THE EFFECTS OF SOCIAL NETWORKS ON THE HEALTH OF OLDER AUSTRALIANS

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ABSTRACT

Background

Over the past three decades, social relationships have been shown to have important effects upon health. However, many different definitions and aspects of social relationships have been considered in the various studies, making comparison of findings difficult. Furthermore, the effects of social relationships upon different health outcomes have rarely been investigated within the same cohort of older people. In addition, there is a paucity of information concerning the effects of social relationships upon health of older Australians.

Aim

This thesis aims to investigate the effects of the structural aspects of social relationships – that is, social networks – on health among older Australians. The three specific health outcomes considered in this thesis were disability, residential care use and death. The specific aims of the thesis were to:

1. Develop a measurement model of social networks.
2. Examine the effects of total and specific social networks upon disability.
3. Determine the effects of total and specific social networks upon use of residential care.
4. Investigate the effects of total and specific social networks upon survival.

An additional aim was to determine if there were threshold effects of social networks on the three specific health outcomes.

Methods

The study drew on six waves of data from 1477 participants in the Australian Longitudinal Study of Ageing. A range of statistical techniques, including binary and

multinomial logistic regression and survival analysis, were used in the analysis of the data. Propensity score adjustment was used to control for the effects of a broad range of covariates that encompassed sociodemographic, health, psychological and lifestyle characteristics of participants.

Results

A measurement model with social networks for children, relatives, friends and confidants was validated using confirmatory factor analysis. A variable that measured total social networks was also derived. Better social networks with relatives were protective against developing mobility disability over the nine year follow-up period (odds ratio (OR) 0.77; 95% confidence interval (95%CI) 0.62 – 0.96). A similar result was found for Nagi disability (OR 0.76; 95%CI 0.62 – 0.93). Other specific social networks did not have significant effects on either measure of disability.

There were no significant effects of social networks on use of low-level residential care overall. There was a significant effect of social networks with confidants and total social networks, such that participants in the upper category of social networks with confidants appeared to be protected against use of high-level residential care (OR 0.53; 95%CI 0.35 – 0.81) compared to participants in the lower category of confidants social networks. Similarly, participants in the upper category for total social networks appeared to be protected against use of high-level residential care (OR 0.68; 95%CI 0.46 – 0.99).

In terms of mortality, better social networks with confidants and with friends appeared to be protective against death during the decade following the Wave 1 interview. The hazard ratio (HR) for participants in the upper category for confidants was 0.74 (95%CI

0.63-0.88) compared to participants in the lower category. For friends networks, the analogous HR was 0.75 (95%CI 0.63-0.89). Better total social networks also appeared to be protective against death over the 10 years of follow-up (HR 0.83; 95%CI 0.70-0.99).

There were few significant effects of social networks with children on the three health outcomes considered. There was little evidence of threshold effects of the specific social networks on the health outcomes.

Discussion

There are important and differing effects of specific social networks on the three health outcomes of disability, residential care and mortality that were considered in this thesis. Policymakers may need to reconsider whether specific kinds of social relationships, beyond spouses and children, have been given adequate weight in current policy frameworks that address the health of older people.

DECLARATION

This work contains no material which has been accepted for the award of any other degree or diploma in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text.

I give consent to this copy of my thesis, when deposited in the University Library, being made available in all forms of media, now or hereafter known.

Lynne Giles

19 December 2007

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Dr Patricia Metcalf of the Department of Statistics in the University of Auckland supervised me in the early stages of this thesis before my return from Auckland to Adelaide. Her willingness to supervise in an area outside of her main interests and her sound advice to publish results early were influential in all stages of this work.

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especially want to thank my husband Simon and children Edward and Grace for making those many trips to the zoo, to Bunnings, and all those other outings that have helped when I needed time to work on this thesis. I will be forever grateful for their support, encouragement and love. I'm looking forward to rejoining in the adventures of our family life, and dedicate this thesis to them.

1 INTRODUCTION

1.1 AN AGEING POPULATION

Worldwide, the population is ageing rapidly. In Australia in 2006, nine per cent of the population was aged 70 years or more, and this is projected to increase to 13 per cent of the population by 2021, and 21 per cent of the population by 2051 (Australian Bureau of Statistics, 2003; 2006). Personal and environmental factors that are associated with ageing well are therefore of great interest, from both individual and societal perspectives.

Over the past 30 or so years, social relationships have been shown to have significant effects upon health. A range of health outcomes has been considered in this work, including mortality, physical function, depression, and cognitive function. Health service use has also been considered, most notably in the form of use of residential care. With the exception of residential care use and cognitive function, the majority of studies have focussed on adults in general. However, given the ageing Australian population, the effects of social relationships upon health in older people remains an important topic.

Many of the epidemiological studies that have examined the effects of social relationships upon health outcomes for older people have used *ad hoc* measures of some aspects of social relationships. Commentators have often remarked on this. Barrera and Ainlay (1983) noted '*The growth of research on social support has led to a comparable proliferation in the ways it is conceptualized and operationalized*' while more recently Berkman and Glass (2000, p. 137) commented that '*When investigators write about the impact of social relationships upon health, many terms are used loosely and interchangeably*…'. However, there has been little rigorous investigation of the

measurement of social relationships within epidemiological studies. Relatively few studies have considered the impact of more objective measures of social relationships upon health. Furthermore, the effects of social relationships upon different health outcomes have rarely been investigated within the same cohort of older people. Finally, there is a paucity of information concerning the effects of social relationships upon health of older Australians.

1.2 OBJECTIVES

This thesis aims to investigate the effects of the structural aspects of social relationships – that is, social networks – on health among participants in the Australian Longitudinal Study of Ageing (ALSA), an epidemiological study of older people living in Adelaide, South Australia. Because health is such a broad concept, it was operationally defined using the three specific outcomes of disability, residential care use and mortality in this thesis. Strictly speaking, use of residential care is a health service utilization outcome rather than a health outcome, but it is considered in the latter sense in this thesis as a key point that can arise in the continuum between good health and death. The specific objectives of the thesis were to:

1. Develop a measurement model of social networks.
2. Examine the effects of total and specific social networks upon disability.
3. Determine the effects of total and specific social networks upon use of residential care.
4. Investigate the effects of total and specific social networks upon survival.

These are outlined more fully at the end of Chapter 2.

1.3 STRUCTURE OF THIS THESIS

An overview of the definitions and models that have been proposed in the measurement of social relationships is presented in the next chapter. Chapter 2 also examines how social relationships may affect health through consideration of the Berkman model (Berkman et al., 2000). A review of the literature concerning the effects of social relationships upon disability, residential care use and mortality is then presented. While the aim of this thesis is to examine the health effects of social networks, related aspects of social relationships were examined so as to review the literature more comprehensively and delineate how this thesis contributes to the conceptual framework.

Chapter 3 outlines the methods used in the ALSA, and presents some simple descriptive statistics concerning the study participants. The statistical methods used in this thesis are presented in Chapter 4. Chapter 5 describes the derivation of the social network variables used in this thesis, and Chapters 6 through 8 present the results and discussion for the studies concerning the effects of social networks upon disability, use of residential care and survival, respectively. The final chapter discusses the results as a whole along with some implications of the results for public policy.

1.4 STATEMENT OF PARTICIPATION

My doctoral research involved the use of data collected as part of the first six waves of ALSA covering the period 1992 through to 2000. The activities for which I was solely responsible under the guidance of my supervisors were:

1. The conceptualization of the specific studies reported here;
2. Data management and analysis; and
3. Presentation and interpretation of results.

From June 1994 to March 1996 I was employed as a Biostatistician for ALSA. During this time, Waves 3 and 4 of ALSA were carried out and I oversaw the field data collection for these waves of the study.

Prizes

In October 2004 I was awarded the Best Student Presentation at the Australasian Epidemiological Association meeting for a presentation concerning the effects of social networks on mortality based on the results presented in Chapter 8. In December 2005 I received a Flinders University Media Award for the 'story with most mentions', concerning press and radio coverage of Giles, Glonek, Luszcz and Andrews (2005).

Publications that have arisen to date from my doctoral studies include:

Peer reviewed journal articles (see Appendix 1)

Giles, L. C., Glonek, G. F. V., Luszcz, M. A., & Andrews, G. R. (2007) Do social networks affect the use of residential aged care among older Australians? *BMC Geriatrics,* 7, 24. [relevant to Chapter 7 in this thesis]

Giles, L. C., Glonek, G. F. V., Luszcz, M. A., & Andrews, G. R. (2005) Effect of social networks on 10-year survival in very old Australians: the Australian Longitudinal Study of Ageing. *Journal of Epidemiology and Community Health,* 59, 574-579. [relevant to Chapter 8 in this thesis]

Giles, L. C., Metcalf, P. A., Glonek, G. F. V., Luszcz, M. A., & Andrews, G. R. (2004) The effects of social networks upon disability in older Australians. *Journal of Aging and Health,* 16, 517-538. [relevant to Chapter 6 in this thesis]

Giles, L. C., Metcalf, P. A., Anderson, C. S., & Andrews, G. R. (2002) Social networks among older Australians: A validation of Glass' model. *Journal of Cancer Epidemiology and Prevention,* 7, 195-204. [relevant to Chapter 5 in this thesis]

Conference Presentations

Giles, L. C., Glonek, G. F. V., Luszcz, M. A., & Andrews, G. R. (2006) *The effects of diverse social networks on residential care use.* Paper presented at the Australian Association of Gerontology conference. November; Sydney, New South Wales.

Giles, L. C., Glonek, G. F. V., Luszcz, M. A., Crotty, M., & Andrews, G. R. (2006) *Effects of social networks on use of residential care: Results from the Australian Longitudinal Study of Ageing.* Poster presented at the New Zealand Statistical Association/Statistical Society of Australia Joint Annual Meeting. July; Auckland, New Zealand.

Giles, L. C., Glonek, G. F. V., Luszcz, M. A., Crotty, M., & Andrews, G. R. (2005) *The effects of social networks on nursing home admission among older Australians.* Paper presented at the Public Health Association (SA Branch) conference. October; Adelaide, South Australia.

Giles, L. C., Glonek, G. F. V., Luszcz, M. A., & Andrews, G. R. (2004) *We get by with a little help from our friends: 10 year survival in the Australian Longitudinal Study of Ageing*. Poster presented at the XVIIth International Biometrics Conference. July; Cairns, Queensland.

Giles, L. C. (2002) *The effects of social networks on physical function in older people.* Paper presented at the Health Policy, Practice and Research conference. May; Auckland, New Zealand.

Giles, L., Metcalf, P., Anderson, C., & Andrews, G. (2001) *The effects of social networks on disability in older people*. Paper presented at the Australasian Biometrics and New Zealand Statistical Association Joint Conference. December; Christchurch, New Zealand.

2 LITERATURE REVIEW

2.1 BACKGROUND

Three decades ago, two reviews of the effects of an individual's social environment on their health were published almost simultaneously (Cassel, 1976; Cobb, 1976). The risk of diseases associated with a range of social relationships and phenomena was summarized by Cassel (1976). He reached the conclusion that epidemiologists had failed to recognize the importance of social relationships to health. Concurrently, Cobb (1976) summarized the protective role that social relationships may play across the life span in many physical and psychological conditions. Cassel (1976) and Cobb (1976) independently concluded that social relationships had direct effects on health status, and also served to buffer the effects of psychosocial and physical stresses on the health and well-being of individuals.

This founding work by Cobb and Cassel, along with influential articles by Berkman and Syme (1979), Syme and Berkman (1976) and Kaplan et al. (1977) led to the formal recognition of the field of 'social epidemiology' (Berkman, 1986). Social epidemiology has been defined as 'the branch of epidemiology that studies the social distribution and social determinants of states of health' (Berkman and Kawachi, 2000 p. 6). The aim is the identification of socio-environmental exposures that may be related to a broad range of physical and mental health outcomes. The socio-environmental exposures that have been considered in an enormous range of studies have encompassed societal level factors – such as culture, socioeconomic status, politics and social change – along with structural and functional aspects of social relationships.

Social epidemiology, although a term coined relatively recently, was strongly influenced by Durkheim's classic analysis of social relationships and suicide

(Durkheim, 1951). Durkheim compared suicide rates among regions in a classic ecological study. According to Durkheim, people have a certain level of attachment to their social groups and institutions, through the family, church, workplace and political structures; Durkheim termed this 'social integration'. He theorized that the level of social integration of the group is related to suicide rates, and that suicide is triggered by the undermining of society's capacity for integration (Berkman et al., 2000).

Despite this original use of the term 'social integration', it has come to be used in a much looser fashion in more recent times. Over the past three decades, numerous studies that have examined the effects of social relationships on the health of individuals have appeared, and almost as many terms as studies have been used to describe social relationships (Antonucci et al., 1996). *Social relationships*, *social integration*, *social networks*, *social support*, *social ties*, *social engagement*, and *social anchorage* have all been used, often interchangeably and hence imprecisely (O'Reilly, 1988). Several authors (Orth-Gomér and Unden, 1987; O'Reilly, 1988; Bowling, 1997; Berkman et al., 2000) have contended that much of the research concerning social relationships has no conceptual definition of terms as a basis. Common definitions and a solid conceptual framework are needed to guide future research endeavours and to synthesise existing findings of the effects of social relationships upon health (Berkman et al., 2000). In Section 2.3 definitions of these commonly used terms are presented and expanded on.

Before this though, the conceptual model presented by Berkman et al. (2000) is described to place various aspects of social relationships in context with respect to each other and to health outcomes. The distinction between structural and functional aspects of social relationships themselves are also reviewed in the next section before the

effects of social relationships on specific health outcomes, with a focus on disability, residential care use and mortality, are reviewed.

