineteen years the separate the beginning of the study from the end of Phase 7, the sample size was reduced from the original 10, 308 civil servants recruited at baseline, to the 6,967 participants interviewed at Phase 7.

The issue of reduction of sample size through time is a common problem in cohort studies, for in between waves of data collection, some participants might die or move elsewhere, some might withdraw from the study altogether and some might simply fail to respond to one or several waves of collection but complete the some of the other phases. The Whitehall II cohort was no different as at Phase 2, only 79% of the original 10,308 participants responded to the questionnaire and the response rate became 76% at Phase 5 and 68% at Phase 7. At each phase the majority of participants who failed to respond had either withdrew or simply did not attend a particular wave of collection, with only a small proportion having deceased from phase to phase (Figure 4.2.1). Further, often participants who did not respond to a particular phase then joined the following one, or the last one before they died thus allowing to collect some information about them even though sporadically. In addition to the number of participants thus lost, a small proportion of individuals were missing information on depression scores, hence further reducing the sample size at each phase. Missing data, although to a lesser extent, were also in measures of support, inclusion in networks and alcohol consumption as well. The issue of missing data will be addressed more thoroughly in Chapter 6.

Figure 4.2.1 *Number of participants lost at each phase due to withdrawal/non-response or death.*



4.2.2 HAPIEE

The HAPIEE prospective cohort study focuses on the adult urban population of Novosibirsk (Russia), Krakow (Poland) and six cities of the Czech Republic (Hradec, Kralove, Jihlava, Kromeriz, Liberec and Usti nad Labem). A full report of the study design and rationale can be found in Peasey et al (2006). Baseline measurements were carried out between 2002 and 2005, when participants, aged 45-69, were randomly selected from the local population registers. Measurements took the form of a structured questionnaire, subjected to participants during an interview, and a short clinic examination. In Poland and the Czech Republic participants were first visited at home for the interview and then invited to a clinic for the examination, while in Russia both interview and examination took place in a clinic for security reasons. The structured questionnaire included items on health, life-style, diet, socio-economic circumstances, and psychosocial factors both outside and within the work place. All items in the questionnaire were translated from English to each of the participants' native languages and then back-translated into English for accuracy.

The planned sample size of the study was 30,000: 10,000 individuals in each of the three countries. The actual sample size was 28,948 individuals. Response rates were 61% in Russia and Poland and 55% in the Czech Republic. In addition, an error in the interviewer protocol in Novosibirsk led to 3,274 depression questionnaires being excluded, thus this analysis was based on 25,674 individuals (12,075 men and 13,599 women) with valid scores for depressive symptoms (Nicholson, Pikhart et al. 2008). Only baseline data of the HAPIEE cohort study was here used in the cross-sectional comparison of the patterns of association

between social support, inclusion in social networks, alcohol consumption and depressive symptoms observed in Central and Eastern Europe and the UK.

4.3 Measures of Depressive Symptoms

4.3.1 Whitehall II: the General Health Questionnaire (GHQ)

In the Whitehall II cohort study, psychiatric disorders were measured through the 30-items General Health Questionnaire (GHQ) (Goldberg 1972), a validated and widely used instrument designed to detect minor psychiatric disorders in the general and clinical population (Stansfeld, Fuhrer et al. 1998; Stansfeld, Head et al. 1998). Following the rationale of Stansfeld et al (1998), four of the items that already formed the depression sub-scale in the 28-items GHQ were picked to form a depression sub-scale in this study (Stansfeld, North et al. 1995). The four items were: 'Have you recently: - thought yourself worthless? –felt life is hopeless? – felt that life isn't worth living? – felt nerves stopped you?' Responses were scored by summing all items scored on a scale from 0 to 3, with individuals scoring 4 or above being considered as clinically depressed (Stansfeld, Head et al. 1998).

4.3.2 HAPIEE: the Centre for Epidemiological Studies Depression Scale (CES-D)

Depressive symptoms were measured using the Centre for Epidemiologic Studies Depression scale (CES-D) (Radloff 1977). This is a self-reported tool composed of twenty items about presence of depressive symptoms in the past week (less than one day; one or two days; three four days; or five to seven days). Responses were scored on a scale from 0 to 3, with 3 being the highest frequency of depressive symptoms. All but two items were negatively formulated; the two positively formulated items were reversely scored so that 3 corresponded to the highest prevalence of depressive symptoms. Thus obtained, the total individual score ranged between 0 and 60. The depression score was calculated if at least

16 of the 20 questions were answered. If fewer than 20 questions (but at least 16) were answered, the score was recalculated to have values between 0 and 60, by taking the mean score of valid answers and multiplying it by 20 (Pikhartova, Chandola et al. 2009). Participants with a score of 16 or above were classified as having depressive symptoms, consistently with a number of studies (Roberts and Vernon 1983; Beekman, Deeg et al. 1997; Lyness, Tamson et al. 1997; Nicholson, Pikhart et al. 2008).

4.3.3 Comparison of the two scales

The four items depression sub-scale of the GHQ has only recently been formally validated against a structured psychiatric interview. A recent study by Head at el (2013) looked at assessing the validity of three self-administered instruments for depression and found that the GHQ depression sub-scale has a good criterion validity to detect generic mental disorder but is not as specific to depression as the CES-D scale. However, there is no formal comparison of the two scales and it could be that they yield slightly different results. Therefore the cross-sectional comparison between the Whitehall II cohort and the HAPIEE cohort was conducted twice. The first time data from Phase 1 of the Whitehall II cohort using the GHQ depression subscale were compared with data from baseline of the HAPIEE cohort, using the CES-D scale. The second time the same analysis was conducted but with data from Phase 7 of the Whitehall cohort, as at Phase 7 the CES-D scale was introduced in Whitehall II. Results of the two sets of analysis were then compared.

4.4 Measures of Inclusion in Social Networks and Social Support

4.4.1 Whitehall II

Information on support and inclusion in networks was collected by asking participants about size of their networks, frequency of social associations, group membership, church attendance and social support in the work place as well as the Close Person Questionnaire (Stansfeld and Marmot 1992), fifteen items of which assess `qualitative' types of support from and to each of the close persons and the questions start with the phrase ``How much in the last 12 months did this person...'' (Fuhrer, Stansfeld et al. 1999). In the validation study of the questionnaire, 3 sub-scales were derived from the 15 items using factor analysis (Stansfeld and Marmot 1992). Seven items constituted the confiding/emotional support sub-scale, 3 items constituted the practical support sub-scale, and 4 items were included in the negative aspects of close relationship sub-scale (Fuhrer, Stansfeld et al. 1999).

The full questions of the three subscales are reported in Table 4.4.1. Responses for each of the questions in the three subscales were assigned Likert-scaled and the score totalled for each close person nominated. A cumulative weighted total score was generated for each subscale across the number of close persons nominated, with the most weight given to the first close person and progressively less weight given to each subsequent close person nominated (Fuhrer, Stansfeld et al. 1999). The score for the first close person was assigned a weight of 1.0; the score for the second close person was assigned a weight of 0.25; and the third and fourth close persons were assigned weights of 0.15 and 0.10 respectively (Fuhrer, Stansfeld et al. 1999). Scores for each type of support were then recoded in three

categories: high, medium and low. These weighted tertile variables were chosen as measures of social support for the present analysis, with the category 'high' taken as the reference category for confiding/emotional and practical support, while for negative support 'low' was the reference category.

Three measures of social networks were derived from questions on frequency and number of contact with friends, relatives and social groups. These were a 'network beyond the household', a friends and a relative scale (Stansfeld, Fuhrer et al. 1998). As for support, these measures were categorised as high, medium and low contact, with 'high' being the reference category.

Table 4.4.1 *Full questions of the support sub-scales of the close person questionnaire*

	Confiding/emotional support
1.	How much in the last 12 months did this person give you information, suggestions and guidance that you found helpful?
2.	How much in the last 12 months did this person make you feel good about yourself?
3.	How much in the last 12 months did you share hobbies, interests and fun with this person?
4.	How much in the last 12 months did you want to confide in (talk frankly, share feelings with) this person?
5.	How much in the last 12 months did you confide in this person?
6.	How much in the last 12 months did you trust this person with your most personal worries and problems?
7.	How much in the last 12 months did he/she talked about their personal problems with you?
	Practical support
1.	How much in the last 12 months did you need practical help from this person with major things?
2.	How much in the last 12 months did this person give you practical help with major things?
3.	How much in the last 12 months did this person give you practical help with minor things when you needed it?
	Negative support
1.	How much in the last 12 months did this person give you worries, problems and stress?
2.	How much in the last 12 months would you have liked to have confided more in this person?
3.	How much in the last 12 months did talking to this person make things worse?
4.	How much in the last 12 months would you have liked more practical help with major things from this person?

4.4.2 HAPIEE

No data on social support were collected in the HAPIEE cohort study. Although, data were collected on the levels of trust participants held into formal and informal networks. Trust was measured through the question: “on whom do you rely first when having a problem?” In Russia and the Czech Republic, the question was repeated nine times (for: friends, relatives, myself, no-one, state, employer, private organizations, public organisations, charities and church), and participants had to answer “yes” or “no.” In Poland, the question was asked once and participants had to choose one out of the nine possible answers. Following the rationale proposed by Bobak et al (1998) and Rose (2000), answers to these questions were grouped into four categories: whether participants relied first on (1) nobody, (2) themselves, (3) friends and relatives (informal networks), or (4) state, employer, charities and church, or private or public organisations (formal networks).

Inclusion in social networks was measured by asking how often participants visited friends and relatives not living in the same household and how many of these were visited at least once a week. Response categories to the items on frequency of contacts were: less than once a month, several times a week, once a week, several times a month, and “I don’t have any” friends or distant relatives. Response categories to the question investigating number of friends or relatives visited at least once a week were: none, 1 or 2, 3 to 5, more than 5, and “I don’t have any” friends or distant relatives. In the Czech Republic, participants were not subjected to the items asking how many friends or distant relatives they visited on a weekly basis. In addition, participants were asked if they were members of clubs or organisations and if so how often did they take part on events organised by said club or

organisation. Response categories were: several times a week, several times a month, about once a month, several times a year and never or almost never.

4.5 Measures of alcohol consumption

4.5.1 Whitehall II

In the Whitehall II cohort study, alcohol consumption was measured in terms of frequency of consumption in the previous 12 months. Responses were: (1) no, (2) only in special occasions, (3) once or twice a month, (4) once or twice a week, (5) almost daily and (6) twice a day or more (Marmot, North et al. 1993). Response 'once or more a week' was taken as the reference category. Because of the reduced number of participants who reported drinking twice or more a day or not drinking the variable was recoded as: (1) drinking once or more a day, (2) drinking once or more a week, (3) drinking once or more a month, (4) drinking in special occasions or not drinking.

In addition, at each phase participants were asked if they had consumed alcoholic beverages in the previous seven days, if the answer was 'yes' they were asked how many alcoholic drinks they consumed in those seven days. Types of alcoholic drinks included 'measures' of spirits, 'glasses' of wine and 'pints' of beer. In the UK, a standard measure of spirit and a glass of wine correspond to 8g of alcohol, while a pint of beer corresponds to 16g of alcohol. In addition, in the UK a standard unit of alcohol corresponds to 8g alcohol, hence, following Britton et al (2009) the amounts reported by participants were converted into units (one unit= 8g alcohol) and divided into seven categories from 0 to 6, as: 0= no

units in the past week; 1= 1-7 units in the past week; 2= 8-14; 3=15-21; 4=22-28; 5= 29-35; 6= 36 + units consumed in the past week (Britton, Marmot et al. 2009).

4.5.2 HAPIEE

In the HAPIEE cohort study alcohol consumption was measured through the graduated frequency questionnaire (GF), a tool developed by Jurgen Rhem (1998) to measure quantity, frequency and volume of alcohol consumed. Through the graduated frequency questionnaire participants were asked how many times in the previous twelve months did they consume a certain amount of alcohol as measured in local units of beer, wine and spirits. The amount of alcohol ranged from 0 to 10 or more drinks, with a drink being defined as 500ml of beer, 200ml of wine or 50ml of spirits (Borbova, West et al. 2010). More specifically, in the three countries of the HAPIEE study, participants were asked how many times in the previous twelve months did they consume: one or two; three or four; five or six; seven or nine; and ten or more drinks. The answers provided to these questions allowed to estimate the mean annual number of drinking occasions, annual alcohol intake, mean dose of alcohol consumed per drinking occasion and the frequency of drinking and binge drinking. In addition, participants were asked separately how often did they alcohol consumed in the previous twelve months, with responses coded in five different categories ranging from (1) never, to (2) almost once a month, to (3) once or more per month, to (4) one to four times a week, to (5) five or more times a week.

For the scope of the present analysis, I used the measure of mean dose of alcohol consumed per drinking session derived from the graduated frequency questionnaire and the measure of alcohol consumption recorded separately from the GF questionnaire as representative of alcohol consumption. The scores of the measure for mean dose of alcohol consumed per drinking occasion were first recorded in millilitres, as a continuous variable, and then divided in 5 categories with different values for men and women. The five categories followed the recommended daily intake of alcohol for men and women, and are described in Table 4.5.2. Very few women reported consuming more than five units per drinking session, therefore they were grouped in the category below. No distinction was made between ex-drinkers and participants who never consumed alcohol.

Table 4.5.1 *Categorisation of volume of alcohol consumed per drinking session by men and women*

	Men	Women
1	Never	Never
2	10-39 ml (up to 4 UK units)	10-29 ml (up to 3 UK units)
3	40-59 ml (between 4 and 6 UK units)	30-49 ml (between 3 and 5 UK units)
4	60-79 ml (between 6 and 8 UK units)	50 + ml (more than 5 UK units)
5	80+ ml (more than 8 UK units)	

4.6 Covariates

4.6.1 Whitehall II

At each phase of the Whitehall II cohort study, information was collected regarding age, sex, marital status, employment grade, smoking status, physical activity and life events experienced in the past year, of participants. Marital status was classified in four categories: (1) married or cohabiting, (2) single, (3) divorced or separated, and (4) widowed. For what concerns employment grade, the British Civil Service identifies 12-employment levels on the basis of salary. These 12 levels differ not only in salary but also in other socio-economic indicators such as education, house tenure, car ownership and father's occupation. For this reason, employment grade was here taken as a proxy for socio-economic circumstances. In this analysis, the 12 employment levels were grouped into three categories: (1) administrative, (2) professional/executive, and (3) clerical/support. Moreover, participants were asked about the frequency and duration of their involvement in (1) mildly energetic (i.e. weeding, general housework, bicycle repair), (2) moderately energetic (i.e. dancing, cycling, leisurely swimming), or (3) vigorous (i.e. running, hard swimming, playing squash) physical activity (Singh-Manoux, Hillsdon et al. 2005). Life events from eight self-report questions concerning experiences in the previous 12 months were also assessed in terms of both number of life events occurred and how upsetting to the participant they were.

4.6.2 HAPIEE

In the HAPIEE cohort study, information on age, sex, marital status, socio-economic circumstances, physical activity and smoking status of participants was also collected. Marital status was classified into four categories: (1) single, (2) married or cohabiting, (3) divorced or separated, and (4) widowed. In countries of Central and Eastern Europe, employment grade alone is not as informative of socio-economic circumstances as it is in the UK, therefore, information on socio-economic circumstances was collected through questions on education, occupation, ownership of household amenities and perceived financial hardship of participants. Education was measured in terms of the highest level of education attained and classified into five categories: (1) none or incomplete formal education, (2) primary, (3) vocational, (4) secondary, and (5) university. A very small proportion of persons reported not having achieved any education so they were grouped with participants who reported having completed primary education. Participants' occupation was classified into eight categories: (1) employed, (2) free-lance, (3) self-employed, (4) entrepreneur, (5) farmer or housewife, (6) employed pensioner, (7) unemployed pensioner, (8) and unemployed.

Material deprivation of participants was measured through a question about which, if any, of twelve luxury items they owned. The list of twelve items included: a microwave, a video recorder, a television, a washing machine, a dishwasher, a car, a freezer, a cottage for holidays or weekends, a video-camera or a camcorder, a satellite or cable TV, a telephone, and a mobile phone. Responses were classified into four different groups: (1) 0-3 items owned, (2) 4-6 items owned, (3) 7-9 items owned, and (4) 10-12 items owned. Self-reported

financial hardship was measured through an item in the questionnaire about how well participants and their partners were doing financially. Responses ranged from (1) “we are managing really well” through (2) “we are managing quite well”, (3) “we are getting by alright”, (4) “we don’t manage very well” and (5) “we have some financial difficulties” to (6) “we are in deep financial trouble.” The proportion of participants who reported managing really well financially was so slim that they were grouped with those who reported managing quite well.

Participants were also asked how many hours of physically demanding activities and of sport did they perform in winter and in summer. Information on smoking status was collected via the question “do you smoke cigarettes?” responses were: (1) yes, regularly (more than a cigarette a day); (2) yes, occasionally (less than a cigarette a day); (3) no, I smoked in the past but I quit; (4) and no, I have never smoked.

4.7 **Ethics**

Ethical approval for the Whitehall II study was obtained from the University College London Medical School committee on the ethics of human research (Ferrie, Shipley et al. 2007). Ethical approval for the HAPIEE study was obtained from the ethics committee at University College London, UK, and from the ethics committees in each participating centre. In addition, all participants of both studies gave informed consent in writing (Peasey, Bobak et al. 2006; Ferrie, Shipley et al. 2007).

5. International Comparison

5

5.1 Introduction

The present chapter is dedicated to the investigation of the associations between measures of inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms at a given point in time, using cross sectional data from Phase 1 of the Whitehall II cohort study and from baseline of the HAPIEE cohort study. More specifically, this chapter aims:

O1: to investigate the association between measures of inclusion in social networks and depressive symptoms;

H1: *individuals who do not have any or never meet their friends and relatives outside the household are more likely to suffer from depressive symptoms than their more socially connected counterparts;*

O2: to investigate the association between alcohol consumption and depressive symptoms;

H2: *both individuals who consume alcohol heavily and frequently and individuals who do not consume alcohol are more likely to suffer from depressive symptoms than moderate drinkers;*

O3: to compare patterns observed in the UK, the Czech Republic, Russia and Poland;

H3: *the patterns of association between inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms would vary across countries;*

O4: to test whether the associations between inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are confounded by age, gender, marital status, occupation, alcohol consumption and inclusion in social networks;

H4: *the associations between inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are not confounded by age, gender, marital status, occupation, alcohol consumption and inclusion in social networks*.

In order to address these research objectives and hypotheses, cross-sectional analysis will be carried out on data from Whitehall II cohort study and from baseline of the HAPIEE cohort study. Details of the statistical analysis carried out are presented in section 5.2 together with a brief description of proportions of missing data in the sample here used. Section 5.3 will present the results of the analysis, including a comparison of the two epidemiological scales used to measure depressive symptoms in the two studies cohorts, using data coming from Phase 7 of the Whitehall II cohort study where both scales were used simultaneously. Further, a comparison of the patterns of association observed in the two cohorts can be found in section 5.4. Finally, section 5.5 offers a summary of the chapter.

5.2 Statistical Analysis

Statistical analysis was as follows. First, missing data for each variable under study were calculated in both cohorts (Table 5.2.1 and Table 5.2.2). The proportion of missing data found in the variables included in the analysis was small to insignificant, with the exception of number of friends and relatives seen every month. In the Whitehall II cohort study, the measure of number of friends seen per month had 26.0% missing data, due to the fact that the questionnaire submitted to participants at Phase 1 was changed to include an item on number of friends seen per month fairly late in the data collection process. In the HAPIEE cohort, the questions regarding number of friends and relatives seen per month was not present at all in the questionnaire submitted to participants from the Czech Republic. Therefore, given the high proportion of data which was not collected rather than simply missing the variables for number of friends and relatives seen per month were not used in this analysis.

Table 5.2.1 *Number and percentage of complete and missing values at Phase 1 of the Whitehall II cohort study*

	Complete		Missing	
	N	%	N	%
Age groups	10,308	100.0	0	0
Sex	10,308	100.0	0	0
Marital Status	10,270	99.6	38	0.4
Employment grade	10,308	100.0	0	0
Number of friends seen a month	7,630	74.0	2,678	26.0
Frequency of contact with friends	10,226	99.2	82	0.8
Number of relatives seen a month	9462	91.8	846	8.2
Frequency of contact with relatives	10,197	98.0	206	2.0
Frequency of drinking in past year	10,278	99.7	30	0.3
Dose of alcohol consumed per week	10,214	99.1	94	0.9
Depressive Symptoms	10,208	99.0	100	1.0

Table 5.2.2 *Number and percentage of complete and missing values at Phase 1 of the HAPIEE cohort study.*

	Complete		Missing	
	N	%	N	%
Age groups	25,674	100.0	0	0
Sex	25,674	100.0	0	0
Marital Status	25,618	99.8	56	0.2
Occupation	25,596	99.7	78	0.3
Number of friends seen a month	17,237	67.1	8,437	32.9
Frequency of contact with friends	25,590	99.7	84	0.3
Number of relatives seen a month	17,341	67.5	8,333	32.5
Frequency of contact with relatives	25,627	99.8	47	0.2
Frequency of drinking in past year	25,453	99.1	221	0.9
Dose consumed per drinking session	25,453	99.1	221	0.9
Depressive Symptoms	25,674	100.0	0	0

Secondly, any possible association between gender and measures of either inclusion in social networks or alcohol consumption were tested through Likelihood of Ratio Tests (LRTs). In addition, in HAPIEE LRTs were used to test the association between country and inclusion in social networks or alcohol consumption. In both cohorts, an association was found between gender and alcohol consumption, hence results relating to alcohol consumption are reported stratified by gender. In addition, in the HAPIEE study an association was found between gender and measures of alcohol consumption and of frequency of contact with friends, and between country and measures of contact with relatives. Therefore, subsequent analysis on the HAPIEE data was stratified by gender and country (Table 5.2.3 and Table 5.2.4).

Table 5.2.3 *Likelihood of ratio test chi square and relative p value for the association between gender and measures of inclusion in networks and alcohol consumption in the Whitehall II cohort.*

	LRT	P
Frequency of contact with friends	4.62	0.329
Frequency of contact with relatives	2.51	0.775
Frequency of alcohol consumption	7.05	0.217
Dose of alcohol consumed	19.63	0.003

Table 5.2.4 *Likelihood of ratio chi square and relative p value for the association between gender, country and measures of inclusion in networks and alcohol consumption in the HAPIEE cohort.*

Gender	LRT	P
Frequency of contact with friends	15.73	0.008
Frequency of contact with relatives	10.97	0.052
Frequency of alcohol consumption	16.67	0.034
Dose of alcohol consumed	30.78	<0.001
Country		
Frequency of contact with friends	6.04	0.302
Frequency of contact with relatives	36.19	<0.001
Frequency of alcohol consumption	11.93	0.154
Dose of alcohol consumed	2.42	0.660

Thirdly, descriptive statistics were calculated in both cohorts to identify what proportion of participants were married, single, divorced or widowed, unemployed or employed and at what level, belonged to more or less socially included groups, consumed alcohol more or less frequently and in what amount. The relative prevalence of depressive symptoms for each group was also calculated and chi-square tests were performed to assess the association between depressive symptoms and each of the exposure variables and covariates.

Fourthly, three logistic regression models were run on each dataset. The first was adjusted only for the effects of age, and gender in the Whitehall II cohort study; the second took into account the possible confounding effects of marital and employment status too; and the third was adjusted for the effects of age, marital status, employment status, and alcohol consumption or inclusion in social networks. These models, and the descriptive statistics, were run first on data from Phase 7 of the Whitehall II cohort study, using both scores from the General Health Questionnaire (GHQ) depression subscale, and from the Centre for Epidemiological Studies Depression scale (CESD), with the intent of comparing results from the two scales.

Further, for the logistic regression models described above, the reference category for number of friends and relatives visited was '1-2 friends or relatives visited once a month' in all three datasets. However, if 'every few months' was the reference category for frequency of contact with friends and relatives in the two phases of the Whitehall II cohort; 'less the once a month' was the reference category for the equivalent measure in the HAPIEE cohort. For what concerns frequency of alcohol consumption, the reference category was 'more than once a week' in the two phases of the Whitehall II cohort and '1-4 times a week' and 'once a month' for men and women respectively in the HAPIEE cohort. Finally, in Phase 1 of the Whitehall II cohort the reference category for mean dose of alcohol consumed per week was 1-7 units; while in the HAPIEE cohort, the reference category for mean dose consumed per drinking session was '20-39ml' for both men and women.

5.3 Results

5.3.1 Comparison of the CESD and GHQ depression scales

The cross-sectional logistic regression analysis presented in this chapter aims not only at investigating the patterns of association between inclusion in social networks, alcohol consumption and depressive symptoms, but also at comparing patterns observed in the UK with those observed in the Czech Republic, Russia and Poland. However, this is rendered difficult by the fact that the Whitehall II and HAPIEE cohort studies used two different scales to measure depressive symptoms among their participants. In fact, while the GHQ scale used in the Whitehall II cohort is designed to detect psychological distress and ability or inability to carry out day to day tasks, the CESD scale used in the HAPIEE cohort was designed to detect depressive symptoms in the general population; and the two scales have never been formally compared. Therefore, this section will attempt to compare the two scales using data from Phase 7 of the Whitehall cohort study, where both scales were used independently to detect depressive symptoms.

Of the original 10,208 participants, 6,943 replied to the questionnaire of Phase 7 and of these 6,768 had complete scores for the GHQ depression subscale, and 6,012 had complete scores for the CES-D scale. Table 5.3.1 and Table 5.3.2 show the prevalence of depressive symptoms according to general characteristics and according to measures of inclusion in social networks and measures of alcohol consumption, as measured separately with the GHQ depression subscale and the CESD scale. Prevalence of depressive symptoms was overall 1.2 times higher when measured with the CESD scale compared to the GHQ scale, and among participants who were last employed in the lower grades of the civil service

prevalence of depressive symptoms was as much as 1.6 times higher when measured with the CESD scale. This discrepancy is likely to be due to the fact that the CESD was designed to detect depressive symptoms specifically rather than psychiatric disorders in general.

Table 5.3.1 *Prevalence of depressive symptoms according to general characteristics, measured with the GHQ and CESD depression scales.*

		GHQ			CESD	
	%	Depr. %	P	%	Depr. %	P
Total	6,768	12.0		6,012	14.9	
Sex			0.023			<0.001
Men	70.6	11.4		71.8	13.1	
Women	29.4	13.4		28.2	19.7	
Age			<0.001			<0.001
<54	18.1	15.2		18.6	19.2	
55-59	29.7	12.8		30.8	15.1	
60-64	21.3	10.6		21.2	12.9	
65-69	21.0	10.3		20.2	13.6	
<74	9.9	10.7		9.2	13.7	
Marital Status			<0.001			<0.001
Married/cohabiting	75.7	11.0		76.0	12.4	
Single	12.5	14.3		12.6	22.5	
Divorced	7.4	15.7		7.1	22.6	
Widowed	4.4	17.0		4.3	24.9	
Employment Grade			0.020			<0.001
Administrative	45.1	11.9		45.0	13.3	
Prof/Executive	43.9	15.7		44.9	18.7	
Clerical/Support	11.0	17.5		10.1	25.9	
Last employment grade			<0.001			<0.001
Administrative	45.6	8.7		46.5	9.6	
Prof/Executive	43.2	12.3		43.4	16.3	
Clerical/Support	11.2	15.3		10.1	25.1	

Table 5.3.2 *Prevalence of depressive symptoms according to measures of contact with friends and relatives and alcohol consumption, measured with the GHQ and CESD depression scales.*

		GHQ			CESD	
	%	Depr. %	P	%	Depr. %	P
Frequency of contact with friends			<0.001			<0.001
Almost daily	9.5	8.7		9.4	8.3	
About once a week	36.0	10.4		36.0	13.0	
About once a month	24.4	11.9		24.4	15.4	
Once every few months	22.1	14.5		22.3	17.5	
Never/almost never	8.0	16.2		7.9	22.9	
Frequency of contact with relatives			<0.001			<0.001
Almost daily	7.9	11.3		7.8	11.8	
About once a week	29.7	10.4		29.8	13.1	
About once a month	24.1	11.2		24.4	13.7	
Once every few months	28.5	12.1		28.4	15.6	
Never/almost never	9.8	18.7		9.5	21.2	
Frequency of alcohol consumption			0.004			<0.001
Once or more a day	46.0	11.4		46.5	12.5	
Once or more a week	29.7	11.1		29.9	13.2	
Once or more a month	8.7	12.5		8.5	17.2	
Never/Special occasions	15.6	15.3		15.0	23.2	
Amount of alcohol consumed per week			<0.001			<0.001
None	17.3	16.2		16.6	22.7	
1-7 units	29.9	10.8		29.6	14.2	
8-14 units	22.4	11.0		22.7	12.0	
15-21 units	13.3	11.7		13.7	12.3	
22-28 units	7.3	9.8		7.5	9.3	
29-35 units	4.0	10.1		3.9	12.0	
36+ units	5.9	13.4		5.8	18.7	

The discrepancy in magnitude between results obtained when using the GHQ scale and results obtained when using the CESD scale was present in logistic regression analysis too. Results from the first logistic regression model, adjusted for age and sex, showed that odds of depressive symptoms were higher among participants who reported never seeing their relatives when measured with both scales. However, when measured with the CESD scale (OR 1.45; 95% C.I. 1.13-1.86) these odds were 1.2 times lower than when measured with the GHQ scale (OR 1.70; 95% C.I. 1.33-2.17). Further, odds of depressive symptoms were lowest among participants who reported seeing their friends daily when, but again they were 1.3 times lower when measure with the CESD scale (OR 0.43; 95% C.I. 0.31-0.60) than when measured with the GHQ scale (OR 0.58; 95% C.I. 0.42-0.79). For what concerns alcohol consumption, odds of depressive symptoms were higher among participants who reported drinking only on special occasions or not at all when measured with both scales, but these odds were 1.3 times higher when measured with the CESD scale (OR 1.91; 95% C.I. 1.54-2.37) than when measured with the GHQ scale (OR 1.48; 95% C.I. 1.18-1.85). Finally, participants who reported not drinking any alcohol in an average week had higher odds of depressive symptoms, and these odds were 1.1 times higher when measured with the CESD scale than when measured with the GHQ scale (Table 5.3.3).

In the second logistic regression model the effects of marital status, employment grade, last employment grade before retirement, smoking status, physical activity were taken into account as well as those of age and sex, which affected the magnitude of the difference between results obtained with the GHQ scale and those obtained with the CESD scale. In fact, in this model, odds of depressive symptoms among participants who reported seeing

their friends daily were 1.5 lower when measured with the CESD scale compared to when measured with the GHQ scale. Odds of depressive symptoms measured with the CESD scale were 1.3 times lower among participants who never saw their relatives, 1.1 times lower among participants who did not consume alcohol on an average week, and 1.1 times lower among those who consumed 36 or more units per week (Table, 5.3.4). In addition, odds of depressive symptoms among participants who reported drinking only in special occasions or not at all (OR 1.60; 95% C.I. 1.21-2.12) and once or more a month (OR 1.56; 95% C.I. 1.11-2.17) were higher than among participants who consumed several times a week when measured with the CESD scale, while the association was not significant when measured with the GHQ scale.

Finally, in the third model, the effects of alcohol consumption were taken into account when looking at the association between frequency of contacts with friends and relatives and depressive symptoms, and conversely the effects of frequency of contacts with friends and relatives were taken into account when looking at the association between alcohol consumption and depressive symptoms. Similarly to what observed in Model two, in this model also, the CESD scale yielded odds of depressive symptoms that were 1.4 times lower among participants who reported visiting their friends almost daily and 1.3 times lower among participants who reported never seeing their relatives. The odds of depressive symptoms were again higher among participants who reported drinking only in special occasions or not at all (OR 1.51; 95% C.I. 1.13-2.01) and once or more a month (OR 1.58; 95% C.I. 1.13-2.22) when measured with the CESD scale, while the association between frequency of alcohol consumption and depressive symptoms was not statistically significant

when measured with the GHQ scale. While the association between amount of alcohol consumed per week and depressive symptoms was only significant when measured with the GHQ scale (Table 5.3.5).

Table 5.3.3 *Model 1: crude odds ratios and 95% confidence intervals for depressive symptoms according to measures of inclusion in social networks and alcohol consumption.*

	GHQ	CESD
	OR (95% C.I.)[1]	**OR (95% C.I.)[2]**
Frequency of contact with friends		
Never/almost never	1.15 (0.87-1.51)	1.41 (1.09-1.83)
Once every few months	1	1
About once a month	0.80 (0.65-0.99)	0.78 (0.64-0.96)
About once a week	0.70 (0.58-0.85)	0.71 (0.58-0.86)
Almost daily	0.58 (0.42-0.79)	0.43 (0.31-0.60)
Frequency of contact with relatives		
Never/almost never	1.70 (1.33-2.17)	1.45 (1.13-1.86)
Once every few months	1	1
About once a month	0.95 (0.77-1.17)	0.89 (0.73-1.10)
About once a week	0.88 (0.72-1.08)	0.85 (0.70-1.04)
Almost daily	1.00 (0.73-1.36)	0.78 (0.57-1.08)
Frequency of alcohol consumption		
Never/Special occasions	1.48 (1.18-1.85)	1.91 (1.54-2.37)
Once or more a month	1.15 (0.87-1.53)	1.34 (1.02-1.75)
Once or more a week	1	1
Once or more a day	1.07 (0.89-1.28)	0.99 (0.83-1.19)
Amount of alcohol consumed per week		
None	1.59 (1.29-1.97)	1.72 (1.40-2.11)
1-7 units	1	1
8-14 units	1.02 (0.82-1.27)	0.86 (0.69-1.06)
15-21 units	1.08 (0.84-1.39)	0.90 (0.70-1.15)
22-28 units	0.89 (0.64-1.24)	0.66 (0.47-0.94)
29-35 units	0.93 (0.61-1.43)	0.91 (0.59-1.39)
36+ units	1.27 (0.91-1.76)	1.51 (1.11-2.06)

[1] Adjusted for age and sex

[2] Adjusted for age and sex

Table 5.3.4 *Model 2: partially adjusted odds ratios and 95% confidence intervals for depressive symptoms according to measures of inclusion in social networks and alcohol consumption.*

	GHQ	CESD
	OR (95% C.I.)[1]	**OR (95% C.I.)[2]**
Frequency of contact with friends		
Never/almost never	1.36 (0.96-1.92)	1.32 (0.94-1.86)
Once every few months	1	1
About once a month	0.93 (0.71-1.22)	0.80 (0.61-1.05)
About once a week	0.75 (0.58-0.97)	0.66 (0.51-0.84)
Almost daily	0.62 (0.43-0.91)	0.41 (0.29-0.62)
Frequency of contact with relatives		
Never/almost never	1.86 (1.37-2.52)	1.38 (1.01-1.88)
Once every few months	1	1
About once a month	1.05 (0.80-1.37)	0.88 (0.68-1.15)
About once a week	0.87 (0.67-1.13)	0.83 (0.65-1.07)
Almost daily	1.17 (0.82-1.68)	0.75 (0.51-1.09)
Frequency of alcohol consumption		
Never/Special occasions	1.29 (0.97-1.72)	1.60 (1.21-2.12)
Once or more a month	1.17 (0.82-1.67)	1.56 (1.11-2.17)
Once or more a week	1	1
Once or more a day	1.20 (0.95-1.51)	1.08 (0.85-1.36)
Amount of alcohol consumed per week		
None	1.43 (1.09-1.86)	1.36 (1.06-1.76)
1-7 units	1	1
8-14 units	1.10 (0.83-1.45)	0.87 (0.66-1.15)
15-21 units	1.19 (0.86-1.65)	0.84 (0.61-1.18)
22-28 units	1.09 (0.73-1.65)	0.82 (0.54-1.25)
29-35 units	1.18 (0.69-2.01)	0.94 (0.53-1.64)
36+ units	1.66 (1.12-2.46)	1.50 (1.01-2.22)

[1] Adjusted for age, sex, marital status, employment grade, last employment grade before retirement, smoking status and physical activity

[2] Adjusted for age, sex, marital status, employment grade, last employment grade before retirement, smoking status and physical activity

Table 5.3.5 *Model 3: fully odds ratios and 95% confidence intervals for depressive symptoms according to measures of inclusion in social networks and alcohol consumption.*

	GHQ	CESD
	OR (95% C.I.)[1]	**OR (95% C.I.)[2]**
Frequency of contact with friends		
Never/almost never	1.36 (0.96-1.92)	1.27 (0.90-1.79)
Once every few months	1	1
About once a month	0.94 (0.72-1.23)	0.80 (0.61-1.04)
About once a week	0.74 (0.57-0.95)	0.66 (0.51-0.84)
Almost daily	0.60 (0.41-0.88)	0.42 (0.28-0.62)
Frequency of contact with relatives		
Never/almost never	1.85 (1.36-2.52)	1.37 (1.00-1.87)
Once every few months	1	1
About once a month	1.05 (0.80-1.37)	0.88 (0.70-1.15)
About once a week	0.87 (0.67-1.13)	0.82 (0.64-1.05)
Almost daily	1.19 (0.83-1.71)	0.75 (0.51-1.10)
Frequency of alcohol consumption		
Never/Special occasions	1.26 (0.93-1.69)	1.51 (1.13-2.01)
Once or more a month	1.18 (0.82-1.69)	1.58 (1.13-2.22)
Once or more a week	1	1
Once or more a day	1.20 (0.94-1.52)	1.07 (0.84-1.37)
Amount of alcohol consumed per week		
None	1.39 (1.06-1.83)	1.28 (0.98-1.66)
1-7 units	1	1
8-14 units	1.08 (0.81-1.44)	0.84 (0.64-1.12)
15-21 units	1.18 (0.85-1.66)	0.88 (0.62-1.23)
22-28 units	1.17 (0.77-1.77)	0.85 (0.55-1.31)
29-35 units	1.26 (0.73-2.15)	1.02 (0.58-1.79)
36+ units	1.63 (1.08-2.44)	1.49 (0.99-2.24)

[1] Adjusted for age, sex, marital status, employment grade, last employment grade before retirement, smoking status, physical activity and measures of alcohol consumption or of frequency of contact with friends and relatives

[2] Adjusted for age, sex, marital status, employment grade, last employment grade before retirement, smoking status, physical activity and measures of alcohol consumption or of frequency of contact with friends and relatives

These differences between the results yielded by the CESD and GHQ depression scales are likely to be due to the fact that the CESD scale was designed to detect depressive symptoms alone in the general population, while the GHQ scale was designed to detect mental disorders in general. Hence, the higher odds of depressive symptoms yielded by the GHQ scale might be a figure that includes odds of depressive symptoms but also of other mental disorders that are detected by the scale; while the lower odds of depressive symptoms yielded by the CESD scale might be a more specific representation of the odds of depressive symptoms in the population. This should be kept in mind when reading the results of the remaining of the analysis presented below, especially for what concerns differences between countries, which might be entirely, or partly due to the two scales used. From the analysis presented in this section, it safe to say that one should expect to find in the Czech Republic, Russia and Poland, slightly lower odds of depressive symptoms according to measures of frequency of contact with friends and relatives and measures of amount of alcohol consumed, and slightly higher odds of depressive symptoms according to measures of frequency of alcohol consumption, compared to the UK, purely because of the difference in the scales used.

5.3.2 Whitehall II descriptive statistics

Descriptive statistics revealed how of the 10, 208 participants of the Whitehall II cohort, 66.1% were men and 33.1% women. Among men, 29.2% were aged 35-39; 80.4% were married or cohabiting and 52.3% were employed in the professional/executive grades of the civil service. In contrast, 31.1% of women were aged 50-55, 61.2% married or cohabiting, and nearly half (49.7%) were employed in the clerical/support grades of the civil service (Table 5.3.1). Depressive symptoms were generally more prevalent among women (14.5%) than among men (12.9%), among non-married rather than married individuals, and among participants who were employed in the lower grades of the civil service (Table 5.3.6).

Further, the majority of participants reported seeing up to five friends and relatives anywhere between once a week and once a month and 42.5% of men and 35.4 % of women reported drinking alcoholic beverages at least once a week, with 45.6% of men consuming between 1g and 80g of alcohol per week, and 42.3% of women consuming up to 48g of alcohol per week (Table 5.3.1). Depressive symptoms were most prevalent among men and women who reported not visiting any friends or relative on a regular basis and among men who reported not drinking or only in special occasions (15.4%) and women who reported drinking at least once a day (15.8%) or more than 80g of alcohol per week (17.0%)(Table 5.3.6).

Table 5.3.6.*Prevalence of depressive symptoms according to general characteristics, in men and women at Phase 1 of the Whitehall II cohort study*.