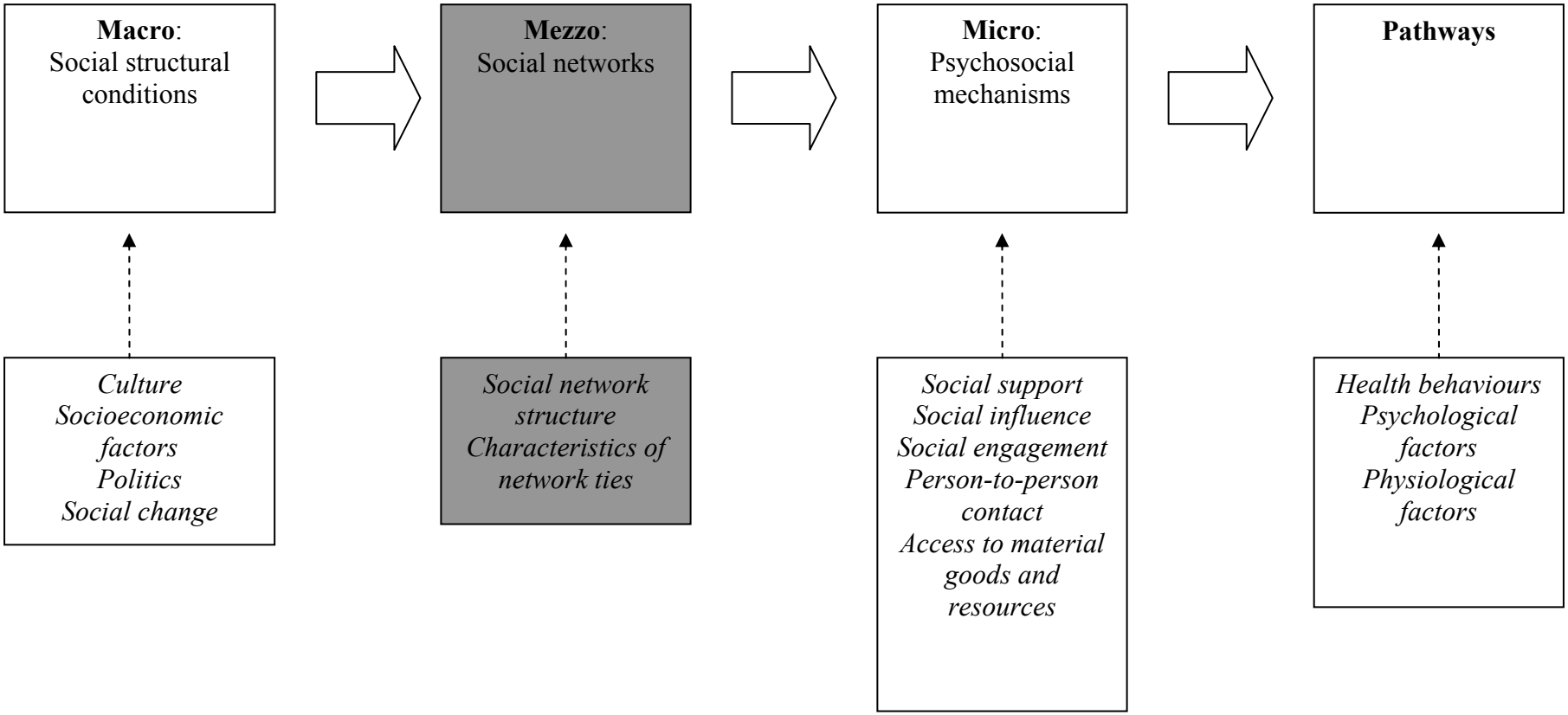
2.2 THE BERKMAN MODEL OF THE EFFECTS OF SOCIAL NETWORKS UPON HEALTH

Although there have been many studies that relate social relationships to health outcomes (as discussed further in Section 2.6), there have been few models that present a conceptualization of how health is affected by social relationships. Recently, Berkman et al. (2000) presented a broad conceptual model of how social networks may impact on health (Figure 2.1). The Berkman model proposes that social relationships have the potential to positively influence health in several ways through psychological, behavioural and physiological processes (Berkman et al., 2000).

The model is framed in terms of a cascading causal process that starts with the macro-social context in which social networks operate. Berkman et al. (2000) termed these 'upstream forces'. These larger social and cultural factors may condition the extent, shape, nature and structure of social networks, and may include socioeconomic status, culture, political forces, and processes of societal change.

The structure of social networks and interaction between members of social networks are described as the 'mezzo' level in Berkman's model. Structural components, such as network size, homogeneity and proximity of network members along with aspects of networks that relate to interaction, such as frequency of contact and reciprocity of contact, shape the definition of social networks employed by Berkman. The primary interest in this thesis concerns the effects of the mezzo level of social networks on health.

Figure 2.1: Conceptual model of the influence of social networks on health



adapted from Berkman et al. (2000); shaded area highlights the focus of this thesis

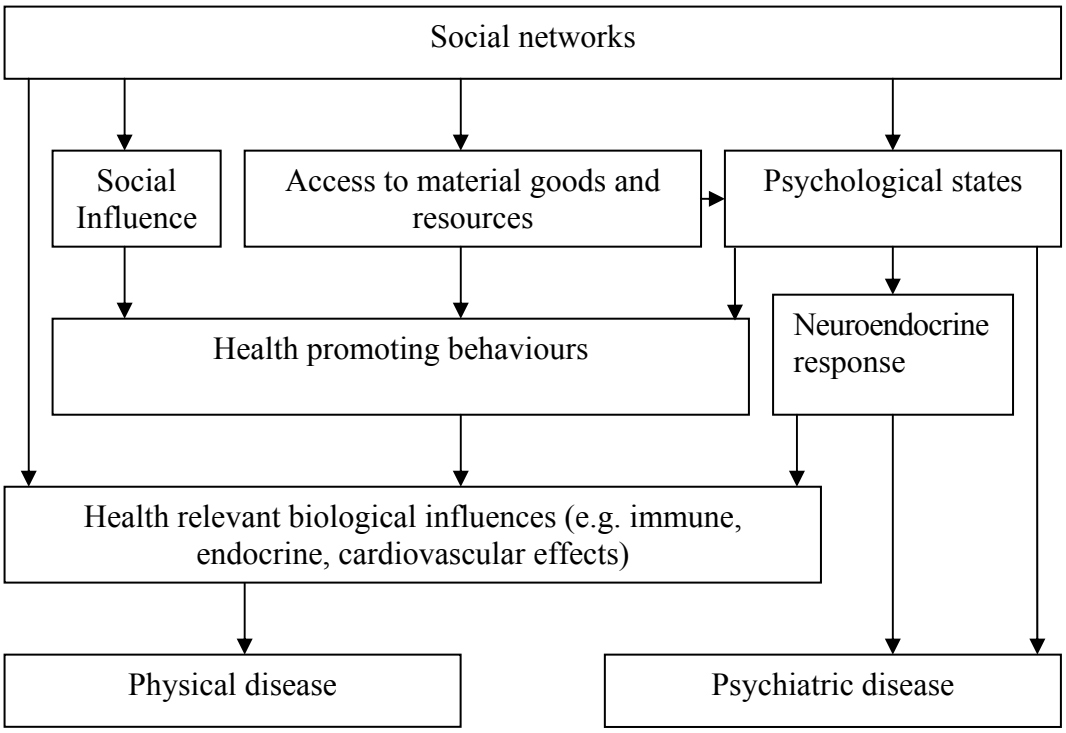
The ‘micro’ level in the model includes the ways in which social networks may function, through the provision of opportunities for social support and engagement in social activity. Berkman’s model contends that social networks operate at the micro level in four main ways, namely i) the provision of social support, ii) social influence, iii) social engagement and iv) access to material goods and resources.

This ‘micro’ level of social network function is then hypothesised to influence health through a number of pathways, and these may include health behavioural, psychological and physiological routes. For example, cognitive and emotional states such as self-esteem, coping, depression and sense of well-being are psychological factors that may be affected by social support. However, there is considerable scope for reciprocity of effects at this level, since these psychological factors may also influence social support and in turn health. Similarly, diet, exercise and help-seeking behaviour are health behavioural pathways that may be influenced by the function of the social networks, and in turn these behaviours can influence health. From a physiological perspective, psychosocial mechanisms may be important due to their influence on immune system function or neuroendocrine activation or allostatic load, as several reviews on this topic led by Seeman (Seeman and McEwen, 1996; Seeman, 2000) and Uchino (Uchino et al., 1996; Uchino, 2006) have shown. Thus the ways in which the micro level of social networks can influence health are complex, and not well understood (Berkman, 1985).

Underpinning the concepts of social relationships and their consequences for health are two broad hypotheses, namely the *direct effects* hypothesis and *buffering effects* hypothesis (House, 1981; Cohen and Wills, 1985; Cohen, 1988). The direct effects hypothesis, also referred to as the main effects hypothesis, suggests that social relationships enhance health and well-being, irrespective of other stressors (Cohen and

Syme, 1985). Cohen's conceptualization of the main effects that social relationships may have on health is illustrated in Figure 2.2 (Cohen et al., 2000, p. 11). In relation to the Berkman et al. (2000) model, it is the mezzo-level social networks that are the foundation for the flow-on direct effects shown in Figure 2.2.

Figure 2.2: Putative pathways through which social relationships have effects on health

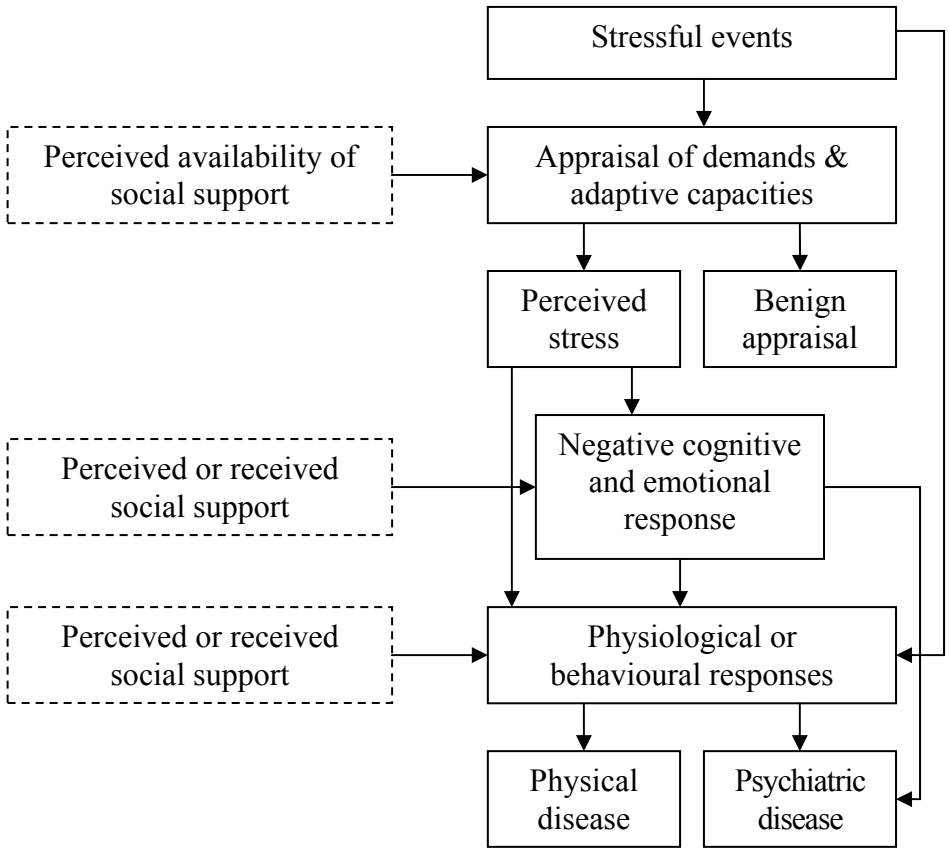


adapted from Cohen et al. (2000, p. 11)

The buffering effects hypothesis offers an alternative explanation to that shown in Figure 2.2. This hypothesis suggests that social relationships have beneficial health effects in the presence of stressors through protecting people from the pathogenic effects of the stressors (Cohen and Syme, 1985). Social support is hypothesised to cushion the impact of negative events or stressors. House (1981) suggested that social relationships may act as buffers against stressors in three ways: i) they may reduce the importance of the perception that a situation is stressful, ii) they may affect the

neuroendocrine system so that the reaction to perceived stress is less, and iii) that social relationships may promote healthy behaviours. Cohen and Syme (1985) suggested that social support acts a stress buffer through the belief that others will provide appropriate aid when needed. Figure 2.3 presents Cohen's conceptualization of how social relationships mediate stressful life events. With reference to the Berkman et al. (2000) model, it is the micro-level – that is, psychosocial mechanisms, specifically in the form of social support – that act to buffer against the effects of stress.

Figure 2.3: Pathways through which social relationships influence responses to stressful life events



adapted from Cohen et al. (2000, p. 12)

The direct and buffering effects hypotheses are not mutually exclusive, and there is evidence that both kinds of effects of social relationships may have positive health

consequences (Cohen and Syme, 1985). Under a long-term or life-span perspective, simultaneous direct and buffering effects of social relationships on health are feasible and possible (Antonucci et al., 1996).

However, in this thesis, the direct effects hypothesis was primarily explored, albeit implicitly. This is because the primary interest was how different types of social networks, at the mezzo level in Berkman's model, might affect health. Measures of social networks can be made more objectively than those of social support, and this led to the decision to concentrate on the former's effect on health in this thesis. To progress understanding of the distinction between social networks and social support, more detailed definitions of social networks, social support and related terms are now presented.

2.3 DEFINITIONS AND MEASUREMENT OF SOCIAL RELATIONSHIPS

As already noted, a wide range of related terms and concepts are used in the literature to describe social relationships. In keeping with the conceptual model described in Section 2.2, the definitions outlined in Berkman et al. (2000) are largely used in this review, although the definitions clearly explicated by Antonucci et al. (1996) have also informed the way these terms are used throughout this thesis.

The term *social networks* is used to describe the structural or objective characteristics of an individual's social relationships. *Social ties* characterize the relationships among people in a social network. *Social support* describes the exchange of resources, goods or services between or among social network members. *Social engagement,* often used interchangeably with the term *social participation,* is used to describe the involvement of people with others, usually via membership of groups or religious organizations.

Others have used the term *social anchorage*, defined as the degree to which an individual belongs to and is anchored within formal and informal groups (Hanson et al., 1989). Kawachi et al. (1996) and Kaplan et al. (1988) have used the term *social connections* in a similar way.