		Men			Women	
	%	Depr. %	P	%	Depr. %	P
Total (10, 208)	**66.9**	**12.9**		**33.1**	**14.5**	
Age groups			0.536			0.063
35-39	29.2	12.5		23.3	14.4	
40-44	27.1	13.3		23.1	17.0	
45-49	19.5	13.7		22.5	12.2	
50-55	24.2	12.2		31.1	14.3	
Marital Status			<0.001			0.001
Married/cohabiting	80.4	10.9		61.2	12.7	
Single	13.9	19.4		21.6	16.0	
Divorced	5.2	24.6		14.1	18.9	
Widowed	0.5	29.4		3.1	19.8	
Employment grade			<0.001			0.192
Administrative	38.4	10.6		11.2	11.9	
Prof/executive	52.3	13.6		39.2	15.5	
Clerical/support	9.3	18.0		49.7	14.3	
Frequency of contact with friends			<0.001			<0.001
Never	9.7	18.7		9.7	21.3	
Every few months	20.1	13.6		20.1	18.0	
Once a month	34.3	12.2		31.5	13.1	
Once a week	32.2	11.5		34.2	11.8	
Daily	3.8	13.6		4.5	15.1	
Frequency of contact with relatives			<0.001			0.001
Never	10.0	21.6		9.5	21.8	
Every few months	24.2	12.6		21.0	15.4	
Once a month	31.7	12.9		27.8	14.8	
Once a week	29.6	10.4		33.6	12.4	
Daily	4.5	12.1		8.1	11.8	
Frequency of drinking in past year			0.092			0.568
Special occasions/Never	12.0	15.4		31.9	14.6	
Once or twice a month	11.7	13.0		14.0	12.8	
Once or twice a week	42.5	12.1		34.3	14.3	
Daily or more	33.8	13.0		19.8	15.8	
Intensity of consumption			0.001			0.003
None	13.0	15.4		29.1	15.8	
1-7 units	34.0	12.4		46.2	13.2	
8-14 units	22.4	12.2		15.0	13.5	
15-21 units	11.9	11.0		5.7	17.3	
22-28 units	7.2	11.6		2.0	20.3	
29-35 units	4.1	11.5		1.0	34.3	
36+ units	7.3	18.2		0.9	6.4	

5.3.3 Whitehall II - Inclusion in Social Networks, Alcohol Consumption and Depressive Symptoms

The first aim of the analysis presented in this section was to investigate the relationship between inclusion in social networks and depressive symptoms, focusing on whether individuals who were more connected with their friends and family would have lower odds of depressive symptoms than their more isolated counterparts. Logistic regression models showed that indeed, at baseline of the Whitehall II cohort study, participants who admitted to never meeting with their friends were more likely to suffer from depressive symptoms (OR 1.29; 95% C.I. 1.05-1.58) while individuals who met with their friends on a weekly basis were the least likely to suffer from depressive symptoms (OR 0.70; 95% C.I. 0.59-0.82). Furthermore, when compared to participants who reported spending time with their relatives only once every few months, participants who admitted to never meet their relatives were 1.56 (95% C.I. 1.28-1.90) times more likely to report depressive symptoms, while participants visited their relatives on a weekly basis were 0.80 (95% 0.68-0.95) times less likely to suffer from depressive symptoms (Table 5.3.7).

The second aim of this section was to investigate the association between alcohol consumption and depressive symptoms, paying particular attention at whether participants who consumed either heavily and frequently or did not at all had higher odds of depressive symptoms than those who drank in moderation. Logistic regression models showed that indeed, compared to those who reported drinking a few times a week, both participants who reported never drinking (OR 1.06; 95% C.I. 0.89-1.26) and participants who reported drinking once or even twice a day (OR 1.13; 95% C.I. 0.97-1.31) had higher odd of

depressive symptoms. However the association was not statistically significant and appeared to be confounded by the effects of age, gender, marital status, employment grade and measures of inclusion in social networks (Table 5.3.7). In the same fashion, high odds of depressive symptoms were found among both men and women who reported consuming 36 or more units per week (OR 1.42; 95% C.I. 1.10-1.83) (Table 5.3.7).

Finally, this section aimed at investigating whether the associations between inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms were net of the effects of age, gender, marital status, employment grade and either inclusion in social networks or alcohol consumption. Likelihood of ratio tests revealed how while the association between social networks and depressive symptoms was net of the effects of confounders, the association between alcohol consumption and depressive symptoms was not so (Table 5.3.7).

Table 5.3.7 *Odds ratios and 95% confidence intervals for depressive symptoms according to measures of inclusion in social networks and alcohol consumption*

	Model 1	Model 2	Model 3
	OR (95% C.I.)[1]	OR (95% C.I.)[2]	OR (95% C.I.)[3]
Frequency of contact with friends			
Never	1.37 (1.13-1.68)	1.28 (1.05-1.57)	1.29 (1.05-1.58)
Every few months	1	1	1
Once a month	0.81 (0.69-0.95)	0.81 (0.69-0.96)	0.81 (0.69-0.95)
Once a week	0.74 (0.63-0.87)	0.70 (0.59-0.83)	0.70 (0.59-0.82)
Daily	0.92 (0.68-1.25)	0.80 (0.59-1.10)	0.80 (0.59-1.09)
Frequency of contact with relatives			
Never	1.78 (1.47-2.16)	1.56 (1.28-1.90)	1.56 (1.28-1.90)
Every few months	1	1	1
Once a month	1.00 (0.85-1.17)	1.02 (0.87-1.20)	1.02 (0.87-1.20)
Once a week	0.79 (0.68-0.94)	0.81 (0.68-0.95)	0.80 (0.68-0.95)
Daily	0.85 (0.64-1.13)	0.85 (0.64-1.12)	0.85 (0.64-1.13)
Frequency of drinking in past year			
Special occasions/Never	1.17 (1.00-1.38)	1.09 (0.92-1.28)	1.06 (0.89-1.26)
Once or twice a month	1.01 (0.84-1.22)	1.01 (0.83-1.22)	0.99 (0.81-1.21)
Once or twice a week	1	1	1
Daily or more	1.09 (0.95-1.25)	1.11 (0.96-1.28)	1.13 (0.97-1.31)
Dose consumed per week (M)			
None	1.29 (1.03-1.61)	1.15 (0.92-1.45)	1.11 (0.88-1.39)
1-7 units	1	1	1
8-14 units	0.98 (0.81-1.20)	0.97 (0.79-1.18)	0.97 (0.80-1.19)
15-21 units	0.87 (0.68-1.12)	0.88 (0.68-1.13)	0.88 (0.68-1.14)
22-28 units	0.93 (0.69-1.26)	0.91 (0.68-1.23)	0.94 (0.69-1.27)
29-35 units	0.92 (0.62-1.36)	0.87 (0.59-1.29)	0.86 (0.47-1.28)
36+ units	1.57 (1.21-2.04)	1.36 (1.05-1.78)	1.39 (1.07-1.82)
Dose consumed per week (W)			
None	1.23 (0.98-1.55)	1.22 (0.97-1.54)	1.15 (0.91-1.45)
1-7 units	1	1	1
8-14 units	1.03 (0.76-1.38)	1.06 (0.79-1.43)	1.09 (0.81-1.47)
15-21 units	1.51 (1.10-2.07)	1.58 (1.13-2.19)	1.63 (1.17-2.27)

[1] Adjusted for age and sex

[2] Adjusted for age, sex, marital status and employment grade

[3] Adjusted for age, sex, marital status, employment grade, frequency and quantity of alcohol consumption

5.3.4 HAPIEE descriptive statistics

Descriptive statistics showed how of the 25,674 participants included in the study, 32.2% were Czech, 26.9% were Russian and 40.9% were Polish. In each country just over half of participants were women, and the male population tended to be slightly older as 23.4% of Czech men and 22.9% of Russian men were aged 65-69 (Table 5.3.8 and Table 5.3.9). The vast majority of participants were married, and while 50.5% of Czech men were still employed, 48.0% of Czech women were pensioners; similarly, in Russia 41.3% of men were still employed while 48.2% of women were pensioners; in Poland on the other hand the highest proportion of both men and women were pensioners (Table 5.3.10).

Prevalence of depressive symptoms was between 1.6 (Poland), 1.7 (Czech Republic) and 2.2 (Russia) times higher among women than among men. In the Czech Republic, depressive symptoms were least prevalent among men and women aged 60 to 64, who were married, and retired but still working (Table 5.3.8). In Russia, lowest prevalence of depressive symptoms was to be found among men aged 50 to 54 and women aged 60 to 64 and among men and women who were married and still employed (Table 5.3.9). Finally, in Poland, depressive symptoms were least prevalent among men and women aged 65 to 69, men who were married and women who were single, men who were retired but still working and women who were still employed (Table 5.3.10).

Moreover, in all three countries between 24.4% (Russian men) and 33.4% (Polish men) of participants reported seeing their friends less than once a month; with the exception of Czech women, 22.2% of whom reported seeing their friends once a month (Table 5.3.8 and

Table 5.3.9). In the Czech republic, 28.0% of men and 42.2% of women reported seeing their distant relatives several times a week; while in Russia 29.3% reported seeing their distant relatives once a week and 30.3% women several times a week; finally in Poland, 30.9% of men reported seeing their distant relatives several times a month, and 25.7 of women less than once a month (Table 5.3.8; Table 5.3.9; Table 5.3.10). Further, in all three countries men consumed alcohol more frequently and more heavily than women. In fact, if a higher proportion of men in all three countries reported consuming alcohol between one and four times a week, the higher proportion of Czech and Russian women reported drinking once a month, and of Polish women reported never drinking (Table 5.3.8; Table 5.3.9; Table 5.3.10). Similarly, while higher proportion of Czech and Russian men reported consuming up to 79ml of alcohol per drinking session and Polish men consumed up to 39ml of alcohol per drinking session; Czech and Russian women consumed on average up to 39ml of alcohol per session and 46.3% of Polish women declared to be abstainers (Table 5.3.8; Table 5.3.9; Table 5.3.10).

Furthermore, patterns of prevalence of depressive symptoms varied markedly between countries and genders. In fact, in the Czech Republic prevalence of depressive symptoms was lower among men and women who visited their friends several times a month and their distant relatives several times a week; among men who consumed alcohol five or more times a week and women who drunk up to four times a week and among men who consumed up to 39ml of alcohol per drinking session and women who consumed up to 19ml 39ml of alcohol per drinking session (Table 5.3.8). In Russia depressive symptoms were least prevalent among men who visited their friends once a week and their distant relatives several times a month, drunk up to four times a week and consumed an average of 80ml or more of alcohol per

drinking session; they were also least prevalent among women who visited their friends several times a month and their distant relatives once a week, consumed alcohol up to four times a week and on average up to 39ml of alcohol per session (Table 5.3.9). Finally, in Poland, depressive symptoms were least prevalent among men who visited their friends and distant relatives several times a month, drunk at least on a monthly basis, and consumed an average of up to 19ml of alcohol per drinking session; they were also least prevalent among women who visited their friends once a month and their distant relatives several times a month, drunk up to four times a week and consumed an average of up to 39ml of alcohol per session (Table 5.3.10).

Table 5.3.8 *Prevalence of depressive symptoms according to general characteristics, in men and women in the Czech Republic.*

	Men			Women		
	%	Depr %	P	%	Depr %	P
Total	**46.6**	**14.1**		**53.4**	**24.1**	
Age, Years			0.093			0.001
45-49	16.0	14.2		18.2	23.9	
50-54	19.1	16.6		20.8	26.9	
55-59	19.8	14.8		18.3	21.5	
60-64	21.8	11.9		23.7	20.8	
65-69	23.4	13.2		18.9	27.6	
Marital Status			<0.001			<0.001
Single	2.9	20.3		2.3	28.4	
Married/Cohabiting	83.9	12.8		68.4	21.4	
Divorced/Separated	9.9	21.3		15.3	26.2	
Widowed	3.2	17.7		14.0	34.0	
Occupation			<0.001			<0.001
Employed	50.5	12.5		41.6	22.1	
Pensioner, still employed	8.3	7.6		7.7	17.8	
Pensioner, unemployed	38.1	16.0		48.0	26.1	
Unemployed	3.1	32.2		2.7	42.9	
Contact with friends			<0.001			<0.001
Don't have any	2.8	36.1		2.6	43.9	
Less than once a month	25.0	16.3		19.8	27.1	
Once a month	22.4	14.2		22.1	22.6	
Several times a month	21.0	9.9		21.9	21.4	
Once a week	18.3	13.0		21.6	23.9	
Several times a week	10.5	12.7		12.0	22.5	
Contact with distant relatives			<0.001			<0.001
Don't have any	1.2	26.1		0.8	32.4	
Less than once a month	13.1	20.8		7.3	29.4	
Once a month	13.6	17.8		8.1	29.0	
Several times a month	18.0	13.0		15.1	25.4	
Once a week	26.1	14.1		26.4	25.4	
Several times a week	28.0	9.3		42.2	22.5	
Drinking Frequency			0.008			<0.001
Never	6.2	21.8		17.8	29.8	
Once a month	16.9	14.3		32.9	24.4	
Once a week	17.6	13.6		24.0	22.5	
1 to 4 times a week	36.7	13.4		20.8	19.7	
5+ times a week	22.6	12.6		4.4	21.0	
Mean dose consumed per drinking session			0.002			<0.001
Non-drinkers	6.2	21.8		17.8	29.8	
10-19 ml	21.1	13.2		32.9	21.4	
20-39 ml	37.9	12.7		38.5	21.9	
40-79 ml	23.3	13.1		7.9	22.9	
80+ ml	11.6	16.6		2.9	41.6	

Table 5.3.9. *Prevalence of depressive symptoms according to general characteristics, in men and women in Russia.*

	Men			Women		
	%	Depr. %	P	%	Depr. %	P
Total	44.9	15.0		55.1	33.6	
Age, Years			0.003			<0.001
45-49	16.4	13.1		18.3	29.6	
50-54	20.5	12.4		18.9	31.3	
55-59	21.2	15.3		22.5	32.8	
60-64	18.9	13.6		17.8	33.4	
65-69	22.9	19.4		22.5	40.0	
Marital Status			<0.001			<0.001
Single	2.3	17.1		4.5	34.9	
Married/Cohabiting	88.5	13.6		60.6	30.2	
Divorced/Separated	5.3	21.1		14.4	37.9	
Widowed	3.9	37.7		20.4	40.7	
Occupation			<0.001			<0.001
Employed	41.3	10.8		32.0	27.6	
Pensioner, still employed	21.4	13.1		16.7	28.7	
Pensioner, unemployed	31.7	20.6		48.2	39.1	
Unemployed	5.6	20.0		3.1	34.7	
Contact with friends			<0.001			<0.001
Don't have any	12.9	28.0		8.5	48.9	
Less than once a month	24.4	16.0		29.0	36.1	
Once a month	18.4	11.4		20.4	33.3	
Several times a month	10.0	14.5		11.4	24.1	
Once a week	15.2	9.8		15.1	27.5	
Several times a week	19.1	12.9		15.6	34.2	
Contact with distant relatives			<0.001			<0.001
Don't have any	3.3	26.0		3.2	54.5	
Less than once a month	21.6	18.0		16.5	38.6	
Once a month	16.4	15.5		13.3	33.5	
Several times a month	10.1	10.5		8.6	31.0	
Once a week	29.3	12.6		28.2	26.6	
Several times a week	19.2	15.1		30.3	36.2	
Drinking Frequency			0.001			0.008
Never	13.5	18.7		16.3	38.2	
Once a month	16.9	16.0		54.3	33.6	
Once a week	23.5	14.8		20.8	32.6	
1 to 4 times a week	41.5	12.6		8.2	26.9	
5+ times a week	4.6	23.1		0.4	46.7	
Mean dose consumed per drinking session			0.007			0.009
Non-drinkers	13.5	18.7		16.3	38.2	
10-19 ml	2.6	25.9		20.0	34.4	
20-39 ml	20.8	14.5		50.1	31.8	
40-79 ml	37.4	13.9		12.1	32.5	
80+ ml	25.7	13.8		1.5	46.4	

Table 5.3.10. *Prevalence of depressive symptoms according to general characteristics, in men and women in Poland.*

	Men			Women		
	%	Depr. %	P	%	Depr. %	P
Total	48.7	20.4		51.3	32.9	
Age, Years			0.329			0.673
45-49	17.3	20.8		19.7	33.0	
50-54	19.8	21.5		21.4	33.9	
55-59	21.4	21.8		20.3	31.6	
60-64	20.4	19.0		19.5	32.0	
65-69	21.0	19.0		19.1	34.0	
Marital Status			<0.001			<0.001
Single	4.1	27.9		7.1	28.4	
Married/Cohabiting	86.5	18.3		66.5	30.0	
Divorced/Separated	5.6	35.9		9.2	46.4	
Widowed	3.8	38.8		17.1	38.7	
Occupation			<0.001			<0.001
Employed	40.7	14.4		36.1	27.3	
Pensioner, still employed	7.7	14.2		5.6	26.8	
Pensioner, unemployed	45.6	24.4		54.0	36.1	
Unemployed	6.0	34.9		4.3	46.7	
Contact with friends			<0.001			<0.001
Don't have any	7.6	33.2		6.8	52.2	
Less than once a month	33.4	21.2		29.8	37.4	
Once a month	24.3	17.2		23.4	28.3	
Several times a month	16.0	16.7		17.2	28.9	
Once a week	12.1	19.7		14.6	28.6	
Several times a week	6.6	24.1		8.1	28.7	
Contact with distant relatives			<0.001			<0.001
Don't have any	3.6	36.2		4.7	53.8	
Less than once a month	30.3	24.0		25.7	38.9	
Once a month	30.9	18.2		17.1	31.2	
Several times a month	17.7	14.1		17.6	27.2	
Once a week	17.3	19.6		20.2	29.1	
Several times a week	10.2	21.1		14.7	29.4	
Drinking Frequency			<0.001			<0.001
Never	21.9	27.3		46.3	36.1	
Once a month	19.2	18.0		27.2	31.4	
Once a week	23.1	18.0		16.5	28.9	
1 to 4 times a week	28.5	18.3		8.9	26.7	
5+ times a week	7.3	22.5		1.0	34.5	
Mean dose consumed per drinking session			<0.001			<0.001
Non-drinkers	21.9	27.3		46.3	36.1	
10-19 ml	23.9	16.8		29.5	30.1	
20-39 ml	32.4	17.8		20.4	27.7	
40-79 ml	14.6	20.4		2.9	36.7	
80+ ml	7.2	24.2		0.8	52.3	

5.3.5 HAPIEE – Inclusion in Social Networks, Alcohol Consumption and Depressive Symptoms

As for with data from the Whitehall II cohort study, the first aim of this analysis was to investigate the relationship between inclusion in social networks and depressive symptoms, focusing on whether individuals who were more connected with their friends and family would have lower odds of depressive symptoms than their more isolated counterparts, and paying attention to differences in the association due to country of origin or gender. To this end, results are presented separately for each country and for men and women.

In the Czech Republic, compared to participants who visited their friends less than once a month, men who reported not having any friends were 2.86 (95% C.I. 1.85-4.42) times more likely to suffer from depressive symptoms; while women who reported not having any friends were 1.97 (95% C.I. 1.31-2.97) times more likely to suffer from depressive symptoms. Conversely, visiting friends several times a month was associated with lower odds of depressive symptoms for both men (OR 0.57; 95% C.I. 0.43-0.76) and women (OR 0.74; 95% C.I. 0.60-0.92). Similarly, being in contact with relatives outside of the household was associated with the lowest odds of suffering from depressive symptoms for both men (OR 0.39; 95% C.I. 0.28-0.52) and women (OR 0.63; 95% C.I. 0.48-0.82) (Table 5.3.11).

The second aim of this analysis was to investigate the association between alcohol consumption and depressive symptoms, paying particular attention at whether participants who consumed either heavily and frequently or did not at all had higher odds

of depressive symptoms than those who drank in moderation and at whether there would be any gender or country difference. Logistic regression showed that Czech men (OR 1.73; 95% C.I. 1.21-2.47) and women (OR 1.31; 95% C.I. 1.08-1.60) who reported being abstainers had higher odds of depressive symptoms than those who consumed alcohol. In addition, Czech women who reported drinking up to four times a week had 0.75 (95% C.I. 0.61-0.93) lower odds of depressive symptoms, compared to women who consumed only once a month. Similarly, compared to participants who reported consuming on average up to 39ml of alcohol per drinking session, both men (OR 1.82; 95% C.I. 1.28-2.60) and women (OR 1.49; 95% C.I. 1.22-1.82) who were abstainers had higher odds of suffering from depressive symptoms. In addition, women who consumed 280ml or more of alcohol per drinking session had 2.58 (95% C.I. 1.76-3.77) higher odds of depressive symptoms (Table 5.3.11).

A similar pattern of association was observed in Russia, where both men (OR 1.81; 95% C.I. 1.34-2.45) and women (OR 1.57; 95% C.I. 1.22-2.02) who reported not having any friends had higher odds of depressive symptoms than their counterparts who visited their friends less than once a month. Further, Russian men who reported visiting their friends once a week had 0.54 (95% C.I. 0.38-0.79) lower odds of depressive symptoms, and Russian women who visited their friends once a month had 0.56 (95% C.I. 0.43-0.72) lower odds of depressive symptoms. Low odds of depressive symptoms were also found among Russian men (OR 0.51; 95% C.I. 0.33-0.77) who visited their relatives once a month, and among Russian women (OR 0.55; 95% C.I. 0.45-0.69) who visited their relatives once a week. Russian women who reported not having any relatives, on the other hand, had 1.82 (95% C.I. 1.23-2.71) higher odds of suffering from depressive symptoms (Table 5.3.12).

For what concerns alcohol consumption, Russian men who reported never drinking were 1.47 (95% C.I. 1.09-1.99) times more likely to suffer from depressive symptoms than their counterparts who drank up to four times a week, but also Russian men who drank five or more times a week were 1.92 (95% C.I. 1.24-3.00) times more likely to suffer from depressive symptoms. While Russian women who did not consume alcohol were 1.39 (95% C.I. 1.02-1.89) times more likely to suffer from depressive symptoms than those who drank up to four times a week. Further, consuming an average of up to 19ml of alcohol per session was associated with higher odds of depressive symptoms among Russian men (OR 2.12; 95% C.I. 1.23-2.64), as drinking 280ml or more of alcohol per session was among Russian women (OR 1.93; 95% C.I. 1.93-3.33) (Table 5.3.12).

Further, data coming from Poland also confirmed the hypothesis that more socially isolated individuals would be more likely to suffer from depressive symptoms. In fact, both men (OR 1.80; 95% C.I. 1.49-2.29) and women (OR 1.79; 95% C.I. 1.48-2.25) who reported not having any friends were almost twice as likely to suffer from depressive symptoms as those who visited their friends as seldom as less than once a month (Table 5.3.10). And the same pattern was observed among both men (OR 1.78; 95% C.I. 1.29-2.47) and women (OR 1. 84; 95% C.I. 1.40-2.42) who reported not having any relatives (Table 5.3.10). When looking at what frequency of contact with friends was associated with the lowest likelihood of suffering from depressive symptoms, the latter were found among both men (OR 0.76; 95% C.I. 0.63-0.92) and women (OR 0.67; 95% C.I. 0.57-0.78) who reported seeing their friends once a month (Table 5.3.10); similarly, the odds of suffering from depressive symptoms for

both men (OR 0.52; 95 % C.I. 042-0.65) and women (OR 0.59; 95% C.I. 0.50-0.71) who reported visiting their relatives several times a month (Table 5.3.13).

The hypothesis that both abstainers and heavy drinkers would be more likely to suffer from the depressive symptoms than moderate drinkers was confirmed by analysis of measures of mean dose of alcohol consumed per drinking session by Polish men and women, but not by measures of frequency of consumption. In fact, Polish men 1.65 (95% C.I. 1.36-2.00) and women (OR 1.22; 95% C.I. 1.06-1.40) who reported never drinking had higher odds of depressive symptoms than men who drank up to four times a week, and women who consumed alcohol once a month (Table 5.3.10). Further, compared to participants who consumed up to 39ml of alcohol per drinking session, odds of depressive symptoms were higher among men (OR 1.72; 95% C.I. 1.42-2.06) and women (OR 1.44; 95% C.I. 1.23-1.70) who did not drink at all, and among men (OR 1.41; 95% C.I. 1.07-1.85) and women (OR 2.60; 95% C.I. 1.41-4.80) who consumed 280ml or more of alcohol per drinking session (Table 5.3.13).

Table 5.3.11 *Odds ratios and 95% confidence intervals for depressive symptoms according to measures of inclusion in social networks and alcohol consumption in the Czech Republic*

	Men			Women		
	Model 1	**Model2**	**Model3**	**Model 1**	**Model 2**	**Model 3**
	OR (95% C.I.)[1]	**OR (95% C.I.)[2]**	**OR (95% C.I.)[3]**	**OR (95% C.I.)**	**OR (95% C.I.)**	**OR (95% C.I.)**
Contact with friends						
Don't have any	2.93 (1.91-4.51)	2.92 (1.90-4.49)	2.86 (1.85-4.42)	2.11 (1.41-3.14)	2.11 (1.42-3.15)	1.97 (1.31-2.97)
Less than once a month	1	1	1	1	1	1
Once a month	0.85 (0.66-1.00)	0.85 (0.66-1.10)	0.88 (0.68-1.14)	0.79 (0.64-0.97)	0.78 (0.63-0.97)	0.78 (0.62-0.96)
Several times a month	0.56 (0.43-0.75)	0.56 (0.42-0.75)	0.57 (0.43-0.76)	0.73 (0.51-0.91)	0.73 (0.51-0.91)	0.74 (0.60-0.92)
Once a week	0.77 (0.58-1.01)	0.77 (0.58-1.01)	0.79 (0.59-1.04)	0.85 (0.69-1.05)	0.85 (0.69-1.05)	0.84 (0.68-1.04)
Several times a week	0.74 (0.53-1.05)	0.74 (0.53-1.04)	0.72 (0.51-1.03)	0.78 (0.61-1.01)	0.78 (0.60-1.00)	0.78 (0.60-1.00)
Contact with distant relatives						
Don't have any	1.85 (0.67-2.69)	1.34 (0.67-2.69)	1.17 (0.56-2.47)	1.15 (0.55-2.38)	1.15 (0.55-2.38)	1.19 (0.57-2.51)
Less than once a month	1	1	1	1	1	1
Once a month	0.83 (0.61-1.13)	0.83 (0.61-1.13)	0.83 (0.61-1.14)	0.98 (0.71-1.37)	0.98 (0.70-1.37)	1.01 (0.72-1.42)
Several times a month	0.56 (0.41-0.77)	0.57 (0.42-0.77)	0.59 (0.43-0.80)	0.82 (0.61-1.10)	0.82 (0.61-1.10)	0.83 (0.62-1.13)
Once a week	0.62 (0.47-0.82)	0.63 (0.47-0.83)	0.63 (0.48-0.84)	0.82 (0.62-1.08)	0.82 (0.62-1.08)	0.83 (0.62-1.10)
Several times a week	0.39 (0.29-0.53)	0.39 (0.29-0.53)	0.39 (0.28-0.52)	0.63 (0.48-0.82)	0.62 (0.48-0.82)	0.63 (0.48-0.82)
Drinking Frequency						
Never	1.87 (1.32-2.65)	1.86 (1.31-2.64)	1.73 (1.21-2.47)	1.32 (1.09-1.62)	1.32 (1.08-1.61)	1.31 (1.08-1.60)
Once a month	1.10 (0.84-1.44)	1.10 (0.84-1.45)	1.05 (0.80-1.38)	1	1	1
Once a week	1.03 (0.79-1.35)	1.03 (0.79-1.36)	1.02 (0.78-1.35)	0.89 (0.74-1.08)	0.89 (0.74-1.08)	0.90 (0.75-1.09)
1 to 4 times a week	1	1	1	0.75 (0.61-0.92)	0.75 (0.61-0.92)	0.75 (0.61-0.93)
5+ times a week	0.94 (0.73-1.22)	0.94 (0.73-1.22)	0.95 (0.73-1.22)	0.82 (0.57-1.19)	0.82 (0.57-1.19)	0.81 (0.56-1.17)
Mean dose of alcohol						
Non-drinkers	1.95 (1.38-2.77)	1.94 (1.37-2.75)	1.82 (1.28-2.60)	1.52 (1.24-1.85)	1.51 (1.24-1.84)	1.49 (1.22-1.82)
10-19 ml	1.05 (0.82-1.36)	1.05 (0.82-1.36)	1.02 (0.79-1.32)	0.97 (0.81-1.15)	0.97 (0.81-1.15)	0.97 (0.81-1.15)
20-39 ml	1	1	1	1	1	1
40-79 ml	1.02 (0.79-1.31)	1.02 (0.79-1.31)	1.05 (0.82-1.35)	1.05 (0.80-1.39)	1.05 (0.80-1.39)	1.04 (0.79-1.38)
280+ ml	1.33 (0.99-1.80)	1.33 (0.99-1.79)	1.34 (0.99-1.81)	2.52 (1.74-3.67)	2.53 (1.74-3.69)	2.58 (1.76-3.77)

[1] Adjusted for age

[2] Adjusted for age, marital status and occupation

[3] Adjusted for age, marital status, employment grade, and frequency and quantity of alcohol consumption or frequency of contact with friends and relatives

Table 5.3.12 *Odds ratios and 95% C.I. according to measures of inclusion in social networks and alcohol consumption in men and women in Russia*

	Men			Women		
	Model 1	**Model 2**	**Model 3**	**Model 1**	**Model 2**	**Model 3**
	OR (95% C.I.)[1]	**OR (95% C.I.)[2]**	**OR (95% C.I.)[3]**	**OR (95% C.I.)**	**OR (95% C.I.)**	**OR (95% C.I.)**
Contact with friends						
Don't have any	1.98 (1.47-2.65)	1.85 (1.37-2.49)	1.81 (1.34-2.45)	1.65 (1.28-2.12)	1.57 (1.22-2.03)	1.57 (1.22-2.02)
Less than once a month	1	1	1	1	1	1
Once a month	0.68 (0.49-0.94)	0.65 (0.47-0.90)	0.66 (0.47-0.91)	0.91 (0.75-1.10)	0.91 (0.75-1.11)	0.92 (0.75-1.11)
Several times a month	0.91 (0.63-1.32)	0.85 (0.58-1.24)	0.85 (0.58-1.25)	0.57 (0.44-0.73)	0.56 (0.43-0.72)	0.56 (0.43-0.72)
Once a week	0.58 (0.41-0.84)	0.55 (0.38-0.79)	0.54 (0.38-0.79)	0.68 (0.54-0.85)	0.66 (0.53-0.83)	0.67 (0.53-0.83)
Several times a week	0.79 (0.58-1.08)	0.73 (0.53-1.00)	0.74 (0.54-1.01)	0.91 (0.74-1.25)	0.88 (0.71-1.08)	0.88 (0.71-1.08)
Contact with distant relatives						
Don't have any	1.54 (0.95-2.49)	1.44 (0.88-2.36)	1.44 (0.88-2.36)	1.89 (1.28-2.80)	1.84 (1.24-2.73)	1.82 (1.23-2.71)
Less than once a month	1	1	1	1	1	1
Once a month	0.85 (0.62-1.15)	0.82 (0.60-1.13)	0.81 (0.45-0.79)	0.81 (0.63-1.03)	0.80 (0.62-1.02)	0.80 (0.62-1.02)
Several times a month	0.53 (0.35-0.79)	0.52 (0.34-0.78)	0.51 (0.33-0.77)	0.70 (0.52-0.93)	0.68 (0.51-0.90)	0.67 (0.51-0.90)
Once a week	0.65 (0.49-0.86)	0.60 (0.45-0.80)	0.59 (0.59-1.12)	0.56 (0.45-0.69)	0.55 (0.45-0.69)	0.55 (0.45-0.69)
Several times a week	0.79 (0.59-1.07)	0.76 (0.56-1.03)	0.75 (0.55-1.01)	0.88 (0.72-1.08)	0.86 (0.71-1.06)	0.86 (0.70-1.06)
Drinking Frequency						
Never	1.56 (1.16-2.10)	1.51 (1.12-2.04)	1.47 (1.09-1.99)	1.52 (1.12-2.05)	1.46 (1.08-1.98)	1.39 (1.02-1.89)
Once a month	1.25 (0.93-1.66)	1.23 (0.92-1.64)	1.21 (0.90-1.62)	1.29 (0.99-1.69)	1.27 (0.97-1.67)	1.22 (0.92-1.60)
Once a week	1.16 (0.89-1.51)	1.12 (0.86-1.47)	1.10 (0.84-1.44)	1.30 (0.97-1.74)	1.31 (0.98-1.76)	1.24 (0.93-1.67)
1 to 4 times a week	1	1	1	1	1	1
5+ times a week	2.12 (1.38-3.23)	2.00 (1.30-3.07)	1.92 (1.24-3.00)	2.35 (0.82-6.70)	2.00 (0.70-5.77)	1.79 (0.62-5.15)
Mean dose consumed						
Non-drinkers	1.42 (1.05-1.91)	1.35 (0.99-1.82)	1.34 (0.99-1.81)	1.24 (1.02-1.50)	1.20 (0.99-1.46)	1.19 (0.98-1.45)
10-19 ml	2.08 (1.22-3.51)	2.12 (1.24-3.61)	2.12 (1.23-3.64)	1.09 (0.91-1.31)	1.11 (0.92-1.32)	1.08 (0.90-1.30)
20-39 ml	1.03 (0.78-1.35)	0.99 (0.75-1.31)	0.99 (0.75-1.31)	1	1	1
40-79 ml	1	1	1	1.06 (0.85-1.32)	1.03 (0.83-1.28)	1.03 (0.83-1.29)
280+ ml	1.01 (0.78-1.31)	0.94 (0.72-1.23)	0.95 (0.73-1.25)	2.02 (1.18-3.45)	1.89 (1.10-3.25)	1.93 (1.12-3.33)

[1] Adjusted for age

[2] Adjusted for age, marital status and occupation

[3] Adjusted for age, marital status, employment grade, and frequency and quantity of alcohol consumption or frequency of contact with friends and relatives

Table 5.3.13 *Odds ratios and 95% confidence intervals for depressive symptoms according to measures of inclusion in social networks and alcohol consumption in Poland.*

	Men			Women		
	Model 1	**Model2**	**Model3**	**Model 1**	**Model 2**	**Model 3**
	OR (95% C.I.)[1]	**OR (95% C.I.)[2]**	**OR (95% C.I.)[3]**	**OR (95% C.I.)**	**OR (95% C.I.)**	**OR (95% C.I.)**
Contact with friends						
Don't have any	1.88 (1.47-2.39)	1.86 (1.46-2.37)	1.80 (1.41-2.29)	1.83 (1.46-2.31)	1.82 (1.45-2.29)	1.79 (1.42-2.25)
Less than once a month	1	1	1	1	1	1
Once a month	0.76 (0.63-0.92)	0.76 (0.63-0.92)	0.76 (0.63-0.92)	0.66 (0.56-0.77)	0.66 (0.56-0.78)	0.67 (0.57-0.78)
Several times a month	0.74 (0.59-0.92)	0.74 (0.60-0.92)	0.77 (0.62-0.96)	0.68 (0.57-0.81)	0.68 (0.57-0.81)	0.69 (0.57-0.82)
Once a week	0.90 (0.72-1.14)	0.90 (0.72-1.14)	0.93 (0.73-1.17)	0.67 (0.56-0.81)	0.67 (0.55-0.80)	0.68 (0.56-0.82)
Several times a week	1.18 (0.89-1.55)	1.17 (0.88-1.54)	1.22 (0.93-1.61)	0.67 (0.53-0.85)	0.67 (0.53-0.84)	0.68 (0.54-0.85)
Contact with distant relatives						
Don't have any	1.81 (1.31-2.50)	1.80 (1.30-2.49)	1.78 (1.29-2.47)	1.83 (1.39-2.40)	1.81 (1.38-2.37)	1.84 (1.40-2.42)
Less than once a month	1	1	1	1	1	1
Once a month	0.70 (0.57-0.85)	0.70 (0.58-0.86)	0.70 (0.58-0.86)	0.71 (0.60-0.85)	0.71 (0.60-0.85)	0.72 (0.60-0.86)
Several times a month	0.52 (0.41-0.64)	0.52 (0.41-0.64)	0.52 (0.42-0.65)	0.59 (0.49-0.70)	0.59 (0.49-0.70)	0.59 (0.50-0.71)
Once a week	0.77 (0.63-0.94)	0.77 (0.63-0.95)	0.78 (0.64-0.96)	0.64 (0.54-0.76)	0.64 (0.54-0.76)	0.64 (0.54-0.76)
Several times a week	0.85 (0.67-1.08)	0.85 (0.67-1.08)	0.86 (0.68-1.10)	0.65 (0.54-0.80)	0.65 (0.54-0.79)	0.65 (0.54-0.78)
Drinking Frequency						
Never	1.76 (1.45-2.13)	1.73 (1.43-2.10)	1.65 (1.36-2.00)	1.25 (1.09-1.44)	1.24 (1.08-1.43)	1.22 (1.06-1.40)
Once a month	1.01 (0.82-1.25)	1.01 (0.82-1.25)	0.97 (0.79-1.20)	1	1	1
Once a week	0.99 (0.81-1.21)	0.99 (0.81-1.21)	0.97 (0.79-1.19)	0.88 (0.73-1.05)	0.89 (0.74-1.06)	0.89 (0.74-1.08)
1 to 4 times a week	1	1	1	0.79 (0.63-0.99)	0.79 (0.63-1.00)	0.81 (0.64-1.03)
5+ times a week	1.29 (0.98-1.71)	1.30 (0.98-1.71)	1.28 (0.97-1.70)	1.14 (0.65-2.01)	1.15 (0.65-2.02)	1.11 (0.63-1.97)
Mean dose consumed						
Non-drinkers	1.80 (1.49-2.16)	1.77 (1.47-2.13)	1.72 (1.42-2.07)	1.51 (1.29-1.77)	1.49 (1.27-1.74)	1.44 (1.23-1.70)
10-19 ml	0.95 (0.78-1.15)	0.94 (0.78-1.16)	0.96 (0.79-1.17)	1.13 (0.96-1.34)	1.13 (0.95-1.34)	1.12 (0.94-1.33)
20-39 ml	1	1	1	1	1	1
40-79 ml	1.18 (0.94-1.46)	1.17 (0.94-1.45)	1.17 (0.94-1.46)	1.51 (1.06-2.14)	1.51 (1.06-2.14)	1.58 (1.11-2.25)
280+ ml	1.45 (1.10-1.90)	1.44	1.41 (1.07-1.85)	2.85 (1.56-5.23)	2.84 (1.55-5.22)	2.60 (1.41-4.80)

[1] Adjusted for age

[2] Adjusted for age, marital status and occupation

[3] Adjusted for age, marital status, employment grade, and frequency and quantity of alcohol consumption or frequency of contact with friends and relatives

5.4 Inclusion in Social Networks, Alcohol Consumption and Depressive Symptoms – Differences and Similarities between the Whitehall II and HAPIEE cohorts

This chapter set off to investigate the associations between inclusion in social networks and depressive symptoms and alcohol consumption and depressive symptoms in the adult urban populations of the UK and of Russia, Poland and the Czech Republic. The analysis here presented aimed at testing whether social isolation was associated with higher odds of depressive symptoms, and whether abstention and heavy alcohol consumption were associated with higher odds of depressive symptoms. In addition, this chapter aimed at comparing the patterns of association observed in the UK and in the three countries of Central and Eastern Europe. This section is dedicated to compare the results obtained from the Whitehall II and HAPIEE cohort studies.

It is important to understand that this comparison is rendered difficult by the fact the two cohorts under study are very different in nature. First of all, baseline measurements for the Whitehall II cohort study were carried out between 1985 and 1988, while measurements for the HAPIEE cohort took place between 2002 and 2005. During the seventeen years passed between these two dates the socio-economic and cultural environment of all four countries involved changed dramatically. Secondly, the population of the HAPIEE cohort was on average ten years older than that of the Whitehall II cohort, which could potentially play a part in the higher prevalence of depressive symptoms observed in the HAPIEE cohort. Lastly, all participants in the UK were employed in the civil service which is known for being a secure job environment with favourable benefits; the population recruited in Russia, Poland and the Czech Republic was employed in a variety of occupations including

entrepreneurial and self-employed job, and the majority of the population reported being retired. This may also play a role in the higher prevalence of depressive symptoms observed in the HAPIEE cohort, through the stressful aspects of more a precarious employment. A final factor that could have played a role in the generally higher prevalence of depressive symptoms observed in the HAPIEE cohort study, is the use of the CESD depression scale as opposed to the GHQ scale. However, while it is important to keep all these factors in mind when approaching this comparison, there are aspects of the association between inclusion in social networks, alcohol consumption and depressive symptoms that cannot be explained by these factors alone.

For example, not only prevalence of depressive symptoms was generally higher in the HAPIEE compared to the Whitehall cohort, but the gender difference in prevalence of depressive symptoms was also higher. In fact, if depressive symptoms were 1.1 times more prevalent among British women than among British men; they were 1.7 times more prevalent among Czech women than among Czech men; 2.2 times more prevalent among Russian women than among Russian men; and 1.6 times more prevalent among Polish women than among Polish men. In addition, while in the UK gender did not play a part in the association between inclusion in social networks, alcohol consumption and depressive symptoms, in countries of Central and Eastern Europe it did and analysis had to be stratified for gender as well as country.

Nevertheless, in all four countries prevalence of depressive symptoms was highest among men and women who reported never being in contact with friends or distant relatives. Further, in the UK and Russia prevalence of depressive symptoms was higher among men who abstained from consuming alcohol and women who consumed frequently and heavily; while in the Czech Republic and Poland, they were more prevalent among who men did not drink and women who never drank or consumed heavily.

Logistic regression models revealed that in all countries participants who did not have any or never met their friends or relatives were more likely to be suffering from depressive symptoms. Further, British and Polish participants as well as Russian women, who reported never seeing their friends or relatives were also more likely to suffer from depressive symptoms. However, the frequency of contact with friends or relatives that proved to be associated with the lowest odds of depressive symptoms varied more from country to country. In fact, if in the UK and Russia participants who visited their friends and relatives once a week were the least likely to suffer from depressive symptoms; in the Czech Republic lowest odds of depressive symptoms were observed among participants who visited their friends several times a month and their relatives several times a week; and finally Polish men and women reporting meeting their friends once a month and their relatives several times a month were the least likely to suffer from depressive symptoms.

When looking at the effects of alcohol consumption on depressive symptoms, in the Czech Republic, Russia and Poland men and women who did not consume alcohol were more likely to suffer from depressive symptoms; in addition, high odds of depressive symptoms

were also found among women in all four countries who reported drinking heavily on a regular basis.

These results show that, despite the differences between the populations of the two cohort studies, and the differences in measurement of depressive symptoms, individuals who are socially isolated are more likely to be suffering from depressive symptoms, even in settings as different as London in the late 1980s and countries of Central and Eastern Europe in the early 2000s. The association between alcohol consumption and depressive symptoms on the other hand, might have been more heavily affected by the differences between the CESD and GHQ scales. In fact, comparison of the two scales showed how when depressive symptoms were measured with the GHQ scale, their association with frequency of alcohol consumption ceased to be significant, which could explain the marked difference between patterns observed in the UK and in the Czech Republic, Russia and Poland. Alternatively this difference could be due to the need to drink as means to cope with a more stressful socio-economic environment, or to a stronger and more embedded drinking culture for which alcohol is a fundamental part of social gathering and abstaining from alcohol might be a sign of social awkwardness and isolation (Peele and Brodksy 2000; Rimal and Real 2005). In which case, more research would be needed on the association between alcohol consumption and depressive symptoms in Central and Eastern Europe.