Social integration has been used somewhat imprecisely in the literature. Berkman et al. (2000) described social integration in terms of embeddedness and involvement with others across the spectrum from intimate to extended social ties. Under this definition, social integration reflects the degree to which an individual feels integrated into the community and society through their social relationships with others. In contrast, and more akin to social engagement, Brissette et al. (2000) defined social integration as participation in a broad range of social relationships. Antonucci et al. (1996) proposed a somewhat different definition, that social integration is an outcome of optimally existing social networks and social support. Each of these authors has used a term that relates to an individual's perspective. Classically, however, social integration was used in a broader 'macro' sense, reflecting societal norms, values and levels of cohesiveness (Durkheim, 1951). Given these varied ways in which social integration has been described, the use of this term is generally avoided in this thesis. *Social relationships* is used in this thesis as an umbrella term capturing social networks, social support and social engagement (Antonucci et al., 1996).

These broad definitions of social networks, social support and social engagement, and examples of their operational use in epidemiological studies, are now further described.

2.3.1 Definition of social networks

Social networks describe the structural or objective characteristics of the social relationships maintained by an individual (Antonucci et al., 1996). Walker et al. (1977) defined social networks as the set of personal contacts through which an individual maintains their social identity, and receives various kinds of social supports. Another definition of social networks is the web of social relationships that surround an individual and the characteristics of those ties (Mitchell, 1969, Fischer, 1982).

As discussed by Hall and Wellman (1985), social networks are commonly defined in terms of a number of structural components, which these authors further dichotomized and described as features of social ties or social networks (as shown in Table 2.1). Hall and Wellman separated the characteristics of the social network itself from the characteristics of the individual social ties.

Other authors have not elaborated the distinction between social ties and social networks, and others still have operationally defined social networks using one or more characteristics drawn from network size, geographic dispersion, strength of ties, density, composition and homogeneity (Mitchell, 1969; Craven and Wellman, 1974; Walker et al., 1977). Irrespective of the specific components used, the focus of such a definition of social networks is on the *structural* properties of the relationships that exist for members of social networks. It is this concept that is the main focus of this thesis.

Table 2.1: Measures of social network characteristics and social tie characteristics[1]

Network characteristics	
Range (size)	Number of network members (nodes)
Density	Extent to which a network is interconnected overall by means of direct ties, measured by comparing the actual number of direct ties between network members with the number of ties that would exist if all members were directly connected
Degree	Extent to which a network member is tied directly to other network members
Boundedness	Proportion of all ties of network members that stay within the network's boundary
Reachability	Average number of ties required to link any two network members
Homogeneity	Extent to which network members have similar personal attributes
Cliques	Portion of networks in which all members are tied directly; has density of 1.0
Clusters	Portions of networks with high density, but defined by less stringent connectivity criteria than cliques
Components	Portion of networks in which all members are tied directly or indirectly
Ties characteristics	
Strength	Quantity of resources characterising a relation
Frequency	Quantity of contact between two network members (i.e. number of times a resource flows between the two)
Multiplexity	Number of different resources exchanged by two network members in a tie (i.e. number of relationships between the two)
Duration	Length of time a tie or relation has existed
Symmetry	Extent to which resources are both given and received
Intimacy	Perceived social closeness of network member by another

1: adapted from Hall and Wellman (1985, p. 28)

2.3.2 Measurement of social networks

2.3.2.1 Social network analysis

There are two distinct ways in which social networks are described and analysed, depending largely on disciplinary orientation. Social network analysis (Wasserman and Faust, 1994), used by quantitative sociologists, uses graph theory to describe the web of social connections between individuals. Generally, this approach is not concerned with the individual characteristics of network members but with the structural properties of these connections. Two main kinds of networks are defined under this paradigm. Personal or egocentric networks describe the ties that surround a single focal individual. In contrast, aggregate or whole networks encompass the entire set of ties among members of a population (Brissette et al., 2000, pp. 70-71; Faber and Wasserman, 2002, p. 32). Studies of AIDS and HIV-infected populations have particularly benefited from the application of social network analysis through the consideration of entire networks (Trotter et al., 1995).

However, this paradigm has not been widely used in epidemiological studies. Instead, measures of social networks that focus on the relationship of a focal individual with others have generally been used. A considerable body of epidemiological research has used measures of social networks based on the numbers of children, friends, other relatives, and/or neighbours and contacts with these people over some defined time period. The viewpoint usually used, although not often stated, in such studies is that of egocentric networks – the set of direct social ties between an individual and others, not the full relational networks that are studied in social network analysis (Mitchell, 1969).

This 'epidemiological' definition of social networks is the focus of this thesis. In this sense, the use of the term *social networks* here is something of a misnomer, and some

have taken umbrage at its use in this way (Hall and Wellman, 1985). However, the techniques of social network analysis have not been widely employed in the epidemiological literature, and in keeping with this body of work and the use of terms in the epidemiological literature, the term social networks is used in the 'metaphoric' sense throughout this thesis.

2.3.2.2 Network typologies

Aside from measures of total social networks, some authors have distinguished between different components of social networks. Two main and distinct definitions of kinds of networks have appeared in the literature: i) the classification of individuals into one of a range of 'network typologies' based on the composition of their social networks (e.g. diverse network, integrated network), and ii) the classification of individuals across a range of specific network types (e.g. some measure of children networks, friends networks, etc. as developed by Glass et al., 1997).

The concept of 'network typology' was first described by Wenger in a series of manuscripts (Wenger, 1984; 1986; 1989; 1991; 1996). Her definition included a range of variables concerning the structure of social networks and the interaction between members of the network. The term 'network typology' has been used by Wenger to describe the dominant composition of an individual's network. Five distinct network typologies have been described by Wenger, namely:

- *family dependent network* that consisted of a small group that relied primarily on close relatives living in the same community;
- *locally integrated network* that included relationships with family, neighbours and friends;

- *local self-contained network* that was small and reflected a household-centred lifestyle with reliance on neighbours if essential;
- *wider community-focused network* that was large, associated with an absence of local kin, and mainly centred on friends and involvement in community groups; and
- *private-restricted network* that was characterized by no kin living nearby and minimal social ties with neighbours and the community.

However, the data used as the basis for this work were drawn from a sample of only 25 Welsh people aged 79 years or more, and thus the generalizability of this approach to other settings is unclear. Furthermore, within each of the network typologies, the relationship of individuals to the focus person is masked.

Mugford and Kendig (1986) described network typologies among 910 older Australians in terms of network size and diversity of linkages between individuals (termed 'multiplexity' in the definition by Hall and Wellman). Mugford and Kendig (1986) suggested that a typical network for an older Australian had about three close relatives, one more distant relative, two friends, and a neighbour or other person. Mugford and Kendig (1986) used their results to construct a network typology among older people, defined by size and multiplexity. These authors hypothesised the existence of five typologies, namely

- *balanced*, which was a network of medium size and with medium multiplexity;
- *attenuated*, a network of low size with low multiplexity;
- *intense*, a low-size network with high multiplexity;
- *diffuse*, a network that is high in size but of low multiplexity; and
- *complex*, a network of high size with high multiplexity.

While Mugford and Kendig (1986) presented case studies of different network typologies, the network typologies do not appear to have been related to any health outcomes subsequent to the publication of this work.

Litwin (2001) also created five network typologies in a sample of 2079 older Jewish people who had lived in Israel for some time. The five typologies were derived from a cluster analysis of variables that reflected social networks and social engagement, namely marital status, proximate children, frequency of contact with children, friends and neighbours, frequency of attendance at a synagogue, and frequency of attendance at a social club. The typologies described in this research were similar to those of Wenger, although labelled somewhat differently:

- *diverse network*, in which the majority of members were married, had on average one child who lived in close proximity, had very frequent contact with children, friends, and neighbours and attended the synagogue a moderate amount;
- *friends network*, which were similar to the diverse network with the exception of little contact with neighbours;
- *neighbours network*, in which there was a higher prevalence of widowhood, and members reported frequent contact with adult children and neighbours but not friends;
- *family network*, in which members had an average of five proximate children, reported very frequent contact with them, attended the synagogue frequently, and had minimal social ties with neighbours and friends; and
- *restricted network*, in which members most likely did not have a spouse, less frequent contact with adult children, and very little contact with friends and neighbours.

Five distinct network typologies were also reported among older people in Finland by Melkas and Jylhä (1996), with the five typologies again similar to those reported by Litwin (2001) and Wenger (1989; 1991).

Common to each of these definitions is the classification of an individual into one of a range of mutually exclusive categories of social networks. However, there is no recognition in this approach of the overlap that may exist between categories. For example, the distinction between diverse and friends networks in the work by Litwin (2001) is based solely on the frequency of contact with neighbours. Thus two individuals may actually be very similar in terms of their overall social networks and yet be classified as having different network typologies.

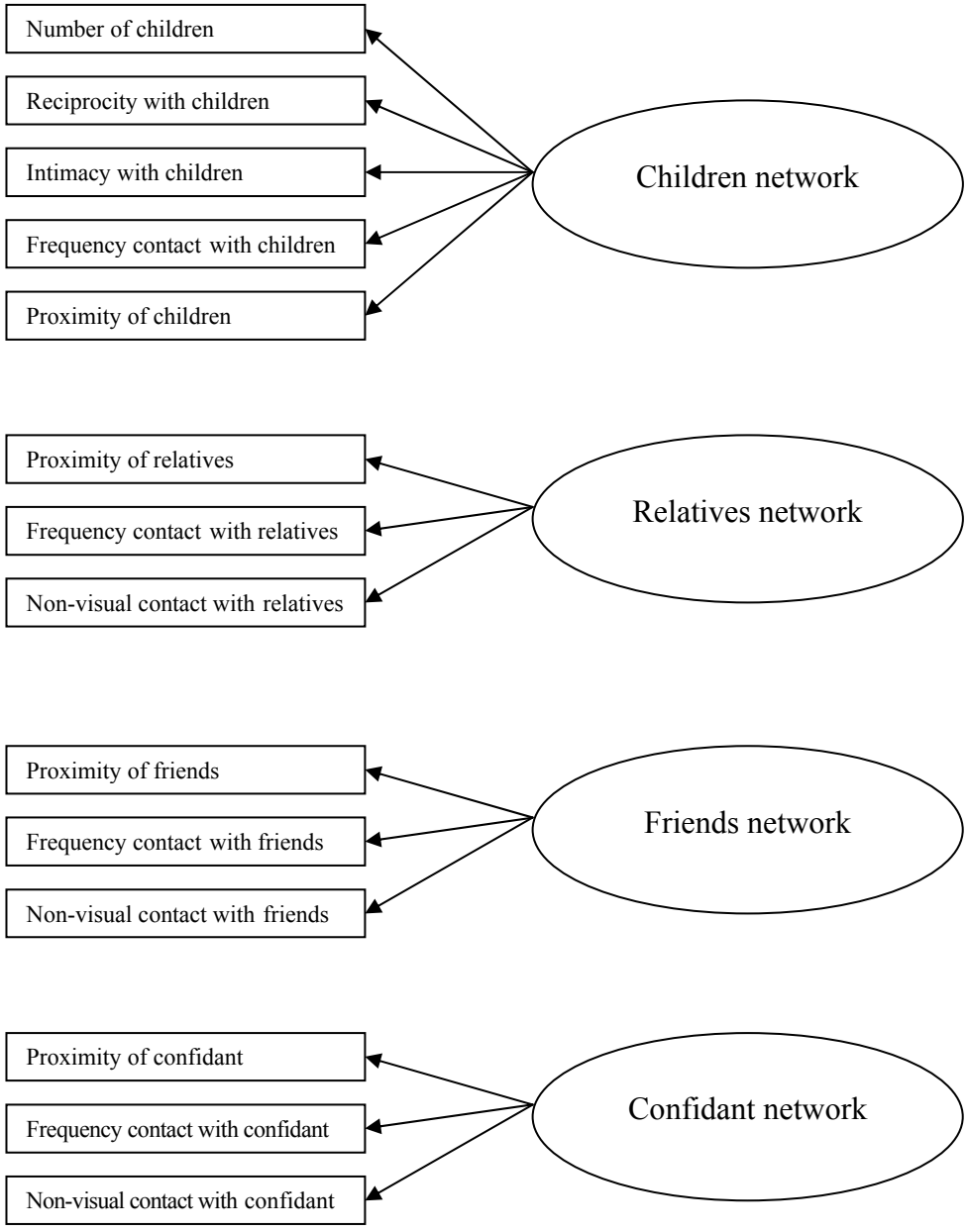
2.3.2.3 Network type

An alternative to the categorization of individuals into a single network type is to classify individuals according to a range of relationships with others. Such an approach was described by Glass et al. (1997), who developed a measurement model of specific social networks and a summary measure of total social networks using data from 2812 participants aged 65 or more from the New Haven, Connecticut site of the Established Populations for Epidemiologic Studies of the Elderly (EPESE).

In their work, Glass et al. (1997) proposed and validated a measurement model of distinct and complementary social networks in four categories – with children, other relatives, friends and confidants – using confirmatory factor analysis. Glass et al. (1997) argued the need to consider the strength of each specific network separately because of the different social roles potentially fulfilled by different relationships with various people (Cohen and McKay, 1984). This role-specific viewpoint is expanded on

in Section 2.4.2. In Glass's model, both the number and frequency of contact with people in each type of network were used to construct summary scores for each specific type of network. A total social network measure was calculated as the sum of the four component network variables. In contrast with the social network typology advocated by Wenger and others, the approach of Glass et al. allows a multi-dimensional view of an individual's social network with family and friends. Importantly, Glass's approach also separates out kin and non-kin relationships. A schema of the model developed by Glass et al. (1997) is presented in Figure 2.4. This highlights how individual variables may be combined in the derivation of multi-dimensional measures of social networks.

Figure 2.4: Conceptual model of social networks



adapted from Glass et al. (1997)

In summary, the objective measurement of social networks has been operationalized in epidemiological studies by authors in a variety of ways, all of which draw on the morphological characteristics of an individual's relationships with other people. The ways in which social networks may operate, at the micro level through social support and social engagement, are now examined.

2.3.3 Definition of social support

As described in Section 2.2, Berkman et al. (2000) hypothesised that social networks exert their influence on health partly through providing the structures from which social support can emanate. Historically, *social support* is a term that has been used very loosely, as reviewed by a number of authors (Orth-Gomér and Unden, 1987; O'Reilly, 1988; Bowling, 1991; 1997). Measures that are purported to be of social support often actually reflect components of social networks or social engagement (O'Reilly, 1988).