5.5 Summary

This chapter set out to investigate the associations between measures of inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms at a given point in time among the adult urban populations of four countries as different as the UK, the Czech Republic, Russia and Poland. More specifically, the analysis here presented focused on investigating whether individuals who are more socially isolated, in the sense that they never or very seldom visit their friends or relatives living outside the household, would be more likely to be suffering from depressive symptoms than their more socially involved counterparts. The analysis was also aimed at investigating whether individuals who abstain from drinking alcohol as well as individuals who drink heavily would be more likely to suffer from depressive symptoms than their more moderate counterparts. And finally, there was a particular interest in comparing patterns of association observed in four different European countries, in the attempt to establish whether these patterns are universal or rather influenced by social and cultural norms.

Logistic regression analysis showed that, indeed, in all four adult urban populations, individuals who reported not having or never seeing their friends and relative were more likely to be suffering from depressive symptoms. Similarly, in all countries but the UK, individuals who reported never drinking were also more likely to be suffering from depressive symptoms, while heavy consumption was associated with increased odds of depressive symptoms among women but not men. The patterns of association here investigated were in fact rather similar across countries, with the exception of alcohol consumption not being associated with depressive symptoms in the UK. Further, these associations were not

affected by age, marital status, employment grade or alcohol consumption and inclusion in social networks. So that we can conclude that social isolation is associated with increased likelihood of suffering from depressive symptoms, as is heavy consumption of alcohol.

6. Social support, Inclusion in Social Networks, Alcohol Consumption and Depressive Symptoms in the Whitehall II cohort study.

6

6.1 Introduction

This chapter is devoted to investigating the magnitude and duration of the effects of social support, inclusion in social networks and alcohol consumption on depressive symptoms through longitudinal analysis of data coming from four phases of the Whitehall II cohort study. At the same time, this chapter presents a first attempt to address the issue of temporality, and an analysis of the effects of possible confounders in the associations of interest. More specifically, the analysis here presented aimed:

O5: to investigate the association between measures of social support and depressive symptoms;

H5: *individuals who receive low levels of confiding/emotional or practical support, and high levels of negative support are more likely to suffer from depressive symptoms*;

O6: To investigate the duration and magnitude of the association between inclusion in social networks, social support and depressive symptoms.

H6: *Individuals who are poorly connected to friends, relatives outside the household or other clubs and societies at Phase 1, will be more likely to be affected by depressive symptoms for many years.*

H7: *Individuals who receive low levels of confiding/emotional or practical support, and high levels of negative support at Phase 1, will be more likely to be affected by depressive symptoms for many years.*

O7: To investigate the magnitude and duration of the association between alcohol consumption and depressive symptoms.

H8: *Individuals who either consume alcohol heavily and frequently or who do not drink at all will be more likely to be affected by depressive symptoms for many years than moderate drinkers.*

O8: to test whether the associations social support, inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are confounded by the effects of age, gender, marital status, employment grade, smoking status, physical activity, and alcohol consumption or social support and inclusion in social networks respectively;

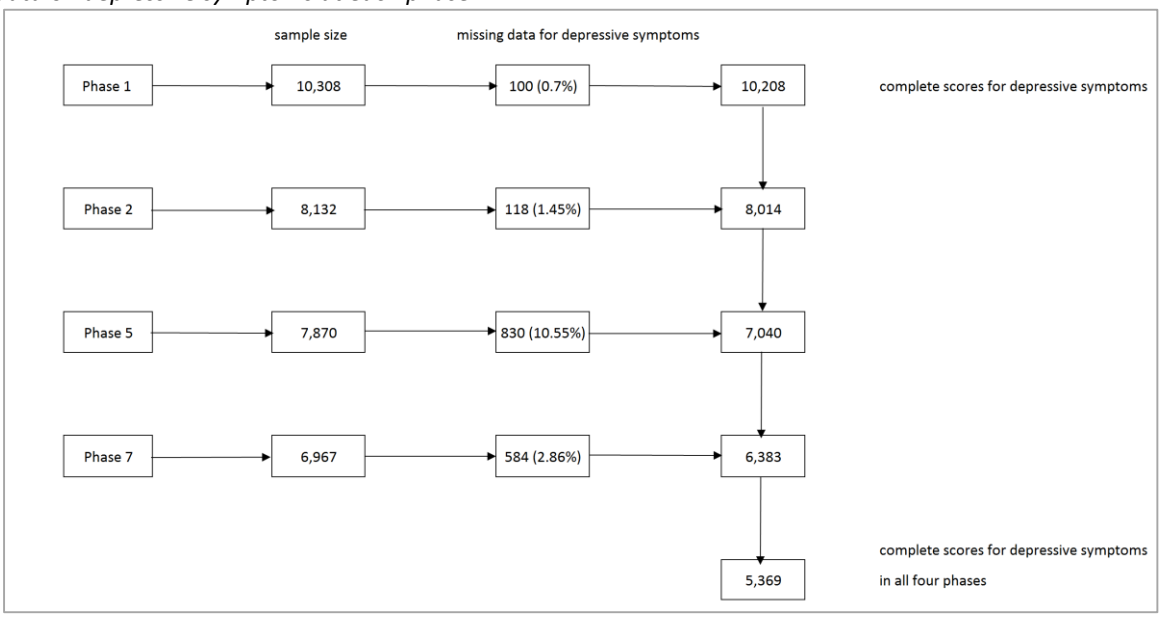
H9: *the associations social support, inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are not confounded by the effects of age, gender, marital status, employment grade, smoking status, physical activity, and alcohol consumption or social support and inclusion in social networks respectively*.

In order to address these objectives and hypotheses logistic regression models were fitted using measures of social support, inclusion in social networks and alcohol consumption to predict depressive symptoms at Phase 2, 5 and 7 of the Whitehall II cohort study, and vice versa. Details of the statistical analysis performed will be provided in section 6.2, while details of how missing data were imputed are to be found in Appendix 2. Sections 6.3 to 6.6 will present the results of this set of analysis addressing each of the research hypotheses stated above. Finally, section 6.7 will provide a summary of the results here presented.

6.2 Statistical Analysis

Statistical analysis was as follows. First, only participants with complete scores for the GHQ depression scale at Phase 1, 2, 5 and 7 of the Whitehall II cohort study were included in the analysis. This was done in order to address the possible issue of selection bias. Selection bias is the name given to the process by which in many cohorts, participants who fall into ill health during the course of the study are more likely to drop out or to fail to respond to a particular phase of measurement, thus biasing the sample towards a healthier population. By reducing the sample size to the 5,369 (Figure 6.2.1) individuals with complete scores in the GHQ depression scale in all the phases here analysed, I have attempted to reduce this bias by including only participants who did not drop out for reasons of mental health. However, the new sample size was almost half the original 10,308 participants included in Phase 1 of the Whitehall II cohort, hence the issue of sufficient statistical power of the new sample presented itself and power calculations were carried out. The estimated statistical power for a one sample t-test of the 5,369 sample size was 0.98 which is well above the conventional 0.80 cut off point taken to signify the ability of a sample to yield statistically significant results. Hence, the sample size for all analysis presented in this chapter was 5,369 individuals.

Figure 6.2.1 *Number and percentage of missing data on depressive symptoms and number of complete data on depressive symptoms at each phase.*



Second, descriptive statistics were performed on the 5,369 sample to detect any possible missing data. These statistics revealed that the percentage of missing data in the three sub-scales measuring social support and in the scale measuring contact with friends at Phase 1 was higher than 25%. While missing data in all other variables were below 5% and in some cases even below 1% (Appendix 1). These disproportionately high percentages of missing values in the measures of support and contact with friends were due to the fact that the close person questionnaire was introduced late in the first phase of data collection. Table 6.2.1 shows the number and percentages of missing values for measures of support, inclusion in social networks and alcohol consumption at Phases 1, 2, 5 and 7.

Table 6.2.1 *Number and percentage of missing data at baseline in the exposure variables*

	Phase 1		Phase 2		Phase 5		Phase 7	
	N	%	N	%	N	%	N	%
Confiding support	1,418	26.4	171	3.2	110	2.0	103	1.9
Practical support	1,415	26.4	154	2.9	100	1.9	102	1.9
Negative support	1,425	26.5	171	3.2	111	2.1	105	2.0
Friends scale	1,369	25.5	46	0.9	373	6.9	47	0.9
Relatives scale	236	4.4	94	1.7	282	5.2	172	3.2
Network scale	40	0.7	44	0.8	224	4.2	79	1.5
Frequency of alcohol consumption	7	0.1	4	0.1	70	1.3	14	0.3
Volume of alcohol consumed	40	0.7	8	0.1	53	1.0	26	0.5

Missing data were then imputed through multiple chained imputation using 'mi impute' in STATA 13. Multiple imputation has long established itself as a convenient and flexible paradigm to analyse data with missing values, and increasingly more sophisticated software for imputation have emerged in recent years. Multiple imputation is a principled simulation-based approach for analysing incomplete data which replaces missing values with multiple sets of simulated values to complete the data and applies standard analysis to each complete dataset while adjusting the obtained parameter estimates for missing-data uncertainty (Marchenko 2011). The aim of multiple imputation is not to predict missing values as close as possible to the true values but to handle missing data in a way that will results in valid statistical inference; where statistically valid refers to an imputation model that is proper and to a primary completed-data analysis that is statistically valid in the absence of missing data (Rubin 1987; Rubin 1996). Multiple imputation is based on the statistical assumptions that the data are missing at random (MAR) and would be normally distributed (Rubin 1996). Following the rationale of Rubin (1996), based on the calculation that a higher number of imputations will produce higher standard errors, 5 complete-data datasets were imputed and subsequently analysed.

Missing values were imputed on the basis of a series of equations including: age; sex; marital status at Phase 1, 2, 5 and 7; employment grade at Phase 1, 2, 5 and 7; last known employment grade at Phase 5 and 7; physical activity at Phase 1, 2, 5 and 7 ; smoking status at Phase 1, 2, 5 and 7; depression at Phase1; confiding/emotional support; practical support; negative support; network index; friends index; relatives index; frequency of alcohol consumption; and volume of alcohol consumed per week. Predictor variables were

chosen because they were correlated with the missing variable and thus would have helped imputing missing values while preserving relationships in the data. In addition, because information about support was collected only half way through Phase 1, support subscales at Phase2 were used as predictors of support at Phase1. Full details of the multiple imputation process are to be found in Appendix 1.

Once missing data had been imputed, Likelihood of Ratio Tests were performed to detect any possible association between measures of social support, inclusion in social networks, alcohol consumption and gender in their association with depressive symptoms. Results are shown in Table 6.2.2. In a second time, descriptive statistics were then performed on imputed data in order to calculate the prevalence of depressive symptoms according to general characteristics and to measures of social support, inclusion in social networks and alcohol consumption at each phase. Because the measures of social support and inclusion in social networks used in this chapter were not part of the analysis presented in Chapter 5, their relative prevalence of depressive symptoms at baseline was presented separately. In addition, for the same reason, logistic regression was carried out cross-sectionally to investigate the original patterns of association between social support, inclusion in social networks and depressive symptoms.

Thirdly, logistic regression models were then performed on imputed data and odds of depressive symptoms at each wave and relative confidence intervals were calculated by measures of support, network index, index of contact with friends and relatives, frequency of alcohol consumption and number of UK units consumed per week, at baseline. For each of the variables above, three regression models were run: (1) a preliminary model adjusted

for sex and age at each phase; (2) a partially adjusted model, accounting for the effects of age, sex, employment grade, last known employment grade for participants who were retired at Phases 5 and 7, marital status, smoking status and physical activity at each wave; (3) a fully adjusted model, accounting for the effects of all variables above plus measures of alcohol consumption or social support and inclusion in social networks. Measures of alcohol consumption were adjusted for when modelling the association between social support or inclusion in social networks and depressive symptoms, while social support and inclusion in social networks were adjusted for when modelling the association between alcohol consumption and depressive symptoms.

Further, each variable measuring social support and inclusion in social networks at Phase 2, 5 and 7 was then recoded as a dichotomous variable scored "high/medium" and "low"; similarly measures of alcohol consumption at Phase 2, 5 and 7 were dichotomised into "hazardous drinking" and "moderate drinking". Hazardous drinkers were participants who had consumed alcohol at least daily in the previous year, and who reported consuming more than 21 standard UK units in the previous week. Moderate drinkers were participants who had consumed alcohol less frequently than daily, and reported drinking 21 or less standard UK units in the previous week. The three logistic regression models were then run again, using depressive symptoms at Phase 1 to predict social support, inclusion in social networks and alcohol consumption in later phases.

Table 6.2.2 *Likelihood of ratio chi square and relative p value for the association between gender and measures of social support, inclusion in networks and alcohol consumption*

	Phase 2		Phase 5		Phase 7	
	LRT	**P**	**LRT**	**P**	**LRT**	**P**
Confiding/emotional support	1.84	0.399	0.05	0.975	2.09	0.159
Practical support	6.46	0.039	5.53	0.063	4.91	0.027
Negative support	5.04	0.080	2.53	0.282	1.55	0.460
Network index	3.38	0.185	0.19	0.909	1.47	0.480
Contact with friends	2.30	0.317	0.03	0.986	1.08	0.582
Contact with relatives	5.50	0.064	1.75	0.416	0.39	0.825
Frequency of alcohol consumption	0.86	0.836	0.04	0.998	3.96	0.555
Dose of alcohol consumed	31.8	0.199	37.38	0.069	6.57	0.255

6.3 Descriptive analysis

Preliminary descriptive statistics revealed how when participants were called back for Phase 2 measurements, 30% of them were aged between 40 and 44 and they remained the most numerous age group through to Phase 7 measurements. Interestingly, at each phase prevalence of depressive symptoms was lowest among the oldest age group (Table 6.3.1). Over three quarters of participants were married at Phase 2, and remained married through to Phase 7 and enjoyed relatively low rates of depressive symptoms, ranging from 12.3% at Phase 2, to 10.3% at Phase 7. Between 80% and 90% of participants were employed in the administrative and professional/executive grades of the civil service, or retired from them as time went by, and the higher the grade of employment, the lower the prevalence of depressive symptoms. Finally, the vast majority of participants were non-smokers or engaged in regular moderate physical activity and enjoyed lower prevalence of depressive symptoms than smokers or more sedentary people (Table 6.3.1).

Further statistics showed how at time baseline measurements of the Whitehall II study were carried out (1985-1988), just over 30% of participants were perceiving themselves as being highly supported by the people closest to them, both emotionally and practically, and 37.4% of participants were little affected by the negative aspects of their social relations (Table 6.3.2). Further, 36.6% of participants reported being highly involved in club and societies, 40.4% to have a very active social life, and 44.7% to be often in contact with their family and distant relatives. Among these highly socially connected and well supported participants prevalence of depressive symptoms was really low, ranging from 7.5% among

participants who enjoyed highly positive relationships to 10.0% among participants who were often in touch with their relatives (Table 6.3.2).

The same patterns were observed in later phases as at each measurement point, participants who were well emotionally and practically supported kept having low rates of depressive symptoms, as did those who suffered only from small levels of negativity in their relationships (Table 6.3.3). Similarly, participants who kept being highly involved in clubs and societies, maintained an active social life and were frequently in contact with their relatives through time, enjoyed low prevalence of depressive symptoms phase after phase (Table 6.3.3). Moreover, at each phase between 39.1% (Phase 2) and 46.6% (Phase 7) of participants reported drinking at least once a day but the lowest prevalence of depressive symptoms was found among those who drunk several times a week; while between 28.3 % (Phase 5) and 37.8 % (Phase 2) of participants reported drinking between 1 and 7 units in the previous seven days, but all phases the lowest prevalence of depressive symptoms was found among those who consumed between 22 and 28 units per week (Table 6.3.3).

Table 6.3.1. *Prevalence of depressive symptoms according to covariates at phase 2, 5 and 7*

	Phase 2		Phase 5		Phase 7	
	%	D%	%	D%	%	D%
Men	71.6	12.8	71.6	11.6	71.6	10.7
Women	28.4	17.7	28.4	13.6	28.4	12.4
Age groups						
1[1]	10.1	13.8	20.6	15.7	17.9	14.0
2	30.0	14.3	28.6	13.3	29.4	11.8
3	23.2	14.1	21.2	11.6	21.2	9.9
4	20.7	14.8	22.0	9.6	21.5	10.1
5	16.0	13.5	7.6	6.8	10.1	9.6
Marital Status						
Married/cohabiting	77.2	12.3	79.6	10.8	76.2	10.3
Single	14.6	19.8	11.4	18.1	12.6	13.9
Divorced	6.9	21.9	6.0	15.7	6.8	11.8
Widowed	1.3	19.7	2.9	15.8	4.4	17.3
Employment grade						
Administrative	39.5	12.1	43.2	11.4	45.6	10.6
Prof/executive	48.1	15.2	45.3	15.1	44.5	14.4
Clerical/support	12.5	16.9	11.6	18.1	9.8	16.6
Last employment grade						
Administrative			46.3	8.7	47.2	8.7
Prof/executive			42.8	11.4	43.0	11.6
Clerical/support			10.9	15.8	9.8	12.9
Smoking status						
Smoker	11.9	16.9	8.7	13.3	6.3	15.0
Non-smoker	88.1	13.8	91.3	12.0	93.7	10.8
Physical activity						
None	0.8	28.6	0.4	15.0	0.9	28.6
Mild	9.0	21.5	8.3	16.9	3.0	16.5
Moderate	90.1	13.3	89.1	11.6	94.2	10.8
Vigorous	0.1	0.0	8.2	16.1	1.8	12.4

[1] Age groups in each phase:

	Phase 2	Phase 5	Phase 7
1	37-39	44-49	50-54
2	40-44	50-54	55-59
3	45-49	55-59	60-64
4	50-54	60-64	65-69
5	55-59	65-69	70-74

Table 6.3.2 *Prevalence of depressive symptoms according to measures of social support and inclusion in social networks at Phase 1.*

	% of participants	% of depressive symptoms
Confiding/emotional		
High	31.0	8.6
medium	39.5	11.9
low	29.5	16.4
Practical		
High	32.0	10.1
medium	35.3	11.9
low	32.6	14.6
Negative		
Low	37.4	7.5
medium	32.6	12.1
High	30.0	18.2
Network		
High	36.6	8.5
medium	36.7	12.4
low	26.7	17.2
Friends		
High	40.4	8.3
medium	35.1	13.3
low	24.6	17.1
Relatives		
High	44.7	10.0
medium	21.3	13.5
low	34.0	14.5

Table 6.3.3 *Prevalence of depressive symptoms according to indicators of social support, inclusion in social networks and alcohol consumption, in men and women.*

	Phase 2		Phase 5		Phase 7	
	%	D%	%	D%	%	D%
Confiding/emotional						
High	23.4	11.1	11.9	7.7	41.3	8.3
medium	38.7	12.9	37.4	9.8	26.5	10.3
low	37.8	17.3	50.7	14.4	32.2	15.0
Practical						
High	25.5	11.7	12.1	10.6	48.8	10.2
medium	36.5	13.5	38.7	10.5	27.7	11.4
low	37.9	16.3	49.2	13.3	23.5	12.1
Negative						
Low	35.0	9.2	40.1	6.4	16.6	5.4
medium	33.2	12.2	35.4	11.7	48.1	7.7
High	31.7	21.6	24.5	21.3	34.9	18.1
Network						
High	35.7	11.5	36.3	7.7	39.8	7.5
medium	35.2	13.5	35.8	12.4	34.8	12.3
low	29.1	18.2	27.8	16.8	25.4	15.6
Friends						
High	38.0	11.5	46.0	8.6	50.4	8.0
medium	35.7	14.2	31.8	13.0	30.2	13.4
low	26.3	17.9	22.2	17.4	19.4	16.3
Relatives						
High	39.1	13.2	46.6	10.4	47.9	9.4
medium	18.6	14.4	27.0	12.3	24.4	12.3
low	42.3	15.3	26.4	14.0	27.6	13.1
Frequency						
>1/day	31.9	14.3	45.3	10.9	46.6	10.8
>1/week	40.1	13.7	32.5	11.4	30.7	10.4
>1/month	12.7	14.9	8.7	11.7	8.6	10.7
Special occasions/never	15.3	14.3	13.5	16.8	14.1	14.3
Units consumed in past week						
None	16.8	15.0	15.0	16.5	16.4	14.8
1-7	37.8	14.1	28.3	10.2	30.3	10.2
8-14	21.0	14.6	22.3	10.3	22.7	10.3
15-21	11.0	11.5	12.4	12.5	13.8	11.4
22-28	5.8	12.5	8.5	9.1	7.2	9.4
29-35	3.1	17.6	5.4	15.7	4.0	9.4
36+	4.4	15.5	8.1	13.5	5.7	11.8

6.4 Social Support and Inclusion in Social Networks, Phase 1

Logistic regression analysis of data from Phase 1 of the Whitehall II cohort study revealed a strong association between measures of social support and inclusion in social networks and depressive symptoms. In fact, compared to those who received high levels of confiding/emotional or practical support, and after accounting for the effects of age, sex, marital status, employment grade, smoking status, physical activity and alcohol consumption, participants who reported receiving low levels of confiding emotional support were 1.89 (95% C.I. 1.72-2.08) times more likely to suffer from depressive symptoms, and participants who received low levels of practical support were 1.1 (95% C.I. 1.08-1.31) times more likely to suffer from depressive symptoms. But the most striking result concerned those participants who suffered from the negative aspects of social associations, who were 2.88 (95% C.I. 2.63-3.15) times more likely to suffer from depressive symptoms as well, than their counterparts who enjoyed better relationships (Tale 6.4.1).

When looking at the effects of inclusion in social networks on depressive symptoms, participants who were little involved in clubs or societies resulted to be 2.16 (95% C.I. 1.97-2.36) times more likely to suffer from depressive symptoms; in the same fashion, participants who reported being little in contact with their friends (OR 2.27; 95% C.I. 2.07-2.49) or relatives (OR 1.46; 95% C.I. 1.35-1.58) were also more likely to suffer from depressive symptoms, even after accounting for the effects of age, sex, marital status, employment grade, smoking status, physical activity and alcohol consumption (Table 6.4.2).

Table 6.4.1 *Odds Ratios and 95% confidence intervals for depressive symptoms by measures of social support at Phase 1.*

	Model 1[1]	Model 2[2]	Model 3[3]
	OR (95% C.I.)	**OR (95% C.I.)**	**OR (95% C.I.)**
Confiding/emotional			
High	1	1	1
medium	1.45 (1.32-1.59)	1.41 (1.28-1.55)	1.41 (1.29-1.55)
low	2.11 (1.93-2.31)	1.89 (1.72-2.08)	1.89 (1.72-2.08)
Practical			
High	1	1	1
medium	1.20 (1.10-1.31)	1.16 (1.06-1.27)	1.16 (1.06-1.27)
low	1.50 (1.37-1.64)	1.19 (1.08-1.31)	1.19 (1.08-1.31)
Negative			
Low	1	1	1
medium	1.72 (1.57-1.88)	1.73 (1.58-1.91)	1.73 (1.58-1.90)
High	2.78 (2.55-3.03)	2.88 (2.64-3.15)	2.88 (2.63-3.15)

[1] Adjusted for age and sex
[2] Adjusted for age, sex, marital status, employment grade, smoking status and physical activity
[3] Adjusted for age, sex, marital status, employment grade, smoking status, physical activity and alcohol consumption

Table 6.4.2 *Odds Ratios and 95% confidence intervals for depressive symptoms by measures of inclusion in social networks at Phase 1*

	Model 1[1]	Model 2[2]	Model 3[3]
	OR (95% C.I.)	**OR (95% C.I.)**	**OR (95% C.I.)**
Network			
High	1	1	1
Medium	1.52 (1.39-1.65)	1.47 (1.34-1.60)	1.47 (1.35-1.61)
Low	2.22 (2.04-2.42)	2.13 (1.95-2.33)	2.16 (1.97-2.36)
Friends			
High	1	1	1
Medium	1.70 (1.56-1.85)	1.66 (1.52-1.81)	1.67 (1.53-1.83)
Low	2.28 (2.09-2.49)	2.22 (2.03-2.43)	2.27 (2.07-2.49)
Relatives			
High	1	1	1
Medium	1.41 (1.29-1.54)	1.41 (1.29-1.54)	1.41 (1.28-1.54)
Low	1.54 (1.43-1.66)	1.46 (1.35-1.58)	1.46 (1.35-1.58)

[1] Adjusted for age and sex
[2] Adjusted for age, sex, marital status, employment grade, smoking status and physical activity
[3] Adjusted for age, sex, marital status, employment grade, smoking status, physical activity and alcohol consumption

6.5 Duration of the effects of social support, inclusion in social networks and alcohol consumption on depressive symptoms

This chapter was designed to establish whether inadequate social support, negative aspects of social relations, social isolation and excessive alcohol consumption would have a deleterious effect on an individual's mental health only at the particular point in time in which they were experienced, or whether their impact would linger in time and if so, for how long. In order to do so, measures of social support, inclusion in social networks and alcohol consumption at Phase 1 were used to predict odds of depressive symptoms at Phase 2, 5 and 7. Results are presented below.

6.5.1 Social Support

Earlier in this chapter, cross-sectional analysis showed how participants who received inadequate confiding/emotional support between 1985 and 1988 were 1.89 times more likely to suffer from depressive symptoms at the same time than their fully supported counterparts. But the deleterious effects of insufficient confiding/emotional support were not confined to the three years of baseline measurement, as they predicted a 1.36 (95% C.I. 1.25-1.48) increase in the odds of depressive symptoms at Phase 2, and a 1.26 (95% C.I. 1.10-1.43) increase at Phase 5 (Table 6.5.1). These results suggest that experiencing a lack of emotional or confiding support at one point in life could still increase individual chances of suffering from depressive symptoms ten years after the experience, albeit with less intensity as times goes by.

Participants who received little practical help from the people closest to them in everyday issues when baseline measurements took place were 1.19 times more likely to suffer from depressive symptoms at the time; 1.06 (95% C.I. 0.93-1.21) times more likely at Phase 2, although the association was not statistically significant; and 1.20 (95% C.I. 1.06-1.37) times more likely at Phase 5 (Table 6.5.1). Thus suggesting that practical support might play a lesser role than confiding/emotional support in affecting depressive symptoms, and one that lasts for shorter spells.

However, the strongest and longest lasting repercussions on mental health were inflected by the negative facets of close relationships. In fact, if at baseline the chances of suffering from depressive symptoms were 2.88 times higher among participants who strongly felt the negative aspects of their relations than among those with a happier social life, at Phase 2 participants who had been unhappy in their close relationship at baseline were still 2.28 (95% C.I. 1.94-2.69) more likely to suffer from depressive symptoms, at Phase 5 they were 1.98 (95% C.I. 1.66-2.38) times more likely and Phase 7 they were 2.29 (95% C.I. 1.96-2.68) times more likely to suffer from depressive symptoms (Table 6.5.1). This suggests that negative support has by far the biggest impact on mental health, and one that lasts through time.

However, as there is evidence to support the notion that individuals suffering from depressive symptoms might perceive the support they receive as inadequate, or perceive disproportionately strong negative aspects of social relations, the analysis was run a second time using depressive symptoms at baseline to predict low confiding/emotional or practical

and high negative support at Phases 2, 5 and 7. This showed that indeed, participants who suffered from depressive symptoms at baseline were 1.58 (95% C.I. 1.48-1.70) times more likely to perceive inadequate levels of confiding/emotional support at Phase 2, and 1.60 (95% C.I. 1.43-1.79) at Phase 5, although the effects had faded out by Phase 7 (Table 6.5.2). Perceived practical support at later phases, on the other hand, did not seem to be affected by depressive symptoms at baseline as the association between the two was not statistically significant. As expected, though, presence of depressive symptoms at baseline was a strong predictor of high levels of perceived negative aspects of social relations at Phase 2 (OR 1.99; 95% C.I. 1.85-2.13), Phase 5 (OR 1.96; 95% C.I. 1.75-2.19) and Phase 7(OR 2.01; 95% C.I. 1.77-2.29), as participants who suffered from depressive symptoms at baseline were around twice as likely to perceive high levels of negative support for the following nineteen years (Table 6.5.2).

Table 6.5.1 *Odds Ratios and 95% confidence intervals for depressive symptoms at Phase 2, 5 & 7 by measures of social support at Phase 1*

	Phase 2[1]	Phase 5[2]	Phase 7[3]
	OR (95% C.I.)	**OR (95% C.I.)**	**OR (95% C.I.)**
Confiding/emotional			
High	1	1	1
medium	1.07 (0.98-1.16)	0.95 (0.84-1.08)	1.15 (0.98-1.36)
low	1.36 (1.25-1.48)	1.26 (1.10-1.43)	1.09 (0.91-1.30)
Practical			
High	1	1	1
medium	1.07 (0.94-1.21)	0.87 (0.77-0.98)	1.02 (0.86-1.20)
low	1.06 (0.93-1.21)	1.20 (1.06-1.37)	0.90 (0.75-1.07)
Negative			
Low	1	1	1
medium	1.70 (1.43-2.01)	2.04 (1.70-2.44)	1.69 (1.44-1.98)
High	2.28 (1.94-2.69)	1.98 (1.66-2.38)	2.29 (1.96-2.68)

[1] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

[2] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

[3] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

Table 6.5.2 *Fully adjusted odds of receiving social support at Phase 2, 5, 7 according to depressive symptoms at Phase 1.*

	Confiding/emot.	Practical	Negative
	OR (95% C.I.)[1]	OR (95% C.I.)[2]	OR (95% C.I.)[3]
Phase 2			
Depressive symptoms			
No	1	1	1
Yes	1.58 (1.48-1.70)	1.07 (0.99-1.15)	1.99 (1.85-2.13)
Phase 5			
Depressive symptoms			
No	1	1	1
Yes	1.60 (1.43-1.79)	1.12 (1.00-1.25)	1.96 (1.75-2.19)
Phase 7			
Depressive symptoms			
No	1	1	1
Yes	1.14 (0.99-1.31)	0.92 (0.78-1.09)	2.01 (1.77-2.29)

[1] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

[2] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

[3] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

6.5.2 Inclusion in Social Networks

Section 6.4 showed how participants who were little involved in clubs and societies at Phase 1 were more than twice more likely to be suffering from depressive symptoms than their highly involved counterparts. Longitudinal analysis showed that the deleterious effects of a scarce social life in the community lasted a long time, as those participants who were little involved in clubs and societies at Phase 1 were 1.40 (95% C.I. 1.29-1.52) times more likely to suffer from depressive symptoms at Phase 2, 1.55 (95% C.I. 1.37-1.76) times more likely at Phase 5 and 1.96 (95% C.I. 1.68-2.229) times more likely at Phase 7. These results suggest that participation in clubs and societies, whether through provision of friendship and support or of sense of identity and belonging, could have a long lasting beneficial effect on individual mental health (Table 6.5.3).

In the same fashion, participants who at baseline reported having few and sparse encounters with their friends were not only more than twice more likely to be suffering from depressive symptoms at the same time, but also 1.65 (95% 1.52-1.79) times more likely at Phase 2, 1.49 (1.32-1.69) more likely at Phase 5 and 1.46 (95% C.I. 1.26-1.70) times more likely at Phase 7. Thus, although the beneficial effects of being part of an active group of friends on mental health somewhat diminish as time goes by, it can be said the mere frequency and number of contacts with friends play a long lasting role in affecting mental health (Table 6.5.3).

Having regular contact with the extended family also appeared to have a long lasting effect on depressive symptoms, as participants who reported being little connected to their relatives at baseline had higher odds of depressive symptoms both at Phase 5 (OR 1.20; 95% C.I. 1.07-1.34) and Phase 7 (OR 1.44; 95% C.I. 1.26-1.65). This suggests that perhaps early frequent engagement with relatives living outside the household might be the foundation for a strong family involvement in preventing loneliness and depressive symptoms later in life (Table 6.5.3)

However, just as in the case of social support, these results could not be taken definitively before investigating the effects of depressive symptoms at baseline on levels of social inclusion later in life, for there is evidence suggesting that individuals suffering from depressive symptoms tend to be more socially isolated as a result of their condition. In fact, participants who did suffer from depressive symptoms at baseline had higher odds of being little involved in clubs and societies at Phase 2 (OR 1.54; 95% C.I. 1.43-1.65), Phase 5 (OR 1.83; 95% C.I. 1.64-2.04) and Phase 7 (OR 1.63; 95% C.I. 1.42-1.85); they also had higher odds of being sparsely in contact with their friends at Phase 2 (OR 1.49; 95% C.I. 1.38-1.68), Phase 5 (OR 1.61; 95% C.I. 1.43-1.81) and Phase 7 (OR 1.53; 95% C.I. 1.39-1.69); and higher odds of being little in contact with their extended families at Phase 2 (OR 1.23; 95% C.I. 1.14-1.31), Phase 5 (OR 1.36; 95% C.I. 1.21-1.53) and at Phase 7 (OR 1.41; 95% C.I. 1.29-1.54) (Table 6.5.4).

Table 6.5.3 *Odds Ratios and 95% confidence intervals for depressive symptoms at Phase 2, 5 & 7 by measures of inclusion in social networks at Phase 1.*

	Phase 2	Phase 5	Phase 7
	OR (95% C.I.)[1]	**OR (95% C.I.)[2]**	**OR (95% C.I.)[3]**
Network			
High	1	1	1
Medium	1.06 (0.99-1.15)	1.21 (1.07-1.36)	1.45 (1.24-1.67)
Low	1.40 (1.29-1.52)	1.55 (1.37-1.76)	1.96 (1.68-2.29)
Friends			
High	1	1	1
Medium	1.30 (1.21-1.41)	1.13 (1.00-1.27)	1.02 (0.88-1.18)
Low	1.65 (1.52-1.79)	1.49 (1.32-1.69)	1.46 (1.26-1.70)
Relatives			
High	1	1	1
Medium	1.08 (0.99-1.17)	1.47 (1.29-1.66)	0.93 (0.78-1.11)
Low	1.07 (0.99-1.15)	1.20 (1.07-1.34)	1.44 (1.26-1.65)

[1] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

[2] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

[3] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

Table 6.5.4 *Fully adjusted odds of being included in social networks at Phase 2, 5, 7 according to depressive symptoms at Phase 1*

	Networks	Friends	Relatives
	OR (95% C.I.)[1]	**OR (95% C.I.)[2]**	**OR (95% C.I.)[3]**
Phase 2			
Depressive symptoms			
No	1	1	1
Yes	1.54 (1.43-1.65)	1.49 (1.38-1.60)	1.23 (1.14-1.31)
Phase 5			
Depressive symptoms			
No	1	1	1
Yes	1.83 (1.64-2.04)	1.61 (1.43-1.81)	1.36 (1.21-1.53)
Phase 7			
Depressive symptoms			
No	1	1	1
Yes	1.63 (1.42-1.85)	1.53 (1.39-1.69)1	1.41 (1.29-1.54)

[1] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

[2] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

[3] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and alcohol consumption

6.5.3 Alcohol consumption

Cross-sectional analysis in Chapter 5 showed how men and women who consumed alcohol heavily at baseline were also more likely to be suffering from depressive symptoms (Table 5.3.7). The scope of this analysis was to investigate how long would the effects of alcohol consumption on mental health last. Longitudinal analysis showed that participants who drank alcohol daily or even twice a day, compared to those who consumed several times a week, had higher odds of depressive symptoms at Phase 2 (OR 1.28; 95% C.I. 1.19-1.38), Phase 5 (OR 1.30; 95% C.I. 1.14-1.48) and Phase 7 (OR 1.32; 95% C.I. 1.12-155). This is all the more interesting as the association was not significant at baseline and the effects of frequent alcohol consumption on depressive symptoms seem to have increased rather than diminish as time went by (Table 6.5.5). Further, participants who reported drinking only in special occasions or not at all at baseline, also had higher odds of depressive symptoms at Phase 5 (OR 1.47; 95% C.I. 1.26-1.73) and Phase 7 (OR 1.47; 95% C.I. 1.20-1.79), but not at Phase 2 (Table 6.5.5).

Similarly, participants who consumed 36 or more standard UK units per week at baseline were more likely to suffer from depressive symptoms at Phase 2 (OR 1.38; 95% C.I. 1.18-1.60) and Phase 5 (OR 1.73; 95% C.I. 1.37-2.19) compared to those who consumed between 1 and 7 UK units a week. High odds of depressive symptoms were also found among participants who used to consume between 22 and 28 UK units at baseline, at Phase 2 (OR 1.37; 95% C.I. 1.19-1.57) and at Phase 5 (OR 1.33; 95% C.I. 1.07-1.66). Finally, participants who did not consume any alcohol at baseline were 1.48 (95% C.I. 1.27-1.73) times more

likely to suffer from depressive symptoms at Phase 5, and 1.30 (95% C.I. 1.08-1.56) times more likely at Phase 7 (Table 6.5.5).

The effects of depressive symptoms at Phase 1 on alcohol consumption in the following nineteen years were then investigated, as there is evidence to suggest that poor mental health triggers heavy alcohol consumption as a form of self-medication. This analysis revealed that the effects of depressive symptoms on alcohol consumption were very short lived as already in Phase 2, after adjusting for age, sex, marital status, employment grade, smoking status, levels of physical activity, and levels of social support and inclusion in social networks, the association between depressive symptoms and alcohol consumption was not statistically significant (Table 6.5.6).

Table 6.5.5 *Odds Ratios and 95% confidence intervals for depressive symptoms at Phases 2, 5 and 7 by measures of alcohol consumption at Phase 1.*

	Phase 2	Phase 5	Phase 7
	OR (95% C.I.)[1]	OR (95% C.I.)[2]	OR (95% C.I.)[3]
Frequency of consumption			
>1/day	1.28 (1.19-1.38)	1.30 (1.14-1.48)	1.32 (1.12-1.55)
>1/week	1	1	1
>1/month	1.08 (0.97-1.19)	1.09 (0.92-1.30)	1.22 (0.99-1.50)
Special occasions/never	0.91 (0.82-1.01)	1.47 (1.26-1.73)	1.47 (1.20-1.79)
Units consumed in past week			
None	0.92 (0.83-1.02)	1.48 (1.27-1.73)	1.30 (1.08-1.56)
1-7	1	1	1
8-14	1.00 (0.91-1.09)	0.82 (0.70-0.95)	0.85 (0.72-1.02)
15-21	1.11 (0.99-1.24)	0.98 (0.82-1.19)	1.12 (0.90-1.39)
22-28	1.37 (1.19-1.57)	1.33 (1.07-1.66)	0.90 (0.68-1.20)
29-35	1.02 (0.83-1.24)	0.74 (0.53-1.04)	1.25 (0.87-1.78)
36+	1.38 (1.18-1.60)	1.73 (1.37-2.19)	0.64 (0.46-0.91)

[1] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and measures of social support and inclusion in social networks

[2] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and measures of social support and inclusion in social networks

[3] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and measures of social support and inclusion in social networks

Table 6.5.6 *Fully adjusted odds of heavily consuming alcohol at Phase 2, 5, 7 according to depressive symptoms at Phase 1.*

	Model 1	Model 2	Model 3
	OR (95% C.I.)[1]	OR (95% C.I.)[2]	OR (95% C.I.)[3]
Phase 2			
Depressive symptoms			
No	1	1	1
Yes	1.04 (0.97-1.11)	1.05 (0.97-1.13)	1.07 (0.99-1.15)
Phase 5			
Depressive symptoms			
No	1	1	1
Yes	1.07 (1.00-1.15)	0.97 (0.86-1.10)	1.05 (0.93-1.18)
Phase 7			
Depressive symptoms			
No	1	1	1
Yes	1.03 (0.97-1.11)	0.98 (0.86-1.11)	0.99 (0.86-1.14)

[1] Adjusted for age and sex

[2] Adjusted for sex, age, marital status, employment grade, smoking status and physical activity

[3] Adjusted for sex, age, marital status, employment grade, smoking status, physical activity and measures of social support and inclusion in social networks

6.6 Confounders

The third and final aim of this analysis was to test that the associations between social support, inclusion in social networks, alcohol consumption and depressive symptoms were not confounded by the effects of other factors that have been shown to affect both risk of depressive symptoms and levels of social inclusion, support received and alcohol consumed. Hence, regression models of increasing complexity were run, adjusting first only for age and sex; secondly for age, sex, marital status, employment grade – both current and at the time of retirement – smoking status, and levels of physical activity; and finally for all the above and for alcohol consumption or measures of social support and inclusion in social networks, depending on the exposure under investigation. The rationale behind this being that, although it is not statistically possible to fully remove the effects of confounders, it is possible to observe whether the introduction of a particular factor in the model would significantly alter the odds ratios in the association of interest. Thus, if Section 6.5 presented fully adjusted models, this section will present results from the simpler models to allow an understanding of how the associations between social support, inclusion in social networks, alcohol consumption and depressive symptoms have been affected by possible confounders.

In Section 6.5 we saw that the fully adjusted odds for depressive symptoms at Phases 2, 5 and 7 among participants who received inadequate confiding/emotional support at baseline were 1.36, 1.26 and 1.09 respectively. Table 6.6.1 shows how at Phase 2 these odds were 1.53 when adjusting only for age sex, and then were decreased to 1.36 by the effects of marital status, employment grade, smoking status and physical activity; at Phase

5, the simplest model yielded odds ratios on 1.47, which were reduced to 1.31 when introducing further confounders, and to 1.26 in the final model; while at Phase 7 the final model's odds ratio of 1.09 was somewhere in between the initial 1.14 and the 1.05 yielded by the introduction of marital status, employment grade, smoking status and physical activity (Table 6.6.1). In the case of practical support on the other hand, at both Phase 2 and Phase 5, the odds of depressive symptoms yielded by the simplest model were the higher and were subsequently gradually decreased by the effects of both covariates and alcohol consumption; while at Phase 7 the final odds ratio of 0.90 was somewhere in between the initial 1.03 and the 0.87 yielded by the second model. Which was quite the opposite of the pattern observed for negative support, where at both Phase 2 and 5, the odds ratio obtained from the final model were somewhere in between the simplest and the second model, while at Phase 7 it was lower than in both simpler models (Table 6.6.1).