Social support was defined by Cobb (1976) as 'information leading the subject to believe that he [sic] is cared for and loved, esteemed and a member of a network of mutual obligations'. Thus the term social support refers to the actual exchange of something within the relationship. Social support has also been referred to as functional support (Berkman, 1985), although this definition has been refined more recently by Due et al. (1999) to include the *level of resources* provided by other persons. The functional support definition recognizes that the existence of a social network does not automatically imply that positive social exchanges derive from it (Rook, 1992, 1997; Antonucci et al., 1998).

Social support is conventionally defined in terms of *emotional*, *instrumental*, *appraisal*, and *informational* support (Weiss, 1974a). Emotional support is related to the amount and receipt of love, caring, sympathy, and understanding from others (Thoits, 1995). Usually, emotional social support is provided by a confidant or network of confidants (Berkman et al., 2000). Instrumental support refers to help, aid or assistance with tangible needs in day to day life, including household shopping, cooking and cleaning. House and Kahn (1985) used the term instrumental support to refer to aid in kind, money or labour. Appraisal support relates to the provision of information for self-

evaluation (House and Kahn, 1985), and may include help in making decisions and giving feedback (Bowling, 1997; Berkman et al., 2000). Finally, informational support is provided through advice, education or instruction about access to services and other resources (House, 1981; Bowling, 1997).

2.3.4 Measurement of social support

Social networks need to be characterized so as to be able to adequately measure support that flows from a network. Remarkably few studies have considered the existence and nature of a social network as a necessary condition from which social support, of any kind, can flow. A range of measures of social support have appeared in the literature, either as stand-alone instruments or developed as part of an epidemiological study. The measures distinguish between the perception and actual receipt of support, although some measures capture both of these aspects of social support. Emotional social support and instrumental social support have most commonly been considered.

Briefly by way of example, Barrera et al. (1981) developed an Inventory of Socially Supportive Behaviours that measured received support. Sarason et al. (1983) developed the Social Support Questionnaire to measure the perceived availability of, and satisfaction with, social support. Henderson et al. (1980) developed the Interview Schedule for Social Integration that assessed both the availability and perception of social support; Unden and Orth-Gomér (1984) subsequently abbreviated this questionnaire for use in epidemiological studies. Koenig et al. (1993) developed a brief version of the Duke Social Support Index that was specifically created for use with older people. This abbreviated questionnaire has been validated for use with older Australians (Goodger et al., 1999). Despite its name, aspects of social networks as well

as social support are included in the Duke Social Support Index. No single measure of social support appears to dominate in the literature.

In a review of 24 measures of social support, O'Reilly (1988) concluded that many of the items that supposedly measured social support were actually measures of social participation, social isolation, or components of social networks. He cautioned that 'something that measures everything ends up measuring nothing'. In a similar vein, Bowling (1997) contended that there had been little attempt to assess measures of social support in terms of validity and reliability.

Orth-Gomér and Unden (1987) reviewed 17 instruments that measure social relationships, of which 13 were clearly measures of social support. The remainder assessed social networks or social engagement. Of the measures of social support, Orth-Gomér and Unden (1987) concluded that they were generally time-consuming to complete and their predictive capacity was not adequately reported. Of particular relevance to studies involving older people, many questions were classed as difficult to understand. Orth-Gomér and Unden (1987) also found the conceptual framework underpinning each of the 13 instruments varied widely.

Thus despite decades of debate and research, the valid and reliable measurement of social support remains contentious. There is no clear consensus on measures that adequately capture the various dimensions of social support. The distinction between perceived and received social support is often not made in epidemiological studies, or different types of social support are combined in one summary measure. The issue of measurement error (Bland and Altman, 1996) in social support is rarely addressed, although such subjective measures may be affected by large measurement error that

correlates with an individual's characteristics and behaviours (Bertrand and Mullainathan, 2001). Even with these noted limitations, many authors have considered the effects of social support on specific health outcomes, as reviewed in Section 2.5.

2.3.5 Definition of social engagement

The term *social engagement* has been used to describe an individual's involvement in the community, usually through membership of religious and other organizations. Social engagement has most commonly been operationalized by counts of organizational relationships or social activity that involves either going somewhere other than usual residence (for example, to the movies or to church) or involves contact with other people. As is the case for social support, it can be argued that the foundation of social networks is necessary for social engagement to take place.

Berkman et al. (2000) discussed social participation and social engagement jointly, and described the two concepts as

> '*result[ing] from the enactment of potential ties in real life activity. Getting together with friends, attending social functions, participating in occupational or social roles, group recreation, church attendance – these are all instances of social engagement.*'

2.3.6 Measurement of social engagement

Glass et al. (1999) measured 'engagement in social activity' through a total frequency measure across 14 types of activity including church attendance, visits to cinema, restaurants or sporting events, day or overnight trips, playing cards, games, bingo, and participation in social groups. Mendes de Leon et al. (2003) derived a related measure of social engagement using data from the same cohort as Glass et al. (1999). These

authors used 11 questions concerning social or productive activities (visits to theatres or sporting events, meal preparation, gardening, shopping, day or overnight trips, game playing, church attendance, participation in groups, paid community work, unpaid community work, paid employment, participation in groups) to derive a measure of social engagement scored from 0 to 23.

Other authors have conceptualized social engagement as organizational relationships. For example, House et al. (1982) examined formal organizational involvements outside of work, such as going to church or meetings of voluntary associations, active and relatively social leisure (such as going to movies) and passive and relatively social leisure (such as watching television). As well as considering a measure of social networks, Jylhä and Aro (1989) developed an index of social engagement based on social activities (e.g. theatre, movies, and travel) in the previous 12 months. Liu et al. (1995) and Simons et al. (1996) examined organizational attendance as a measure of social engagement.

Despite its name, the Social Network Index (SNI) developed by Berkman and Syme (1979) is a blend of items that cover both social networks and social engagement. From the Berkman model, it is clear that the SNI is a mixture of items at two levels – the mezzo level (networks) and micro level (engagement). The SNI is based on a weighted sum of indicators of marital status, contacts with close friends and relatives, church membership, and informal and formal group membership. Many subsequent studies have used a measure based on the SNI (e.g. Schoenbach et al. 1986; Seeman et al., 1987; Michael et al., 1999; Berkman et al., 2004).

However, all of the abovementioned measures of social engagement may be problematic in studies that examine their effects on health. For example, attendance at church generally requires some minimum level of physical and cognitive health. A measure of social engagement that includes church attendance is therefore likely to be confounded with health status. Similarly, attendance at club or group meetings generally requires a certain minimum of physical and cognitive ability.

Indeed, Dean et al. (1994) described serious concerns with the face and construct validity of the SNI in particular. These authors concluded that significant item bias in the variables from which the SNI is composed render it invalid as a measure in investigations of the influence of social relationships on health. However, similar problems are evident in other measures of social engagement. For example, many of the items in the measures developed by Glass et al. (1999) and Mendes de Leon et al. (2003) involve incident physical activity (e.g. gardening, paid employment). Thus confounding may be a significant issue in analyses concerning the effects of social relationships on health, and this appears to be inadequately addressed in many studies in this area. This point is returned to in Section 2.5 when individual studies that examine the effects of social relationships on health are reviewed.

2.3.7 Summary

The measurement of social support has often lacked a conceptual basis and can be difficult to measure in practice. The measurement of social engagement is also difficult, as it may be strongly correlated with both physical and psychological health. Social networks can be measured more objectively than either social support or social engagement, and the obvious problem of confounding with physical and cognitive health status is avoided to some extent. Consideration of different network types may

provide additional insight into the effects of social networks on health that measures that do not differentiate between different network types cannot provide. Furthermore, social networks may be less correlated with health and psychological status than micro level measures of social support and social engagement. For these reasons, social networks are the focus of this thesis, rather than social support or social engagement.

Before reviewing the literature concerning social networks and specific health outcomes in Section 2.5, a greater examination of how social relationships might operate independent from health outcomes may be salutary. Thus models of social relationships are described briefly in Section 2.4.

2.4 MODELS OF SOCIAL RELATIONSHIPS

The model proposed by Berkman et al. (2000) draws on elements of several smaller conceptual models that cross several disciplinary boundaries. Several theoretical models of social networks and social support have been proposed and these are outlined in this section. For social networks, the work by Hall and Wellman (1985) in describing a structural model (as described in Section 2.3.2) and Cohen and McKay (1984) in relation to a role-specific model have been influential. Functional aspects of social relationships have been described in models concerning 'Convoys through the life course' (Kahn and Antonucci, 1980), socio-emotional selectivity theory (Carstensen, 1991; 2003) and the functional-specificity model (Simons, 1983-1984; Weiss, 1974b). These three models provide a foundation that helps to understand how social support for older people may operate. Theoretical models that focus on the receipt and provision of social support have also been proposed and are outlined in this section. This is done in the interest of a comprehensive review of the literature, although it is noted that these are not directly relevant to the focus on structural aspects of social relationships in this

thesis. Among these latter models are the hierarchical-compensatory model (Cantor, 1979), the substitution model (Shanas, 1979), and the task-specificity model (Litwak, 1985).

2.4.1 Structural model

According to Hall and Wellman (1985), the morphology of the social network described by the characteristics in Table 2.1 provides the optimal description of the social network. Hall and Wellman (1985) argued for more comprehensive network analysis that moved away from an individual view to a structural view. Studies that simply use one aspect of social networks (e.g. network size) have been criticized for being uni-dimensional (Glass et al., 1997) and psychologically naïve (Antonucci, 1986 cited in Glass et al. 1997).

However, there are few epidemiological studies that have operationalized the structural approach advocated by Hall and Wellman (1985). The research described by Glass et al. (1997) is an exception to this. The structural model of Hall and Wellman is consistent with the direct effects hypothesis, as it is the resources that derive from the social networks, and the networks themselves, that are of primary importance in this approach.

2.4.2 Role-specificity model

Cohen and McKay (1984) presented an alternative way of characterizing social networks that focussed on the role-specificity of particular network ties. Under this model, social networks are defined according to the nature of social roles to which ties are attached, such as ties with children, relatives and friends (Glass et al., 1997; Argyle, 1992). The structural model (Hall and Wellman, 1985; Mitchell, 1969) and the role-

specific model (Cohen and McKay, 1984) can be used concurrently by considering the structural components of social networks with respect to specific types of relationships with kin and non-kin, as outlined in Section 2.3.2.3.

2.4.3 Hierarchical-compensatory model

The hierarchical-compensatory model was put forward by Cantor (1979) to describe the way in which family and others are turned to by older people for social support. In this model, social support is given according to the relationship of the support provider to the recipient. Under this model, the primary providers of support were defined as the children living nearby followed by other relatives. Cantor (1979) demonstrated that it was the proximate children and relatives who were viewed as the most appropriate providers of social support. However, the type of social support provided was not clearly delineated in this research. Friends and neighbours were classed as secondary support providers, and were turned to next if primary support was unavailable. Finally, formal organizations were turned to for help if primary or secondary sources of support were not available. Cantor (1979) argued that if the initially preferred support provider was absent, then other groups stepped in and acted to compensate as a replacement. In this way, the hierarchy of preferred social support was demonstrated, with other network members compensating if a particular form of social support was not available.

2.4.4 Substitution model

Around the same time as the hierarchical-compensatory model was proposed, Shanas (1979) proposed a model that has subsequently been described as a substitution model (Campbell et al., 1999). Her research showed that widowed or never married older persons are especially dependent on siblings for social support. In the case of widowhood, Shanas (1979) argued that siblings assumed some of the responsibilities

that were formerly derived from the spouse. She also contended that nieces, nephews and other relatives were important network members. In essence, Shanas posited that relatives in particular substituted for each other in the absence of a spouse. The substitution model can be viewed as similar to the hierarchical-compensatory model, but without the implicit ordering of providers of social support.

2.4.5 Task-specificity model

The task-specificity model considers the match between the nature of the task and the characteristics of a particular relationship or individual (Litwak, 1985). By way of example, instrumental support for tasks that require someone nearby, such as cooking, are more likely to be carried out by a proximal member of the social network whereas emotional social support generally requires a member of the social network who is both proximate and companionable (Campbell et al., 1999). The task-specificity model suggests that particular kinds of social support needs are better suited to some specific types of social relationships than others (Litwak, 1985). Thus, predicting the most appropriate giver of social support requires the nature of the support required to also be taken into account. The model reinforces the importance of disaggregating social networks into specific social network types.

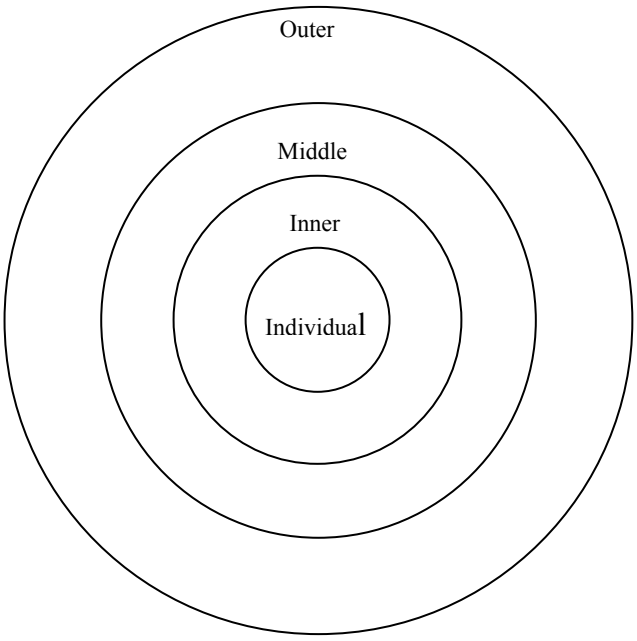
2.4.6 Convoy model

Kahn and Antonucci (1980, p. 269) state that 'each person can be thought of as moving through the life cycle surrounded by a set of people to whom he or she is related by the giving or receiving of social support'. The people that make up the social network of an individual is the convoy, and this can vary at different points in time across the life span. As individuals progress through life, their convoy is hypothesised to provide them with a foundation that has positive effects on psychological and physical health

outcomes. Antonucci and Akiyama (1987) described the convoy as the protective layer of family and friends who surround the individual and help in the successful negotiation of life's challenges.

Kahn and Antonucci (1980) used a schema of concentric circles, as exemplified in Figure 2.5, to demonstrate the relationships that surround an individual. Each circle represents a different level of closeness to the focal person, and the nature and amount of support offered by people in each of the layers or circles of the convoy differs (Antonucci and Akiyama, 1987). Those in the inner circle might give and receive many different kinds of social support, whereas those in the outer circle might be close to the focal person but in a very specific way and offer informational support (e.g. work colleagues whose relationship does not extend outside of the workplace; Antonucci and Akiyama, 1987).

Figure 2.5: Convoys through the life course social support model



2.4.7 Socio-emotional selectivity theory

Carstensen (1991; 1992; 1995; 2003) proposed the theory of socio-emotional selectivity that also takes a life span perspective of social relationships. Socio-emotional selectivity theory proposes that as people age, they become increasingly selective about the specific persons with whom they maintain a close social relationship. Reduced rates of social interaction among older people are viewed as the strategic selection over the life span of social networks that maximize social and emotional gains, and minimize social and emotional risks (Carstensen, 1991). In this light, socio-emotional selectivity theory can be argued to be a means through which the direct and buffering effects of social relationships are optimized as people age.

2.4.8 Functional-specificity model

The functional-specificity model derives from work by Weiss (1974b) and Simons (1983-1984). Weiss (1974b) focussed on middle-aged couples and the findings informed the development of the study of the social networks of 299 older Iowans by Simons (1983-1984). Simons showed that the spouse and adult children of older people served as a primary source of instrumental support, and the spouse acted as a primary source of an intimate, confiding relationship. However, if children and a spouse were absent, their roles in providing security were filled by confidant friends or siblings. There is thus some overlap with the hierarchical-compensatory model. Similar to the convoy perspective (Kahn and Antonucci, 1980), the functional-specificity model contends that social networks and social support are products of time and circumstance so that differences in who provides particular types of support at various times in the life span are expected (Simons, 1983-1984). For example, childless, unmarried older people may receive social support from their siblings because of how this relationship

has developed over time rather than as substitution or compensation for lack of a spouse and children (Connidis and Davies, 1992; Campbell et al., 1999).

2.4.9 Summary

The various models can be divided into three categories that are differentiated by their emphasis on: i) the structure of social relationships, ii) the function of social relationships, and iii) changes in social relationships across the course of life. The first of these categories draws together the structural model (Hall and Wellman, 1985) with the role-specificity model (Cohen and McKay, 1984). This led to the formulation of the specific social networks defined according to the type of relationship (Glass et al., 1997). The second category of models describe ways in which an individual's social network may function, be it through compensating (Cantor, 1979) or substituting (Shanas, 1979) for apparent deficiencies in the composition of social networks or through the recognition of specific needs being matched to the provider of support (Litwak, 1985). The final category draws on a life span perspective of social relationships, that have been conceptualized in terms of socio-emotional selectivity theory (Carstensen, 1991; 1992; 1995; 2003), the convoy model (Kahn and Antonucci, 1980; Antonucci and Akiyama, 1987) and the functional specificity model (Weiss, 1974b, Simons, 1983-1984), recognizing that the composition of an individual's social networks changes with age. Across the life span, it has been hypothesised that members of the social network are selected – either explicitly (Carstensen, 1995) or implicitly (Simons, 1983-1984) – to optimize the likelihood of positive emotional effects (Carstensen, 1991, 1995) or functional effects (Simons, 1983-1984) of the social network.

The focus of this thesis concerns the mezzo level of social relationships, and thus the first of these categories of models is most relevant. Nonetheless, the interpretation of effects of the social networks on health must also be cognizant of how social networks can function and change with time. Thus the models in the second and third categories will help to inform the discussion of the study results.

2.5 SOCIAL RELATIONSHIPS AND HEALTH OUTCOMES

Over the past thirty years, the effects of social relationships on a wide variety of health outcomes have been examined. For example, the effects of social relationships on psychological outcomes, such as cognition (Bennett et al., 2006; Fratiglioni et al., 2000), depression (Oxman et al., 1992; Glass et al. 2006), and morale or self-esteem (Litwin, 2001; Lee and Shehan, 1989) have been examined. Self-rated health was investigated in relation to social networks in older people by Litwin (2006) and Zunzunegui et al. (2004). The effects of social relationships on cardiovascular disease have also been extensively studied (e.g. Reed et al., 1983; Blumenthal et al., 1987; Seeman and Syme, 1987). The relationship between neuroendocrine function and an individual's social milieu was investigated by Seeman et al. (1994) and reviewed by Seeman and McEwen (1996). A broad review of the effects of social relationships on physiological processes was published by Uchino et al. (1996), with an updated review published recently (Uchino, 2006).

The most commonly considered outcome in the gerontological and epidemiological literature with respect to social relationships is that of all-cause mortality. For older people, disability and entry to residential care are also particularly important health outcomes. Disability and residential care use may also be markers of decline prior to death (although many older people die living in the community with little physical

disability). The effects of social networks on disability, residential care use, and mortality are therefore of considerable interest, and are the focus of this thesis. These three outcomes are important key points along the continuum from optimal health to death, and they will be considered in the natural order. The literature relevant to each of these three outcomes is reviewed in the subsequent sections of this chapter. Results are presented largely in their chronological order so that the incremental advances in knowledge concerning the effects of social networks on disability, residential care use and mortality are apparent.

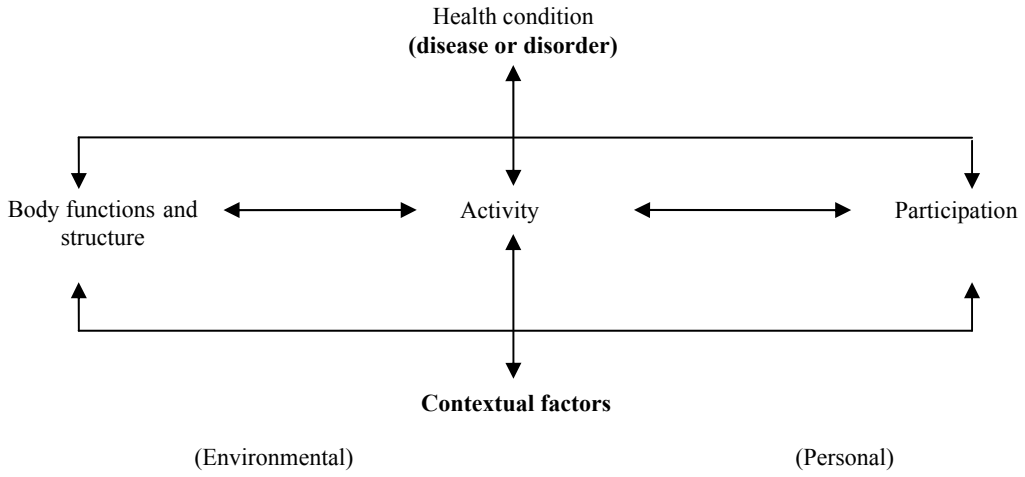
In this literature review, a range of cross-sectional and longitudinal studies are considered. The longitudinal studies with two occasions of measurement are discussed separately from the longitudinal studies with three or more time points. Attrition of participants is a notable feature of longitudinal studies, as is the issue of missing item response. If a study has a large proportion of missing values, then this can lead to response bias and affect the inferences drawn from the study in question. This feature is commented on as appropriate in the literature review.

2.5.1.1 Social relationships and disability

Understanding the factors that are associated with disability in older persons has been deemed a critically important public health issue, since disability may limit an individual's ability to function independently in the community (Guralnik et al., 1996). Several theoretical models have been put forward to explain differing levels of disability (e.g. Pope and Tarlov, 1991; Verbrugge and Jette, 1994; World Health Organization, 1980, 2002). In each of these models, disability is conceptualized as the result of a complex relationship between an individual's health, environment, personal attributes and psychosocial factors.

The most recent of these models, the International Classification of Functioning, Disability and Health (ICF; World Health Organization, 2002) has gained wide acceptability in recent years. The ICF defines health domains and health-related domains from two perspectives: i) body functions and structures and ii) activities and participation. In the ICF, disability is an umbrella term used to encompass impairments, activity limitations or participation restrictions. The ICF classification is particularly useful because it allows a broad conceptualization of disability, placing it within the context of the personal and other factors that surround an individual, as illustrated in Figure 2.6.

Figure 2.6: The International Classification of Functioning, Disability and Health



adapted from World Health Organization (2002)

A wide range of instruments have been used to assess disability in older persons, including activities of daily living (ADL; Katz et al., 1963; Katz et al., 1970), instrumental activities of daily living (IADL; Lawton and Brody, 1969), mobility (Rosow and Breslau, 1966), and Nagi physical function tasks (Nagi, 1976). The ADL scale, usually composed of seven items that are self-reported, covers basic personal self-

maintenance tasks of eating, personal grooming, toileting, bathing, dressing, walking across a small room, and transferring from a bed to a chair (Katz et al., 1970). The IADL scale is usually composed of ten items that cover higher level instrumental tasks than ADL. The ten items typically included in IADL scales are washing clothes, light housework, heavier housework, home maintenance including gardening, meal preparation, use of telephone, management of financial affairs, writing letters, using public transport, and grocery shopping (Lawton and Brody, 1969). Questions concerning mobility usually include whether the respondent reports they can walk some specified distance (often 'half a mile'), and climb stairs without help (Rosow and Breslau, 1966). In contrast, the Nagi tasks address self-reported difficulty in moving large objects, stooping, carrying heavy weights, lifting arms above shoulder level, and fine joint movement (Nagi, 1976).

Disability is often defined in epidemiological studies as the inability to carry out some specified number of items or tasks in one of the abovementioned scales without the use of aids or devices, such as a walking frame or handrails, or help from another person. For example, a person would be classified as having a mobility disability if there were unable to climb a flight of stairs, or walk 800m without help. A person who was unable to use the toilet with handrails would be classified as having an ADL disability.

Disabilities in mobility and Nagi tasks usually precede IADL and ADL disability, while IADL disability will usually occur before ADL disability (Verbrugge and Jette, 1994; Jette et al., 1998). A significant cognitive component is inherent in the measurement of IADLs, and thus IADLs are generally regarded as more complex than ADL tasks. Thus disability in IADL may not reflect physical disability *per se* (Guralnik and LaCroix, 1992). The measurement of ADL disability in community-dwelling samples has been

criticized because it will identify only the most severely limited individuals (Guralnik and LaCroix, 1992; Clark and Bond, 1995). If disability can be identified at earlier points in the disablement process, then there may be more avenues for intervention to prevent disability or promote recovery from disability.

2.5.1.2 Cross-sectional studies

A relatively modest number of studies have considered the effects of social relationships on disability, as summarized in Table 2.2 (pp. 56-60). Several studies have examined cross-sectional associations between disability and social relationships. For example, Berkman et al. (1993) studied 1354 community-dwelling participants aged 70 to 79 years who took part in a US-based study of successful ageing. In this study, Berkman et al. (1993) classified individuals' as high (n=1192), medium (n=80) or low functioning (n=82). Their measure of function was composed of ADL items, mobility items, Nagi items and a clinical test of balance, and thus high function corresponds to no or little disability. These authors demonstrated no significant association between social networks and disability nor social support and disability. However, the results must be interpreted with some caution given the striking imbalance in size of the three groups, and the dominance of people with little or no disability in the sample. Furthermore, only univariate analyses were considered in the report by Berkman et al. (1993).

C. Berkman and Gurland (1998) considered the cross-sectional association between social networks and disability in a sample of 1570 New York residents aged at least 65 years, and adjusted for a range of covariates in the analysis including demographic and psychological variables. These authors reported that those with greater disability had larger networks. Berkman and Gurland (1998) argued that disability led to larger

networks, which in turn increased the availability of support. However, these conclusions cannot be supported from the cross-sectional evidence, and the assumption that larger social networks are correlated strongly with better social support is not universally accepted (e.g. Antonucci et al., 1998; Rook, 1992, 1997).

In a cross-sectional study of 2079 Israelis aged 60 years or more, Litwin (2003) defined disability based on Nagi items. Unlike other authors, Litwin (2003) used the network typologies defined in Litwin (2001) as the dependent variable and examined the effects of demographic variables and disability on network typology. He showed that individuals classified as having a restricted network typology were more likely to have high or moderate levels of Nagi disability. Furthermore, those classified as belonging to the neighbours and family network types had higher levels of disability than those with diverse or friends networks.

2.5.1.3 Longitudinal studies with two time points

Many more studies have examined the effects of social relationships on disability using longitudinal data with two times of measurement. An early longitudinal study in this area was reported by Harris et al. (1989). These authors used a subset of data from the US Longitudinal Study of Aging (LSOA) from 1791 community-dwelling participants with a two-year follow-up period. The authors based a measure of disability at follow-up on a hybrid of four items that captured aspects of mobility and Nagi disability, and they defined social networks through telephone contact with friends or neighbours in the previous fortnight. Their work showed no effects of better social networks on development of disability for males (odds ratio (OR) 0.7, 95% confidence interval (CI) 0.4 – 1.4) or females (OR 1.7; 95%CI 0.8 – 3.3). This may signal that telephone contact alone is not particularly helpful in the face of physical disability.

In a sample of 1745 LSOA participants aged between 70 and 74 years at baseline, Mor et al. (1989) examined the effects of social engagement and social networks on mobility disability over two years of follow-up. Among those who were not initially disabled (n=852), Mor et al. (1989) found no significant effects of church or temple attendance (OR 1.02; 95%CI 0.82 – 1.26) nor personal contact with relatives (OR 0.95; 95%CI 0.21 – 1.25) on disability status at two year follow-up.

Kaplan et al. (1993) used data from 356 community-dwelling participants aged 65 to 95 years in the Alameda County Study that began in California in the mid-1960s. In their study, Kaplan et al. (1993) derived a disability score out of 72 based on ADL, IADL mobility and Nagi items, with higher scores indicating less physical disability. Kaplan et al. (1993) based a measure of social relationships on the Berkman and Syme (1979) SNI which, as noted in Section 2.3.6, measures a mixture of social networks and social engagement. After adjusting for the number of comorbid conditions, Kaplan et al. (1993) reported a significant protective effect of better social relationships against increasing disability over a six year follow-up period, although quantification of the size of the effect was not possible from the presentation of Kaplan et al.'s results.