Moreover, Table 6.6.2 shows the effects of possible confounders on the association between measures of inclusion in social networks and depressive symptoms. Participants who were little involved in clubs and societies at Phase 1 had 1.44 higher odds of suffering from depressive symptoms at Phase 2 when adjusting only for age and sex, but these odds were reduced to 1.40 when accounting for marital status, employment grade, smoking status and physical activity, but not affected by alcohol consumption; the odds of suffering from depressive symptoms among the same participants were 1.85 in the simplest model, reduced to 1.56 in Model 2, and further – although minimally – reduced by alcohol consumption to 1.55 in Model 3; the same pattern was observed at Phase 7 where the original odds of 2.01, were progressively reduced by the introduction of other confounders

to 1.96 (Table 6.6.2). Further, when looking at the odds of depressive symptoms among participants who had scarce and few encounters with their friends at Phase 1, if the variation in odds ratios between the three models was negligible at Phase 2 and Phase 7, at Phase 5 the original odds ratio of 1.85 was reduced to 1.56 by the effects of marital status, employment grade, smoking status and physical activity, and even further reduced to 1.49 by alcohol consumption. The original odds of depressive symptoms among participants who had scarce contact with their extended families at Phase 1 were 1.14 at Phase 2 when only adjusting for age and sex, and were reduced by 0.07 by the effects of marital status, employment grade, smoking status and physical activity and not affected by alcohol consumption; at Phase 5 the odds of depressive symptoms were 1.18 in Model 1, unaltered in Model 2, and increased by 0.02 by the effects of alcohol consumption; at Phase 7 the odds of depressive symptoms were 1.22 in Model 1, increased by 0.24 by the effects of marital status, employment grade, smoking status and physical activity, and then decreased to 1.44 by alcohol consumption (Table 6.6.2).

Finally, participants who at baseline consumed alcohol at least once a day, at Phase 2 had 1.25 higher odds of depressive symptoms than those who consumed several times a week after adjusting for age and sex, these odds were not altered by the effects of marital status, employment grade, smoking status and physical activity, and were only marginally increased – to 1.28 – by the effects of measures of social support and inclusion in social networks; at Phase 5, they had 1.18 higher odds which were increased by 0.01 by the effects of demographic covariates, and drastically increased to 1.30 by the effects of social support and inclusion in social networks; at Phase 7 they had 1.21 higher odds which were

decreased to 0.90 lower odds by demographic covariates in Model 2, and dramatically increased to 1.32 by social support and inclusion in social networks (Table 6.6.3). At the same time, when adjusting only for age and sex, participants who at baseline reported never drinking or indulging only in special occasions, at Phase 5 had 1.55 higher odds of depressive symptoms, which turned into 1.60 higher odds of depressive symptoms when accounting for marital status, employment grade, smoking status and physical activity and into 1.47 when adjusting for measures of social support and inclusion in social networks; at Phase 7 these same participants had 1.32 higher odds which increased to 1.42 by demographic covariates and further to 1.47 by the effects of social support and inclusion in social networks (Table 6.6.3). Social support and inclusion in social networks also had a great impact on the odds of depressive symptoms according to amount of alcohol consumed per week, for adjusting for their effects altered dramatically the odds observed when adjusting for age and sex or for age, sex marital status, employment grade, smoking status and physical activity (Table 6.6.3). These results lead to think that measures of support and inclusion in social networks might somewhat mediate the association between alcohol consumption and depressive symptoms in time.

Table 6.6.1 *Logistic regression models for odds of depressive symptoms at Phase 2, 5 and 7 according to measures of social support at Phase 1*

	Phase 2		Phase 5		Phase 7	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
	OR (95% C.I.)[1]	OR (95% C.I.)[2]	OR (95% C.I.)[3]	OR (95% C.I.)[4]	OR (95% C.I.)[5]	OR (95% C.I.)[6]
Confiding/emotional						
High	1	1	1		1	1
medium	1.12 (1.04-1.22)	1.07 (0.99-1.16)	1.11 (1.02-1.22)	0.97 (0.86-1.10)	1.03 (0.90-1.17)	1.12 (0.96-1.32)
low	1.53 (1.42-1.67)	1.36 (1.25-1.47)	1.47 (1.35-1.60)	1.31 (1.15-1.49)	1.14 (0.99-1.30)	1.05 (0.88-1.25)
Practical						
High	1	1	1		1	1
medium	1.25 (1.15-1.36)	1.19 (1.10-1.29)	0.91 (0.83-0.99)	0.90 (0.79-1.02)	1.04 (0.95-1.14)	0.99 (0.84-1.16)
low	1.41 (1.30-1.53)	1.16 (1.06-1.27)	1.30 (1.20-1.42)	1.25 (1.11-1.42)	1.03 (0.94-1.12)	0.87 (0.73-1.03)
Negative						
Low	1	1	1		1	1
medium	1.72 (1.46-2.04)	1.70 (1.44-2.01)	1.88 (1.60-2.21)	2.01 (1.69-2.40)	1.56 (1.42-1.71)	1.69 (1.45-1.98)
High	2.35 (2.00-2.77)	2.27 (1.93-2.67)	1.88 (1.59-2.21)	2.00 (1.67-2.39)	2.34 (2.14-2.55)	2.34 (2.01-2.74)

[1] Adjusted for age and sex
[2] Adjusted for sex, age, marital status, employment grade, smoking status and physical activity
[3] Adjusted for age and sex
[4] Adjusted for sex, age, marital status, employment grade, smoking status and physical activity
[5] Adjusted for age and sex
[6] Adjusted for sex, age, marital status, employment grade, smoking status and physical activity

Table 6.6.2 *Logistic regression models for odds of depressive symptoms at Phase 2, 5 and 7 according to measures of inclusion in social networks at Phase 1*

	Phase 2		Phase 5		Phase 7	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
	OR (95% C.I.)[1]	OR (95% C.I.)[2]	OR (95% C.I.)[3]	OR (95% C.I.)[4]	OR (95% C.I.)[5]	OR (95% C.I.)[6]
Network						
High	1	1	1	1	1	1
Medium	1.08 (1.00-1.17)	1.06 (0.98-1.15)	1.32 (1.22-1.44)	1.26 (1.12-1.41)	1.54 (1.41-1.68)	1.45 (1.25-1.67)
Low	1.44 (1.34-1.56)	1.40 (1.29-1.51)	1.85 (1.70-2.02)	1.56 (1.38-1.76)	2.01 (1.84-2.20)	1.99 (1.71-2.31)
Friends						
High	1	1	1	1	1	1
Medium	1.30 (1.20-1.40)	1.30 (1.20-1.40)	1.20 (1.11-1.31)	1.15 (1.02-1.29)	1.24 (1.13-1.35)	1.03 (0.89-1.19)
Low	1.64 (1.51-1.78)	1.63 (1.50-1.77)	1.79 (1.65-1.95)	1.50 (1.33-1.70)	1.82 (1.67-1.99)	1.50 (1.29-1.74)
Relatives						
High	1	1	1	1	1	1
Medium	1.09 (1.00-1.19)	1.08 (1.00-1.18)	1.29 (1.18-1.41)	1.42 (1.25-1.61)	1.12 (1.02-1.23)	0.91 (0.76-1.08)
Low	1.14 (1.06-1.22)	1.07 (1.00-1.16)	1.18 (1.10-1.28)	1.18 (1.06-1.32)	1.22 (1.13-1.32)	1.46 (1.28-1.67)

[1] Adjusted for age and sex
[2] Adjusted for sex, age, marital status, employment grade, smoking status and physical activity
[3] Adjusted for age and sex
[4] Adjusted for sex, age, marital status, employment grade, smoking status and physical activity
[5] Adjusted for age and sex
[6] Adjusted for sex, age, marital status, employment grade, smoking status and physical activity

Table 6.6.3 *Logistic regression models for odds of depressive symptoms at Phase 2, 5 and 7 according to measures of alcohol consumption at Phase 1*

	Phase 2		Phase 5		Phase 7	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
	OR (95% C.I.)[1]	OR (95% C.I.)[2]	OR (95% C.I.)[3]	OR (95% C.I.)[4]	OR (95% C.I.)[5]	OR (95% C.I.)[6]
Frequency of consumption						
>1/day	1.25 (1.16-1.34)	1.25 (1.16-1.35)	1.18 (1.09-1.28)	1.19 (1.06-1.34)	1.21 (1.11-1.31)	0.90 (0.77-1.04)
>1/week	1	1	1	1	1	1
>1/month	1.12 (1.01-1.23)	1.08 (0.98-1.20)	1.10 (0.98-1.22)	1.12 (0.96-1.31)	1.03 (0.92-1.15)	0.91 (0.75-1.11)
Special occasions/never	1.03 (0.93-1.13)	0.95 (0.86-1.05)	1.55 (1.40-1.71)	1.60 (1.39-1.85)	1.32 (1.19-1.46)	1.42 (1.20-1.67)
Units consumed in past week						
None	1.01 (0.92-1.11)	0.95 (0.86-1.05)	1.54 (1.40-1.70)	1.63 (1.42-1.86)	1.28 (1.16-1.42)	1.44 (1.22-1.71)
1-7	1	1	1	1	1	1
8-14	0.98 (0.90-1.07)	0.98 (0.90-1.07	0.91 (0.83-1.01)	0.81 (0.70-0.93)	0.99 (0.89-1.09)	0.85 (0.72-1.01)
15-21	1.05 (0.94-1.17)	1.07 (0.95-1.19)	1.14 (1.01-1.28)	0.91 (0.77-1.09)	1.24 (1.10-1.39)	1.11 (0.91-1.37)
22-28	1.37 (1.20-1.57)	1.37 (1.20-1.57)	1.45 (1.26-1.67)	1.15 (0.93-1.42)	1.63 (1.42-1.88)	0.85 (0.65-1.13)
29-35	0.95 (0.78-1.15)	0.94 (0.77-1.15)	0.77 (0.62-0.97)	0.72 (0.53-0.98)	1.06 (0.86-1.31)	1.15 (0.83-1.59)
36+	1.35 (1.17-1.57)	1.22 (1.05-1.41)	1.36 (1.17-1.59)	1.33 (1.07-1.65)	1.37 (1.17-1.61)	0.64 (0.46-0.88)

[1] Adjusted for age and sex
[2] Adjusted for sex, age, marital status, employment grade, smoking status and physical activity
[3] Adjusted for age and sex
[4] Adjusted for sex, age, marital status, employment grade, smoking status and physical activity
[5] Adjusted for age and sex
[6] Adjusted for sex, age, marital status, employment grade, smoking status and physical activity

6.7 Summary

The analysis presented in this chapter aimed at investigating the magnitude and duration of the effects of social support, inclusion in social networks and alcohol consumption on depressive symptoms among the adult urban population of the UK, with a particular focus on the deleterious effects of receiving inadequate support, experiencing negative social relations, being socially isolated or regularly consuming alcohol in excess. A secondary aim of this chapter was to preliminary address the issue of temporality in the associations between social support, inclusion in social networks, alcohol consumption and depressive symptoms, as there is evidence to support the notion that individuals who suffer from depressive symptoms could perceive disproportionately high levels of negativity in their relationships, inadequate levels of positive support, become socially isolated and engage in heavy alcohol consumption as a form of self-medication, as a result of their condition (Cohen 2004; Brugha, Weich et al. 2005; Boden and Fergusson 2011; Thoits 2011; Bell and Britton 2014). The third aim of this chapter was to investigate whether the associations between social support, inclusion in social networks, alcohol consumption and depressive symptoms were influenced by the effect of age, sex, marital status, employment grade, smoking status and physical activity and whether alcohol consumption played a role in the association between social support, inclusion in social networks and depressive symptoms, and whether social support and inclusion in social networks played a role in the association between alcohol consumption and depressive symptoms.

Longitudinal regression analysis showed how participants who reported receiving inadequate levels of confiding emotional or practical support at baseline, were more likely to be suffering from depressive symptoms than their better supported counterparts, not only at baseline but also at Phase 2 (for confiding/emotional support) and Phase 5. Further, participants who at baseline experienced negative social relationships were more than twice more likely to be suffering from depressive symptoms at the same time and continued to be doubly at risk for the following nineteen years, until Phase 7 measurements. Similarly, participants who at baseline were little involved in clubs and societies, reported having little contact with friends, or being scarcely in contact with their extended families were more likely to be suffering from depressive symptoms at the time and at all subsequent phases of data collection included in this analysis.

However, these results did not throw any light on the issue of temporality in the association as participants who were considered as suffering from depressive symptoms at baseline were much more likely to receive inadequate confiding/emotional or practical support, to perceive strong negative aspects of social relations and to be little involved in clubs or society, and scarcely in contact with friends and extend families at Phases 2, 5 and 7. Hence, the issue of temporality in the association between social support, inclusion in social networks and depressive symptoms will be further investigated with more advanced statistical techniques in the next chapter.

The effects of alcohol consumption on depressive symptoms were also long lived. In fact, participants who at baseline reported drinking at least once a day were more likely to suffer from depressive symptoms than those who consumed alcohol several times a week, at Phase 2, Phase 5 and Phase 7. Conversely, participants who at baseline reported never drinking or indulging only in special occasions, were also more likely to suffer from depressive symptoms at Phase 5 and Phase 7. In fact, they had the highest odds of depressive symptoms. This is consistent with the existing literature documenting a U shaped association between alcohol consumption and mental health.

Further, participants who at baseline reported drinking between 22-28 and 36 or more UK units per week had high odds of depressive symptoms at both Phases 2 and 5 but not Phase 7; while participants who did not consume any alcohol at baseline had high odds of depressive symptoms at Phases 5 and 7 but not 2. This pattern of association also resembles a U, albeit a delayed in time one. Interestingly the association between alcohol consumption and depressive symptoms was not quite a strong and clear cut in the cross-sectional analysis of Chapter 5, suggesting a possible time lag between the hazardous drinking behaviour adopted by an individual and the onset of its effects on mental health.

Moreover, depressive symptoms at baseline were not a predictor of increased odds of hazardous drinking behaviour later in life, which is surprising as the existing literature seems to suggest mental health has as strong influence on drinking behaviours (Boden and Fergusson 2011; Bell and Britton 2014). Again, the issue of temporality in this association will be further explored in the next chapter. Finally, the odds of depressive symptoms

according to alcohol consumption were substantially affected by the effects of social support and inclusion in social networks, suggesting a possible association, which will be further investigated in the next chapter.

7. Trajectories of change in Social Support, Inclusion in Social Networks, Alcohol Consumption and Depressive Symptoms

7

7.1 **Introduction**

This chapter sets out to further investigate the associations between social support, inclusion in social networks, alcohol consumption and depressive symptoms through time. More specifically, as Chapter 6 highlighted how social support and inclusion in social networks both affected and were affected by depressive symptoms in time, this Chapter aims to throw further light on the direction of the association through the use of parallel growth curve models which will allow to investigate how changes in social support and inclusion in social networks co-vary with depressive symptoms over time. Parallel growth curves will also be used to further investigate the association between alcohol consumption and depressive symptoms in time. In other words, this chapter aims:

O9: To investigate the developmental trajectories of change in the association between social support, inclusion in social networks, alcohol consumption and depressive symptoms through time.

H10: *Individual variation in depressive symptoms through time is associated with individual variation in experienced levels of social support and inclusion in social networks.*

H11: *Individual variation in depressive symptoms through time is associated with individual variation in alcohol consumption – measured in terms of frequency of drinking sessions.*

H12: *The effects of social support and inclusion in social networks on depressive symptoms vary when the effects of alcohol consumption are taken into consideration and vice versa.*

These hypotheses will be addressed by investigating the individual developmental trajectories of change in the association between social support, inclusion in social networks and alcohol consumption and depressive symptoms through time using latent growth curve modelling (LGCM), a relatively recent statistical technique that provides a means of modelling development as a factor of repeated observations over time. Latent growth curve modelling is based on structural equation modelling and thus shares many of the strengths and weaknesses of the latter. In addition, LGCM has the peculiar ability to allow to test the adequacy of the hypothesized growth form, to incorporate time-varying as well as invariant covariates and to develop a common development trajectory from the data, thus ruling out cohort effects. LGCM will be discussed in more detail in section 7.2 while in section 7.3 I will introduce the details of the statistical analysis here used and present the latent growth curve models employed in the analysis. Section 7.4 will present the results and 7.5 will provide a summary of the chapter.

7.2 Statistical Analysis

7.2.1 Introduction to Latent Growth Curve Modelling (LGCM)

Statistical analysis in this chapter took the form of eight latent growth curve models of increasing complexity. Latent growth curve modelling is a relatively new framework for the analysis of growth and developmental processes (Duncan and Duncan, 2009). Latent growth curve modelling differs from more traditional frameworks for longitudinal analysis, in that it allows for more flexibility to examine inter- and intra- individual variation over time. In addition, latent growth curve models can accommodate multivariate or higher order specifications, multiple populations, multilevel of hierarchical structures and complex relations (Duncan and Duncan, 2009). In fact, while more traditional methods for longitudinal analysis, such as ANOVA or multiple regression, analyse only mean changes and treat differences among individuals as error variance, latent growth curve models use random coefficients to capture individual differences in growth over time (Duncan and Duncan, 2009).

These models are somewhat similar to confirmatory factor analysis, except that LGCMs use repeated measures as raw-score data and hence the latent factors are interpreted as chronometric common factors representing individual differences over time (McArdle, 1988; Duncan, 2006). In addition, LGCMs take into account both factor means and variances, a combination that renders them a unique technique. Growth curve methodology is formed of two stages, first a regression curve is fitted to the repeated measures of each individual in the sample, and in a second time the parameters for an individual's curve become the focus of the analysis instead of the original measures.

The simplest form of growth curve models sees a regression curve modelling two latent factors modelled: *intercept* and *slope* of developmental trajectories in time being fitted to the repeated individual measures. The *intercept* is a constant for all individuals across time, and therefore it has fixed values for the factor loadings on the repeated measures. The intercept loadings are conventionally fixed to 1. In the model, the intercept for any given individual has the same meaning of the intercept of a straight line on a coordinate system: it is the point in which the line crosses the vertical axis. The intercept latent factor represents information about the mean and variance of all intercepts in the sample.

The *slope* represents the slope of an individual trajectory and it has a mean and variance across the whole sample that, as for the intercept mean and variance, can be estimated from the data. Unlike the intercept though, the slope loadings can be rescaled to vary across time.

In more complex forms of growth curve models, multiple growth curves can be modelled in parallel, so that two or more fitted Intercepts and Slopes are allowed to co-vary in time. This enables to model the effects of change of one variable through time, on to the change of another. In the case of the present analysis, baseline levels (Intercept) of social support, inclusion in social networks and alcohol consumption will be allowed to co-vary with the initial levels of depressive symptoms (Intercept). And subsequent changes (Slope) in social support, inclusion in social networks and alcohol consumption will be allowed to co-vary with changes in depressive symptom (Slope) through. Allowing the slopes to co-vary will reveal whether it is change in social support, inclusion in social networks and alcohol consumption to affect change in depressive symptoms through time or the other way round, as the strongest effect will prevail.

More specifically, this analysis will present eight LGCMs, divided in three groups of increasing complexity. The first group of LGCMs will be composed of four baseline models inferring the trajectory of change through time of depressive symptoms, social support, inclusion in social networks and alcohol consumption independently of each other. The second group of models will include three parallel growth models in which social support, inclusion in social networks and alcohol consumption will, in turn, be allowed to covariate with changes in depressive symptoms. The final model will incorporate the previous three parallel models, allowing social support, inclusion in social networks, alcohol consumption and depressive symptoms to co-vary through time.

7.2.2 Statistical Analysis

All statistical analysis related to LGCM was carried out in STATA 13, using the SEM Model Builder, a software tool that allows the user to create path diagrams for Structural Equation Models (SEM) and Generalized Structural Equation Models (GSEM), fit those models, and show results on the path diagram. In the Model Builder, path diagrams can be selected from the menu or manually drawn. The meanings of specific symbols in these path diagrams are presented in Table 7.2.1.

Table 7.2.1 *Meanings of specific symbols in path diagrams*

Symbol	Meaning
	Observed Variable
	Latent Variable
	Path
	Generalized Response Variable
	Multilevel Latent Variable

In the eight models here presented, depressive symptoms were here used as continuous variable, rather than a dichotomous one, using all the observed values measured with the GHQ depression scale. This was done in order to be able to measure individual trajectories in growth of depressive symptoms, which would not have been possible with a dichotomous variable. Measures of confiding/emotional, practical and negative support were also used as continuous variables and combined into one overarching variable named 'support', scored on a scale from 0 to 12 where 0 corresponds to the lowest levels of support and 12 to the highest. In order to create this variable, the continuous scores for negative support were reversed so that the highest levels of negative support are scored as 0.

Similarly, measures of inclusion in networks outside the household, and of contact with relatives and friends were also taken as continuous variables and combined into one overarching variable for inclusion in social networks, named 'inclusion', scored on a scale from 0 to 23 , where 0 corresponds to the lowest levels of inclusion and 23 to the highest. Further, as the analysis presented in Chapter 5 and Chapter 6 revealed a lack of statistical association between amount of alcohol consumed per week and depressive symptoms, only measures of frequency of alcohol consumption were used for this part of the analysis. The measure of frequency of alcohol consumption here used is composed of six categories: (1) never, (2) in special occasions, (3) once or more a month, (4) once or more a week, (5) daily , or (6) twice or more a day. All analysis was run on original data, as LGCM allows to take into account missing data in the estimation on the trajectories of change. Details of the individual LGC models are given in the next sections.

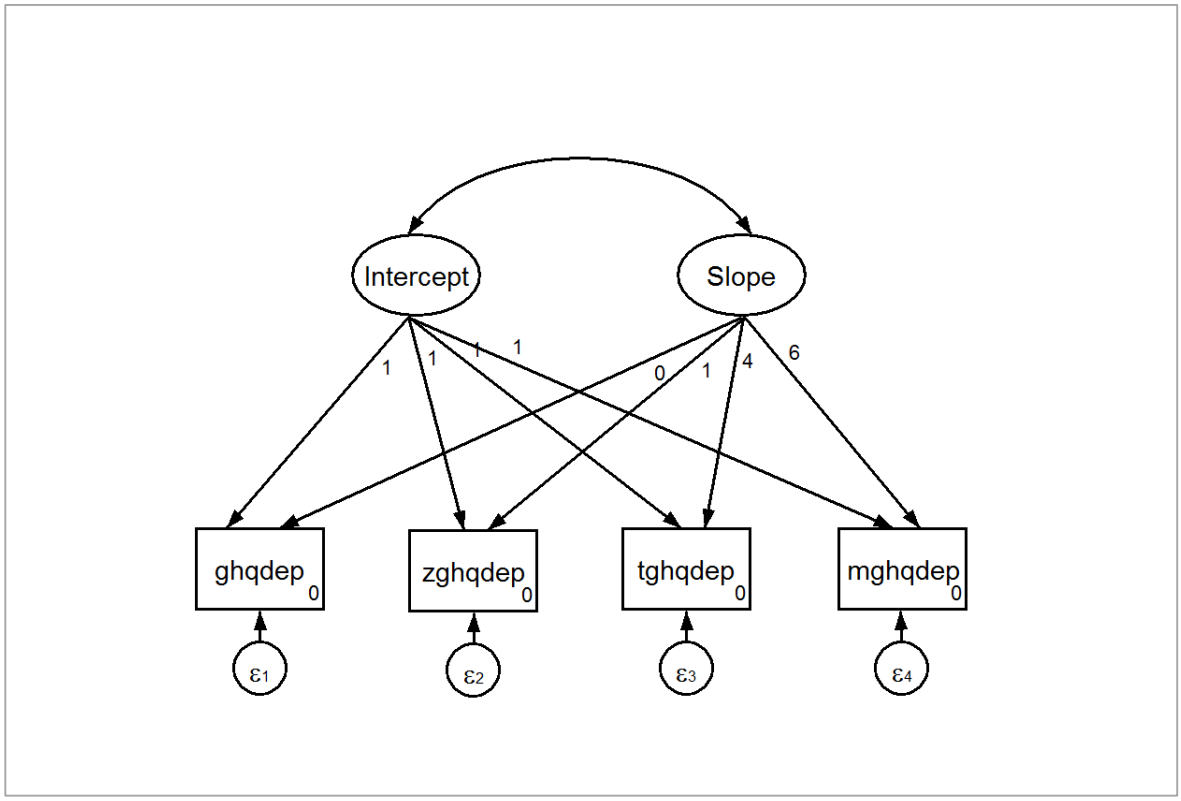
Moreover, the eight latent growth curves were modelled on the original data sample of the Whitehall II cohort study and missing data were handled by default through maximum likelihood (ML) in STATA 13. Maximum Likelihood is an advanced missing data method in which in missing values are not replaced or imputed, as in the case of multiple imputation, but are handled within the analysis model. Maximum Likelihood is based on the likelihood function, which expresses the probability of the data as a function of the data and of the unknown parameter values. Just like multiple imputation, described in Chapter 5, maximum likelihood is based on the assumption that data will be missing at random (MAR). This could present a problem in that, if data are not missing at random the estimates produced may not be accurate, however recently some methodologists have argued that routine departures from MAR may not be large enough to cause serious bias in the estimates produced by maximum likelihood or multiple imputation (Baraldi and Enders, 2010; Schafer and Graham, 2002). In addition, Maximum Likelihood has often been preferred to multiple imputation, on the basis that it is a simpler technique and that it provides more accurate standard errors (Larsen, 2011). One downfall of maximum likelihood is that when applied to small sample sizes it loses some of its accuracy of estimation (Baraldi and Enders, 2010), however in the present study the sample sizes are large enough for maximum likelihood to produce accurate estimates of the missing data.

7.2.3 Depressive Symptoms

The first of the latent growth curve models run in this analysis is concerned with modelling the growth of change in depressive symptoms through time. Growth among measures of depressive symptoms at Phases 1, 2, 5, 7 of the Whitehall II cohort study, will be modelled. A regression curve will be fitted to the individual repeated measures, modelling the intercept and slope of the whole sample. Factor loadings for the intercept will be fixed at 1, while factor loadings for the slope will mirror the time point at which measures of depressive symptoms were taken. Because individual phase measurements in the Whitehall II cohort study were taken at roughly regular intervals of 2.6 years, phases were taken as units of time, in this and all other models. Therefore the factor loadings for the slope will be 0 for the first Phase of the Whitehall study, 1 for the second, 4 for the fifth, and 6 for the seventh (Figure 7.2.1).

Figure 7.2.1 depicts the model for depressive symptoms. The square boxes represent the observed values of depressive symptoms at Phase 1, 2, 5 and 7 of the Whitehall II cohort study, labelled with 'ghqdep' which is the name of the variable for depressive symptoms in the dataset, preceded by the letters z-, t- or m- which are the Phase-specific prefixes. The round boxes represent the latent variables that are being modelled, in this case the Intercept and Slope of the growth curve for depressive symptoms through time. The arrows connecting the latent variables with the observed variables represent the paths in this path diagram. The numbers next to them are the factor loadings for the Intercept (1) and Slope (0,1,4,6) respectively. The ε in the round boxes at the bottom of the diagram represent residual errors in the measurements.

Figure 7.2.1 *LGCM modelling the intercept and slope of change in depressive symptoms through time.*



7.2.4 Social Support and Inclusion in Social Networks

The second and third latent growth curve models are simple growth curve models fitting the Intercept and Slope of growth in social support and inclusion in social networks, respectively (Figure 7.2.2 and Figure 7.2.3). In both models the individual repeated measures of social support or inclusion in social networks are displayed in the square boxes, each representing data from a subsequent phase of the Whitehall II cohort study. The four boxes are each connected to the Intercept and Slope by arrows representing the paths connecting individual measures with the latent elements of the growth curve.

In Figure 7.2.2 and Figure 7.2.3, the pathways connecting individual measures of social support and inclusion in social networks to the intercept of their growth curve, are marked by four 1s. This signifies that the value of the Intercept is fixed at 1. On the other hand, the paths connecting individual measures of social support and inclusion in social networks to the Slope of their growth curve are marked by the numbers 0, 1, 4, 6. These represent the four phases included in the model, with 0 corresponding to baseline measures, 1 to Phase 2 measures, 4 to Phase 5 measures and 6 to Phase 7 measures. The arched arrow connecting Intercept and Slope signifies that they are allowed to co-vary (Figure 7.2.2 and Figure 7.2.3).

Figure 7.2.2 *LGCM modelling the intercept and slope of change in social support through time*

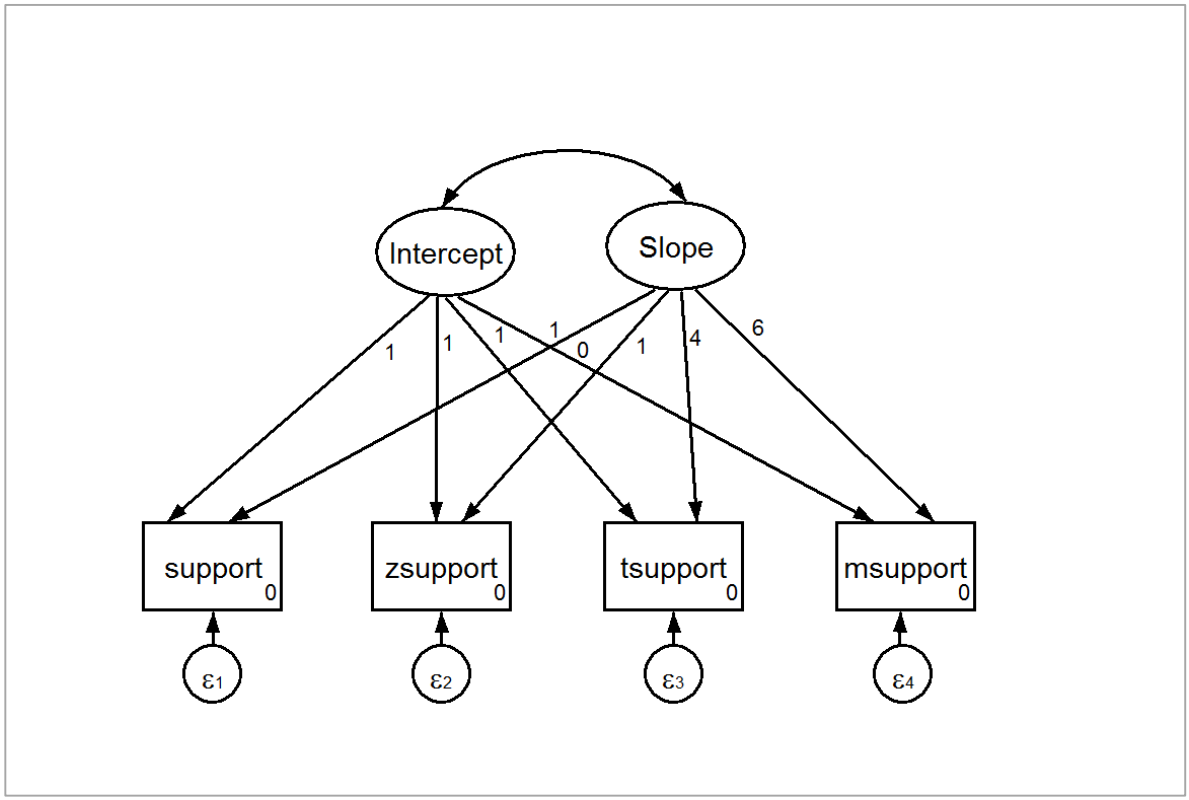
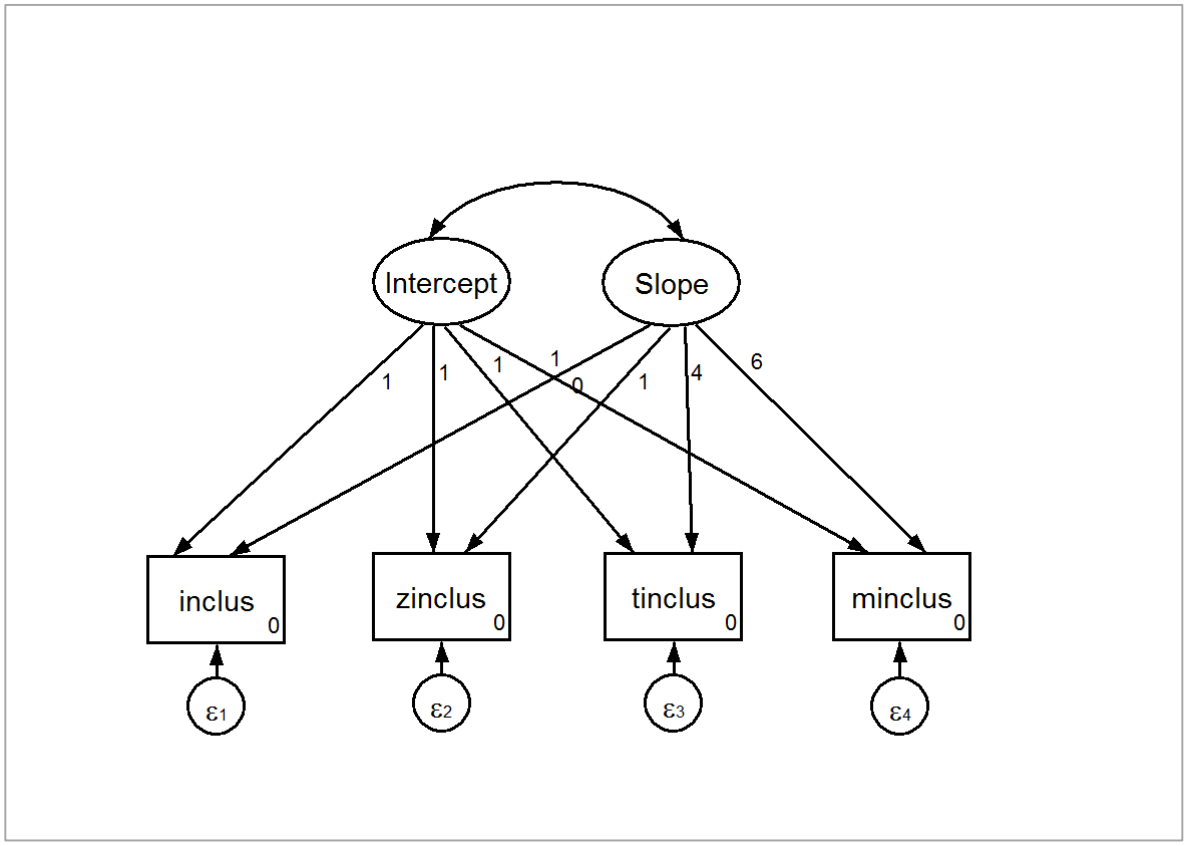


Figure 7.2.3 *LGCM modelling the intercept and slope of change in inclusion in social networks through time.*

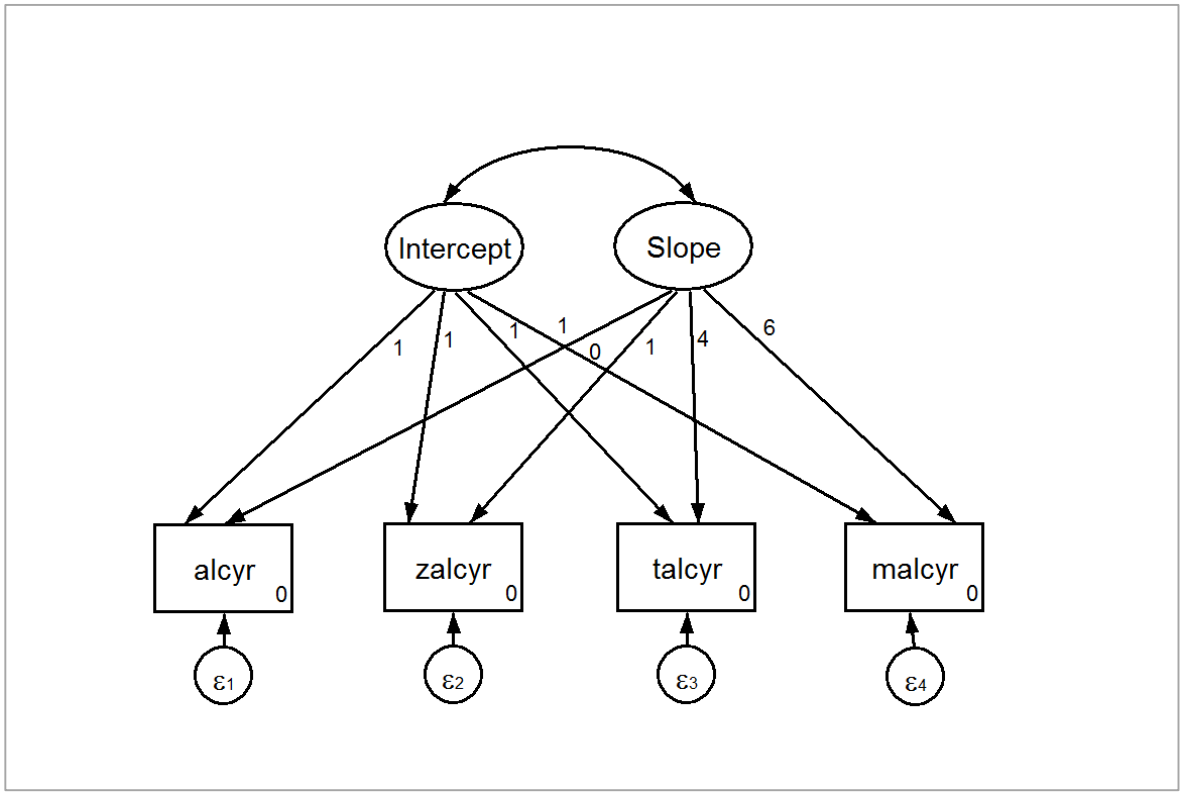


7.2.5 Alcohol Consumption

The fourth latent growth curve model is designed to model the growth of change in alcohol consumption from Phase 1 to Phase 7 of the Whitehall II cohort study. Since the only measure of alcohol consumption here included is frequency of consumption, this model will be very similar to the first three models. For, in this model too, a regression curve will be fitted to the individual repeated measures, modelling the intercept and slope of change in frequency of alcohol consumption.

This model very closely resembles the three models previously described. In fact, here too, the repeated individual measures of frequency of alcohol consumption are represented in four square boxes, one for each phase of the Whitehall II cohort study, connected by arrow paths to the Intercept and Slope of their growth through time. The value of the Intercept is fixed at one, while the values of the paths connecting the measures with their slope of growth represent the four points in time included in the model (0= Phase 1; 1=Phase2; 4=Phase5; 6=Phase7). Finally, the Intercept and Slope are allowed to co-vary (Figure 7.2.4).

Figure 7.2.4 *LGCM representing the intercept and slope of frequency of alcohol consumption through time*

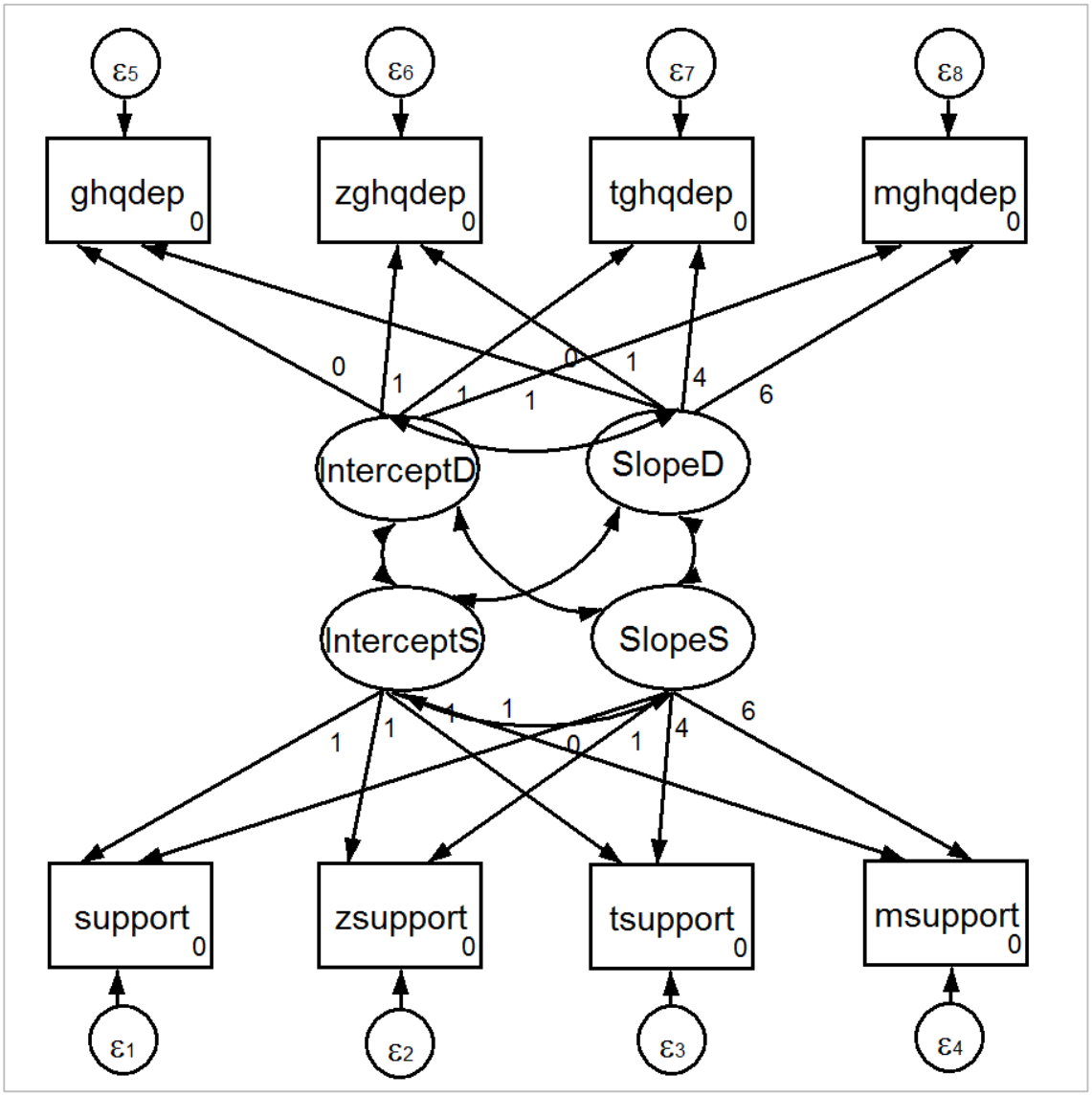


7.2.6 Social Support and Depressive Symptoms

In the first of the parallel growth models I will allow the intercept and slope of growth in social support to co-vary with the intercept and slope of growth in depressive symptoms through time. In order to do so, the model will be a combination of the simpler models for depressive symptoms and social support, where two separate regression curves will be fitted to the individual repeated measures for depressive symptoms and social support so as to calculate the intercepts and slopes for depressive symptoms and social support respectively, for the whole sample (Figure 7.2.5). Moreover, the variation among individuals in the Intercept and Slope for social support will be allowed to be associated with the individual-level variation in the Intercept and Slope for change in depressive symptoms (Figure 7.2.5).