While the focus of the research by Boult et al. (1994) was on chronic conditions that led to ADL disability, these authors also considered the effects of social networks on disability. In a four-year follow-up of 5210 participants in the LSOA aged 70 years or more, Boult et al. (1994) demonstrated that participants with better social networks, measured by telephone or personal contacts with friends or relatives during the previous fortnight, were significantly less likely to develop ADL disability over the follow-up period than those with poorer social networks (OR 0.4; 95% CI 0.3 – 0.8). A range of

covariates that covered demographic, lifestyle and psychological characteristics were included in the analyses. This contrasts with the findings by Harris et al. (1989) who showed telephone contact alone was not protective against the development of disability, albeit Harris' work only included a subsample of participants considered by Boult et al.

A study of the effects of social networks, social support and social engagement on disability was carried out by Liu et al. (1995) in a sample of 2200 Japanese people aged at least 60 years. Liu et al. (1995) considered the effects of social relationships on the transitions between states of disability at baseline and follow-up three years later. Disability in this study was defined using a measure that included bathing (ADL) and stair climbing and walking a short distance, in this case 200-300 metres (mobility). After controlling for demographic status, Liu et al. (1995) concluded that participants with better social engagement and better emotional social support were more likely to recover from disability at baseline during the follow-up period. These authors also found that more frequent social contacts were associated with disability that continued from baseline to follow-up.

As part of a study designed to examine whether cognitive impairment precedes ADL disability, Moritz et al. (1995) used data from 1856 community-dwelling participants in the Yale Health and Aging Project, who were aged 65 years or more and with no ADL disability at baseline. The study had a follow-up period of three years. Moritz et al. (1995) considered the number of instrumental social supports (the number of persons available to help with basic daily chores), social networks (frequency of visual contact with friends or relatives), and social engagement (participation in hobbies, reading, and going to the movies, restaurants, or sporting events). After adjusting for baseline

demographic variables, cognitive function, comorbid conditions and mobility disability, the authors showed significant protective effects of better social networks for males (OR 0.5; 95% CI 0.3 – 0.9) and females (OR 0.5; 95%CI 0.3 – 0.8) on the development of ADL disability. The presence of more instrumental supports at baseline was a risk factor for the development of disability (males OR 2.4; 95%CI 1.2 – 4.6 and females OR 2.1; 95%CI 1.2 – 3.8). Overall, the social engagement items were not significant predictors of disability, although there was a marginally significant protection against disability for males who participated in hobbies in the previous month (OR 0.6; 95%CI 0.3 – 1.0). The risk of disability associated with more instrumental social supports in this study highlights that ADL disability may not be sensitive to important changes in function for older adults. It is possible that disability in mobility or Nagi tasks were present at baseline in this study, but this was not reported by Moritz et al. (1995).

Data from the same cohort analysed by Kaplan et al. (1993) were used in an analysis by Strawbridge et al. (1996). These authors dichotomized individuals based on their ability to carry out all ADL and IADL items independently and with no difficulty, and with no more than a little difficulty in each of the five Nagi items. Using this definition, Strawbridge et al. (1996) classified people as 'ageing successfully' or 'not ageing successfully', although their sole reliance on daily living activities and Nagi items make it appropriate to consider this as a measure of disability. These authors classified social networks on the basis of less than or at least five close personal contacts, although a time frame for contact with these network members was not stated. After adjusting for demographic variables, baseline disability status, and number of chronic conditions, Strawbridge et al. (1996) showed that no disability at follow-up was predicted by more social contacts at baseline (OR 0.57; 95%CI 0.33 – 0.98).

Two articles by Seeman and colleagues (Seeman et al., 1995; 1996) investigated the effects of different aspects of social relationships on disability. In the first of these articles, Seeman et al. (1995) examined a cohort of 1189 high-functioning men and women aged between 70 and 79 years who took part in the MacArthur Studies of Successful Aging. It is not clear from this article why there were three fewer participants than in the original Berkman et al. (1993) publication. These authors considered the size of the social network (measured as the total number of children, other relatives and friends) and the frequency and perceived adequacy of emotional and instrumental social support. Seeman et al. (1995) also described 'a summary score reflecting the average frequency of demands and criticism from network members', but did not elaborate on its derivation. The validated measure of disability used in this study was based on a timed assessment of gait, chair stands, balance, foot raps, and manual ability (Seeman, Charpentier et al., 1994). Demographic, health variables, cognitive function, and hospitalizations were used as covariates in the analyses. Change in disability status across the three years of follow-up was the outcome in this study. The results showed no effect of social network size or measure of network conflict/demand on change in disability. However, more frequent emotional support had a significant, protective effect against disability. Furthermore, there was a significant interaction between emotional and instrumental social support such that the effect of emotional support was greatest in those with low instrumental support.

In a subsequent study with the same cohort and length of follow-up, Seeman et al. (1996) examined the onset of ADL disability in relation to the social networks, social support, and the frequency of criticism and excessive demands. A subset of the covariates considered in Seeman et al. (1995), that nonetheless covered the same major domains as in the earlier work, were included in Seeman et al. (1996). For male

participants, more ties with children was a significant risk factor for developing ADL disability (adjusted OR 1.24; 95%CI 1.03 – 1.49). There was no significant effect of the social network variables for female participants. Receipt of more instrumental support was also a risk factor for developing ADL disability among males (OR 4.72; 95%CI 1.71 – 13.02) but not females. However participants in the MacArthur Studies of Successful Aging were selected to be high functioning at baseline. Thus the generalizability of findings from Seeman's studies to the wider population of older people may be quite limited.

Hays et al. (1997) examined the effects of aspects of social networks and social support on three measures of ADL disability, mobility disability and Nagi disability. Data were drawn from 3240 community-dwelling participants in the Duke EPESE, and follow-up data were collected after one year. Hays et al. (1997) scored ADL, mobility and Nagi disability as counts of items with ranges of 0 – 7, 0 – 3 and 0 – 5 respectively. The results showed that the size of the social network was not significantly related to any of the measures of disability. The receipt of instrumental support was a strong predictor of all types of disability, while providing instrumental social support was protective against disability. The availability of a confidant was protective against ADL and mobility disability, but not Nagi disability. However, for the latter form of disability the perceived adequacy of social interaction was protective. Multiple linear regression of each disability measure on the social relationship variables was carried out, thus precluding the derivation of odds ratios from this study. Additional insights into the relationship between the social variables and disability in this study may have been gleaned through the consideration of categorizations of the disability measure in this model. This approach would also have allowed for the conduct of logistic regression

analyses, and thus greater comparability between Hays et al. (1997) and the extant literature would have been possible.

The effects of a hybrid measure of social networks and social engagement on mobility disability in a sample of 1483 community-dwelling people aged 70 years or more living in Hong Kong were examined by Ho (1997). The measure was based on Berkman and Syme's SNI and was composed from questions regarding participants' contact with friends, relatives, and neighbours and participation in community or religious activities. After controlling for a range of baseline covariates including demographic variables, self-rated health, comorbid conditions, and lifestyle variables, the effect of social relationships on mobility disability after 18 months of follow-up was not significant. However, the adjusted odds ratios were not presented by Ho (1997) so the size of the effect can not be presented.

Unger et al. (1999) also used data from the MacArthur Studies of Successful Aging, albeit with a seven year follow-up period and a smaller sample (n=850), to examine the effects of social networks and social support on changes in Nagi disability status. The reported analyses controlled for demographic variables, health status, and health behaviours. Unger et al. (1999) showed that larger social networks at baseline were protective against development of Nagi disability, and this effect was greater in men than in women. The authors also reported a stronger effect of larger social networks for those participants who had poorer physical ability at study baseline, providing some evidence of a buffering effect of social networks. The effects of emotional and instrumental support on Nagi disability were not significant. Although this is one of the few studies to specifically consider Nagi disability, the findings must be again interpreted with the same caveats as apply to Seeman et al. (1995) and Seeman et al.

(1996). Furthermore, the way in which change in Nagi disability was scored (a six point scale) and analysed (using linear regression) make the findings from this study difficult to compare to other studies in this area.

More recently, Avlund et al. (2004a) considered the effects of social networks, instrumental social support and social engagement on a measure of ADL disability in 651 people aged 75 years at baseline in Finland and Denmark who lived in the community or in residential care. In their analyses, they adjusted for living status, comorbid conditions, cognition and depression. Notably, covariates such as alcohol and tobacco consumption that reflect lifestyle were not included in the analyses. Follow-up data were collected after five years. Their results showed that better social networks at baseline, measured by at least weekly telephone contact with social network members, was associated with lower levels of disability at follow-up in both men (OR 0.40; 95%CI 0.20 – 0.77) and women (OR 0.56; 95%CI 0.32 – 0.91). Avlund et al. (2004a) also showed that social engagement at baseline was not significantly associated with disability at follow-up. At odds with many other studies, instrumental social support was not a risk factor for incident disability in either gender. The authors suggested that their findings may be partly due to the limited social relationships variables that were available to them for analysis.

A second study by Avlund et al. (2004b) also considered the effects of social networks, social support and social engagement on the onset of mobility disability. A sample of 1396 community dwelling Danes aged 75 or 80 years who were initially not disabled in mobility were followed over 18 months. Avlund et al. (2004b) reported that low diversity of social networks (based on the frequency of contact with children, grandchildren and great-grandchildren, siblings, other relatives and friends) was a

significant risk factor for incident disability among 75 year old men. However the large point estimate of the adjusted odds ratio and associated confidence interval (OR 11.5; 95%CI 1.5 – 86.7) coupled with the lack of an effect in 80 year old men and in women suggests this finding must be interpreted with some caution. There was some evidence of an effect of lower social engagement on development of disability in 75 year old men and women, but this effect was not significant after adjustment for covariates.

2.5.1.4 Longitudinal studies with three or more time points

Several authors have considered more than two time points in their analyses of social relationships and disability. For example, Camacho et al. (1993) analysed three distinct time points. Recent studies led by Mendes de Leon have considered the effects of social relationships upon disability using multiple waves of data (Mendes de Leon et al., 1999; 2001; 2003).

In the study by Camacho et al. (1993), the effects of social networks and social engagement on a composite disability measure were examined in a small subset (n=91) of participants in the Alameda County Study. Follow-up interviews with the participants aged 60 years or more at baseline were carried out at two times – 10 and 20 years – after the baseline interview. Social networks were measured by the number and frequency of contacts with close friends and relatives. Social engagement was measured by church and other group membership. The same composite disability score developed in Kaplan et al. (1993) was investigated. Camacho et al. (1993) found that after controlling for age, gender and chronic conditions, there was a significant protective effect of better social networks against higher disability scores. The effect of social engagement on disability was not significant. However, the power to detect effects in this study was probably low due to the small sample size.

Three studies by Mendes de Leon and colleagues have examined the effects of different aspects of social relationship on disability using multiple waves of data. In the first of these studies, Mendes de Leon et al. (1999) examined the effects of the measures of social support and specific social networks with children, other relatives, friends and confidants (as derived by Glass et al., 1997) on ADL disability and mobility disability. Data from 2812 participants aged at least 65 years in the New Haven EPESE were available at baseline, and eight waves of follow-data for disability status were available. After adjustment for demographic, health and cognitive status, significant protective effects of total social networks and networks with relatives on the development of ADL disability were found (total social network: OR 0.84; 95%CI 0.75 – 0.94; relatives: OR 0.82; 95%CI 0.77 – 0.93). Recovery from ADL disability was predicted by better total social networks (OR 1.41; 95%CI 1.14 – 1.74), better relatives networks (OR 1.25; 95%CI 1.09 – 1.43), and better friends networks (OR 1.28; 1.05 – 1.54).

Emotional social support was found to have no significant effect on ADL disability. However, the receipt of greater instrumental social support at baseline was predictive of developing ADL disability (OR 1.85; 95%CI 1.39 – 2.56). This suggests that disability at an earlier stage in the disablement process (such as mobility disability or Nagi disability) may have been present at baseline. However, Mendes de Leon et al. (1999) found no significant effects of specific or total social networks on mobility disability. An alternative explanation is that the provision of instrumental support to older participants in this study led to the relinquishment of abilities and thus incident ADL disability developed.

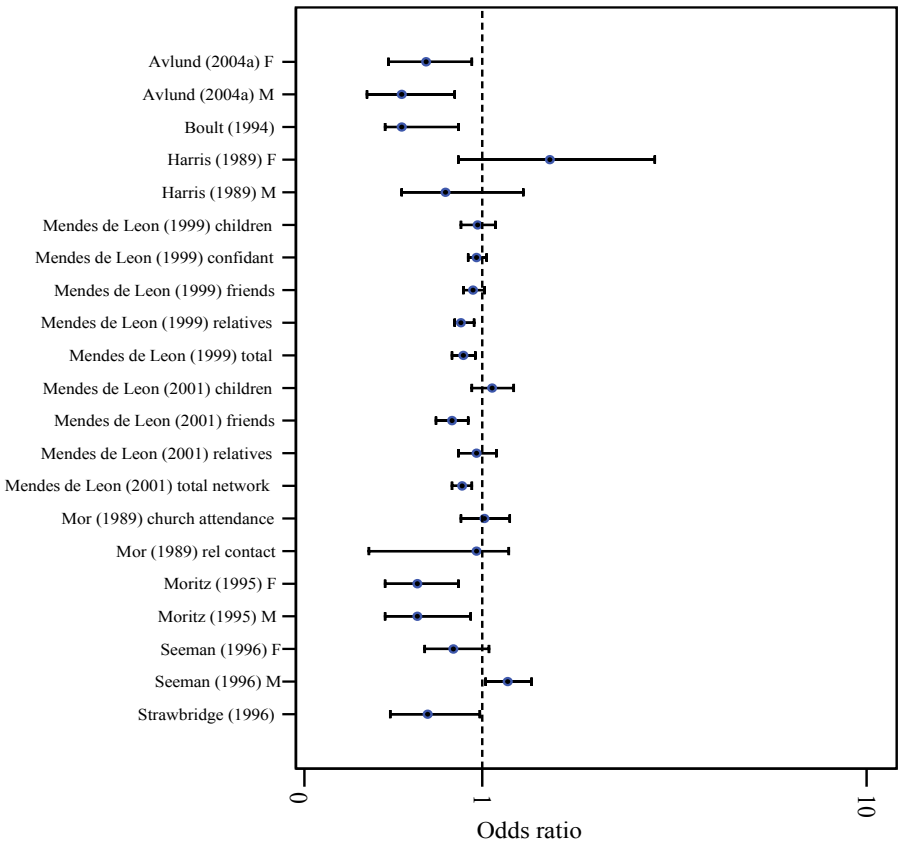
Mendes de Leon et al. (2001) undertook similar analyses to those described above using data from 4136 people aged 65 years or more who took part in the Duke EPESE study. Mendes de Leon et al. (2001) considered the size of the social network, the number of children, relatives and friends seen at least monthly, and a hybrid measure of social networks and engagement. These authors also derived measures of instrumental and emotional support. ADL disability and mobility disability status were available at baseline and in six subsequent annual waves of data collection. After adjustment for a range of demographic and health related covariates, the results showed significant protective effects of greater social network size against ADL disability (OR 0.85; 95%CI 0.74 – 0.97) and mobility disability (OR 0.83; 95%CI 0.75 – 0.91). Greater social engagement was also protective for both types of disability (ADL OR 0.77; 95%CI 0.67 – 0.88 and mobility OR 0.80; 95%CI 0.73 – 0.89). In contrast with the findings of Mendes de Leon et al. (1999), larger friends networks were protective against not only ADL disability (OR 0.67; 95%CI 0.54 – 0.82) but also mobility disability (OR 0.75; 95%CI 0.63 – 0.88). Greater instrumental social support was a significant risk factor for ADL disability (OR 1.18; 95%CI 1.13 – 1.24) and mobility disability (OR 1.12; 95%CI 1.08 – 1.16). There were no significant effects of children and relatives networks or emotional social support on disability status.