This model is depicted in Figure 7.2.5, where it can be observed that just like Models 1 and 2, square boxes represent the observed values of depressive symptoms (top) and social support (bottom), labelled with the variables names at each phase. These are connected by paths, to the respective latent variables, labelled 'InterceptS and SlopeS' for social support, and 'InterceptD and SlopeD' for depressive symptoms. As in previous models, the factor loading for both intercepts are fixed at 1, while factor loadings for the slope will mirror the time point at which measures of frequency of alcohol consumption were taken, with 0 representing the first Phase of the Whitehall study, 1 for the second, 4 for the fifth, and 6 for the seventh (Figure 7.2.5). In addition, in this model, covariance paths connect 'InterceptS' and 'SlopeS' , 'InterceptD' and 'SlopeD', 'InterceptS' and 'InterceptD', 'SlopeS' and 'SlopeD', 'InterceptS' and 'SlopeD', and 'InterceptD' and 'SlopeS'.(Figure 7.2.5).

Figure 7.2.5 *LGCM representing the intercept and slope of social support then used to model the intercept and slope of depressive symptoms.*



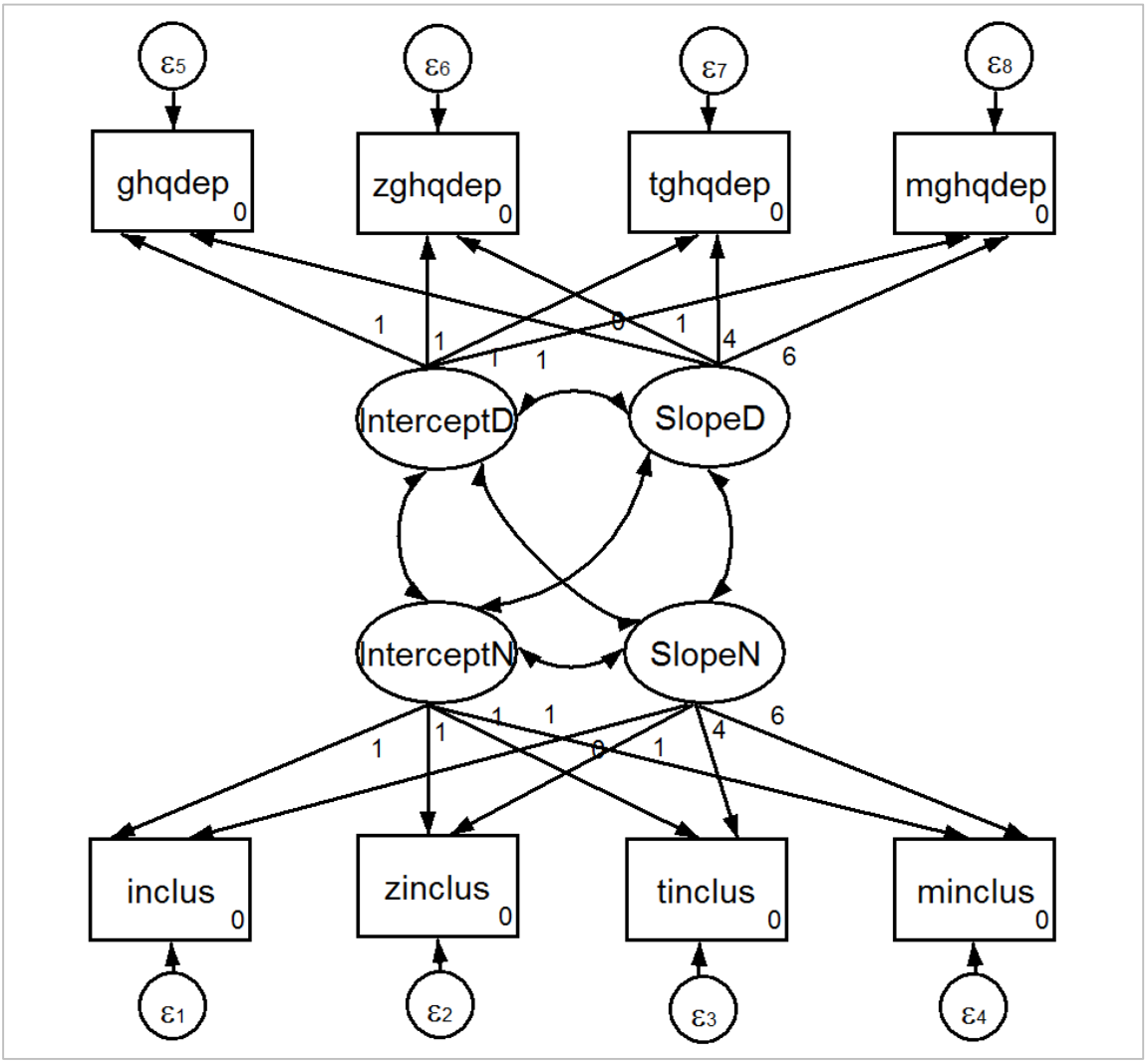
7.2.7 Inclusion in Social Networks and Depressive Symptoms

The second of the parallel growth models will be very similar to the previous one, only this time, the intercept and slope of growth in inclusion in social networks will be allowed to covariate the growth in depressive symptoms through time. As in the case of Model 5, a regression curve will be fitted to the individual measures of depressive symptoms, and one to the individual measures of inclusion in social networks in order to be able to calculate the respective intercept and slope for the whole sample (Figure 7.2.6). In a second time, the Intercept and Slope for inclusion in social networks will be allowed to affect the variation in the Intercept and Slope for change in depressive symptoms (Figure 7.2.6).

This model is depicted in Figure 7.2.6, where it can be observed that just like Model 5, square boxes at the top and bottom of the figure represent the observed values of depressive symptoms and inclusion in social networks, respectively, with prefixes z- , t- and m- to indicate Phase 2, 5 and 7 respectively. These square boxes are connected to their relative latent variables, labelled 'InterceptN' and 'SlopeN' for inclusion in social networks, and 'InterceptD and SlopeD' for depressive symptoms, through paths in the shape of arrows. As in previous models, the factor loading for both intercepts are fixed at 1, while factor loadings for the slope will mirror the time point at which measures of frequency of alcohol consumption were taken, with 0 representing the first Phase of the Whitehall study, 1 for the second, 4 for the fifth, and 6 for the seventh (Figure 7.2.6). In addition, in this model like in the previous one, covariance arrows connected the 'InterceptN' and 'SlopeN' of inclusion in social networks, the 'InterceptD' and 'SlopeD' of depressive symptoms, but also the 'InterceptN' and 'SlopeD', the 'InterceptD' and the 'SlopeN', the 'InterceptN' and

the ‘InterceptD’, and the ‘SlopeN’ and ‘SlopeD’, symbolizing the fact that inclusion in social networks and depressive symptoms are allowed to affect each other (Figure 7.2.6).

Figure 7.2.6 *LGCM representing the intercept and slope of inclusion in social networks then used to model the intercept and slope of depressive symptoms*



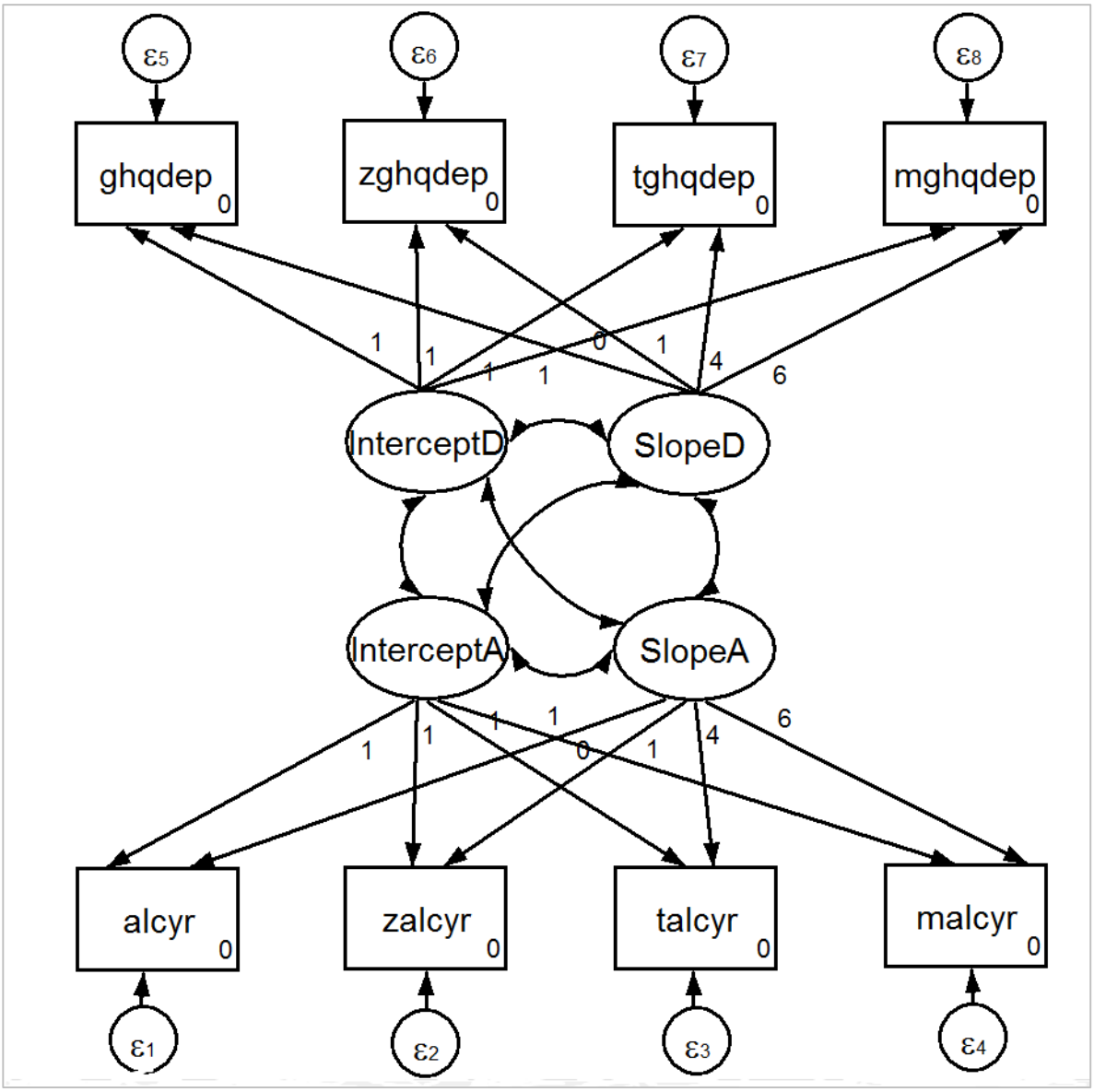
7.2.8 Alcohol Consumption and Depressive Symptoms

This model will be very similar to the previous two parallel growth models, and the intercept and slope of growth in alcohol consumption will be allowed to affect the intercept and slope of depressive symptoms though time. As in the case of the previous two parallel growth models, a regression curve will be fitted to the individual measures of depressive symptoms, and one to the individual measures of alcohol consumption in order to be able to calculate the respective intercept and slope for the whole sample (Figure 7.2.7). In a second time, the Intercept and Slope for alcohol consumption will be allowed to affect the variation in the Intercept and Slope for change in depressive symptoms (Figure 7.2.7).

This model is depicted in Figure 7.2.7, where it can be observed that just like Model 5 and Model 6, square boxes at the top and bottom of the figure represent the observed values of depressive symptoms and inclusion in social networks, respectively, with prefixes z- , t- and m- to indicate Phase 2, 5 and 7 respectively. These square boxes are connected to their relative latent variables, labelled 'InterceptA' and 'SlopeA' for alcohol consumption, and 'InterceptD and SlopeD' for depressive symptoms, by paths in the shape of arrows. As in previous models, the factor loading for both intercepts are fixed at 1, while factor loadings for the slope will mirror the time point at which measures of frequency of alcohol consumption were taken, with 0 representing the first Phase of the Whitehall study, 1 for the second, 4 for the fifth, and 6 for the seventh (Figure 7.2.7). In addition, in this model like in the previous two models, covariance arrows connecting 'InterceptA' with 'SlopeA' , 'InterceptD' and 'SlopeD', but also 'InterceptD' with 'SlopeA', 'InterceptA' with 'SlopeD',

'InterceptA ' with 'InterceptD', and 'SlopeA' with 'SlopeD' symbolizing the fact that alcohol consumption and depressive symptoms are allowed to affect each other (Figure 7.2.7).

Figure 7.2.7 *LGCM representing the intercept and slope of alcohol consumption then used to model the intercept and slope of depressive symptoms*



7.2.9 Social support, Inclusion in Social Networks, Alcohol Consumption and Depressive Symptoms

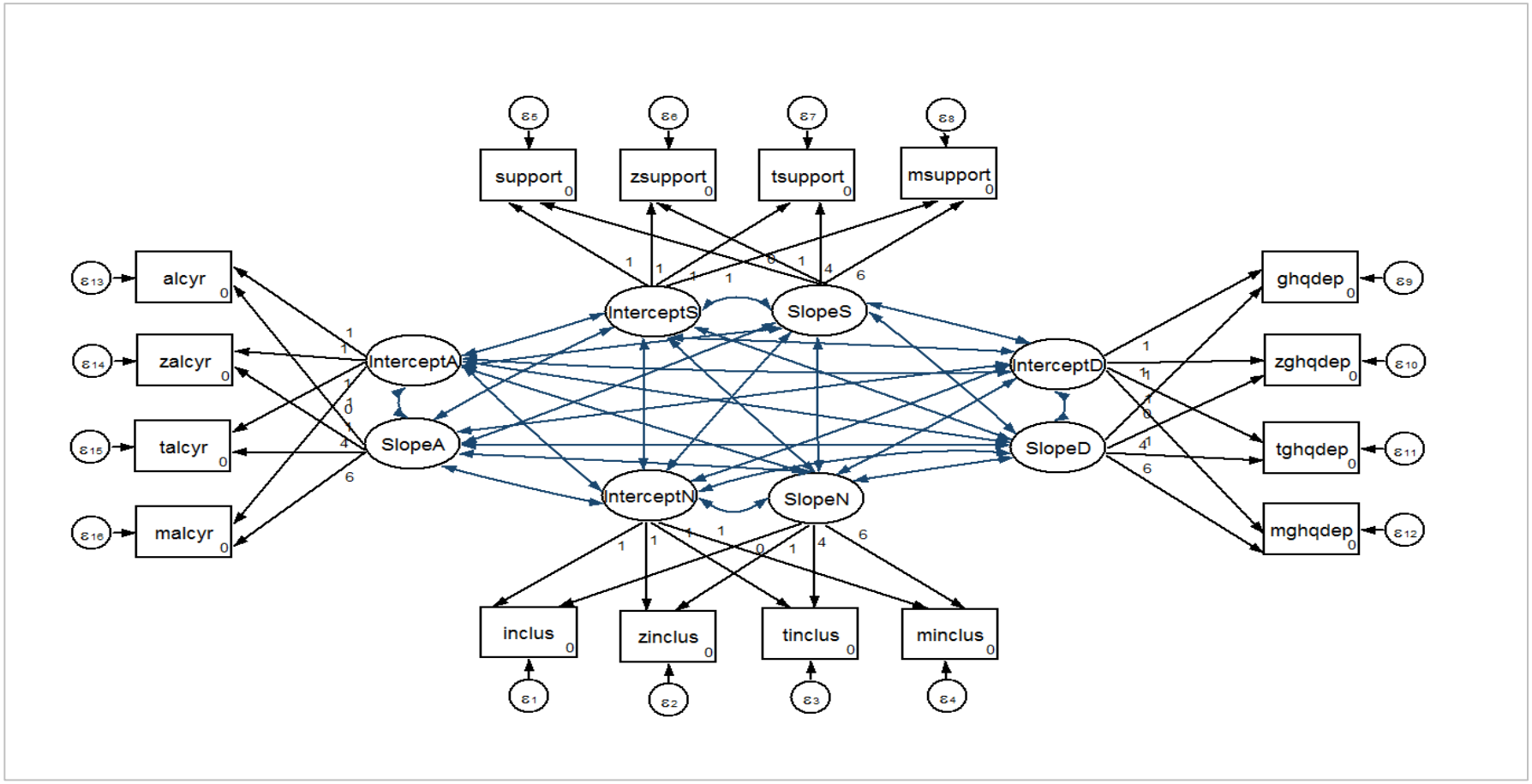
In this final model, individual changes through time in social support, inclusion in social networks and alcohol consumption will be allowed to co-vary with each other as well as with depressive symptoms. The model is, therefore, a combination of all previous models and it is illustrated in Figure 7.2.8. From left to right, the first element in Figure 7.2.8 is the fourth model presented earlier, in which measures of alcohol consumption are used to calculate the Intercept(A) and Slope(A) of change in individual consumption through time. Intercept(A) and Slope(A) are in turn allowed to co-vary. In the central upper section of Figure 7.2.8 is the model that infers the Intercept(S) and Slope(S) of individual change in social support through time. As in the case of alcohol consumption the Intercept(S) and Slope(S) are allowed to co-vary. In the lower central section of the figure, measures of inclusion in social networks are used to model the Intercept(N) and Slope(N) of individual change in social inclusion through time. Intercept(N) and Slope(N) that are, in turn, allowed to co-vary. On the right hand side of Figure 7.2.8, measures of depressive symptoms are used to model the Intercept(D) and Slope(D) of individual change in depressive symptoms through time, allowing the Intercept(D) and Slope(D) to co-vary.

In the centre of the diagram, covariation paths (in blue) connect the four sets of latent variables. So that, from the left, the Intercept(A) and Slope(A) for variation in alcohol consumption are allowed to co-vary with: (1) the Intercept(S) and Slope(S) for variation in social support; (2) the Intercept(N) and Slope(N) for variation in inclusion in social networks; and (3) with the Intercept(D) and Slope(D) of growth in depressive symptoms. Similarly, the

Intercept(S) and Slope(S) of individual variation in social support are allowed to co-vary with: (1) the Intercept(A) and Slope(A) of growth in individual alcohol consumption; (2) the Intercept(N) and Slope(N) of individual growth in inclusion in social networks; and (3) the Intercept(D) and Slope(D) of individual variation in depressive symptoms.

Further, the Intercept(N) and Slope(N) of growth in individual inclusion in social networks were allowed to co-vary with: (1) the Intercept(A) and Slope(A) of change in alcohol consumption; (2) the Intercept(S) and Slope(S) of change in social support; and (3) the Intercept(D) and Slope(D) of growth in individual depressive symptoms. Finally, the Intercept(D) and Slope(D) of individual change in depressive symptoms through time were allowed to co-vary with all the Intercepts and Slopes of social support, inclusion in social networks and alcohol consumption.

Figure 7.2.8 *LGCM representing how individual variation through time in alcohol consumption, social support, inclusion in social networks or depressive symptoms can affect the growth of any of these factors.*

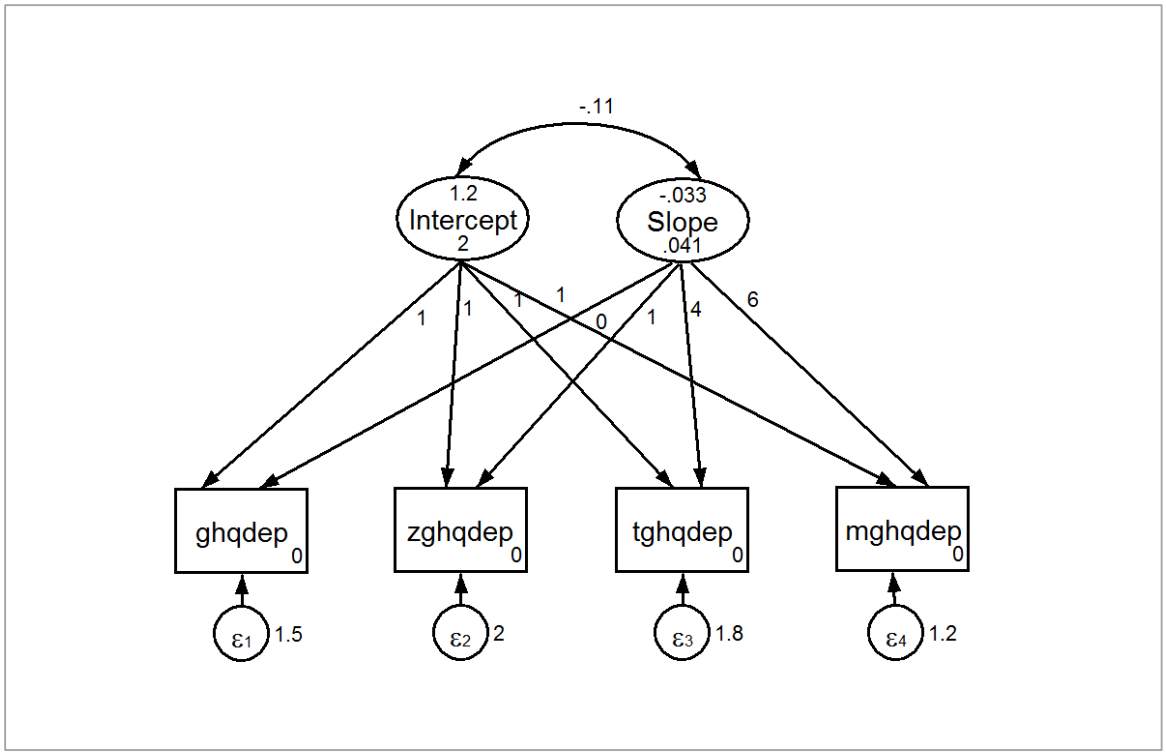


7.3 Results

7.3.1 Depressive Symptoms

Figure 7.3.1 shows the trajectory of change in depressive symptoms among participants of the Whitehall II cohort study from 1985-1988 to 2002-2004. The model here presented was designed to calculate the intercept and slope of the growth curve in depressive symptoms on 10289 observations. The intercept was 1.22 (95% CI 1.18 to 1.25), while the estimate of the slope was -0.03 (95% CI -0.04 to -0.03) indicating that average levels of depressive symptoms decreased between Phase 1 and Phase 7 of the Whitehall II cohort study. Further, the model revealed a significant correlation coefficient between intercept and slope of depressive symptoms (β = -0.11, 95% CI -0.12 to -0.09). The fact that the β coefficient is negative indicates that persons who scored higher in the GHQ depression scale underwent the greatest reductions GHQ scores between phases. In other words, participants who suffered from the strongest depressive symptoms experienced greater improvement in their condition between the four phases of the Whitehall II cohort study included in the present analysis.

Figure 7.3.1 *LGCM modelling the intercept and slope of change in depressive symptoms through time (sample size 10289).*



7.3.2 Social Support and Inclusion in Social Networks

Model 2 and Model 3 were designed to model the intercept and slope of change in individual levels of social support and inclusion in social networks, respectively, through time among participants of the Whitehall II cohort study. The number of observations included in this model was 9866. Figure 7.3.2 illustrates Model 2, in which the value of the intercept for growth in social support is revealed to be 15.44 (95% CI 15.36 to 15.51) with a slope decreasing by -0.29 (95% CI -0.30 to -0.27). Further, the correlation coefficient between intercept and slope (β) was negative (β= -0.58, 95% CI -0.65 to -0.50), indicating that persons who enjoyed the highest levels of support underwent the greatest reduction in support experienced between phases.

Figure 7.3.3 on the other hand illustrates Model 3, inferring the trajectory of growth in inclusion in social networks over time. The number of observations for this model was 10823. The intercept of the growth curve is shown to equal 9.7 (95% CI 9.65 to 9.81) with a decreasing slope at an angle of -0.43 (95% CI -0.45 to -0.42). Further, the correlation coefficient β between intercept and slope was negative (β= -1.28, 95% CI -1.36 to -1.20), indicating that persons who were most socially involved underwent the greatest reduction in their social participation between phases.

Figure 7.3.2 *LGCM modelling the intercept and slope of change in social support through time (sample size 9866).*

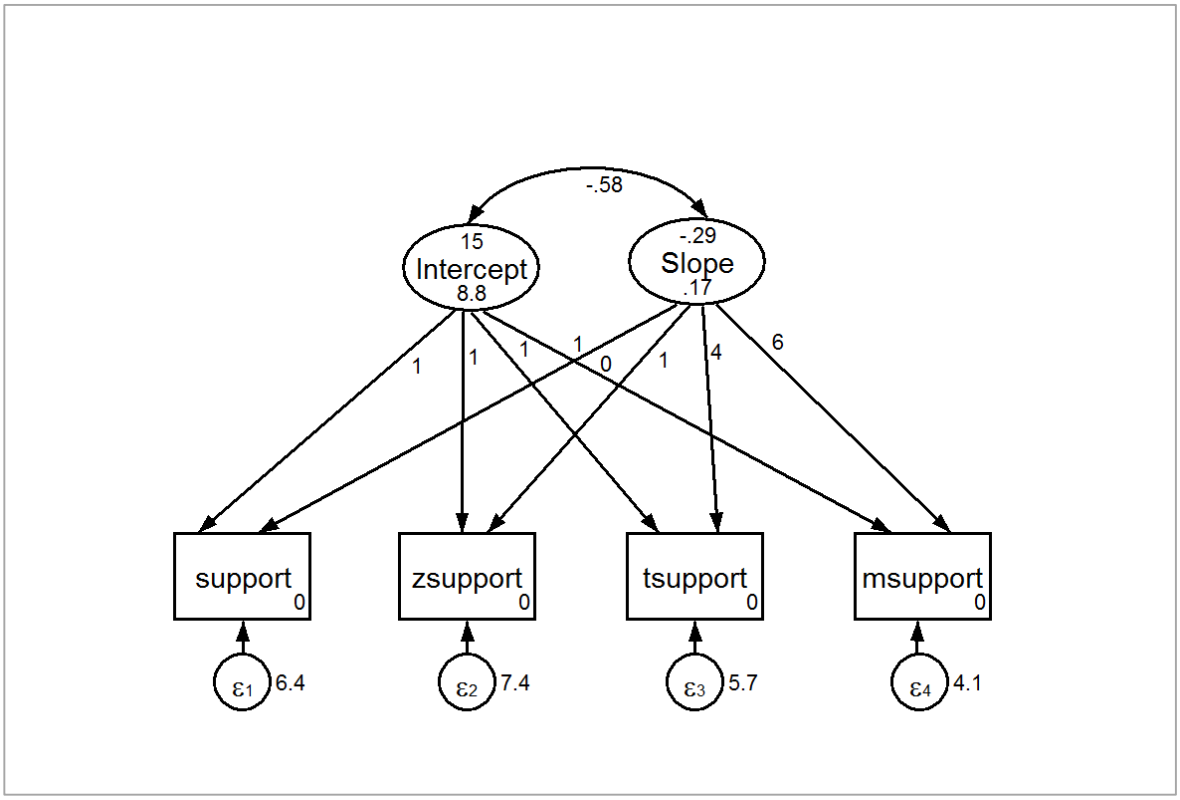
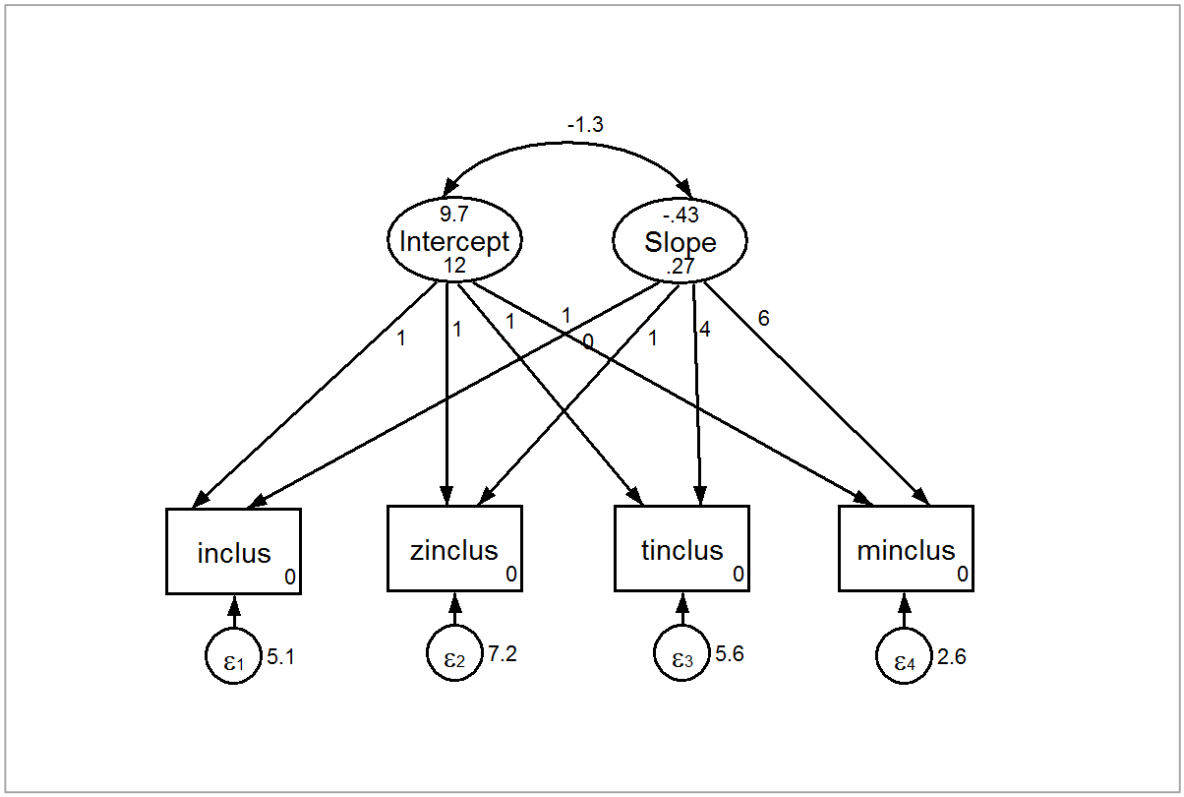


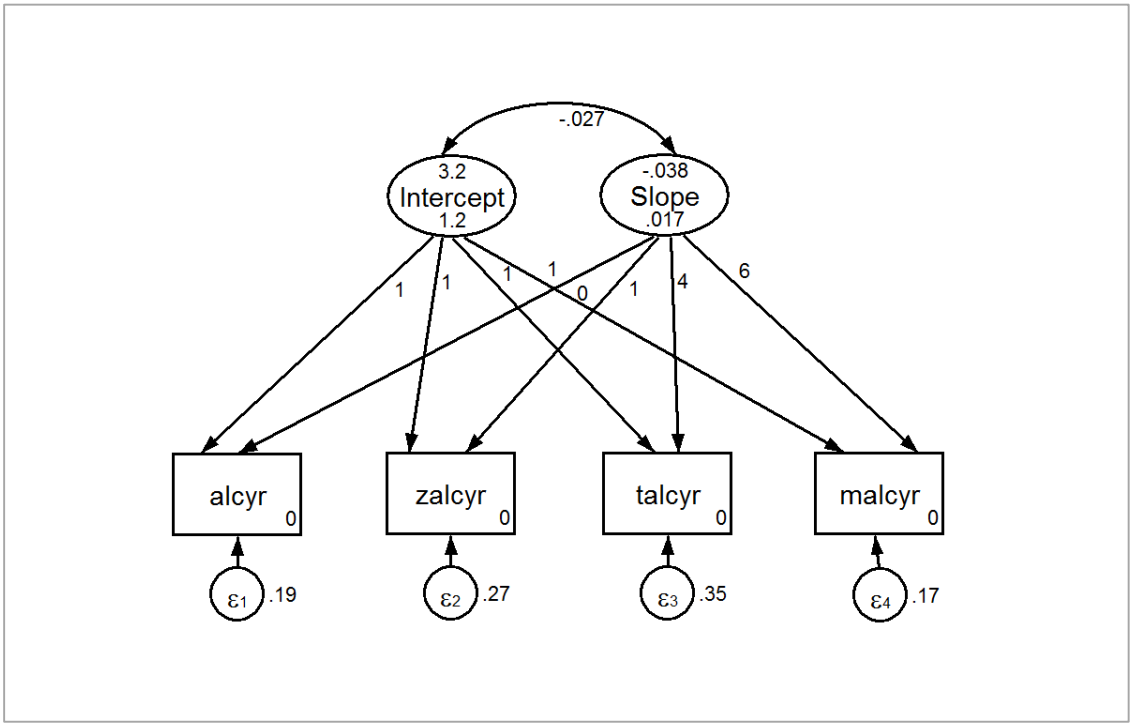
Figure 7.3.3 *LGCM modelling the intercept and slope of change in inclusion in social networks through time (sample size 10823).*



7.3.3 Alcohol Consumption

Figure 7.3.4 shows the trajectory of change in individual alcohol consumption among participants of the Whitehall II cohort between 1985-1988 and 2002-2004. The model was designed to calculate the intercept and slope of the growth curve of change in alcohol consumption on 10305 observations. Results revealed an intercept value of 3.78 (95% CI 3.76 to 3.80) and a slope increase through time of 0.04 (95% CI 0.03 to 0.04). In addition, a significant and negative correlation coefficient between intercept and slope was found (β= -0.03, 95% CI -0.03 to -0.02) for alcohol consumption. The fact that the β coefficient is negative means that individuals drinking less often made greater reductions in their alcohol consumption between phases.

Figure 7.3.4 *LGCM representing the intercept and slope of frequency of alcohol consumption through time (Sample size 10305).*



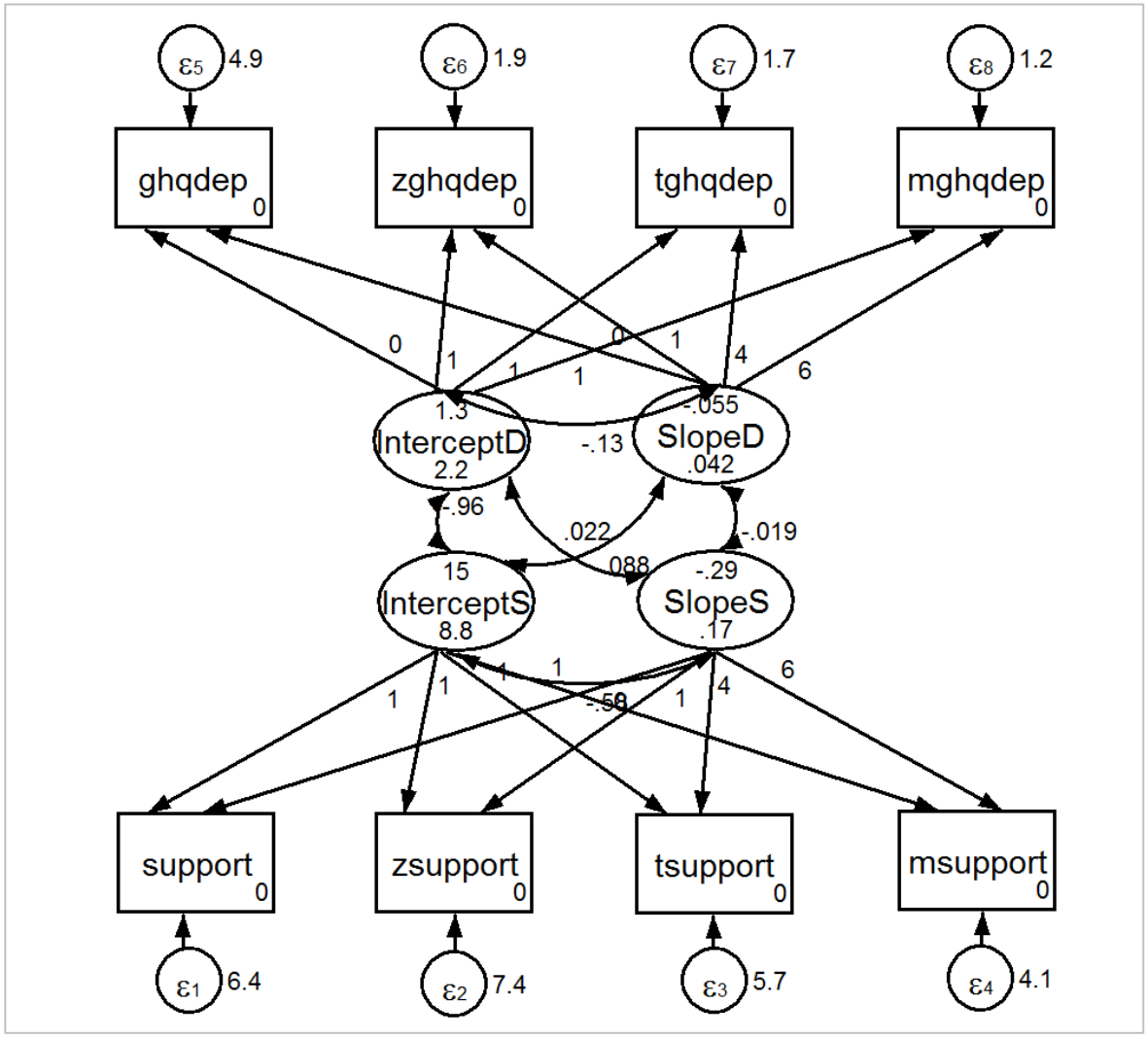
7.3.4 Social Support and Depressive Symptoms

The first parallel growth model here presented was designed to investigate how change in individual social support and change in individual depressive symptoms affected each other through time. The parallel growth model was fitted to jointly model the two growth curves and at the same time to allow the intercepts and slopes of growth in depressive symptoms and in social support to be associated with each other. This model was fitted on 10929 observations. The values intercept and slope for both social support and depressive symptoms were unaltered from Model 1 and Model 2. Similarly, of the five covariation paths here included, the values of the two connecting 'InterceptS' and 'SlopeS', and 'InterceptD' and 'SlopeD' were unaltered from those observed in Model 1 and Model 2. In addition, the estimates of the remaining three covariation paths between the intercepts and slopes indicated that both level of and change in social support were associated with change in depressive symptoms (Figure 7.3.5).

In fact, the three new β coefficients produced by this model were: (1) the negative co-variation between the intercept for depressive symptoms and the intercept for social support (β= -0.89, 95% CI -1.04 to -0.74); (2) the positive co-variation between the slope for depressive symptoms and the intercept for social support (β= 0.08, 95% CI 0.04 to 0.11); and (3) the negative co-variation between the slope for depressive symptoms and the slope for social support (β= -0.01, 95% CI -0.02 to -0.01). This means that: (1) the intensity of depressive symptoms was inversely correlated with the levels of social support experienced, so that the more intense the symptoms the lower the social support; (2) the change in intensity of depressive symptoms through time is directly correlated with social

support, so that higher levels of social support are associated with greater changes in depressive symptoms ; and (3) the slopes for depressive symptoms and social support are negatively correlated so that the greater the changes in depressive symptoms, the smaller the changes in social support between phases. This model was a relatively good fit as the root mean square error of approximation statistic (RMSEA) was 0.053 (Table 7.3.1).

Figure 7.3.5 *LGCM representing the intercept and slope of social support then used to model the intercept and slope of depressive symptoms (sample size 10929).*



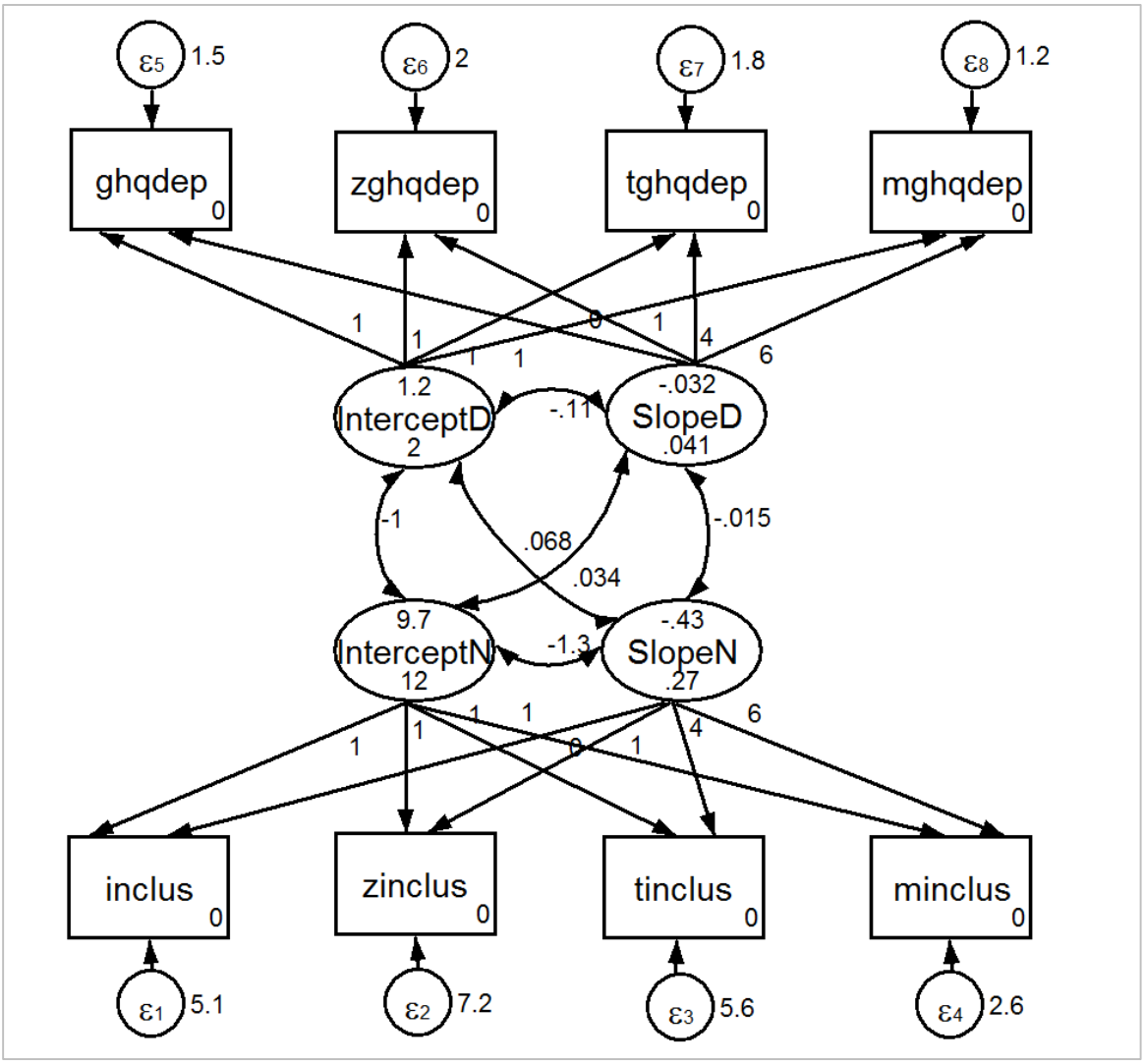
7.3.5 Inclusion in Social Networks and Depressive Symptoms

This parallel growth model was a combination of the earlier ones modelling the growth of depressive symptoms and inclusion in social networks separately on 10299 observations. Hence, the values intercept and slope of both inclusion in social networks and depressive symptoms were the same as when they were modelled separately. Further, of the five β coefficients included in this model, the two linking 'InterceptN' and 'SlopeN' and 'InterceptD and 'SlopeD' were unvaried from Model 1 and Model 3 (Figure 7.3.6).

The remaining four co-variation β coefficients here produced were: (1) the negative effect of the intercept of depressive symptoms on the intercept of inclusion in social networks (β= -0.99, 95% CI -1.13 to -0.86), meaning that the intensity of depressive symptoms was inversely correlated with the levels of inclusion in social networks experienced, so that the more intense the symptoms the lower the social inclusion in social networks; (2) the positive co-variation between the slope of depressive symptoms and the intercept of inclusion in social networks (β= 0.034, 95% CI -0.005 to -0.063), meaning that the variation in depressive symptoms between phases directly affects the levels of inclusion in social networks at each phase; (3) the positive co-variation between the intercept of depressive symptoms and the slope of inclusion in social networks (β= 0.068, 95% CI 0.042 to 0.094), meaning that the intensity of depressive symptoms at baseline directly affects the levels of inclusion in social networks is subsequent years; and (4) the negative correlation between the slope of depressive symptoms and the slope of inclusion in social networks (β= -0.015, 95% CI -0.02 to -0.001), meaning that the greater the changes in depressive symptoms, the smaller the changes in inclusion in social networks between phases (Figure 7.3.6). However,

overall fit statistics revealed this model to be a not particularly good one as the root mean square error of approximation statistics was close to one (RMSEA = 0.090; Table 7.3.1).

Figure 7.3.6 *LGCM representing the intercept and slope of inclusion in social networks then used to model the intercept and slope of depressive symptoms (sample size 10299).*



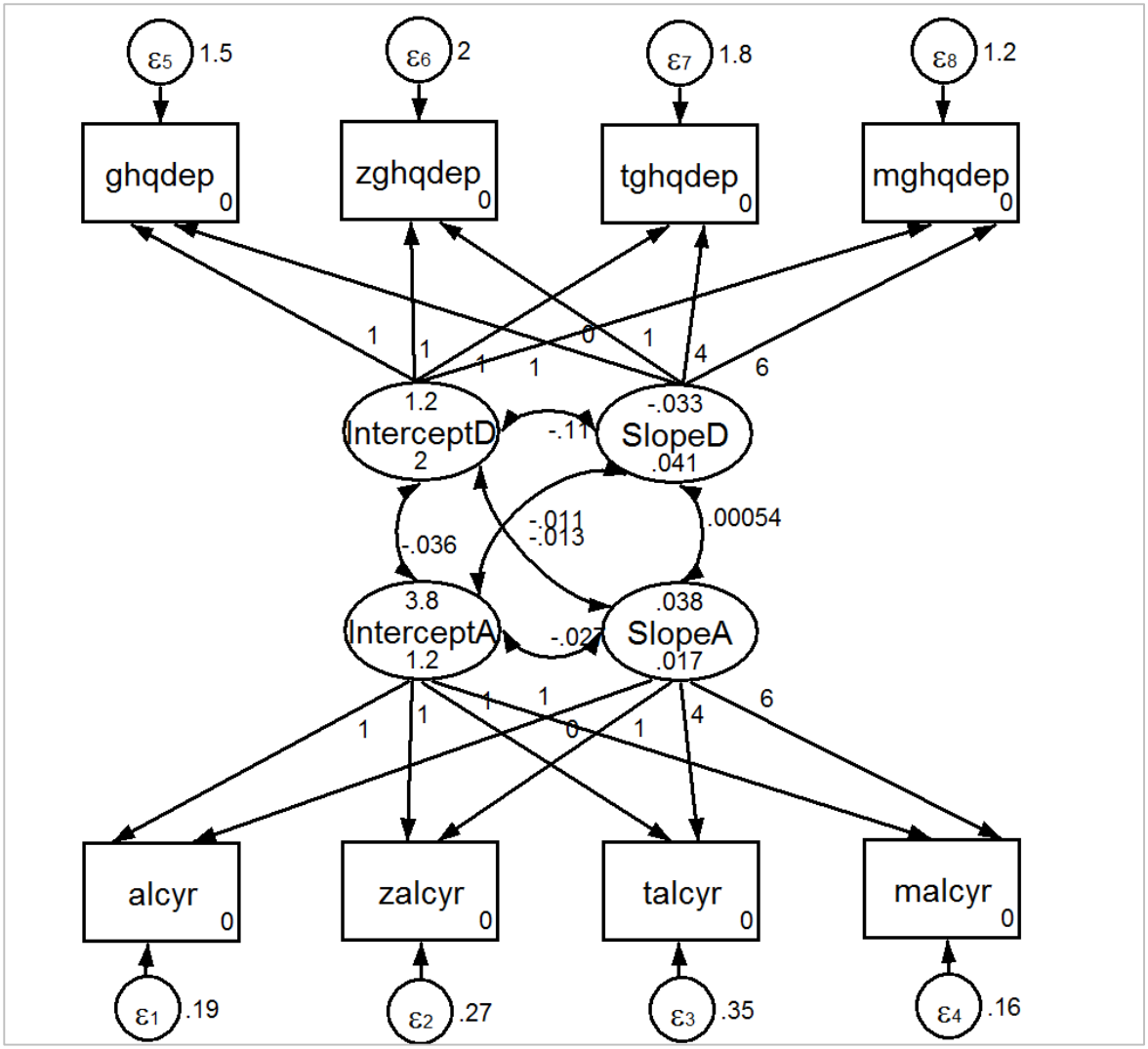
7.3.6 Alcohol Consumption and Depressive Symptoms

The third parallel growth model here presented allowed the simpler models for the growth of depressive symptoms and the growth of alcohol consumption to covariate. As such the model is composed of the intercepts and slopes for alcohol consumption and depressive symptoms already presented in sections 7.3.1 and 7.3.4, which remain unvaried, and the two coefficients β generated by allowing the two curves to covariate, which also remained unvaried from Model 1 and Model 4. This model was fitted on a sample of 10307 observations (7.3.7), and revealed how frequency of alcohol consumption was indeed associated with depressive symptoms.

The remaining four β coefficients in this model highlighted: (1) the negative but not statistically significant correlation between the intercept for depressive symptoms and the intercept of alcohol consumption (β= -0.036, 95% CI -0.076 to 0.004), meaning that the intensity of depressive symptoms was inversely correlated with the levels of alcohol consumption, so that the more intense the symptoms the lower the frequency of alcohol consumption; (2) the negative correlation between the slope of depressive symptoms and the intercept of alcohol consumption (β= -0.011, 95% CI -0.019 to -0.002), meaning that the variation in depressive symptoms between phases negatively affects the levels of alcohol consumption at each phase; (3) the negative correlation between the intercept of depressive symptoms and the slope of alcohol consumption (β= -0.013, 95% CI -0.019 to -0.006), meaning that people affected by more intense depressive symptoms at baseline were less likely to alter their alcohol consumption between phases; (4) the negative, but not statistically significant, correlation between the slope of depressive symptoms and the

slope of alcohol consumption (β= 0.001, 95% CI -0.002 to 0.000), meaning that the greater the changes in depressive symptoms, the smaller the changes in alcohol consumption between phases, even if only slightly so (Figure 7.3.7).

Figure 7.3.7 *LGCM representing the intercept and slope of alcohol consumption then used to model the intercept and slope of depressive symptoms (sample size 10307).*



7.3.7 Social Support, Inclusion in Social Networks, Alcohol Consumption and Depressive Symptoms

The final and most complex parallel growth model was designed to model the growth curves of social support, inclusion in social networks, alcohol consumption and depressive symptoms, at the same time allowing the Intercepts and Slopes of all four factors to co-vary with each other. Once fitted, on a sample of 10308 observations, the model revealed a complex pattern of association, which is described below and illustrated in Figure 7.3.8. The statistically significant correlations resulting from this model are discussed below.

First, the Intercept(S) of growth in social support was positively correlated with the Intercept(N) of change in inclusion in social networks (β = 1.75, 95% CI 1.46 to 2.06), but negatively correlated with the Slope(N) of inclusion in social networks (β= -0.097, 95% CI -0.153 to -0.042). This means that at baseline, individuals who enjoyed high levels of social support also experienced high levels of inclusion in social networks, but that as time went by, great changes in social support were associated with small changes in inclusion in social networks. The Intercept(S) of growth in social support was also negatively correlated with the Intercept(D) of depressive symptoms (β= -0.67, 96%CI -1.10 to -0.83) and positively correlated with the Slope(D) of depressive symptoms (β=0.09, 95% CI -0.06 to -0.12). Which means that at baseline, individuals who enjoyed high levels of support were also very little affected by depressive symptoms and, as time went by, changes in social support were mirrored by changes in depressive symptoms. Finally, the Intercept(S) of social support was positively correlated with the Intercept(A)for alcohol consumption (β= 0.13, 95%CI 0.04 to

0.22).This means that at baseline individuals who were highly supported were more likely to indulge in frequent drinking.

Further, the Slope(S) of growth in social support was positively correlated with the Slope(N) of change in inclusion in social networks (β= 0.02, 95% CI 0.01 to 0.03), positively correlated with the Intercept(D) of depressive symptoms (β=0.03, 95%CI 0.01 to 0.06), and negatively correlated with the Slope(D) of change in depressive symptoms (β= -0.02, 95%CI -0.03 to -0.02). This means that individuals who underwent great changes in the levels of social support over time also suffered from intense depressive symptoms at baseline, experienced great changes in inclusion in social networks, and underwent small changes in their depressive symptoms over time.

Secondly, the Intercept(N) of the growth curve of inclusion in social networks in time was negatively correlated with the Intercept(D) of growth in depressive symptoms (β= -0.99, 95% -1.13 to -0.86), positively correlated with the Slope(D) of depressive symptoms (β= 0.03, 95%CI 0.00 to 0.06), and positively correlated with the Intercept(A) of growth in alcohol consumption (β= 0.37, 95% CI 0.28 to 0.47). In other words, individuals who led a more social life at baseline also reported very low levels or no depressive symptoms, indulged in frequent drinking and underwent great changes in depressive symptoms over time.

Further, the Slope(N) of the curve of change in inclusion in social networks over time was positively correlated with the Intercept(D) of change in depressive symptoms (β= 0.0, 95% CI -0.04 to 0.09), negatively associated with the Slope(D) of depressive symptoms (β= -0.01,

95%CI -0.02 to -0.01), and negatively associated with the Intercept(A) for alcohol consumption (β= -0.05, 95% CI -0.07 to -0.03). This means that individuals who underwent great changes in inclusion in social networks through time were less likely to experience changes in their mental health, less likely to have consumed alcohol frequently at baseline and more likely to have experienced little depressive symptoms at baseline.

Finally, the Intercept(D) for the growth curve of depressive symptoms was negatively correlated with the Slope(A) of the growth curve of alcohol consumption (β= -0.01, 95% CI -0.02 to -0.01), while the estimated Slope(D) of the curve of change in depressive symptoms through time was negatively associated with the Intercept(A) of alcohol consumption (β= -0.01, 95% CI -0.02 to -0.00). This means that individuals who suffered from strong depressive symptoms at baseline were less likely to change their alcohol consumption over time, while individuals who underwent great changes in depressive symptoms over time were less likely to have drunk frequently at baseline.

Figure 7.3.8 *LGCM representing how individual variation through time in alcohol consumption, social support, inclusion in social networks or depressive symptoms can affect the growth of any of these factors (sample size 10308).*

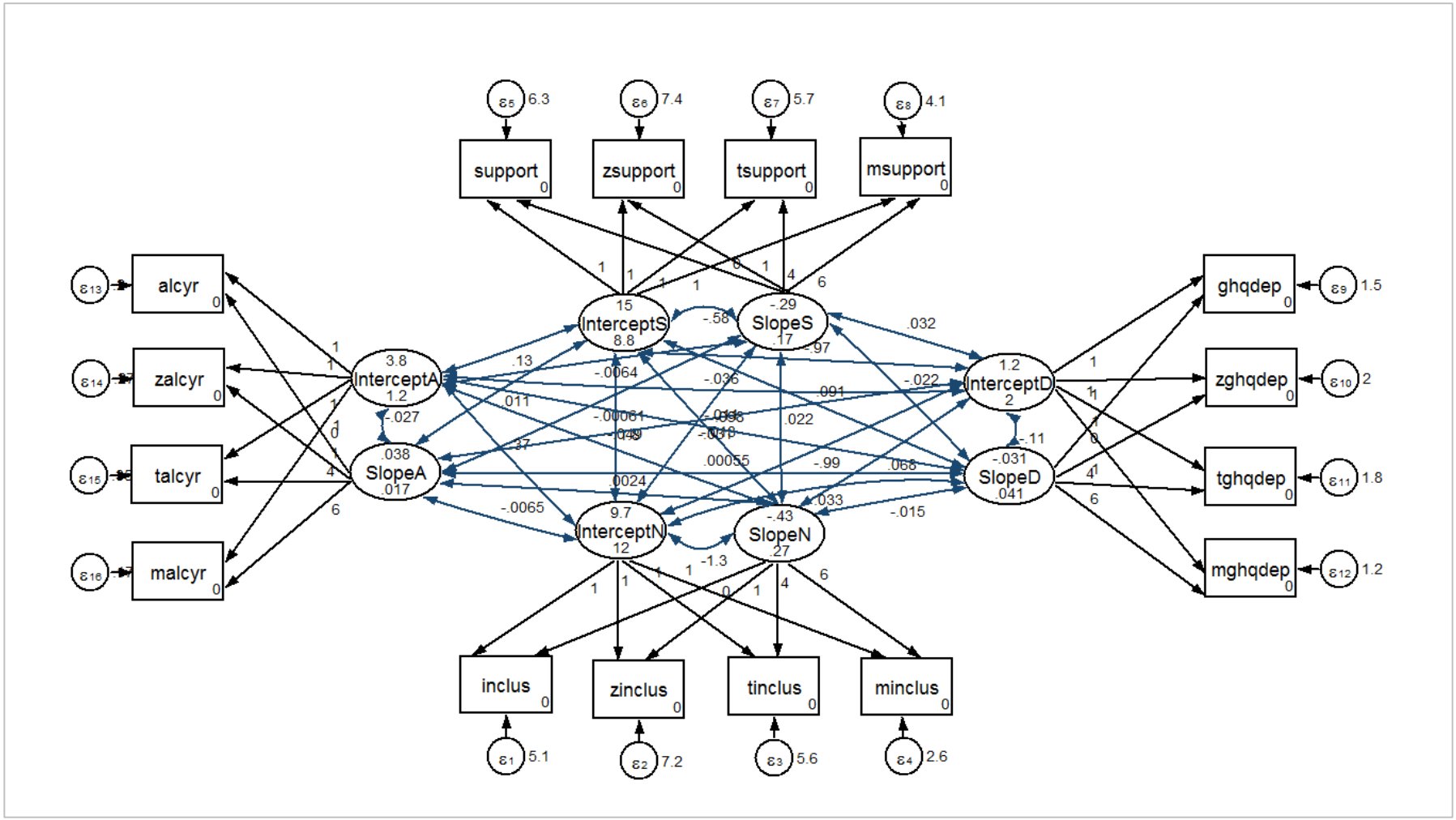


Table 7.3.1. *Goodness of fit statistics for LGC models.*

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
Fit Statistics								
X2 (df)	69.084 (5)	595.921 (5)	1804.785 (5)	169.490 (5)	693.829 (23)	1946.08 (23)	268.95 (23)	2886.9(98)
RMSEA[1]	0.035	0.109	0.187	0.057	0.053	0.090	0.032	0.053
AIC[2]	124738.297	150947.354	166623.429	81371.553	275314.997	291008.521	206087.975	522651.012
BIC[3]	142803.446	151012.126	166688.573	81436.716	275467.019	291160.557	206240.027	523042.009
CFI[4]	0.991	0.918	0.818	0.993	0.953	0.887	0.992	0.942
TLI[5]	0.989	0.901	0.781	0.991	0.943	0.863	0.990	0.929

Df: degrees of freedom; RMSEA: root mean square error of approximation; AIC: Akaike information criterion; BIC: Bayesian information criterion; CFI: comparative fit index; TLI = Tucker-Lewis index.

[1] RMSEA: the closer to 0 the better the fit, the higher the value the worst the fit of the model
[2] AIC: the minimum value is preferred as better fit
[3] BIC: the minimum value is usually preferred as the better fit
[4] CFI: a model scoring 0.90 or greater is considered a good fit
[5] TLI: a model scoring 0.95 or higher is considered a good fit

7.4 Summary

This chapter set out to investigate whether the developmental trajectories of change in social support, inclusion in social networks and alcohol consumption were associated with the developmental trajectories of depressive symptoms through time. More specifically, this chapter addressed whether individual variation in depressive symptoms is associated with individual variation in social support, inclusion in social networks or alcohol consumption through time and whether social support, inclusion in social networks and alcohol consumption would affect each other and their relationship with depressive symptoms through time.

In order to address these research questions, eight latent growth curve models were fitted. The first four simply modelled the developmental trajectories of change in each of the four factors under study individually. The fifth, addressed the association between variation in social support and variation in depressive symptoms; the sixth modelled the co-variation in change between inclusion in social networks and depressive symptoms; the seventh focused on the association between alcohol consumption and depressive symptoms; and the last model addressed the question of whether social support, inclusion in social networks and alcohol consumption affect each other in their association with depressive symptoms.

As expected, the parallel modelling of the two growth curves revealed a strong association between social support and depressive symptoms. In fact: at baseline participants who suffered from more intense depressive symptoms were also poorly supported; individuals who underwent greater variation in depressive symptoms between phases enjoyed higher levels of support at each phase; and great variation in depressive symptoms through was associated with little variation in social support.

A similar pattern of association was found between inclusion in social networks and depressive symptoms, as participants who suffered from more severe symptoms of depression at baseline were also more likely to be more socially isolated at the time but also to undergo greater changes in inclusion in social networks over time. Further, co-variation between the growth curve of depressive symptoms and the growth curve of alcohol consumption revealed that participants who at baseline suffered from intense depressive symptoms were more likely to undergo only small changes in their drinking habits over time, while individuals who experienced great variation in depressive symptoms were less likely to drink frequently at each phase.

The analysis presented in this chapter has several novel features that set it apart from the existing literature on the topic. Firstly, here for the first time the growth curves of social support, inclusion in social networks, alcohol consumption and depressive symptoms were modelled in parallel and allowed to co-vary with each other. In fact, Model 5 allowed the curves of social support and depressive symptoms to co-vary with each other in order to in order to detect which one would have a stronger effect on the other. Results showed that

indeed it was intensity of depressive symptoms at baseline and magnitude of variation in depressive symptoms to affect both levels of social support at baseline and variation in social support in time. Similar results were observed when the growth curves of inclusion in social networks and alcohol consumption were modelled in parallel to the growth curve in depressive symptoms.

The second novel feature of the present analysis is the last and more complicated growth model. Here, for the first time, the growth curves of social support, inclusion in social networks and alcohol consumption were allowed to co-vary with each other as well as with the growth curve depressive symptoms. This was done in order to establish whether the relationship between each of the three factors and depressive symptoms would be affected by the presence of the other two factors. Indeed, when inclusion in social networks and alcohol consumption were added to the model, the association between social support and depressive symptoms changed and it was baseline levels of social support and variation in social support between phases to affect levels and variation in depressive symptoms over time. A similar change was observed in the association between inclusion in social networks and depressive symptoms, for now, it was initial levels and between levels variation in inclusion in social networks to affect depressive symptoms. On the other hand, the association between alcohol consumption and depressive symptoms remained unvaried.

These results are not entirely unexpected as, since social support is one of the pathways through which inclusion in social networks affect health, it could be that the two factors operate as a buffer against depressive symptoms only when acting together. In other

words, depressive symptoms resulted to be affecting levels and variation in social support because social support was modelled independently of inclusion in social networks. Conversely, depressive symptoms resulted to be affecting levels and variation in inclusion in social networks because they were modelled independently of the effects of social support.

8. Discussion and Conclusion

8

8.1 Introduction

This research set off to provide answers to some of the questions still unanswered in the existing literature about social determinants of depression. For, as prevalence and burden of depression keep growing the world over, accounting for 8.3% and 13.0 % (Ustun, Ayuso-Mateos et al. 2004) of years lost to disability in men and women respectively, prescription and consumption of antidepressants still provide an improvement for only 50% to 55% of patients with severe depression and no help for patients with mild or medium depression (NHS 2013). The burden and cost of depression are so high, that prevention of this disorder has come to be on top of the public health agenda of the European Union, in order to not only improve the life of mental patients but also to reduce the exorbitant cost of antidepressants in Europe (Sobocki, Jonosson et al. 2006; Saxena 2011; Wahlbeck, Anderson et al. 2011). The first step towards preventing depression is collecting evidence on social factors that either increase or reduce the risk of depression, in order to draft informed and effective policies. Three such factors are social support, inclusion in social networks and alcohol consumption (Cohen and Wills 1985; Cohen 2004; Fergusson, Boden et al. 2009; Boden and Fergusson 2011; Thoits 2011).

In fact, during the past thirty years a vast body of literature has provided evidence supporting the beneficial role of positive social support, both emotional and practical, and deleterious effects of the negative aspects of support on mental health (Cohen and Wills

1985; Berkman, Glass et al. 2000).This model was then extended, including the positive effects of being included in a social networks which provides individuals with social control, behavioural guidance, sense of identity and purpose as well, self-esteem, companionship and sense of belonging, as well as with social support (Berkman and Glass 2000; Cohen 2004; Thoits 2011). Conversely, there is evidence that more socially isolated individuals, because they do not enjoy all the benefits of social life, are more at risk of depression (Brugha, Weich et al. 2005; Chan and Lee 2006). However, there is also evidence to support the fact that individuals who suffer from depression are more likely to perceive more negative aspects of support and isolate themselves more from their social life because of the nature of the disorder (Segrin, Powell et al. 2003; Maher, Mora et al. 2006; Lasgaard, Goossens et al. 2011). Hence, the question remains of whether it is actually social isolation and lack of positive support that increases the risk of depression or whether it could be depression that plays a role in isolating patients from their social networks.

In the same fashion, alcohol consumption has been proven to be associated to depression in different ways. Heavy alcohol consumption is in itself considered a mental disorder, and often coexists with depression, but it is unclear which triggers the other as alcohol is both a depressant and a powerful means used for self-medication by depressed patients (Boden and Fergusson 2011). Further, moderate alcohol consumption has been proven to be protective against depression, while abstention seems to be associated with increased risk of depression (Lipton 1994; Power, Rodgers et al. 1998; Caldwell, Rodgers et al. 2002; Rodgers, Parslow et al. 2007; Lucas, Windsor et al. 2010). Moreover, alcohol consumption and inclusion in social networks have been shown to influence each other as social

networks influence consumption through norms and behavioural examples and variation from the approved levels of consumption could result in social isolation, but also, as individuals might consume alcohol as a social lubricant to make more friends in new situations (Peele and Brodksy 2000; Room and Makela 2000; Rimal and Real 2005; Ahern, Galea et al. 2008). However, very little research has been carried out on the association between inclusion in social networks, social support, alcohol consumption and depression (Peirce, Frone et al. 2000; Allgower, Wardle et al. 2001) and no study to my knowledge has researched the association between social networks, social support and alcohol consumption in their effects on depression. Hence, this thesis has tried to answer the questions still pending existing in the literature and provide new evidence that could help drafting policies to prevent depression effectively. More specifically, this research had three main sets of objectives and hypotheses, which are outlined below:

8.1.1 Cross-sectional objectives and hypotheses

O1: To investigate the association between measures of inclusion in social networks and depressive symptoms.

H1: *Individuals who do not have any or never meet their friends and relatives outside the household are more likely to suffer from depressive symptoms than their more socially connected counterparts.*

O2: To investigate the association between alcohol consumption and depressive symptoms.

H2: *Both individuals who consume alcohol heavily and frequently and individuals who do not consume alcohol are more likely to suffer from depressive symptoms than moderate drinkers*.

O3: To compare patterns observed in the UK, the Czech Republic, Russia and Poland.

H3: The patterns of association between inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms would vary across countries.

O4: To test whether the associations between inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are confounded by age, gender, marital status, occupation, alcohol consumption and inclusion in social networks.

H4: *The associations between inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are not confounded by age, gender, marital status, occupation, alcohol consumption and inclusion in social networks*.

8.1.2 Longitudinal objectives and hypotheses

O5: To investigate the association between measures of social support and depressive symptoms

H5: *Individuals who receive low levels of confiding/emotional or practical support, and high levels of negative support are more likely to suffer from depressive symptoms*.

O6: To investigate the duration and magnitude of the association between inclusion in social networks, social support and depressive symptoms.

H6: *Individuals who are poorly connected to friends, relatives outside the household or other clubs and societies at Phase 1, will be more likely to be affected by depressive symptoms for many years.*

H7: *Individuals who receive low levels of confiding/emotional or practical support, and high levels of negative support at Phase 1, will be more likely to be affected by depressive symptoms for many years.*

O7: To investigate the magnitude and duration of the association between alcohol consumption and depressive symptoms.

H8: *Individuals who either consume alcohol heavily and frequently or who do not drink at all will be more likely to be affected by depressive symptoms for many years than moderate drinkers.*

O8: To test whether the associations between social support, inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are confounded by the effects of age, gender, marital status, employment grade, smoking status, physical activity, and alcohol consumption or social support and inclusion in social networks respectively.

H9: *The associations between social support, inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms are not*

*confounded by the effects of age, gender, marital status, employment grade, smoking status, physical activity, and alcohol consumption or social support and inclusion in social networks respecti*vely.

8.1.3 Temporality and Association

O9: To investigate the developmental trajectories of change in the association between social support, inclusion in social networks, alcohol consumption and depressive symptoms through time.

H10: *Individual variation in depressive symptoms through time is associated with individual variation in experienced levels of social support and inclusion in social networks.*

H11: Individual variation in depressive symptoms through time is associated with individual variation in alcohol consumption – *measured in terms of frequency of drinking sessions.*

H12: *The effects of social support and inclusion in social networks on depressive symptoms vary when the effects of alcohol consumption are taken into consideration and vice versa.*

This chapter will present a summary of the results obtained through statistical analysis of data coming from the Whitehall II and HAPIEE cohort studies (section 8.2) providing answers to the above objectives and hypotheses; followed by a discussion of the limitations of this study and of the problems encountered during analysis (section 8.3), followed by a discussion of how the present results compare with and complete the existing literature on the topic (section 8.4), followed by policy recommendations based on the evidence gathered in the analysis (section 8.5), and a conclusion (section 8.6).

8.2 Summary of results

8.2.1 International Comparison

In Chapter 5 of this thesis I presented the results of cross-sectional regression analysis carried out on data coming from baseline measurements of both the Whitehall II and HAPIEE cohort studies. Such cross-sectional analysis was designed to investigate the patterns of associations between frequency of contact with friends or relatives living outside the household and depressive symptoms; and between alcohol consumption and depressive symptoms in four very different European countries. In particular, the analysis focused on testing two main hypotheses: first, that more socially isolated individuals, who therefore did not have any or never visited their friends and relatives, would be more likely to suffer from depressive symptoms than their more sociable counterparts; and secondly, that both individuals who did not consume alcohol at all and individuals who consumed heavily and frequently would more likely suffer from depressive symptoms than those who consumed in moderation. Further, this cross-sectional analysis aimed at providing a comparison of patterns observed in different countries. Finally, given that the two cohort studies used different scales to measure depressive symptoms, Chapter 5 offered a comparison of the two scales using data coming from Phase 7 of the Whitehall II cohort study, where both scales were used to detect depressive symptoms. The comparison showed how although the odds ratios yielded by the CES-D scale and GHQ were slightly different, they were still comparable, apart from the odds ratios for depressive symptoms according to measures of alcohol consumption.

Results revealed that indeed individuals who reported not having any or never seeing their friends or relatives were more likely to be suffering from depressive symptoms in all four countries under study, regardless of their age, gender, marital status, occupation or drinking habits. And similarities across countries were found regarding alcohol consumption as well, as in the Czech Republic, Russia and Poland individuals who reported never drinking, and women who consumed high amounts of alcohol, were more likely to be suffering from depressive symptoms than their more moderate counterparts. The UK was the only country to stand out as the association between alcohol consumption and depressive symptoms was not statistically significant. These results lead to think that while social isolation is universally deleterious for mental health, the association between alcohol consumption and depressive symptoms might be influenced by country specific cultural norms regulating drinking.

8.2.2 Social Support, Inclusion in Social Networks, Alcohol Consumption and Depressive Symptoms in the Whitehall II cohort

The primary scope of Chapter 6 was to investigate the duration of the effects of social support, inclusion in social networks and alcohol consumption on depressive symptoms. Its secondary scope was to attempt to address the issue of temporality in these associations; and the third was to assess whether the relationships between social support, inclusion in social networks, alcohol consumption and depressive symptoms were affected by age, sex, marital status, employment grade, smoking status and physical activity and whether social support, inclusion in social networks and alcohol consumption might also play a role in each other's associations with depressive symptoms.

Results showed a strong cross-sectional association between measures of confiding/emotional, practical and negative support and depressive symptoms, with participants who reported receiving inadequate levels of confiding/emotional or practical or high levels of negative support having higher odds of suffering from depressive symptoms. This association was so strong that participants who experienced inadequate levels of confiding/emotional, practical and negative support or high levels of negative support at baseline still had higher odds of depressive symptoms at Phase 2 and Phase 5. However, when addressing the issue of temporality by using depressive symptoms at baseline to predict subsequent odds of receiving inadequate confiding/emotional or practical support, and of experiencing negative social relationships later in life, depressive symptoms turned out to be a strong predictor. Hence it was, at this stage, impossible to reliably infer temporality in the association.

Similarly, participants who at baseline reported being little involved in clubs and societies or being scarcely in contact with their friends and extended families had high odds of depressive symptoms not only at baseline itself but also at Phase 2, Phase 5 and Phase 7. Thus suggesting that social isolation could have a deleterious effect on mental health that could last up to nineteen years. However, depressive symptoms at baseline turned out to be a strong predictor of social isolation through time all the way to Phase 7 as well; again making it impossible at this stage to infer temporality in the association between inclusion in social networks and depressive symptoms.

Investigating the links between alcohol consumption and depressive symptoms yielded unexpected and interesting results. First, if the cross-sectional association between the two presented in Chapter 5, was weak and hardly significant, the odds of suffering from depressive symptoms at Phases 2, 5 and 7 were between 1.28 and 1.32 times higher among participants who reported drinking hazardously frequently at baseline. Thus, suggesting a possible delayed effect of risky alcohol consumption on mental health. Odds of depressive symptoms at Phase 5 and 7, were also high among participants who reported not drinking at baseline, resembling the theorised U shaped association between alcohol consumption and mental health. This U shape was observable in the relation between amount of alcohol consumed per week, but only at Phase 5, when highest odds of depressive symptoms were found among participants who did not consume or consumed heavily at baseline.

Secondly, when using depressive symptoms at baseline to predict subsequent hazardous consumption the association completely lost in statistical significance. Which is surprising, as a wide body of literature supports the self-medication hypothesis; that is heavy alcohol is seen as a result of an attempt to self-medicate from individuals who suffer from poor mental health. And finally, the odds of depressive symptoms according through alcohol consumption through time appeared to be strongly influenced by the effects of social support and inclusion in social networks thus leading to think there might be a possible association; that is alcohol consumption could affect depressive symptoms differently according to the levels of social support and social inclusion an individual is experiencing.

8.2.3 Trajectories of change in Social Support, Inclusion in Social Networks, Alcohol Consumption and Depressive Symptoms

The scope of Chapter 7 was to investigate the association between developmental trajectories of change in social support, inclusion in social networks and alcohol consumption with the developmental trajectory of change in depressive symptoms over time. In particular, three parallel growth curve models were fitted to address the question of whether the developmental trajectories of change in social support, inclusion in social networks and alcohol consumption are associated with the trajectory of change in depressive symptoms. A fourth model was fitted to address whether the curves of change in social support, inclusion in social networks and alcohol consumption affected each other in their relationship with depressive symptoms.

The parallel growth models revealed a correlation between change in depressive symptoms over time and change in social support, inclusion in social networks and alcohol consumption. In fact, the intensity of depressive symptoms was directly correlated with both social support and inclusion in social networks, as participants who suffered the most from depressive symptoms were found to be the least supported and the more socially isolated. However, changes in the condition of persons suffering from depressive symptoms were not readily followed in changes in their social situation as great variation in the former were accompanied by small variations in the latter. Moreover, persons experiencing strong depressive symptoms were more likely to drink sporadically and participants who consumed alcohol at least daily at baseline were less likely to undergo great changes in their symptoms of depression.

When a model was fitted allowing social support, inclusion in social networks, alcohol consumption and depressive symptoms all to affect each other through time an interesting pattern of association appeared. In fact, while when modelled individually depressive symptoms affected both initial levels and between phases variation of social support and inclusion in social networks, when all factors were included in the model it was social support and inclusion in social networks to affect levels and variation of depressive symptoms. This is likely to be due to the fact that social support and inclusion in social networks are highly interconnected, with each boosting the effect of the other on mental health. The association between alcohol consumption and depressive symptoms, on the other hand, remained unvaried thus suggesting a direct effect of depressive symptoms on frequency of alcohol consumption, regardless of the levels of social support or inclusion in social networks experienced by individuals.

8.3 Methodological Issues

Even though this research has provided new and meaningful insights into the associations between social support, inclusion in social networks and depressive symptoms and between alcohol consumption and depressive symptoms, there were a number of methodological issues which deserve some discussion. These issues broadly fall into two categories: (1) issues related to the section of the population sampled by the two cohort studies; (2) issues related to the measures used in two studies, and in particular to the epidemiological tools used to measure depressive symptoms. Both types of issues will be addressed below.

8.3.1 Methodological issues in the cohorts

The statistical analysis presented in this thesis was performed on data coming from the Whitehall II and the HAPIEE prospective cohort studies, both of which recruited their participants among the adult population of major cities. Thus, the population samples here studied are not representative of the whole population of the UK, the Czech Republic, Russia or Poland, as the rural part of the populations of these countries might significantly differ in socio-economic circumstances and patterns of inclusion in social networks or of alcohol consumption. Further, participants of the Whitehall II cohort study were recruited among civil servants because the strong hierarchical structure of the civil service is thought to reflect the class system of the UK (Marmot and Brunner 2005). However, the civil service is a secure work environment that rewards its employees with bonuses and support, and

thus does not reflect the more precarious and stressful job circumstances which many inhabitants of London might find with different employers.

A further issue of the two datasets that needs to be addressed is the problem of non-response. In fact, the original target population for the Whitehall II cohort study was all men and women aged 35 to 55, working in twenty different departments of the civil service as of 1985. Participants were invited by letter, and the response rate was 73% (74% among men and 71% among women) yielding a total sample size of 10,308. Response rates were higher among the highest grades (81%) than they were in the lowest grades of the civil service (68%). However, the true response rates might have been higher, as 4% of those on the list of employees had either moved prior to the beginning of the study or were not eligible (Marmot, Davey Smith et al. 1991). The fact that there was such a difference in response between higher and lower grades of employment may reflect a tendency among lower-grade employees to be unwilling to take part in the study for reasons that could potentially include health issues, in which case there would be a bias in the sample towards healthier individuals, the so called 'healthy volunteer bias' that needs to be kept in mind when interpreting these results.

In addition to the original potential 'healthy volunteer' bias, during the course of the nineteen years that separate the beginning of data collection in 1985 from the end of collection of Phase 7 in 2004, many participants either did not respond to the letters of one or several phases, or dropped out of the Whitehall II cohort study all together. So much so, that of the 10,308 original participants, 79% took part in Phase 2, 76% took part in Phase 5,

and 70% took part in Phase 7. The reasons behind this pattern are manifolds. First, a proportion of participants left the study each year because of death. However, mortality among individuals who did not respond to a particular phase or dropped out of the study was followed, and Jane Ferrie and colleagues (2009) found mortality rates to be double among participants who did not respond to baseline measurements or to phases that involved a clinical examination. Further, individuals who failed to respond to one or two phases but completed the last one before they died had a 38% excess risk of mortality, and individuals who failed to take part in the last phase before their death as well as one or two previously had a 127% excess mortality risk (Ferrie, Kivimaki et al. 2009).

In addition, among those who failed to respond to a particular phase, a greater probability of not-response was found among men, older participants, individuals employed in the lower grades of the civil service, participants who did not own a home, did not have a long standing illness, had higher levels of education and were still employed. Interestingly, married women were also more likely to non-respond (Mein, Johal et al. 2012). The above characteristics were also found to be associated with greater probability of withdrawal, with the notable addition of participants who reported taking part in fewer social activities (Mein, Johal et al. 2012). This could mean that the sample here under study was biased towards participants who were indeed more socially connected. The issue of missing data was addressed through multiple imputation (Chapter 6) and maximum likelihood (Chapter 7), the two most accurate techniques of estimating the values of missing data. Both these techniques are based on the assumption that data is missing at random, which as discussed above might not entirely be the case in the Whitehall II sample. However, it has been

postulated that if departures from missingness at random are small enough they will not cause serious bias in the resulting estimates (Schafer and Graham, 2002).

The HAPIEE cohort encountered similar issues regarding non-response to the invitation to take part in the study. In fact, the cohort was originally designed to include 10,000 participants from each of the three countries involved for a total of 30,000 participants. In order to achieve the planned sample size, letters were sent to high number of people in each country, but only 59% of them were answered on average. More specifically, the response rate was 61% in Poland and Russia, and 55% in the Czech Republic. However, in all urban centres involved in the study, small questionnaires were collected from those who had refused participation and the information thus obtained allowed to compare participants with non-respondents (Peasey, Bobak et al. 2006). Two main patterns appeared from this comparison. First: a great proportion of non-respondents had either moved or died before the start of the study and were therefore non eligible, so the actual response rate is likely to have been higher, in fact close 68% in Krakow, at least 71% in Novosibirsk and over 60% in the Czech Republic; and second: participation rates were lower among men, younger individuals and persons with lower levels of education, worse self-rated health and higher prevalence of smoking. Hence, in the HAPIEE study too there is evidence to support a 'healthy volunteer' bias (Peasey, Bobak et al. 2006).

8.3.2 Issues in measurements

A different type of issues intrinsic in the data analysed in this thesis reside in the tools used to measure inclusion in social networks, social support, alcohol consumption and depressive symptoms in both the Whitehall II and HAPIEE cohort studies. In fact, in both cohorts all these factors were measured from the answers participants gave to items in a structure questionnaire. Therefore, the self-reported nature of the data thus obtained allows the possibility of a number of biases that shall be discussed below.

In both cohort studies, inclusion in social networks was measured in terms of frequency of contact with friends and relatives living outside the household and how many of these were visited on a regular basis; in addition, in the Whitehall II study information was collected for level of participation in clubs and societies. Even though this information was self-reported, unless participants deliberately lied about their social lives, it should be fairly objective and unbiased. However, it is important to remember that participants who were included in the studies because they responded to the invitation letter, might have been more socially connected then those who failed to respond (Mein, Johal et al. 2012), thus creating a 'more socially included volunteer' bias.