In their third manuscript, Mendes de Leon et al. (2003) examined the association between social engagement and ADL disability, mobility disability and Nagi disability. The same cohort described in Mendes de Leon et al. (1999) was used in this study, with nine waves of data in total. Social engagement was defined on the frequency of participation in 11 types of activities, including sporting events, shopping, group participation, and church attendance. The analyses were adjusted for demographic, health and social networks variables, although results for the latter were not reported. A

significant protective effect of better social engagement on each of the types of disability was found (ADL disability OR 0.71; mobility disability OR 0.81; Nagi disability OR 0.85; 95%CIs not calculable).

A synthesis of the size of the effects for measures of social networks on disability is given in the forest plot shown Figure 2.7. ADL disability has most commonly been studied, and therefore in Figure 2.7 the effects of social networks on ADL disability only are summarized. Those few studies that included mobility disability or Nagi disability are not included in the forest plot. Furthermore, the many different definitions of social networks that have been used in the various studies must be borne in mind. A third point to consider is that several studies did not report their results in terms of odds ratios with a confidence interval, and so these studies could not be included in the forest plot. Finally, only the first author has been listed for each study in the figure to conserve space, and alphabetical ordering by first author has been used. With these caveats in mind, the figure highlights the lack of consistency across studies, although a trend towards a protective effect of social networks on disability is apparent in Figure 2.7.

Figure 2.7: Forest plot of effects of social networks on ADL disability[1]



1: M=males; F=females

Table 2.2 presents an overview of the main components of each study described in Section 2.5.1, and summarises findings with respect to social networks, social support and social engagement. A brief summary and discussion of the studies concerning social relationships and disability then follows in Section 2.5.2.

Table 2.2: Summary of studies that examined effects of social relationships on disability

Author	Country	Baseline age	n	Sample	# Time points	Follow-up length	Disability definition	Covariates considered	Effect of social relationship adjusted for covariates	Main statistical method	Comments
Avlund, et al. (2004a)	Finland	75	651	Community & residential care	2	5 yrs	Incident ADL disability	Demographic Health Psychological	Network * ↓ Engagement NS Support NS	Logistic regression	
Avlund, et al. (2004b)	Denmark	75 or 80	1396	Community	2	1.5 yrs	Incident mobility disability	Demographic Health	Network * ↑ [for 75yo M only] Support NS Engagement NS	Logistic regression	Sparse data – only one younger man with high social diversity had onset of disability
Berkman et al. (1993)	US	70-79	1192 + 162	Community	1	-	Composite ADL, IADL, mobility, Nagi disability	N/A	Network NS Support NS	Chi-square or one-way ANOVA	Cross-sectional, no multivariate analyses presented
C. Berkman and Gurland (1998)	US	65+	1570	Probability sample of Medicare recipients in 5 boroughs of New York City	1	-	Composite of ADL, IADL disability, getting around outside home	Demographic Psychological	Network NS	Multiple linear regression	Cross-sectional

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); NS not significant; ↑ Risk factor; ↓ Protective factor

Table 2.2 (continued): Summary of studies that examined effects of social relationships on disability

Author	Country	Baseline age	n	Sample	# Time points	Follow-up length	Disability definition	Covariates considered	Effect of social relationship adjusted for covariates	Main statistical method	Comments
Boult et al. (1994)	US	70+	5210	Community	2	4 yrs	ADL disability	Demographic Health Psychological	Network * ↓	Logistic regression	Social contacts not explicit focus; chronic conditions main point of article
Camacho et al. (1993)	US	60+	91	Community	3	20 yrs	Composite ADL, IADL, mobility, Nagi disability	Demographic	Network + ↓ Engagement NS	Multiple linear regression	
Harris et al. (1989)	US	80+	1791	Community	2	2 yrs	Composite mobility and Nagi disability	Demographic Health	Network NS	Logistic regression	
Hays et al. (1997)	US		3240	Community	2	1 yr	ADL disability Mobility disability Nagi disability	Demographic Health Psychological	Network NS Receive support * ↑ Give support * ↓	Linear regression	? appropriateness of linear regression with counts of difficulty in ADL, mobility and physical performance items
Ho (1997)	Hong Kong	70+	1483	Community	2	1.5 yrs	Incident mobility disability	Demographic Health	Network-Engagement hybrid NS	Logistic regression	

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); NS not significant; ↑ Risk factor; ↓ Protective factor

Table 2.2 (continued): Summary of studies that examined effects of social relationships on disability

Author	Country	Baseline age	n	Sample	# Time points	Follow-up length	Disability definition	Covariates considered	Effect of social relationship adjusted for covariates	Main statistical method	Comments
Kaplan et al. (1993)	US	65-95	356	Community	2	6 yrs	Composite of ADL, IADL, mobility, Nagi disability	Health	Network-Engagement hybrid * ↓	Linear regression	
Litwin (2003)	Israel	60+	2079	Community	1	-	Nagi disability	Demographic	Network type * less restricted ↓	Multinomial logistic regression	Treated network type as dependent variable and examined effects of demographic variables and disability on network type
Liu et al. (1995)	Japan	60+	2200	Not stated but probably community and residential	2	3 yrs	Composite of 1 ADL (bathing) and mobility disability	Demographic	Network * ↓ Support * ↓ Engagement * ↓	Multinomial logistic regression	Emotional support only considered as instrumental support highly correlated with emotional support
Mendes de Leon et al. (1999)	US	65+	2812	Community	9	9 yrs	ADL disability Mobility disability	Demographic Health Psychological	ADL: Total network * ↓ Relatives network * ↓ Receive support * ↑ Mobility: Network NS	Logistic regression separate for development and recovery of disability	Instrumental support risk factor for development of ADL disability; not considered for mobility disability

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); NS not significant; ↑ Risk factor; ↓ Protective factor

Table 2.2 (continued): Summary of studies that examined effects of social relationships on disability

Author	Country	Baseline age	n	Sample	# Time points	Follow-up length	Disability definition	Covariates considered	Effect of social relationship adjusted for covariates	Main statistical method	Comments
Mendes de Leon et al. (2001)	US	65+	4136	Community	7	7 yrs	ADL disability Mobility disability	Demographic Health Psychological Lifestyle	ADL: Network size * ↓ Friends network * ↓ Receive support * ↑ Engagement * ↓ Mobility: Network size * ↓ Friends network * ↓ Receive support * ↑ Engagement * ↓	Proportional odds model with GEE approach	
Mendes de Leon et al. (2003)	US	65+	2812	Community	9	9 yrs	ADL disability Mobility disability Nagi disability	Demographic Health Psychological	Engagement * ↓ for ADL, Mobility and Nagi disability	Proportional odds model with GEE approach	Network results not reported
Mor et al. (1989)	US	70-74	1745	Community	2	2 yrs	Composite of mobility and Nagi disability	Demographic Health	Network-Engagement hybrid NS	Logistic regression	Restricted to those with no initial disability
Moritz et al. (1995)	US	65+	1856	Community	2	3 yrs	Incident ADL disability	Demographic Health Psychological	Network * ↓ Support * ↑ Engagement NS	Logistic regression	Instrumental support increased risk of disability

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); NS not significant; ↑ Risk factor; ↓ Protective factor

Table 2.2 (continued): Summary of studies that examined effects of social relationships on disability

Author	Country	Baseline age	n	Sample	# Time points	Follow-up length	Disability definition	Covariates considered	Effect of social relationship adjusted for covariates	Main statistical method	Comments
Seeman et al. (1995)	US	70-79	1015	Community	2	2 yrs	Summary of timed measures of balance, gait, chair stands, foot taps, and manual ability.	Demographic Health Psychological	Network NS Support * ↓	Linear regression for change scores and logistic regression for ↓ vs. ↑	High functioning cohort; classifies marital status as social variable; More frequent emotional support positive effect on better physical performance among those with low instrumental support
Seeman et al. (1996)	US	70-79	1012	Community	2	2 yrs	Incident ADL disability	Demographic Health Psychological	Network * ↑ [M only] Support * ↑ [M only]	Logistic regression	High functioning cohort with low incident disability; classifies marital status as social variable; Instrumental support significant for M not F
Strawbridge et al. (1996)	US	65-95	356	Community	2	6 yrs	Composite of ADL, mobility and Nagi disability	Demographic Health	Network * ↓	Logistic regression	"Successful ageing": same items as (Kaplan et al., 1993)
Unger et al. (1999)	US	70-79	850	Community	2	7 yrs	Incident Nagi disability	Demographic Health	Network * ↓ Support NS		High functioning cohort at baseline

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); NS not significant; ↑ Risk factor; ↓ Protective factor

2.5.2 Summary and conclusions

ADL, IADL, mobility and Nagi disability were considered separately in the majority of reviewed studies (Boult et al., 1994; Moritz et al., 1995; Seeman et al., 1996; Hays et al., 1997; Ho, 1997; Mendes de Leon et al., 1999; Unger et al., 1999; Mendes de Leon et al., 2001; Mendes de Leon et al., 2003; Litwin, 2003; Avlund, et al. 2004a, b). However, many studies used composite measures of disability (Harris et al., 1989; Mor et al., 1989; Berkman et al., 1993; Camacho et al., 1993; Kaplan et al., 1993; Liu et al., 1995; Strawbridge et al., 1996; C. Berkman and Gurland, 1998). It is unclear which aspect of disability is being measured in these studies. For example, from a composite score of 50, it is impossible to determine if an individual has some mobility disability and ADL disability in several tasks or no mobility disability but ADL disability in all tasks. This is important because assessing the effects of social relationships on composite measures of disability provides little insight into which aspects of disability are most affected by which kinds of social relationships.

Social networks were generally shown to protect against ADL disability (Boult et al., 1994; Moritz et al., 1995; Seeman et al., 1996; Mendes de Leon et al., 1999; Hays et al., 2001; Mendes de Leon et al., 2003; Avlund et al. 2004a). The evidence concerning mobility disability and Nagi disability was less clear, as these two forms of disability were considered as distinct outcomes in far fewer studies. Avlund et al. (2004b) and Mendes de Leon et al. (2001) reported significant protective effects of social networks on mobility disability, while the effect of social networks on mobility disability was not significant in the studies reported by Mendes de Leon et al. (1999) and Hays et al. (1997). The effect of social networks on Nagi disability was considered in two studies and these had contrasting findings (Unger et al. 1999; Hays et al. 1997). The differing follow-up times in the two studies may partially account for the different results.

Most studies that considered social support implemented different definitions of instrumental and emotional social support, and these were based variously on the perception, receipt or satisfaction with support. This hampers comparability between studies. The relationship between social engagement and disability was most likely confounded by the effects of physical and psychological health status on social engagement. Across the different kinds of social relationships considered, very few studies adjusted for a wide range of covariates that encompassed demographic, health, lifestyle and psychological characteristics of participants.

The majority of studies reported a dichotomous outcome, most commonly incident ADL disability, and used logistic regression to analyse the effects of baseline social relationships on development of disability by the time of follow-up. Mendes de Leon and colleagues extended this approach with multiple waves of data and accounted for the correlation between repeated observations on the same individual in their analyses. Some authors analysed 'continuous' measures of disability using multiple linear regression (Camacho et al., 1993; Kaplan et al., 1993; Seeman et al., 1996; Hays et al., 1997; C. Berkman and Gurland, 1998; Unger et al., 1999), and this made comparison of their results to the wider literature difficult.

In summary, several unanswered questions concerning social relationships and disability remain. The effects of specific social networks with kin and non-kin on mobility disability have been considered only by Mendes de Leon et al. (1999; 2001). Thus there is a need to consider the effects of specific social networks in a broader range of studies and settings. A second point is that the issue of confounding appears to have been addressed inadequately in the majority of reports. Indeed, few of the

reviewed studies have adjusted for a broad range of demographic, health, psychological and lifestyle variables. Finally, no studies were found that examined the effects of specific social networks on Nagi disability using longitudinal data and adjusting for a broad range of covariates. This means that current knowledge concerning the effects of social relationships on the disablement process, particularly with respect to the effects of specific social networks, is incomplete.

2.5.3 Social relationships and use of residential care

Around six per cent of the older Australian population are living in some form of residential care currently (Australian Institute of Health and Welfare, 2002; Australian Bureau of Statistics, 2003), and the demand for residential care will increase as the population grows older. From both individual and societal perspectives, there are high personal and financial costs associated with admission to residential care (Shapiro and Tate, 1988; Gaugler et al., 1999). Indeed, the costs of supplying aged care services in Australia are forecast to increase from $7.8 billion in 2002-2003 to $106.8 billion in 2042-2043 (Hogan, 2004).

Two levels of care are provided in the Australian residential care setting, namely low-level and high-level care (Australian Institute of Health and Welfare, 2002). Residential care facilities that provide low-level care for older people are often referred to as hostels in the Australian setting, and indeed this was the preferred term until the mid 1990s. High-level care is traditionally referred to as nursing home care in the international literature. Nursing home residents generally have greater disability and, as the Australian nomenclature suggests, require higher levels of nursing and medical care than residents in hostels.