Information on levels of confiding/emotional, practical or negative support was collected through the Close Person Questionnaire, an epidemiological tool validated by Stansfeld and Marmot (1992), on a random subsample of the original population of the Whitehall II cohort. The close person questionnaire is designed to assess social support received from marriage partners, up to three very close others, children and confidents not already

covered (see Chapter 4 for full questions), from the point of view of each participant answering the questionnaire. Thus, the close person questionnaire provides information on perceived support, that is to say an individual's generalised perception of everyday, mostly invisible, supportive exchanges occurring over time between the individual and members of their closest social network (Uchino 2004; Hobfoll 2009; Thoits 2011). This could potentially be an issue as depressive symptoms have been shown to influence perceived support by decreasing perception of beneficial support and increasing perception of negative aspects of relationships (Segrin, Powell et al. 2003; Maher, Mora et al. 2006). However, the fact that low levels of confiding/emotional support and high levels of negative support at baseline proved to be strong predictors of depressive symptoms nineteen years later, suggest an independent and strong effect of support on depressive symptoms.

Information on alcohol consumption was collected slightly differently in the two studies. In fact, if in both cohorts participants were asked how often did they consume alcohol in the previous twelve months; in the Whitehall II study participants were asked how many drinks of wine, spirits or beer had they consumed in the previous seven days, while in the HAPIEE study participants were asked how many drinks of wine, spirits or beer they regularly consumed per drinking session. The measures used in the analysis were then derived from the information thus collected. Despite the difference in the information on dose of alcohol consumed, both these measures allow for potential underreporting bias. Indeed, there is evidence that while frequency of alcohol consumption is a more objective measure of drinking patterns, individuals tend to underreport the amount of alcohol they consume

whether because they are afraid of judgement in admitting drinking more than sociably acceptable, or because they simply cannot remember the exact amount they have consumed (Boniface and Shelton 2013).

Finally, there are limitations with the scales used to measure depressive symptoms in the two cohorts. In fact, the CES-D scale is a widely internationally used and extensively validated scale, but it is not a perfect instrument. As Mulrow and colleagues (1995) have pointed out, the low specificity of the scale makes it perfect for measuring depressive symptoms and minor depression, but not very precise for detecting clinical depression (Mulrow, Williams et al. 1995; Bobak, Pikhart et al. 2006; Nicholson, Pikhart et al. 2008; Head, Stansfeld et al. 2013). The conventional cut-off point of scores sixteen and above used in this study is therefore more a measure of depressives symptoms, minor distress states, anxiety disorders or individual negativity than of the clinical disease (Nicholson, Pikhart et al. 2008). Furthermore, the CES-D scale has previously been successfully used in Poland (Dojka, Gorkiewicz et al. 2003), Russia (Andriushchencko, Drobizhev et al. 2003) and the Czech Republic (Oseka 1999); however, to my knowledge, the translations here used were never formally validated in Russia (Bobak, Pikhart et al. 2006; Nicholson, Pikhart et al. 2008). In theory this could represent an issue in that the Russians might report depressive symptoms differently from other nationalities. However the internal consistency of the CES-D scale and the similarity of the distribution of depressive symptoms in the three countries, suggest that this is unlikely to be the case (Bobak, Pikhart et al. 2006; Nicholson, Pikhart et al. 2008).

The GHQ depression subscale used in the Whitehall cohort study is yet a different tool. In fact, while the CES-D scale was designed specifically to detect depressive symptoms in the general population, the GHQ scale was created to quantify the risk of psychiatric disorders in general by assessing the ability, or indeed inability, to carry out normal tasks and the appearance of distress (Goldberg 1972; Goldberg and Hiller 1979). In addition, while the GHQ scale was compared to the CISR depression scale used in Phase 11 of the Whitehall II cohort study, it has never been formally compared to the CES-D scale.

8.4 Comparison with the literature

The scope of the analysis presented in this thesis was to attempt to provide answers to some of the issues still unresolved in the existing literature on the topic of social support, inclusion in social networks, alcohol consumption and depressive symptoms and their complex relationship. Particularly, I addressed the issue of cultural variation in the association between inclusion in social networks, alcohol consumption and depressive symptoms by analysing and comparing data from four different European countries. Secondly, I addressed the issue of magnitude and duration of the effects exerted by social support, inclusion in social networks and alcohol consumption on depressive symptoms through time, with an investigation of the effects that suffering from depressive symptoms might have on an individual's levels of perceived support, social inclusion and alcohol consumption. And finally, I addressed the issue of how social support and especially inclusion in social networks, might interact with alcohol consumption to affect depressive symptoms in a different way from that they would individually. The results obtained from these statistical investigations were partly to be expected on the basis of previous existing literature, partly surprising in view of the existing literature, and partly new and meaningful insights into those areas still unaddressed by the literature. In this section I will report and discuss how the results presented in this thesis compare with the existing literature, and how do they offer new and valuable basis for informed policies aimed at preventing depressive symptoms.

8.4.1 Cultural variation

In recent years great efforts have been deployed to map the prevalence and burden of depressive symptoms in the world. A large number of studies, both independently and as part of the Global Burden of Disease Project or the Mental Health Survey, have been devoted to collect data on depressive symptoms in a range of different countries (Skapinakis and Lewis 2001; Weich and Araya 2004; Bromet, Andrade et al. 2011; Ferrari, Somerville et al. 2013; Whiteford, Degenhardt et al. 2013). However, the information thus collected presented a rather fragmented picture of the distribution of depressive symptoms as many of the studies on the topic focused on different aspects of prevalence and burden of the disorder and measured them with different scales (Weich and Araya 2004). For example: Bromet and colleagues (2011), investigated on the 12-month prevalence of major depressive episodes (MDE) as measured with the World Health Organization Composite International Diagnostic Interview (CIDI), which is designed to detect the symptoms of eight different syndromes (Kessler, Andrews et al. 2006; Croezen, Peasey et al. 2011); while Whiteford and colleagues (2013) focused on the burden of mental and substance use disorders in general, measuring the global Disability Adjusted Life Years (DALYs) due to depressive symptoms as well as to other disorders; and Paykel and colleagues (2005), reviewed the existing literature on the 12-month prevalence of depressive symptoms in Western Europe, including in their review only studies that had used the CIDI scale. A more recent meta-analysis of existing literature by Ferrari and colleagues (2013) aimed at reviewing evidence on both 12-month prevalence and burden of depressive symptoms worldwide, but it included studies that measured depressive

symptoms with a variety of epidemiological scales, using results obtained with the CIDI scale as the reference category for the meta regression (Ferrari, Somerville et al. 2013).

The data included in this thesis provide evidence for life-long prevalence of depressive symptoms, which is not often the focus of surveys or reviews as 12-month prevalence and burden seem to take prevalence in the existing literature. In addition, data here analysed from the HAPIEE cohort study provide information on prevalence of depressive symptoms in Central and Eastern Europe, which has been reported to be an area requiring further attention, as most of the studies of European data focus on Western Europe (Paykel, Brugha et al. 2005; Ferrari, Somerville et al. 2013). The analysis here presented showed that prevalence of depressive symptoms is affected by age, gender, marital status, socio-economic circumstances and indeed inclusion in social networks and alcohol consumption. For, in all three countries of Central and Eastern Europe and in the UK, depressive symptoms were more prevalent among women, older, unmarried, and in socio-economic adversity individuals, which is consistent not only with what highlighted by Ferrari and colleagues (2013) who also found a direct effect of age, gender, marital status and socio-economic circumstances on depressive symptoms in the studies they reviewed, but also with studies carried out directly on data coming from the Whitehall II and HAPIEE cohort studies. In fact, many a study based on both the Whitehall II and the HAPIEE cohort studies have reported psychiatric disorders in general and depressive symptoms in particular to be more prevalent among women, non-married individuals and participants employed in the lower grades of the civil service (Marmot, Davey Smith et al. 1991; Stansfeld and Marmot

1992; Stansfeld, Head et al. 1998; Fuhrer, Stansfeld et al. 1999; Bobak, Pikhart et al. 2006; Nicholson, Pikhart et al. 2008; Pikhartova, Chandola et al. 2009).

These similarities in prevalence of depressive symptoms were found in the two cohort studies despite the different populations they were based on and the different scales they used to measure depressive symptoms. In fact, as the two depression scales used in the HAPIEE and Whitehall II cohorts not only measure different things; the CES-D scale measuring depressive symptoms in particular, the GHQ scale measuring psychiatric disorders in general; but also had never been formally compared, they were here compared through analysis of data from Phase 7 of the Whitehall II cohort. This comparison showed how, when measured with the CES-D scale, prevalence and odds of depressive symptoms were slightly higher than when measured with GHQ scale, but the essence of the association between depressive symptoms and their predictors was unaltered. This was welcome news, as it allowed for comparison between the two cohorts, but unexpected as several studies have discussed the issues and complications stemming from the inability to compare results from two different depression scales (Skapinakis and Lewis 2001; Weich and Araya 2004).

Results of this statistical comparison of patterns of association between inclusion in social networks, alcohol consumption and depressive symptoms showed that in all four countries prevalence of depressive symptoms was higher among more socially isolated individuals, while there was some country variation in prevalence of depressive symptoms according to alcohol consumption. Logistic regression showed the same pattern, as odds of

depressive symptoms were universally higher among socially isolated individuals, while odds of depressive symptoms according to alcohol consumption showed some country and gender variation. This is consistent with the existing literature, as a vast body of evidence has linked social inclusion, or lack of thereof to depressive symptoms in a number of settings from the UK (Brugha, Conroy et al. 1982; Paykel, Brugha et al. 2005), to China (Chan and Lee 2006), to the United States (Wildes, Harkness et al. 2002), thus suggesting a universal deleterious effect of social isolation on mental health.

The literature on the effects of alcohol consumption on depressive symptoms, on the other hand, leads to expect both frequent and heavy consumption and abstention to be most deleterious for mental health, but also a cultural and gender variation. In fact, the U or J shaped association between alcohol consumption and depressive symptoms has long been established (Lipton 1994; Power, Rodgers et al. 1998; Rodgers, Korten et al. 2000; Rodgers, Korten et al. 2000; Rehm, Greenfield et al. 2001; Caldwell, Rodgers et al. 2002; Rodgers, Parslow et al. 2007; Lucas, Windsor et al. 2010), as is the notion that patterns of alcohol consumption and their effects on depressive symptoms are influenced by social norms and shared social behaviours which affects men and women differently (Ahlstrom, Bloomfield et al. 2001; Bloomfield, Gmel et al. 2001; Bloomfield, Stockwell et al. 2003; Rimal and Real 2005; Makela, Gmel et al. 2006; Rahav, Wilsnack et al. 2006; Ahern, Galea et al. 2008; Le, Ahern et al. 2010; Ahern and Galea 2011). This was partly observed in the present analysis too, as in Czech Republic, Russia and Poland, while odds of depressive symptoms were higher among men who did not consume any alcohol, they were higher among women who did not consume or consumed heavily. This could be explained with the fact that in

countries of Central and Eastern Europe, there still is a social stigma on women who consume too much alcohol as females are traditionally considered to be less prone to risky behaviours and to provide care for their partners (Ahlstrom, Bloomfield et al. 2001; Bloomfield, Gmel et al. 2001; Makela, Gmel et al. 2006; Shelton and Savell 2011). In the UK on the other hand, higher odds of depressive symptoms were found among both men and women who reported drinking heavily, thus suggesting less strong gender norms on drinking, however the number of women drinking as heavily as men was minimal thus suggesting that social norms might not influence the association between alcohol consumption and depressive symptoms but they do influence how much women drink.

8.4.2 Effects of inclusion in social networks on depressive symptoms

A large body of research has been devoted to throwing light on just what it is about social ties that is so beneficial for individual health, and mental health in particular (Landrine, Richardson et al. 1994; Berkman and Glass 2000; Berkman, Glass et al. 2000; Brissette, Cohen et al. 2000; Kawachi and Berkman 2001; Thoits 2003; Cohen 2004; Uchino 2004; Umberson and Montez 2010; Thoits 2011). Despite focusing on different aspects of social relations, the underline common finding of many of these studies seems to be that being a part of a social network goes well beyond just having a membership to a club or seeing relatives at Christmas, but involves not only regular participation in social activities with people considered affine to the self, but also the creation of a number of cognitive components that have a strong impact on mental health (Brissette, Cohen et al. 2000; Cohen 2004; Thoits 2011).

The cognitive components which are thought to derive from participation in a social network are: social influence and comparison, when individual behaviour is influenced by comparison with others in the group; social control, when members of the group actively attempt to modify an individual's behaviour; behavioural guidance, purpose and meaning, refer to the social roles that come with specific ties, such as husband, mother, friend; self-esteem, a by-product of social role identities which is influenced by individual performances in such role; sense of control or mastery, also a by-product of social role identities, is the sense of being 'on top of one's game'; sense of belonging and companionship, which the sense of acceptance, safeness and affinity that stems from being a member of a close group; and social support, which will be explored separately (Thoits

2011). These components are thought to be provided by different types of social groups individuals belong to; for example, social influence and comparison is thought to derive largely from a larger, perhaps less intimate, or secondary group such as peers, and in fact it has been shown that the strongest predictor of taking up smoking among teenagers is smoking prevalence among peers (Landrine, Richardson et al. 1994). Sense of belonging and companionship, on the other hand, is stronger when provided by a smaller, more intimate, or primary, social group which will provide support in times of crisis but perhaps more importantly companionship in day to day activities (Hagerty and Williams 1999; Nolen-Hoeksema and Harrell 2002; Cacioppo, Hughes et al. 2006).

If being part of a social network provides individuals with so many cognitive tools to maintain a balanced mental health, it has also been shown that individuals who are not part of a social network will lack the self-esteem and self-worth that stem from successfully perform a social role, the sense of self-mastery that allows to have confidence enough to overcome obstacles, and, more especially, will lack companions with whom to engage in activities thus leading to loneliness which is also deleterious for mental health (Hagerty and Williams 1999; Nolen-Hoeksema and Ahrens 2002; Heikkinen and Kauppinen 2004; Tiikkainen and Heikkinen 2005; Cacioppo, Hughes et al. 2006). Hence, the first step towards understanding how social ties affect mental health, and in this case depressive symptoms, is to establish whether individuals are involved in social networks, and if so how closely. The most common way of doing so is to measure frequency and number of contacts with friends, relatives and other networks such as clubs and societies or neighbourhood associations (Brugha, Conroy et al. 1982; Berkman, Glass et al. 2000; Wildes, Harkness et

al. 2002; Brugha, Weich et al. 2005; Chan and Lee 2006; Haines, Beggs et al. 2011; Litwin 2011; Schwarzbach, Luppa et al. 2013; Barger, Messerli-Burgy et al. 2014; van Beljouw, van Exel et al. 2014).

The results here presented are consistent with the existing literature supporting the notion that individuals with smaller social networks feel more lonely, which in turn makes them more at risk of depressive symptoms (Brugha, Weich et al. 2005; Chan and Lee 2006; Haines, Beggs et al. 2011; van Beljouw, van Exel et al. 2014). In fact, despite the variety of settings and populations under study, Brugha and colleagues (2005), Chan and Lee (2006), Haines et al (2011) and van Beljouw et al (2014) all found that less socially connected individuals were more likely to suffer from depressive symptoms than more socially integrated ones. Further, in his cross-sectional study of the structure of social networks influences depression, Howard Litwin (2011) highlighted how involvement in more socially dynamic groups such as friendship groups, clubs or societies was associated with a smaller likelihood of suffering from depressive symptoms than involvement in networks composed of members of the family such as children or distant relatives. It was beyond the scope of this thesis to investigate the levels of dynamism of a particular social network, but it is true that participants who were little involved in clubs or societies or with friends were here found to have higher odds of depressive symptoms than participants who were little in contact with their extended families.

Finally, all of the studies investigating the links between social networks and depressive symptoms have done so through cross-sectional analysis or with one follow up. To my knowledge no study to date has attempted to investigate the duration of the effects of social isolation on depressive symptoms over the course of many years. Interestingly, results of this thesis have shown how persons who are socially isolated at one point in time are more likely to suffer from depressive symptoms later in life, regardless of the structure of the networks they are involved with, and even after adjusting for background characteristics and alcohol consumption.

8.4.3 Effects of social support on depressive symptoms

Social support is one of the cognitive components of being part of a social network, but because of its strong, active influence on mental health it is also considered as a functional rather than a structural characteristic of social relations (Cohen and Wills 1985; Cohen 2004; Thoits 2011; Schwarzbach, Luppa et al. 2013) and as such, it is often investigated separately. Two main types of support are identified in the literature: confiding/emotional support, which refers to the love, empathy and understanding usually provided by close and intimate persons; and instrumental or practical support, which refers to the tangible help with practical issues that could be provided by a number of different people (Berkman, Glass et al. 2000; Grav, Hellzen et al. 2011; Thoits 2011; Schwarzbach, Luppa et al. 2013). In addition, negative support, which refers to the psychological strain of stressful aspects of and hardships in relationships, has been shown to play role in affecting mental health (Berkman and Glass 2000; Kawachi and Berkman 2001; Ibarra-Rovillard and Kuiper 2011; Litwin 2011; Schwarzbach, Luppa et al. 2013).

Of these three types of support, negative support is considered to be the one with the biggest impact on mental health, as the strain and stress that could derive from social relations has been shown to take their toll on mental health as much or perhaps even more than loneliness, as they create emotional stress (Berkman and Glass 2000; Kawachi and Berkman 2001; Ibarra-Rovillard and Kuiper 2011; Litwin 2011; Schwarzbach, Luppa et al. 2013). The cross-sectional results presented here are consistent with these observations, as participants who experienced high levels of negative support at baseline were found to

be almost three times more likely to suffer from depressive symptoms than those whose social relations were more positive and less stressful.

Confiding/ emotional support has also been theorised to have strong effect on mental health, both in the form of routine demonstration of love, caring and understanding that we receive daily from people closer to us and that contribute to form the subconscious knowledge of mattering to one another; and perhaps more importantly, in the form of allowing us to vent the little frustrations stemming from impending problems at home, at work or in other social roles , with persons who are close, understanding and caring, so as to prevent these frustrations to escalate intro major stressors (Cohen and McKay 1984; Thoits 1985; Taylor and Aspinwall 1996; Uchino 2004; Gleason, Mausmi et al. 2008; Thoits 2011). The cross-sectional results presented in this thesis show a similar pattern, as participants who reported not being able to confide in the people closer to them as much as they would have liked or needed to where twice as likely to be suffering from depressive symptoms than those who had that opportunity.

Unlike negative or confiding/emotional support which are almost always provided by very close, intimate persons, everyday help with practical issues such as baby-sitting or money landing can come from anybody in the social networks and contribute to create a sense of safeness given by the knowledge that we are not alone and in case of crisis we will have a safety net on which to fall (Bolger, Zuckerman et al. 2000; Cohen 2004; Bolger and Amarel 2007; Thoits 2011). However, this kind of support is thought to have less of an impact on mental health than negative or confiding/emotional support for although it provides relief from the stress stemming from practical issues it does not directly allow to vent worries

and concerns (Bolger, Zuckerman et al. 2000; Cohen 2004; Bolger and Amarel 2007; Thoits 2011). In fact, the cross-sectional results here presented a similar pattern, with participants who reported not receiving enough help with practical issues being more likely to be suffering from depressive symptoms than those who were fully supported, but as much as those who received inadequate confiding/emotional support or experienced unhealthy relationships.

Finally, in this thesis I have attempted something that, to my knowledge, has not been attempted to date, that is investigating the duration of the effects of support on mental health. This is important for fully understanding the association between support and mental health. Here I asked the question of, for example, for how long will a stressful relationship, such as a divorce, with somebody close will be a risk for depressive symptoms? For the first time, I was able to show that the effects of practical support had an immediate impact on depressive symptoms, but this did not last in time. Inadequate confiding/emotional support on the other hand, kept being associated with increased likelihood of depressive symptoms ten years later; and experiencing stressful relationships still had an effect fourteen years later. These findings could have great repercussions in drafting prevention policies for depression.

8.4.4 Effects of alcohol consumption on depressive symptoms

The effects of alcohol consumption on depressive symptoms have long been the subject of a vast body of literature, focusing primarily on the comorbidity of alcohol use disorders and depressive symptoms in a number of different settings (Lipton 1994; Power, Rodgers et al. 1998; Wang and Patten 2001; Holahan, Holahan et al. 2003; Rehm, Sempos et al. 2003; Holahan, Holahan et al. 2004; Fergusson, Boden et al. 2009; Boden and Fergusson 2011; Felnsborg-Madsen 2011; Boschloo, Vogelzangs et al. 2012; Bulloch, Lavorato et al. 2012; Bell and Britton 2014; Briere, Rohde et al. 2014; Skule, Dallavara Lending et al. 2014). This is because alcohol use disorders classify as substance abuse disorders which are often researched together with mood disorders, such as depressive symptoms or anxiety, as both these groups of disorders affect principally the brain and only secondarily the body (Whiteford, Degenhardt et al. 2013). In addition, alcohol use disorders can present a symptomatology very similar to that of depression and have been shown to be often comorbid with the latter as both disorders increase the risk of onset of the other (Boden and Fergusson 2011).

Indeed, if we focus on consumption of alcohol that is so heavy and frequent to induce dependency and classify as a mental disorder in its own right, an unsolved dilemma appears in the existing literature: is it alcohol use disorders that trigger depressive symptoms? Or rather, is it depressive symptoms to induce heavy consumption? Boden and Fergusson (2011) answered this question through a narrative review of the existing literature and concluded a causal link between alcohol use disorders and depressive symptoms in which the former caused the latter. However these results have since been challenged both on

theoretical and empirical grounds. In fact, Felnsborg-Madsen (2011) argued that the review in question not only was based primarily on a previous paper by the same authors but also started from the notion that comorbidity implies causality, which is a questionable assumption as the two disorders may in fact just present common symptoms, without necessarily sharing a causal link too. Further, Steven Bell and colleagues (2014) found that indeed, among British Civil Servants, mental health was the leading force behind changes in alcohol consumption; more specifically, individuals with better mental health were more likely to reduce their alcohol intake in time. A further study by Steven Bell and colleagues (2014) showed how among the urban population countries of Central and Eastern Europe hazardous drinking is associated with a two-fold increase in the odds of depressive symptoms. However, a recent Canadian study presented the opposite results, with major depression increasing the risk of alcohol abuse in the general population but not the other way around (Bulloch, Lavorato et al. 2012). The results presented in this thesis, are consistent with those presented by Bulloch and colleagues (2012) and Boden and Fergusson (2011), as excessive alcohol consumption was found to predict increased odds of depressive symptoms later in life. However, this was purely a temporal finding, as inferring causality in the association was beyond the scope of this thesis.

Furthermore, alcohol use disorders are but one extreme type of consumption, but what about the effects on depressive symptoms of light, moderate and heavy but not pathological consumption or abstention? Since the pioneering work of Lipton (1994), a vast body of literature has supported the idea of a J or U shaped association between alcohol consumption and health. In other words, light or moderate consumption has been shown

to be beneficial for mental health while both abstention and heavy consumption have been shown as detrimental. Although, the mechanisms behind this non-linear association remain unclear, several studies over the years have confirmed the U or J shaped relationship (Lipton 1994; Power, Rodgers et al. 1998; Peele and Brodksy 2000; Rodgers, Korten et al. 2000; Rodgers, Korten et al. 2000; Caldwell, Rodgers et al. 2002; Rodgers, Parslow et al. 2007; Lucas, Windsor et al. 2010; Bulloch, Lavorato et al. 2012). The results presented in this thesis are somewhat consistent with the literature, as although there was no clear association between the two measures of alcohol consumption and depressive symptoms at baseline, participants who consumed alcohol at least once a day or did not consume alcohol at all were found to be more at risk of depressive symptoms at Phase 5 and Phase 7. This U curve was observed among measures of units consumed per week, as persons who at baseline consumed 36 or more units per week or did not drink at all, also had higher odds of depressive symptoms at Phase 5. This suggests delayed effects of consumption on depressive symptoms that had not been observed before. In addition, the results presented here showed a distinct action change in the obtained odds of depressive symptoms when adjusting for measures of inclusion in social networks and of social support. This is consistent with the notion that one of the possible mechanisms behind the beneficial effects of moderate alcohol consumption would be the fact the moderate drinkers are also social drinkers and therefore better socially connected than both heavy drinkers and abstainers and thus enjoying all the beneficial effects of more and better social relations (Peele and Brodksy 2000; Peters and Stringham 2006; Lucas, Windsor et al. 2010).

8.4.5 Growth Curves

If a wide body of research has thrown light on the effects of inclusion in social networks, social support, alcohol consumption on depressive symptoms, considerably less studies have focused on the ways in which depressive symptoms might cause persons who suffer from them to be more socially isolated, feel less supported and engage in more hazardous drinking patterns (Segrin and Powell 2003; Maher and Mora 2006; Lasgaard and Gossans, 2011; Khantzian 1997; Markou, Kosten et al. 1998; Dixit and Crum 2000; Bolton, Robinson et al. 2009; Bell and Britton, 2004).

Inclusion in social networks has been identified in the literature has as a driving force in protecting against depressive symptoms (Thoits, 2011; Berkman and Glass, 2000; Berkman et all, 2000) through seven different pathways. One of the seven pathways is social support, which has been found to be so powerful in affecting both physical and mental health that it has often been studied independently of inclusion in social networks (Thoits, 2011; Cohen and Wills, 1985; Cohen, 2004). However, other studies have reported depressive symptoms to play a role in deteriorating the quality of human relationships. In fact, Segrin and colleagues (2003) investigated how depressive symptoms affected the quality of relationship in dating couples and reported that indeed as the intensity of the symptoms grew the quality of the relationship decreased and, further, loneliness increased (Segrin et al, 2003). Similarly, perceived social support and demand were found to be affected by the cognitive aspects of depressive symptoms, as the disorder causes persons affected to feel they necessitate more and more support and to perceive their needs are not met (Maher and Mora, 2006).

The result presented in this thesis draw a picture which is consistent with the existing literature, although with some variation. In fact, when social support and inclusion in social networks modelled individually against growth of depressive symptoms, the latter affected levels and variation in both factors. In other words, depressive symptoms were the driving force behind increasing social isolation decreasing levels of support over time. However when, in the final model, social support and inclusion in social support were allowed to co-vary, they were the force behind improvement in depressive symptoms. This suggests that perhaps social support and inclusion in social networks provide a successful buffer against depressive symptoms only when taken as two aspects of one powerful social determinant.

The literature highlighting the role of depressive symptoms in influencing individual alcohol consumption is somewhat more consistent. Since the late Nineties, when Edward Khantzian (1997) published his 'self-medication' hypothesis, a number of studies have confirmed that persons suffering from depressive symptoms do tend to indulge in heavy alcohol consumption as a means of self-medication, for alcohol is known to temporarily soften mental defences and ameliorate states of isolation and emptiness that are characteristics of depression (Khantzian 1997; Markou, Kosten et al. 1998; Dixit and Crum 2000; Bolton, Robinson et al. 2009).

Merely three years after Khantzian work was published, Dixit and Crum (2000) reported that North American women with a history of depression were 2.6 times more likely to engage in hazardous drinking then women who never suffered from the disorder. Further, among women who did suffer from depression, an increase in the frequency of symptoms

was associated with an increase in the risk of heavy alcohol use (Dixit and Crum 2000). Bolton and colleagues (2009) also highlighted a distinct pattern of self-medication, as 24.1% of patients with mood disorders included in their study engaged in heavy alcohol consumption, and 41.0% of self-medicating drinking occurred among patients suffering from depression (Bolton, Robinson et al. 2009). More recently, Steven Bell and colleagues (2014) examined the relationship between symptoms of depression and anxiety and alcohol consumption among participants of the Whitehall II cohort study, using parallel growth models. In fact, the models presented by Bell et al (2014) closely resemble the ones presented in this thesis depicting the co-variation between alcohol consumption and depressive symptoms. Although in this thesis, anxiety was not considered. Not surprisingly the results obtained were very similar, for in both studies depressive symptoms were found to influence alcohol consumption, as variations in intensity of depressive symptoms caused variations in frequency of alcohol consumption.

To my knowledge, no research has yet attempted to model the growth of social support, inclusion in social networks, alcohol consumption and depressive symptoms in parallel. This despite there being some evidence of strong ties linking these four factors (Pierce et al, 2000). The parallel growth model here presented highlighted a strong association between social support and depressive symptoms and inclusion in social networks and depressive symptoms, both at baseline and in their trajectory of change. This is consistent with the literature highlighting the effects of social support and inclusion in social networks on depressive symptoms (Berkman, Glass et al, 2000; Thoit, 2011). While depressive

symptoms were found to affect levels of alcohol consumption, which is consistent with the self-medication hypothesis and with what found by Bell et al (2014).

8.5 Policy Recommendations

Mental disorders, led by depression, are the plague of our time as their prevalence and burden are steadily growing. Depression is, indeed, the most common and most burdensome of mental disorders and it has been estimated to become the leading cause of disability worldwide in the next five years (Murray and Lopez 1997; Mathers and Loncar 2006). A report published by the King's Fund in 2008 (McCrone et al, 2008) revealed the estimated cost of depression in England in 2007, as follows: the average cost of services for depression for those in contact with services was £2,085 person, while the average cost of lost employment was £9,311 per person. Thus bringing the total cost of services for depression at £1.7 billion, and the total cost of loss of employment due to depression to £7.5 billion (McCrone et al, 2008).

These rather exorbitant and ever growing costs of depression could be curbed if effective preventive measures were to be put in place. Such prevention strategies will have to be based on research highlighting the social determinants responsible for increasing individual risk of depression. In this thesis I have presented new evidence corroborating the notion that inclusion in social networks and social support provide a buffer capable of reducing the odds of suffering from depressive symptoms. The can be used as a foundation for evidence based policies aimed reducing the risk of depressive symptoms in the adult population of the UK.

In fact, while it would difficult to design policies aimed at improving the quality and quantity of support experienced by an individual, it is possible to design policies aimed at

improving individuals' social connections. Once individuals will be more socially connected the natural properties of being part of a social network will enhance social support received and contribute to provide a buffer against depression.

Interventions aimed at increasing participation in social networks could take a number of forms. First, it is important to remember that many organisations promoting social interactions already exist in the UK in the form of: neighbourhood community groups, local charities, church groups, local orchestras or drama societies, sport clubs and so on. Hence, a policy aimed at increasing people's inclusion in social networks would simply need to enhance participation in these existing networks. One way of doing so would be to arrange a system of tax return if individuals can prove their membership and attendance to a local network. This however could prove complicated and expensive. A simpler way would be to provide extra funding to local organisations such as sport clubs, community groups and charities so that they will be able to provide more services and attract more people to take part in their activities.

8.6 Conclusion

Mental disorders, led by depression, are the plague of our time as their prevalence and burden are steadily growing. Depression is, indeed, the most common and most burdensome of mental disorders and it has been estimated to become the leading cause of disability worldwide in the next five years (Murray and Lopez 1997; Mathers and Loncar 2006). However, there is not yet a single fully effective treatment for depression as drugs are expensive and often lead to debilitating side effects (Whitaker 2010). Further, diagnosed cases of depression are but a fraction of the actual problem as many patients refuse to acknowledge their condition due to fear of stigma and social isolation. Hence, population surveys using bespoke scales to measure symptoms of depression usually give a better estimate of the magnitude of the issue. Therefore, this thesis set off to throw light on the association between depression and three of its main risk factors, as identified in the literature, so as to be able to provide evidence to support new strategies for prevention of depression.

Given the reluctance among patients to admit to their mental issues, epidemiological scales designed to detect depressive symptoms in the general population play a very important role in monitoring the spread of depression across countries, but these scales are very seldom formally compared so that the results produced can be quite confusing. In this thesis, two such epidemiological scales, the general health questionnaire (GHQ) and the centre for epidemiological studies depression scale (CES-D), were formally compared for the first time. This comparison revealed that the CES-D scale yields a slightly higher prevalence of depressive symptoms than the GHQ scale, however this difference did not

affect the nature of the associations between depressive symptoms, inclusion in social networks and alcohol consumption observed in the Czech Republic, Russia, Poland and the UK.

In fact, when examined at the cross-sectional level, persons who are more socially isolated, receive less confiding/emotional and practical support or high levels of negative support and indulge in heavy and frequent alcohol consumption appear to be at a higher risk of suffering from depressive symptoms. Regardless of their age, gender, marital status, socio-economic circumstances, physical activity, country of origin or of the scale used to measure depressive symptoms. This provides grounds for interventions that would focus on increasing the levels of connectivity and integration within communities so as to provide individuals with a sense of belonging and aggregation, with group norms on what levels of alcohol consumption are socially acceptable and with support both of in times of crisis and on a day to day basis, which could help preventing the onset of depression.

Longitudinal analysis on data coming from the Whitehall II cohort study showed the same pattern of association, although the effects of inclusion in social networks, social support and alcohol consumption on depressive symptoms tended to fade as time went by. However, depressive symptoms also appeared to influence individual levels of inclusion, social support and consumption through time, which raised the question of the direction of the association. In other words, could it be that persons who already suffer from depressive symptoms tend to be more socially isolated, to perceive themselves receiving inadequate support and to drink heavily in order to self-medicate?

Growth curve models revealed strong patterns of association between social support, inclusion in social networks and depressive symptoms, as individuals who were better connected and more socially supported at baseline were less likely to suffer from depressive symptoms. However, this was observed only when social support and inclusion in social networks were allowed to co-vary, hence suggesting that the effect of one or the other on its own is not enough to provide a buffer against depressive symptoms In addition, individuals who suffered from depressive symptoms were found to be more likely to engage in frequent drinking.

Finally, the results here presented could be draw upon by health practitioners and policy makers in order to implement interventions - such as community groups, local team sports, support groups, neighbourhood associations - aimed at promoting the social inclusion of individuals who are at risk of developing depressive symptoms as these could prove particularly effective in preventing depressive symptoms but also at containing hazardous alcohol consumption in the target population.

9. References

Abbagnano, N. and G. Fornero (2003). Itenerari di Filosofia. Milano, Mondadori Editori.

Ahern, J. and S. Galea (2011). "Collective Efficacy and Major Depression in Urban Neighborhoods." American Journal of Epidemiology **173**(12): 1453-1452.

Ahern, J., S. Galea, et al. (2008). ""Culture of Drinking" and Individual Problems with Alcohol Use." American Journal of Epidemiology **167**(9): 1041-1049.

Alati, R., D. Lawlor, et al. (2005). "Is there really a 'J-shaped' curve in the association between alcohol consumption and symptoms of depression and anxiety?Findings from the Mater-University Study of Pregnancy and its outcomes." Addiction(100).

Allgower, A., J. Wardle, et al. (2001). "Depressive Symptoms, Social Support, and Personal Health Behaviours in Young Men and Women." Health Psychology **20**(3): 223-227.

Angst, J. and A. Dobler-Mikola (1984). "The definition of depression." Journal of Psychiatric Research **18**: 401-406.

APA (2000). Diagnostic and statistical manual of mental disorders IV. A. P. Association, American Psychiatric Publications.

Badr, H., L. Acitelli, et al. (2001). Weaving social support and relationships together. New York, John Wiley & Sons.

Baraldi, A.N and Enders, C. K. (2010). "An introduction to modern missing data analyses." Journal of School Psychology **48**: 5-37.

Barrera, M. J. (2000). Social Support Research in Community Psychology Handbook of Community Psychology. J. Rappaport and E. Seidman. New York, Kluwer Academic/Plenum**:** 215-245.

Baumeister, R. F., J. D. Campbell, et al. (2003). "Does Self-Esteem Cause Better Performance, Interpersonal Success, Happiness, or Healthier Lifestyles?" Psychological Science in the Public Interest **4**: 1-44.

Beekman, A. T., D. J. H. Deeg, et al. (1997). "Criterion validity of the Center for Epidemiological Studies Depression scale (CES-D): results from a community sample of older subjects in the Netherlands." Psychological Medicine **27**: 231-235.

Bell, S. and A. Britton (2014). "An exploration of the dynamic longitudinal relationship between mental health and alcohol consumption: a prospective cohort study." BMC Medicine **12**(91).

Bell, S., Britton, A., Kubinova, R., Malyutina, S., Pajak, A., Nikitin, Y., & Bobak, M. (2014).Drinking Pattern, Abstention and Problem Drinking as Risk Factors for Depressive Symptoms: Evidence from Three Urban Eastern European Populations. PLoS ONE.

Bellman, S., N. Forster, et al. (2003). "Gender differences in the use of social support as a moderator of occupational stress." Stress and Health **19**: 45-58.

Berkley, S., J.-L. Bobadilla, et al. (1993). World development report 1993: investing in health. World Development Report.

Berkman, L. and T. Glass (2000). Social Integration, Social Networks, Social Support and Health. Social Epidemiology. L. Berkman and I. Kawachi. Oxford, Oxford University Press.

Berkman, L., T. Glass, et al. (2000). "From social integration to health: Durkheim in the new millennium." Social Science & Medicine **51**: 843-857.

Bloomfield, K., G. Gmel, et al. (2001). "Investigating Gender Convergence in Alcohol Consumption in Finland, Germany, The Netherlands, and Switzerland: A Repeated Survey Analysis." Substance Abuse **22**(1): 39-53.

Bloomfield, K., U. Grittner, et al. (2006). "Social Inequalities in Alcohol Consumption and Alcohol-related Problems in the Study Countries of the EU Concerted Action 'Gender, Culture and Alcohol Problems: a Multi-national Study'." Alcohol & Alcoholism **41**(1): i26-i36.

Bobak, M., H. Pikhart, et al. (1998). "Socioeconomic factors, perceived control and self-reported health in Russia. A cross-sectional survey." Social Science & Medicine **47**(2): 269-279.

Bobak, M., H. Pikhart, et al. (2006). "Depressive symptoms in urban population samples in Russia, Poland and the Czech Republic." The British Journal of Psychiatry **188**: 359-365.

Boden, J. M. and D. M. Fergusson (2011). "Alcohol and depression." Addiction **106**: 906-914.

Boffetta, P. and M. Hashibe (2006). "Alcohol and cancer." The Lancet **7**(2): 149-156.

Bolger, N. and D. Amarel (2007). "Effects of Support Visibility on Adjustment to Stress: Experimental Evidence." Journal of Personality and Social Psychology **92**: 458-475.

Bolger, N., A. Zuckerman, et al. (2000). "Invisible Support and Ajustment to Stress." Journal of Personality and Social Psychology **79**: 953-961.

Bolton, J. M., J. Robinson, et al. (2009). "Self-medication of mood disorders with alcohol and drugs in the National Epidemiologic Survey of Alcohol and Related Conditions." Journal of Affective Disorders **115**(367-375).

Bonin, M. F., D. R. McCreary, et al. (2000). "Problem drinking behaviour in two community-based samples of adults: Influence of gender, coping, loneliness, and depression." Psychology of Addictive Behaviors **14**(2): 151-161.

Borbova, N., R. West, et al. (2010). "Gender Differences in Drinking Practices in Middle Aged and Older Russians." Alcohol & Alcoholism **45**(6): 573-580.

Bordieu, P. (1986). The forms of capital. Handbook of Theory and Research for the Sociology of Education. J. Richardson. New York, Greenwood**:** 241-258.

Borsari, B. and K. B. Carey (2001). "Peer influebnces on college drinking: A review of the research." Journal of Substance Abuse **13**(4): 391-424.

Bourdieu, P. (1986). The forms of capital. Handbook of Theory and Research for the Sociology of Education. J. Richardson. New York, Greenwood**:** 241-258.

Briscoe, M. E. (1982). "Sex differences in psychological well-being." Psychological Medicine Monograph **1**: 1-46.

Britton, A. (2002). "Doeas it matter what you drink? Differential health effetcs by beverage type." Public Health Medicine **4**(2): 37-42.

Britton, A. (2006). "How much and how often should we drink? Interpret with caution new evidence on frequency and amount of men's drinking." BMJ **332**: 1224-1225.

Britton, A., M. Marmot, et al. (2009). "How does variability in alcohol consumption over time affect the relationship with mortality and coronary heart disease? ." Addiction **105**: 639-645.

Britton, A. and M. McKee (2000). "The relation between alcohol and cardiovascular disease in Eastern Europe: explaining the paradox." Journal of Epidemiology and Community Health **54**: 328-332.

Britton, A., A. Singh-Manoux, et al. (2004). "Alcohol Consumption and Cognitive Function in the Whitehall II Study." American Journal of Epidemiology **160**(3): 240-247.

Bromet, E., L. H. Andrade, et al. (2011). "Cross-national epidemiology of DSM-IV major depressive episode." BMC Medicine **9**: 90.

Brown, G. W. and T. Harris (1978). Social origins of depression: A study of psychiatric disorder in women. New York, Free Press.

Brugha, T., R. Conroy, et al. (1982). "Social networks, attachments and support in minor affective disorders: a replication." The British Journal of Psychiatry **141**: 249-255.

Brugha, T., S. Weich, et al. (2005). "Primary group size, social support, gender and future mental health status in a prospective study of people living in private household throughout Great Britain." Psychological Medicine **35**: 705-714.

Buu, A., W. Wang, et al. (2011). "Chabges in women's alcoholic, antisocial, and depressive symptomatology over 12 years: A multilevel work of individual, familial, and neighborhood influences." Development and Psychopathology **23**: 325-337.

Cacioppo, J. T., M. E. Hughes, et al. (2006). "Loneliness as a specific risk-factor for depressive symptoms: cross-sectional and longitudinal analyses." Psychology and Ageing **21**: 140-151.

Caldwell, T. M., B. Rodgers, et al. (2002). "Patterns of association between alcohol consumption and symptoms of depression and anxiety in young adults." Addiction **97**: 583-594.

Carney, R. and K. Freedland (2000). Depression and Medical Illness. Social Epidemiology. L. Berkman and I. Kawachi. Oxford, Oxford University Press.

Chan, Y. K. and P. L. Lee (2006). "Network Size, Social Support and Happiness in Later Life: A Comparative Study pf Beijing and Hong Kong." Journal of Happiness Studies **7**(1): 87-112.

Chou, K. and I. Chi (2004). "Childlesseness and psychological well-being in Chinese older adults." International Journal of Geratric Psychiatry **19**: 449-457.

Clerkin, E. M. and N. Barnett (2012). "The separate and interactive effects pf drinking motives and social anxiety symptoms in predicting drinking outcomes." Addictive Behaviours **37**(5): 674-677.

Cobb, S. (1976). "Social Support as a Moderator of Life Stress." Psychosomatic Medicine **38**(5): 300-314.

Cohen, S. (2004). "Social Relationships and Haelth." American Psychologists **59**(Special Issue): 676-684.

Cohen, S. and G. McKay (1984). Social Support, Stress and the Buffering Hypothesis: A Theoretical Analysis. Handbook of Psychology and Health. A. Baum, S. E. Taylor and J. E. Singer. Hillsdale, NJ, Lawrence Erlbaum.

Cohen, S. and T. A. Wills (1985). "Stress, Social Support and the Buffering Hypothesis." Psychological Bulletin **98**: 310-357.

Coleman, J. S. (1988). "Social Capital in the creation of Human Capital." American Journal of Sociology **94**: S95-S120.

Conrad, J. (1904). Nostromo, A Tale of the Seaboard, Dover Publications.

Cornia, A. (2000). Short-Term, Long-Term and Hysteresis Mortality Models: A Review. . The Mortality Crisis in Transitional Economics. A. Cornia and R. Paniccia. Oxford, Oxford University Press.

Croezen, S., A. Peasey, et al. (2011). Report on critical appraisal of existing measurements of ageing-related outcomes in the CHANCES cohorts and in slected major studies of ageing WP11: Health Module. London, University College London.

Croezen, S., H. S. J. Picavet, et al. (2012). "Do positive or negative experiences of social support related to current and future health? Results from the Doetinchem Cohort Study." BMC Public Health **12**: 65.

Dalgard, O. S., C. Dowrick, et al. (2006). "Negative life events, social support and gender difference in depression. A multinational community survey with data from the ODIN study." Social Psychiatry and Psychiatric Epidemiology **41**: 444-451.

Deelstra, J. T., M. C. W. Peeters, et al. (2003). "Receiving Instrumental Support at Work: When Help Is Not Welcome." Journal of Applied Psychology **88**: 324-331.

Dierker, L. C., S. Avenenoli, et al. (2002). "Smiking and Depression: An Examination of Mechanisms of Comorbidity." American Journal of Psychiatry **159**(6): 947-953.

DiTommaso, E. and B. Spinner (1997). "Social and emotional loneliness: a re-examination of Weiss' typology of loneliness." Personality and Individual Differences **22**(3): 417-427.

Dixit, A. R. and R. M. Crum (2000). "Prospective Study of Depression and the Risk of Heavy Alcohol Use in Women." American Journal of Psychiatry(157).

Dobson, M. (2008). Diseasae: The extraordinary stories of history's deadliest killers, Sterling.

Doty, P. and H. de Wit (1995). "Effect of setting on the reinforcing and subjective effects of ethanol in social drinkers." Psychopharmacology **118**: 19-27.

Dunbar, R. I. M. (1998). "The Social Brain Hypothesis." Evolutionary Anthropology **6**: 178-190.

Dunbar, R. I. M. (2003). "The social brain: mind, language and society in evolutionary perspective." Annual Review of Anthropology **32**: 163-181.

Dunbar, R. I. M. and S. Schultz (2007). "The evolution of the social brain: anthropoid primates contrast with othe rvertrebates." Proceedings of the Royal Society **B 274**: 2429-2436.

Duncan, T. E. and Duncan, S. C. (2009). "The ABC's of LGM: An Introduction guide to Latent Variable Growth Curve Modeling." Soc Personal Pyschology Compass. **1; 3(6):** 979-991.

Durkheim, E. D. (1897 [1951]). Suicide: a study in sociology. New York, Free Press.

Euripides (2000). Bacchae. Cambridge, Cambridge University Press.

Falk, D. E., H. Y. Yi, et al. (2008). "Age of onset and temporal sequencing of lifetime DSM-IV alcohol use disorders relative to comorbid mood and anxiety disorders." Drug and Alcohol Dependence **94**(234-245).

Fergusson, D. M., J. M. Boden, et al. (2009). "Tests of Causal Links Between Alcohol Abuse or Dependence and Major Depression." Arch Gen Psychiatry **66**(33): 260-266.

Ferrari, A. J., A. J. Somerville, et al. (2013). "Global variation in the prevalence and incidence of major depressive disorder: a systematic review of the epidemiological literature." Psychological Medicine **43**: 471-481.

Ferrie, J. E., M. J. Shipley, et al. (2007). "A Prospective Study of Change in Sleep Duration: Associations with Mortality in the Whitehall II Cohort." Sleep **30**(12): 1659-1666.

Fischer, C. S. (1982). "What do we mean by 'friend'? an inductive study." Social Networks **3**(4): 287-306.

Fone, D. L., D. M. Farewell, et al. (2013). "Socioeconomic patterning of excess alcohol consumption and binge drininking: a cross-sectional study of multilevel associations with neighbourhood deprivation." BMJ Open **3**: 1-9.

Freud, S. (1917 [1964]). Mourning and Melancholia. The Standard Edition of the Complete Psychological Works of Sigmund Freud. J. Strachey. Oxford, Macmillian. **XIV: On the History of the Psycho-Analytic Movement, Papers on Metapsychology and Other Works:** 237-258.

Fuhrer, R., S. A. Stansfeld, et al. (1999). "Gender, social relations and mental health: prospective findings from an occupational cohort (Whitehall II study)." Social Science & Medicine **48**: 77-87.

Gilman, S. E. and H. D. Abraham (2001). "A longitudinal study of the order of onset of alcohol dependence and major depression." Drug and Alcohol Dependence **63**: 277-286.

Gleason, M. E. J., I. Mausmi, et al. (2008). "Receiving Support as a Mixed Blessing: Evidence for Dual Effects of Support on Psychological Outcomes." Journal of Personality and Social Psychology **94**(5): 824-838.

Goldberg, D. P. (1972). The Detection of Psychiatric Illness by Questionnaire. London, Oxford University Press.

Goldberg, D. P. and V. F. Hiller (1979). "A scaled version of the General Health Questionnaire." Psychological Medicine **9**: 139-145.

Goodwin, F. K. and K. R. Jamison (1990). Suicide, in manic-depressive illness. . New York, Oxford University Press.

Goodwin, R. D. and I. H. Gotlib (2004). "Gender differences in depression: the role of personality factors." Psychiatry Research **126**(2): 135-142.

Grav, S., O. Hellzen, et al. (2011). "Association between social support and depression in the general population: the HUNT study, a cross-sectional survey." Journal of Clinical Nursing **21**(1-2): 111-120.

Hagerty, B. M., J. Lynch-Sauer, et al. (1992). "Sense of Belonging: A Vital Mental Health Concept." Archives of Psychiatric Nursing **6**(3): 172-177.

Hagerty, B. M. and A. R. Williams (1999). "The effects of sense of belonging, social support, conflict and loneliness on depression." Nursing Research **48**: 215-219.

Hagerty, B. M., A. R. Williams, et al. (1996). "Sense of belonging and indicators of social and psychological functioning." Archives of Psychiatric Nursing **10**(4): 235-244.

Haines, V. A., J. J. Beggs, et al. (2011). "Neighborhood Disadvantage, Network Social Capital, and Depressive Symptoms." Journal of Health and Social Behaviour **52**(1): 58-73.

Head, J., P. Martikainen, et al. (2002). Work environment, alcohol consumption and ill-health: the Whitehall II Study, HSE Books.

Head, J., S. A. Stansfeld, et al. (2013). "use of self-administered instruments to assess psychiatric disorders in older people: validity of the General Health Questionnaire, the Center for Epidemiologic Studies Depression Scale and the self-completion version of the revised Clinical Interview Schedule." Psychological Medicine: 1-8.

Hedley, D. and R. Young (2006). "Social comparison processes and depressive symptoms in children and adolescnets with Asperger syndrome." Autism **10**(2): 139-153.

Heikkinen, R. and M. Kauppinen (2004). "Depressive symptoms in late life: A 10 year follow-up." Archives of Gerontology and Geriatrics **38**: 239-250.

Henderson, S. (1977). "The social network, support and neurosis. The function of attachment in adult life." The British Journal of Psychiatry **131**: 185-191.

Henderson, S. (1980). "Social relationships, adversity and neurosis: an analysis of prospective observations." The British Journal of Psychiatry **138**: 391-398.

Hobfoll, S. E. (2009). "Social Support: The Movie." Journal of Social and Personal Relationships **26**: 93-101.

Holmila, M. and K. Raitasalo (2005). "Gender differences in drinking: why do they still exist?" Addiction **100**: 1763-1769.

House, J. S., R. L. Kahn, et al., Eds. (1985). Measures and concepts of social support. Social support and health. San Diego, Academic Press.

Ibarra-Rovillard, M. S. and N. A. Kuiper (2011). "Social support and social negativity findings in depression: Perceived responsiveness to basic psychological needs." Clinical Psychology Review **31**: 342-352.

Jamal, M., A. J. W. Van der Does, et al. (2012). "Association of smoking and nicotine dependence with severity and course of symptoms in patients with depressive or anxiety disorder." Drug and Alcohol Dependence **126**: 138-146.

Joosten, J., R. Knibbe, et al. (2009). "Criticism of drinking ad informal social control: a study in 18 countries." Contemporary Drug Problems **36**(1-2): 85-109.

Kahn, R. L. and T. C. Antonucci (1980). Convoys over the life-course: Attchments, roles, and social support. Life-span development and behaviour. P. B. Baltes and O. Brim. New York, Academic Press. **3**.

Kawachi, I. and L. F. Berkman (2001). "Social ties and mental health." Journal of Urban Health **78**(3): 458-467.

Kendler, K., S., J. Meyers, et al. (2005). "Sex Differences in the Relationship Between Social Support and Risk for Major Depression: A Longitudinal Study of Opposite-Sex Twin Pairs." American Journal of Psychiatry **162**(250-256).

Kessler, R. C., G. Andrews, et al. (2006). "The World Health Organization Composite International Diagnostic Interview Short-Form (CIDI-SF). ." International Journal of Methods in Psychiatric Reserach **7**(4): 171-185.

Keyes, K. M. and D. S. Hasin (2008). "Socio-economic status and problem alcohol use: the positive relationship between income and the DSM-IV alcohol abuse diagnosis." Addiction **103**(7): 1120-1130.

Khantzian, E. J. (1997). "The Self-Medication Hypothesis of Substance Use Disorders: A Reconsideration and Recent Applications." Harvard Review of Psychiatry **4**(4): 231-244.

Kierkergaard, S. (1849 [1983]). Sickness Onto Death, Princeton University Press.

Klatsky, A. L. (2009). "Alcohol and cardiovascular diseases." Expert Reviews **7**(5): 499-506.

Klibansky, R., E. Panofsky, et al. (1964). Saturn and melancholy: studies in the history of natural philosophy, religion and art. New York, Basics Books.

Koppes, L. L. J., J. W. R. Twisk, et al. (2001). "Personality characteristics and alcohol consumption: Longitudinal analyses in men and women followed from ages 13 to 32. ." Journal of Studies on Alcohol **62**(4): 494-500.

Korn, M. E. and J. L. Maggs (2004). "Why Drink Less? Diffidence, Self-Presentation Styles, and Alcohol Use Among University Students." Journal of Youth and Adolescence **33**(3): 201-211.

Kuehener, G. (2003). "Gender differences in unipolar depression: an update of epidemiological findings and possible explanations." Acta Psychiatrica Scandinava **108**: 163-174.

Kuntsche, E., R. Knibbe, et al. (2005). "Why do young people drink? A review of drinking motives." Clinical Psychology Review **25**(7): 841-861.

Kuntsche, E., M. von Fischer, et al. (2008). "Personality factors and alcohol use: A mediator analysis of drinking motives." Personality and Individual Differences **45**: 796-800.

Kuntsche, S., R. Knibbe, et al. (2009). "Social roles and alcohol consumption: A study of 10 industrialised countries." Social Science & Medicine **68**: 1263-1270.

Landrine, H., J. L. Richardson, et al. (1994). "Cultural diversity in the predictors of adolescent cigarette smoking: The relative influence of peers." Journal of Behavioural Medicine **17**(3): 331-346.

Lasgaard, M., L. Goossens, et al. (2011). "Loneliness, Depressive Symptomatology, and Suicide Ideation in Adolescence: Cross-Sectional and Longitudinal Analyses." Journal of Abnormal Child Psychology **39**: 137-150.

Larsen, R. (2011). "Misssing Data Imputation versus Full Information Maximum Likelihood with Second-Level Dependencies." Structural Equation Modeling: A Multidisciplinary Journal **18:4,** 649-662.

Lee, H., J.-A. Lee, et al. (2014). "Physical activity and depressive symptoms in older adults." Geriatric Nursing **35**: 37-41.

Leon, D. A., L. Saburova, et al. (2007). "Harzardous alcohol drinking and premature mortality in Russia: a population based case-control study." The Lancet **369**(9578): 2001-2009.

Levine, H. G. (1992). Temperance Cultures: Concern about Alcohol Problems in Nordic and English-speaking Cultures The Nature of Alcohol and Drug Related Problems. M. Lader, G. Edwards and D. C. Drummond. New York City, Oxford University Press**:** 15-36.

Lewis, B. A. and H. K. O'Neill (2000). "Alcohol expectancies and social deficits relating to problem drinking among college students." Addictive Behaviours **25**(2): 295-299.

Lin, N., X. Ye, et al. (1999). "Social Support and Depressed Mood: A Structural Analysis." Journal of Health and Social Behaviour **40**(4): 344-359.

Lipton, R. I. (1994). "The Effect of Moderate Alcohol Use on the Relationship between Stress and Depression." American Journal of Public Health **84**(12): 1913-1917.

Litwin, H. (2011). "The association between social netwokr relationships and depressive symptoms among older Americans: what matters most?" International Psychogeriatrics **23**(6): 930-940.

Lorant, V. D., D., W. Eaton, et al. (2003). "Socioeconomic Inequalities in Depression: A Meta-Analysis. ." American Journal of Epidemiology **157**(2): 98-112.

Loukas, A., J. L. Krull, et al. (2000). "The Relation of Personality to Alcohol Abuse/Dependence in a High-Risk Sample." Journal of Personality **68**(6): 1153-1174.

Lucas, N., T. D. Windsor, et al. (2010). "Psychological Distress in Non-Drinkers: Associations with Previous Heavy Drinking and Current Social Relationships." Alcohol & Alcoholism **45**(1): 95-102.

Lui, J. H., D. V. Hansen, et al. (2011). "Development and Evolution of the Human Neocortex." Cell **156**(1): 18-36.

Lyness, J. M., K. N. Tamson, et al. (1997). "Screening for depression in elderly primary care patients. A comparison of the Center for Epidemiological Studies - Depression Scale and the Geriatric Depression Scale." Archives of Internal Medicine **157**(4): 449-454.

Lyvers, M., P. Hasking, et al. (2010). "Drinking motives, drinking restraint and drinking behaviour among young adults." Addictive Behaviours **35**(2): 116-122.

MacAndrew, C. and R. B. Edgerton (1969). Drunken Comportment: A Social Explanation. Chicago, Aldine.

Maher, M. J., P. A. Mora, et al. (2006). "Depression as a Predictor of Perceived Social Support and DEmand: A Componential Approach Using a Prospective Sample of Older Adults." Emotion **6**(3): 450-458.

Makela, K. and H. Mustonen (2000). "Relationships of drinking behaviour, gender and age with reported negative and positive experience related to drinking." Addiction **95**(5): 727-736.

Makela, P., G. Gmel, et al. (2006). "Drinking patterns and their gender differences in Europe." Alcohol & Alcoholism **41**(1): i8-i18.

Marchenko, Y. (2011). Chained equations and more in multiple imputation in Stata 12, Nordic and Baltic Stata Users Group Meeting.

Markman Geisner, I., M. E. Larimer, et al. (2004). "The relationship among alcohol use, related problems, and symptoms of psychological distress: Gender as a moderator in a college sample." Addictive Behaviours **29**: 843-848.

Markou, A., T. R. Kosten, et al. (1998). "Neurobiological similarities in depression and drug dependence: a self-medication hypothesis." Neuropsychopharmacology **18**: 135-174.

Marmot, M. (2004). The Status Syndrome. How Your Social Standing Directly Affects Your Health. . London, Bloomsbury Publishing.

Marmot, M. and M. Bobak (2000). Psychosocial and biological mechanisms behind the recent mortality crisis in Central and Eastern Europe. The mortality crisis in transitional economies A. Cornia and R. Paniccia. Oxford, Oxford University press**:** 127-148.

Marmot, M. and E. Brunner (2005). "Cohort Profile: The Whitehall II study." International Journal of Epidemiology **34**: 251-256.

Marmot, M., F. North, et al. (1993). "Alcohol consumption and sickness absence: from the Whitehall II study." Addiction **88**(3): 369-382.

Marmot, M., F. North, et al. (1993). "Alcohol consumption and sickness absence: from the Whithall II study." Addiction **88**(3): 369-382.

Mathers, C. D. and D. Loncar (2006). "Projections of Global Mortality and Burden of Disease from 2002 to 2030." PLoS Medicine **3**(11): 2011-2030.

McCollam, A., C. O'Sullivan, et al. (2008). Mental Health in the EU Key Facts, Figures and Activities

McCrone, P. et al (2008) "Paying the Price. The cost of mental health care in England to 2026." King's Fund.

Merikangas, K. R., R. L. Metha, et al. (1998). "Comorbidity of substance use disorders with mood and anxiety disorders: results of the Inaternational Consortium in Psychiatric Epidemiology." Addictive Behaviours **23**(893-907).

Michel (2009). The structure of social networks: examining gender differences and effects on social support and psychological distress.

Miller, P. M. and J. G. Ingham (1976). "Friends, Confidants and Symptoms." Social Psychiatry **11**: 51-58.

Mirowsky, J. and C. Ross (2003). Social Causes of Psychological Distress. New York, Aldine de Gruyter.

Monahan, J. L. and P. J. Lannutti (2000). "Alcohol as social lubricant. Alcohol myopia theory, social sel-esteem, and social intereaction." Human Communication Research **26**(2): 175-202.

Mulligan Rauch, S. A. and J. Becker Bryant (2000). "Gender and Context Differences in Alcohol Expectancies." Journal of Social Psychology **140**(2): 240-253.

Mulrow, C. D., J. W. J. Williams, et al. (1995). "Case-finding instruments for depression in primary care settings." Annals of Internal Medicine **122**(12): 913-921.

Mumenthaler, M. S., J. L. Taylor, et al. (1999). "Gender Differences in Moderate Drinking Effects." Alcohol Research and Health **23**(1).

Murray, C. J. and A. D. Lopez (1997). "Alternative Projections of Mortality and Disability: 1990-2020 Global Burden of Disease Study." The Lancet **349**: 1498-1504.

Nabi, H., A. Singh-Manoux, et al. (2009). "Hostility and depressive mood: results from the Whitehall II propsective cohort study." Psychological Medicine **July**: 1-9.

NHS (2012). "Drinking and alcohol." Retrieved 14/05/2014, 2014, from http://www.nhs.uk/Livewell/alcohol/Pages/Effectsofalcohol.aspx.

NHS (2013). "NHS Choices Antidepressants." 2014, from http://www.nhs.uk/Conditions/Antidepressant-drugs/Pages/Introduction.aspx.

Nicholson, A., H. Pikhart, et al. (2008). "Socio-economic status over the life-course and depressive symptoms in men and women in Eastern Europe." Journal of Affective Disorders **105**(1): 125-136.

Nietzsche, F. W. (1872 [2000]). The birth of tragedy. Oxford, Oxford University Press.

Nolen-Hoeksema, S. (2004). "Gender differences in risk factors and consequences for alcohol use and problems." Clinical Psychology Review **24**: 981-1010.

Nolen-Hoeksema, S. and C. Ahrens (2002). "Age differences and similar-ties in the correlated of depressive symptoms." Psychology and Ageing **17**: 116-124.

Nolen-Hoeksema, S. and Z. A. Harrell (2002). "Rumination, Depression and Alcohol Use: Tests of Gender Differences." Journal of Cognitive Psychotherapy: An International Quarterly **16**(4): 391-402.

Parker, G. and H. Brotchie (2010). "Gender differences in depression." International Review of Psychiatry **22**(5): 429-436.

Pattenden, S., K. Nanchachal, et al. (2008). "Self-reported never-drinkers in England 1994-2003: Charactersitics and trends in adults aged 18-54." Alcohol & Alcoholism **43**(1): 91-96.

Paykel, E. S., T. Brugha, et al. (2005). "Size and burden of depressive disorders in Europe." European Neuropsychopharmacology **15**: 411-423.

Peace, A. (1992). No finishing without drinking: The construction if social identity in rural Ireland. Alcohol, Gender and Culture. D. Gefou-Madianou. London, Routledge**:** 167-180.

Peasey, A., M. Bobak, et al. (2006). "Determinants of cardiovascular disease and other non-communicable diseases in Central and Eastern Europe: Rationale and design of the HAPIEE study." BMC Public Health **6**: 255-264.

Peele, S. and A. Brodksy (2000). "Exploring psychological benefits associated with moderate alcohol use: a necessary corrective to assessments of drinking outcomes? ." Drug and Alcohol Dependence **60**(3): 221-247.

Peirce, R. S., M. R. Frone, et al. (2000). "A Longitudinal Model of Social Contact, Social Support, Depression, and Alcohol Use." Health Psychology **19**(1): 28-38.

Peters, B. L. and E. Stringham (2006). "No booze? You may lose: Why drinkers earn more than nondrinkers." Journal of Labour Research **27**(3): 411-421.

Piccinelli, M. and G. Wilkinson (2000). "Gender differences in depression: Critical review." The British Journal of Psychiatry **177**: 486-492.

Pikhartova, J., T. Chandola, et al. (2009). "Neighbourhood socioeconomic indicators and depressive symptoms in the Czech Republic: a population based study. ." International Journal of Public Health **54**: 283-293.

Pliner, P. and H. Cappell (1974). "Modification of affective consequences of alcohol: A comparison of social and solitary drinking." Journal of Abnormal Psychology **83**: 418-425.

Poschl, G. and H. K. Seitz (2004). "Alcohol and Cancer." Alcohol & Alcoholism **39**(3): 155-165.

Power, C., B. Rodgers, et al. (1998). "U-shaped relation for alcohol consumption and health in early adulthood and implications for mortality." The Lancet **352**(9131): 857-877.

Putnam, R. D. (1993). Making Democracy Work: Civil Traditions in Modern Italy. Princeton, NJ, Princeton University Press.

Putnam, R. D. (1995). "Bowling Alone: America's Declining Social Capital." Journal of Democracy **6**(1): 65-78.

Putnam, R. D. (2000). Bowling Alone: The Collapse and Revival of American Community. New York, Simon and Schuster.

Radloff, l. S. (1977). "The CES-D sclae: a self-report depression scale for research in the general population." Applied Psychological Measurements **3**: 385-432.

Rahav, G., R. Wilsnack, et al. (2006). "The influence of societal level factors on men's and women's alcohol consumption and alcohol problems." Alcohol & Alcoholism **41**(1): i147-i155.

Rehm, J. (1998). "Measuring Quantity, Frequency and Volume of Drinking." Alcoholism: Clinical and Experimental Research **22**(S2).

Rehm, J., T. K. Greenfield, et al. (2001). "Average Volume of Alcohol Cosnumption, Patterns of Drinking, and All-Cause Mortality: Results from the US National Alcohol Survey." American Journal of Epidemiology **153**(1): 64-71.

Rehm, J., R. Room, et al. (2003). "The relationship of average volume of alcohol consumption and patterns of drinking to burden of disease: an overview." Addiction **98**: 1209-1228.

Rehm, J., C. T. Sempos, et al. (2003). "Alcohol and cardiovascular disease--more than one paradox to consider. Average volume of alcohol consumption, patterns of drinking and riks of cornoray heart disease -- a review." Journal of Cardiovascular Risk **10**(1): 15-20.

Rimal, R. N. and K. Real (2005). "How Behaviours are Influenced by Perceived Norms: A Test of the Theory of Normative Social Behaviour." Communication Research **32**(3): 389-414.

Roberts, R. E. and S. W. Vernon (1983). "The Centre for Epidemiological Studies Depression Scale: its use in a community sample." American Journal of Psychiatry **140**: 41-46.

Rodgers, B., A. E. Korten, et al. (2000). "Risk factors for depression and anxiety in abstainers, moderate drinkers and heavy drinkers." Addiction **95**(12): 1833-1845.

Rodgers, B., A. E. Korten, et al. (2000). "Non-linear relationships in associations of depression and anxiety with alcohol use." Psychological Medicine **30**: 421-432.

Rodgers, B., R. Parslow, et al. (2007). "Affective disorders, anxiety disorders and psychological distress in non-drinkers." Journal of Affective Disorders **99**(1-3): 165-172.

Room, R. and K. Makela (2000). "Typologies of the cultural position of drinking." Journal of Studies on Alcohol **61**(3): 475-483.

Rose, R. (1995). "Russia as an hour-glass society: a constitution without citizens." East European Constitutional Review **4**: 34-42.

Rose, R. (2000). "How much does social capital add to individual health? A survey study of Russians." Social Science & Medicine **51**: 1421-1435.

Rosenbaum, S., A. Tiedemann, et al. (2014). "Physical activity interventions: an essential component in recovery from mental illness." British Journal of Sports Medicine **0**(0): 1-2.

Rubin, D. B. (1987). Multiple Imputation for Nonresponse in Surveys. New York.

Rubin, D. B. (1996). "Multiple imputation after 18+ years." Journal of the American Statistical Association **91**: 473-489.

Sarason, B. R., I. G. Sarason, et al., Eds. (1990). Social Support: An associational view. Wiley deries on personality processes. Oxford, John Wiley & Sons.

Saxena, S. (2011). Mental Health Atals 2011. Mental Health Atals. S. Shekhar. Geneva, WHO.

Schafer, J. L. and Graham, J. W. (2002). "Missing data: our view of the state of the art." Psychological Methods **7:** 147-177.

Schwarzbach, M., M. Luppa, et al. (2013). "Social relations and depression in late life - A systematic review." International Journal of Geriatric Psychiatry **29**: 1-21.

Segrin, C., H. L. Powell, et al. (2003). "Symptoms of depression, relational quality, and loneliness in dating relationships." Personal Relationships **10**: 25-36.

Shakespeare, W. (1599 [2011]). Hamlet. Prince of Denmark. London, William Collins.

Singh-Manoux, A., M. Hillsdon, et al. (2005). "Effects of Physical Activity on Cognitive Functioning in Middle Age: Evidence From the Whitehall II Prospective Cohort Study." American Journal of Public Health **95**(12): 2252-2258.

Skapinakis, P. and G. Lewis (2001). "Epidemiology in community psychiatric research: common use and methodological issues." Epidemiologica and Psichiatria Sociale **10**(18-26).

Skogen, J. C., S. B. Harvey, et al. (2009). "Anxiety and depression among abstainers and low-level alcohol consumers. The Nord-Trondelag Health Study." Addiction **104**: 1519-1529.

Smith, R. C., E. S. Parker, et al. (1975). "Alcohol and affect in dyadic social association." Psychosomatic Medicine **37**: 25-40.

Sobocki, P., B. Jonosson, et al. (2006). "Cost of depression in Europe." Journal of Mental Health Policy Econ. **9**(2): 87-98.

Stansfeld, S. A., R. Fuhrer, et al. (1998). "Types of social support as predictors of psychiatric morbidity in a cohort of British Civil Servants (Whitehall II Study)." Psychological Medicine **28**: 881-892.

Stansfeld, S. A., J. Head, et al. (2003). "Social inequalities in depressive symptoms and physical functioning in the Whitehall II study: exploring a common cause explanation." Journal of Epidemiology and Community Health **57**: 361-367.

Stansfeld, S. A., J. Head, et al. (1998). "Explaining social class differences in depressionand well-being." Social Psychiatry and Psychiatric Epidemiology **33**: 1-9.

Stansfeld, S. A. and M. Marmot (1992). "Deriving a survey measure of social support: the reliability and validity of the close person questionnaire." Social Science & Medicine **35**(8): 1027-1035.

Stansfeld, S. A., F. North, et al. (1995). "Work characteristics and psychiatric disorder in civil servants in London." Journal of Epidemiology and Community Health **49**: 124-130.

Stansfeld, S. A., E. G. S. Rael, et al. (1997). "Social Support and psychiatric sickness absence: a prospective study of British civil servants." Psychological Medicine **27**: 35-48.

Sullivan, P. F., M. C. Neale, et al. (2000). "Genetic epidemiology of major depression: a review and meta-analysis." American Journal of Psychiatry **157**: 1552-1562.

Taylor, S. E. and L. G. Aspinwall, Eds. (1996). Mediating and Moderating Processes in Psychosocial Stress: Appraisal, Coping, Resistance and Vulnerability. Pyschosocial Stress: Perspectives on Structure, Theory, Life Course and Methods. San Diego, CA, Academic Press.

Taylor, S. E. and A. L. Stanton (2007). "Coping Resources, Coping Processes and Mental Health." Annual Review of Clinical Psychology **3**: 377-401.

Thoits, P., Ed. (1985). Social Support and Psychosocial Well-Being: Theoretical Possibilities. Social Support: Theory, Research and Applications. Dordrecht, Netherlands, Martinus Nijhof.

Thoits, P. (2003). Personal Agency in the Accumulation of Multiple Role-Identities. Advances in Identity Theory and Research. P. J. Burke, T. J. Owens, R. Serpe and P. Thoits. New York, Kluwer Academic/Plenum**:** 179-194.

Thoits, P. (2011). "Mechanisms linking social ties and support to physical and mental health." Journal of Health and Social Behaviour **52**(2): 145-161.

Tomkins, S., L. Saburova, et al. (2007). "Prevalence and socio-economic distribution of hazarduos pattern of alcohol drinking: study of alcohol consumption in men aged 25-54 in Izhevsk, Russia." Addiction **102**(4): 544-553.

Uchino, B. N. (2004). Social Support and Physical Health: Understanding the Health Consequences of Relationships. New Haven, CT, Yale University Press.

Umberson, D. and J. K. Montez (2010). "Social Relationships and Health: A Flashpoint for Health Policy." Journal of Health and Social Behaviour **51**(Special Issue): S54-66.

Ustun, T. B., J. L. Ayuso-Mateos, et al. (2004). "Global burden of depressive disorder in the year 2000." British Journal of Psychiatry **184**: 386-392.

van Beljouw, I. M. J., E. van Exel, et al. (2014). ""Being all alone makes me sad": loneliness in older adults with depressive symptoms." International Psychogeriatrics **26**(09): 1541-1551.

Vassiliou, A. (2008). Together for Mental Health and Wellbeing. S. P. o. t. E. Union. Brussels.

Vilhajalmsson, R. (1993). "Life stress, social support, and clinical depression: A reanalysis of the literature." Social Science & Medicine **37**(3): 331-342.

Wahlbeck, K., P. Anderson, et al. (2011). Impact of economic crises on mental health WHO Regional Office for Europe Publications, WHO.

Wahlbeck, K. and M. Makinen (2008). Prevention of depression and suicide. Consensus paper. . European Communities. Luxembourg.

Wang, L., I.-M. Lee, et al. (2010). "Alcohol Consumption, Weight Gain, and Risk of Becoming Overwight in Middle-aged Older Women." Archives of Internal Medicine **170**(5): 453-461.

Wannamethee, S. G. and A. G. Shaper (2003). "Alcohol, body weight, and weight gain in middl-aged men." American Journal of Clinical Nutrition **77**(5): 1312-1317.

Weich, S. and R. Araya (2004). "International and regional variation in the prevalence of common mental disorders: do we need more surveys?" British Journal of Psychiatry **184**(289-290).

Weiss, R. S. (1973). Loneliness: the experience of emotional and social isolation. Cambridge, MA, MIT Press.

Whitaker, R. (2010). Anatomy of an Epidemic: Magic Bullets, Psychiatric Drugs, and the Astonishing Rise of Mental Illness in America, Crown Publishing Group.

Whiteford, H. A., L. Degenhardt, et al. (2013). "Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010." The Lancet **382**: 1575-1586.

WHO (2001). The World Health Report 2001. Mental Health: New Understanding, New Hope. The World Health Report. WHO. Geneva, World Health Organization.

WHO (2004). "Prevalence, Severity, and Unmet Need for Treatment of Mental Disorders in the World Health Organization World Mental Health Surveys." JAMA **291**: 2581-2590.

Wildes, J. E., K. L. Harkness, et al. (2002). "Life events, number of social relationships, and twelve-month naturalistic course of major depression in a community sample of women." Depression and Anxiety **16**: 104-113.

Williams, P. and L. Barclay (2004). "Social Support in Context: A Necessary Step in Improving Research, Intervention, and Practice." Qualitative Health Research **14**(7): 942-960.

Wilsnack, R. W., N. D. Vogeltanz-Holm, et al. (2000). "Gender differences in alcohol consumption and adverse drinking consequences: cross-cultural patterns." Addiction **95**(2): 251-265.

Wilsnack, R. W., S. C. Wilsnack, et al. (2009). "Gender and alcohol consumption: patterns from the multinational GENACIS project." Addiction **104**: 1487-1500.

Witheford, H. A., L. Dagenhardt, et al. (2013). "Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010." Lancet **382**: 1575-1586.

Wupperman, P. and C. S. Neumann (2006). "Depressive symptoms as a function of sex-role, rumination and neuroticism." Personality and Individual Differences **40**(2): 189-201.

Yesavage, J. A., T. L. Brink, et al. (1983). "Development and validation of a geriatric depression rating scale: a preliminary report." Journal of Psychiatric Research: 17-27.

Zung, W. W. K., C. B. Richards, et al. (1965). "Salf-Rating Depression Scale in an Outpatient Clinic Further Validation of the SDS." Arch Gen Psychiatry **13**(6): 508-515.

Zunzunegui, M. V., N. Minicuci, et al. (2007). "Gender differences in depressive symptoms among older adults: a cross-national comparison. The CLESA project." Social Psychiatry and Psychiatric Epidemiology **42**(3): 198-207.

10. Appendix

Number and percentage of missing data were calculated for all variables included in the analysis, including covariates, and are presented in Table A.1. The percentage of missing values was below 5% in most variables, but measures of support, the contact with friends index and volume of alcohol consumed per week.

Table A.1 *Number and percentage of complete and missing data in each variable.*

	Complete N	%	Missing N	%
Confiding support	3,590	73.43	1,299	26.57
Practical support	3,590	73.43	1,299	26.57
Negative support	3,584	73.31	1,305	26.69
Friends scale	3,630	74.25	1,259	26.75
Relatives scale	4675	95.62	214	4.38
Network scale	4,856	99.33	33	0.67
Frequency of alcohol consumption	4,882	99.86	7	0.14
Volume of alcohol consumed per week	4,299	96.70	590	12.07
Age	4,889	100.00		
Sex	4,889	100.00		
Marital Status	4,878	99.78	11	0.22
Employment Grade	4,889	100.00		
Smoking Status	4,887	99.96	2	0.04
Mild Physical Activity	4,840	99.00	49	1.00
Moderate Physical Activity	4,813	98.45	76	1.55
Vigorous Physical Activity	4,777	97.71	112	2.29

Secondly, the assumption that data are missing at random (MAR) was investigated. MAR is not formally testable (reference). However, since MAR assumes that the probability of missing-ness is influenced only by observed values, missing-ness was represented by a dummy and the structure of missing data explored by logistic regression. If variables were missing less than 5% of data, the sample size proved to be too small for regression to be significant. Of all covariates used as predictors of missing-ness for measures of support, inclusion in social networks and alcohol consumption, sex, marital status, employment grade and measures of physical activity proved to predict missing-ness in confiding/emotional, practical and negative support and for the contact with friends index. Results of this analysis are presented in Table A.2.

At this point, the number of datasets to impute was decided. Even though there might not be no definite consensus on the number of imputations that should be run, generally the larger the proportion of missing data, the larger the number of imputations needed. Graham et al (2007) run simulations to assess the loss of power due to different number of imputations run and recommended to perform 20 imputations for 10% to 30% missing information. Hence, given that highest percentage of missing data in my sample was 26.75% for the contact with friends index, 20 imputations we here run.

Missing data were imputed using the MI command package on STATA 12, on the basis of equations including: age; sex; marital status at Phase 1, 2, 5 and 7 ; employment grade at Phase 1, 2, 5 and 7; last known employment grade at Phase 5 and 7; physical activity at Phase 1, 2, 5 and 7; smoking status at Phase 1, 2, 5 and 7; depression at Phase1;

confiding/emotional support; practical support; negative support; network index; friends index; relatives index; frequency of alcohol consumption; volume of alcohol consumed a week by men and women. Predictor variables were chosen because they were correlated with the missing variable and thus would have help imputing missing values while preserving relationships in the data. In addition, because information about support was collected only half way through Phase 1, support subscales at Phase2 were used as predictors of support at Phase1. The STATA command used is presented below:

" mi impute chained (mlogit) conf1t pract1t neg1t netwt netfrndt netrelt alcyr galcoholm galcoholw ghqdepg sex age status smoke grlump physicat zage zsmoke zstatusx zgrlump zphysicat zalcyr zgalcoholm zgalcoholw tstatusx tgrlump tsmoke tphysicat talcyr tgalcoholm tgalcoholw tlrgrlmp mstatusx mgrlump mlrgrlmp msmoke mphysicat malcyr mgalcoholm mgalcoholw zconf1t zpract1t zneg1t znetfrnt, add (20)"[1]

[1]

Conf1t	Confiding/emotional support
Pract1t	Practical support
Neg1t	Negative support
Netwt	Network index
Netfrndt	Contact with friends
Netrelt	Contact with relatives
Alcyr	Frequency of alcohol consumption
Galcoholm	Dose of alcohol regularly consumed by men
Galcoholw	Dose of alcohol regularly consumed by women
Ghqdepg	Depressive symptoms
Sex	
Age	
Smoke	Smoking status
Physicat	Physical activity
Grlump	Employment grade
Tlrgrlmp	Last known employment grade
z- t- m-	z- Phase 2; t-Phase 5; m-Phase 7 measurements

Once the imputed datasets were obtained, percentages of participants in each category of the variables under study were calculated on imputed data and compared to those in the un-imputed dataset. Prevalence of depressive symptoms were also calculated for each category and compared between original and imputed data. Results are shown in Table A3 and Table A.4.

Table A.2. Odds Ratios and relative 95% confidence intervals for missing data in confiding/emotional, practical, negative support and contact with friends index.

	Confiding		Practical		Negative		Friends	
	OR	**95% C.I.**	**OR**	**95% C.I.**	**OR**	**95% C.I.**	**OR**	**95% C.I.**
Gender	1.41	1.20-1.67	1.43	1.21-1.69	1.41	1.19-1.66	1.54	1.30-1.82
Marital Status	1.10	1.03-1.18	1.10	1.03-1.18	1.10	1.03-1.18	1.03	0.97-1.11
Employment Grade	0.76	0.68-0.85	0.76	0.69-0.86	0.77	0.69-0.86	0.71	0.64-0.80
Mild Physical activity	1.28	1.16-1.41	1.28	1.15-1.40	1.28	1.16-1.42	1.25	1.13-1.38
Moderate Physical activity	0.84	0.77-0.91	0.84	0.77-0.91	0.83	0.77-0.90	0.81	0.74-0.88
Vigorous Physical activity	0.78	0.73-0.84	0.79	0.74-0.84	0.79	0.73-0.84	0.78	0.73-0.83

Table A.3. *Percentage of participants and prevalence of depression in each measure of social support and inclusion in social networks.*

	Original		Imputed	
	%	Depression %	%	Depression %
Confiding/emotional				
High	30.81	8.68	30.84	8.57
medium	39.36	11.75	30.18	11.93
low	29.83	16.06	29.98	16.31
Practical				
High	32.67	9.63	32.38	9.93
medium	35.10	12.46	34.98	12.40
low	32.23	14.09	32.64	14.25
Negative				
Low	37.19	6.98	37.49	7.09
medium	32.70	12.29	32.65	12.20
High	30.11	18.26	29.86	18.64
Network				
High	36.92	8.53	36.95	8.53
medium	36.88	12.28	36.87	12.30
low	26.19	17.14	26.18	17.27
Friends				
High	40.63	8.20	40.48	8.36
medium	34.71	13.49	35.34	13.35
low	24.66	17.09	24.18	16.99
Relatives				
High	44.34	10.13	44.74	10.05
medium	21.03	13.73	21.03	13.61
low	34.63	14.21	34.22	14.18

Table A.4. *Percentage of participants and prevalence of depression in each measure of alcohol consumption.*

	Original		Imputed	
	%	Depression %	%	Depression %
Frequency				
>1/day	30.32	12.77	30.33	12.75
>1/week	42.75	12.07	42.74	12.10
>1/month	13.03	11.32	13.03	11.30
Special occasions/never	13.91	12.22	13.90	12.22
Dose consumed (M)				
Never	2.16	13.04	2.16	13.04
1-80 g/week	69.04	11.27	69.04	11.27
81-160 g/week	18.68	11.87	18.68	11.87
161-240 g/week	5.69	15.38	5.69	15.38
>241 g/week	4.44	13.38	4.44	13.38
Dose consumed (W)				
Never	7.10	19.23	7.10	19.23
1-48 g/week	64.94	12.62	64.94	12.62
49-80 g/week	14.30	14.65	14.30	14.65
81-160 + g/week	13.66	16.00	13.66	16.00
Drink most when bored				
No	97.21	12.12	97.10	11.85
Yes	2.79	27.05	2.90	25.81
Drink most under pressure				
No	96.01	11.87	95.87	11.60
Yes	3.99	29.14	4.13	27.68
Drink most in social settings				
No	5.64	11.81	6.72	16.93
Yes	94.36	17.29	93.28	11.85
Drink most when upset				
No	94.22	11.33	94.03	11.08
Yes	5.78	32.50	5.97	30.74