A substantial body of research, dominated by studies carried out in the US, has identified factors such as increasing age, female gender, lack of a marital partner, lack of home ownership, more comorbid conditions, poorer self-rated health, prior nursing home use, more physical disability, and poorer cognitive status as significant predictors of residential care use, as reviewed recently by Gaugler et al. (2007) and Miller and Weissert (2000). The effect of higher income has been reported as a risk factor for admission to residential care by some authors (Greenberg and Ginn, 1979; Palmore, 1976; Liu et al., 1991; Gaugler et al., 2000) and protective by others (Vicente et al., 1979; Kelman and Thomas, 1990). The findings have been drawn from cross-sectional and longitudinal studies of varying follow-up durations. Relatively few Australian studies in this area have been carried out. Two recent Australian studies have been reported by Wang et al. (2001) and McCallum et al. (2005).

Both direct and buffering effects models have been empirically investigated in the literature concerning residential care utilization, although the former has received more attention (Newman et al., 1990). Under the buffering effects hypothesis, social networks may be particularly important in providing care to older people, and may thereby delay or prevent admissions to residential care. The argument put forward by adherents to the direct effects hypothesis is that individuals with better social networks maintain better health status, and are consequently less likely to need residential care. The distinction between the hypotheses is not made in the discussion of the articles reviewed here unless it was an explicit focus of the article under consideration. Table Table 2.3 (pp. 78-81) summarizes the literature that is reviewed in this section concerning social relationships and their effects on the use of residential care.

2.5.3.1 Cross-sectional studies

The earliest cross-sectional study in this area was reported by Kraus et al. (1976). In this study, the characteristics of 193 applicants to long-term residential care were compared with those of 141 persons living independently in the community in a Canadian province. The applicants to care reported smaller social networks with children and others. The applicants also had greater receipt of social support than their contemporaries living in the community. However, no statistical tests were undertaken by Kraus et al. (1976), and the cross-tabulations he presented do not permit any conclusions to be drawn regarding the effects of relationships on actual residential care use. Furthermore, it is not possible to discern the difference between networks with children and networks with others from their analyses.

In another early US study in this area, Greenberg and Ginn (1979) examined the effects of networks with children and relatives and receipt of social support in a cross-sectional comparison of 139 persons who had entered nursing homes in the previous three months with 129 people living at home and receiving community services. Although not specified, instrumental support was most probably assessed. The authors found significant differences in the size of the children networks for nursing home residents (mean 1.2, SD 1.2) compared with those at home (mean 2.3, SD 1.9), and in receipt of social support (nursing home residents 19% versus community 47%). However, the retrospective questionnaires in this study were completed by staff of the nursing home or community service providers, and may therefore not accurately reflect an individual's social circumstances. The effect of this potential recall bias on results cannot be estimated.

2.5.3.2 Longitudinal studies with two time points

The majority of research in this area has prospectively investigated changes in place of residence between two time points. The first longitudinal study was reported by Palmore (1976). Over a twenty year follow-up period, Palmore tracked the residential care use prior to death of 207 volunteers aged at least 60 years in a study conducted in North Carolina. Univariate analyses showed that volunteers with more children were less likely to use residential care. However, when adjusted for other demographic variables including marital status, the effect of number of children on residential care use was not significant.

Relatively few studies have differentiated between the effects of social networks with family and those with friends on residential care use. One of the earliest studies to consider the effects of specific social networks on any use of residential care during a three year follow-up period was reported by Wan and Weissert (1981). In their US study of 1119 people with a mean age of 76 years, social networks were measured by the frequency of contact with spouse, children, grandchildren, siblings, other relatives and friends during a three month period. Social networks with children and grandchildren were found to be protective against use of residential care, and social networks with siblings were apparently a risk factor for use of residential care. However, the size of the effects cannot be quantified from the analyses presented by Wan and Weissert (1981).

A subset of 521 decedents aged 55 years or more in the Alameda County Study were the basis for an analysis by Vicente et al. (1979). In this study, better social networks reported at baseline were a significant protective factor against use of residential care in the subsequent decade prior to death; a quantification of this result was not reported.

However, Vicente et al. (1979) based their measure of social network on marital status and household composition, items which are not necessarily reflective of the wider social network with which an individual interacts. Furthermore, among older people marital status may be strongly confounded with age and gender. With this in mind, the findings reported by Vicente et al. (1979) may have been confounded by other items that were not adjusted for in the analyses.

The effects of social networks with children, other relatives and friends on residential care use in a national sample of 4884 community-dwelling persons aged 65 or older who received an aged pension were investigated in another early study by McCoy and Edwards (1981). Persons who had contact with friends at least a few times per week (OR 0.66; 95%CI not given) and contact with relatives daily (OR 0.54; 95%CI not given) were significantly less likely to be admitted to residential care during the one year follow-up period than those with less frequent contact. Suggestive of a buffering effect, the proximity of children had a marginally protective significant effect on residential care use (OR 0.80; 95%CI not given) among the subgroup (n=1789) with high levels of ADL disability, but not in the total cohort.

Another early prospective study was reported by Branch and Jette (1982), who drew data from a cohort of 1625 participants in a longitudinal study of people aged 65 years or more who lived in Massachusetts. Unlike earlier studies in this area, Branch and Jette (1982) adjusted for many demographic, health and psychological variables in the analyses. Branch and Jette (1982) used a single variable, the proximity of relatives, to capture social networks. The authors reported no effect of relatives living nearby on admission to residential care in the six years of follow-up (OR 0.96; 95%CI 0.54 – 1.72). However, this single variable was unlikely to accurately reflect the wider social

network of an individual, and did not allow effects of children versus siblings or other relatives to be ascertained.

Greene and Ondrich (1990) examined the effects of instrumental social support on residential care use in a national sample of 3332 US people aged 65 years or more. In their analysis, Greene and Ondrich (1990) separately considered the risk factors for the transition from the community to the nursing home, and from nursing home to the community over one year. Their analyses adjusted for demographic, health, psychological and lifestyle covariates that also encompassed regional-level variables such as bed availability. Greene and Ondrich (1990) demonstrated that participants who received more instrumental social support at baseline were marginally less likely to make the transition from nursing home to the community (each hour of support received HR 0.99; $P<0.10$; 95%CI not given) while the receipt of instrumental social support was not a significant predictor of transition from the community to nursing home. However, whether the providers of the support were family or others in the social network remains unclear from Greene and Ondrich (1990). Furthermore, the possibility that the effect of informal care hours on residential care use may be non-linear was not explored in the analyses.

Hanley et al. (1990) analysed the risk of nursing home admission over a two year period for 18777 community-dwelling people aged 65 or more who took part in the US National Long-Term Care Survey (NLTCS). After adjustment for a range of demographic and health variables and cognition in their analyses, Hanley et al. (1990) concluded neither social networks with children nor receipt of unpaid instrumental support were significant predictors of nursing home admission, although there was a

marginally significant protective effect of children living within a 30 minute proximity (OR 0.87; 95%CI 0.74 – 1.02) on nursing home admission.

The direct versus buffering effects hypotheses of social relationships on risk of residential care use were compared by Newman et al. (1990). Using data from 5580 participants in the NLTCS, Newman et al. (1990) examined the effects of receipt of instrumental social support and the existence, relationship and proximity of a primary caregiver on permanent admission to residential care over a two year follow-up period. The authors found the receipt of paid instrumental support was a risk factor for admission to residential care (OR 1.52; 95%CI 1.13 – 2.04), but unpaid support was not a risk factor. Newman et al. (1990) also stated that there was a marginally significant effect of whether the spouse was a caregiver or whether a caregiver was nearby, but did not present these results. Newman et al. (1990) concluded there was more evidence for the direct effects than the buffering effects hypothesis, although this was not substantiated by their results.

Data from 5151 participants in the LSOA were used by Speare et al. (1991) in their analysis of the effects of a range of variables on entry to residential care over two years of follow-up. Social network was classified into one of three categories, namely no living children, sons only, and at least one daughter. The analyses adjusted for a range of health, demographic and psychological variables. The authors found no effect of social networks with sons (OR 0.95; 95%CI 0.70 – 1.29) or daughters (OR 0.86; 95%CI 0.70 – 1.29) on moving to residential care in the follow-up period.

Liu et al. (1991) examined the effects of instrumental support on admission to nursing home and length of stay in a nationally representative cohort of 5795 chronically ill US

residents aged 65 or more. After adjustment for a range of demographic, health, psychological and regional level variables, their analysis showed that receipt of instrumental support was a significant protective factor against entry to residential care over the two years of follow-up (HR 0.64; 95%CI 0.51 – 0.80). This finding is interesting given that the literature concerning ADL disability and instrumental social support suggests that, in general, receipt of instrumental social support is a significant risk factor for incident ADL disability at follow-up (e.g. Moritz et al. 1995; Mendes de Leon et al., 2001), which in turn is a risk factor for residential care entry.

Using data from the same cohort as Speare et al. (1991), Wolinsky et al. (1992) examined the effects of social relationships, separated as kin and non-kin, on admission to residential care. Although they referred to their measure as social support, Wolinsky et al. (1992) measured a hybrid of social networks and social engagement with kin and with non-kin. The non-kin variable was composed of volunteer work, personal and telephone contacts with friends and neighbours, church attendance, and attendance at a group event, such a movie, sports or classes. Kin social networks were measured through the frequency of personal and telephone contact with relatives. Over the four years of follow-up, the participants with better non-kin social relationships were less likely than their contemporaries with poorer non-kin social relationships to enter a nursing home (OR 0.87; P=0.003; 95%CI not given). Participants with better versus poorer kin social relationships were not at increased risk of nursing home entry, although the size of the effect for this network type was not presented by Wolinsky et al. (1992).

Steinbach (1992) also used data from 5151 LSOA participants in her examination of the effects of social networks and social engagement on use of residential care over two

years of follow-up. She derived a social network variable from the frequency of telephone and personal contact with friends and relatives. Social engagement was measured by attendance at a senior's centre, church, movies or sport, and voluntary work. The analyses adjusted for a range of health and demographic variables, although cognitive status was omitted from the covariates. The results showed that better social engagement was a significant protective factor against admission to residential care (adjusted OR 0.58; 95%CI 0.41 – 0.82), but the effect of social networks was not significant.

In the same year, Pearlman and Crown (1992) conducted a similar study to that of Newman et al. (1990) and compared evidence for the buffering versus direct effects hypotheses on use of residential care. Pearlman and Crown (1992) examined the effects of social network type defined as including a spouse, children with no spouse, or paid helpers only. These authors also considered the effects of received instrumental social support. Their analyses controlled for a range of demographic, health and psychological covariates. Social networks that included a spouse reduced the relative risk of an admission to residential care during the follow-up period (OR 0.68; 95%CI 0.51 – 0.89), as did a network that comprised children with no spouse (OR 0.71; 95%CI 0.58 – 0.88). These authors found the receipt of instrumental support did not have a significant effect on use of residential care (OR 1.28; 95%CI 0.89 – 1.86). Pearlman and Crown (1992) concluded there was evidence for both direct and buffering effects of social networks on nursing home use. Furthermore, these authors concluded that instrumental social support had little impact on use of residential care.

The number of children and perceived availability of emotional and instrumental social support in relation to admission to residential care was investigated by Salive et al.

(1993). Using data from the North Carolina EPESE (n=4074) and adjusting for demographic, health and cognitive status variables, these authors showed that having more children was protective against admission to residential care for Caucasian (OR 0.84; 95%CI 0.75 – 0.95) and African-American (OR 0.94; 95%CI 0.88 – 1.01) respondents. Similarly, a high perceived availability of instrumental and emotional social support protected against residential care use in both ethnic groups (Caucasian: OR 0.80; 95%CI 0.66 – 0.95; African-American: OR 0.82; 95%CI 0.72 – 0.93). However, from the combined measure of emotional and instrumental social support availability it is difficult to interpret how the different types of social support may serve to protect against residential care use.

Freedman et al. (1994) examined the effects of specific social networks on nursing home admission based on data from 2812 participants in the New Haven EPESE. These authors adjusted for a broad range of health, demographic and psychological covariates. The results of this study showed that the composition and size of the network of relatives and children had little effect on the risk of nursing home entry over three years of follow-up for men. For older women, the risk of entry for women with no contact with children or other relatives was 2.9 times that of those who had contact with exactly one relative (95%CI 1.5 – 5.1). However, other comparisons of network size (1 versus 2, 2 versus 3+) were not significant. It was unclear whether the effect of network size was significant overall. For men, the 95%CI associated with the 0 versus 1 network size comparison was 0.1 to 8.4, and this width of interval suggests that the rationale for the network size categories should have been re-examined.

Data from 7527 community-dwelling participants in the LSOA were used by Coward et al. (1996) in an examination of the effects of social networks and social support on use

of nursing homes over a six year follow-up period. In this study, the number of living children was used as the measure of social networks, and receipt of unpaid help from other individuals was used to measure instrumental social support. Similar to Greene and Ondrich (1990) and Liu et al. (1991), Coward et al. (1996) also included regional level variables concerning bed availability and accessibility of health services. After adjustment for demographic, self-rated health and psychological characteristics, receipt of instrumental social support (OR 1.32; 95%CI 1.06 – 1.64) and fewer children (OR 1.08; 95%CI 1.04 – 1.12) were significant risk factors for residential care use.

Wilmoth (2000) analysed living arrangement transitions within the community and from the community to residential care over a six year follow-up period in 2713 people aged 60 years or more living in the community at baseline. The data used in this study were drawn from a nationally representative sample of US households. The study considered the effects of social networks with children and the effects of unbalanced social exchanges, the latter measured by the amount of assistance given to and received from children, other relatives and friends, on residential transitions. Wilmoth (2000) showed that there was no significant effect of the number of children nor unbalanced social exchanges with relatives or friends on admission to residential care. However, participants who had phone or visual contact with children at least weekly were marginally less likely to move to residential care (OR 0.69; 95%CI 0.45 – 1.07). Unbalanced social exchange with children in favour of the older person was a marginally significant risk factor for use of residential care (OR 1.20; 95%CI 0.99 – 1.47). While many demographic variables were adjusted for in the analyses, only self-rated health and ADL disability were considered as health covariates, and no indicator of cognitive status nor lifestyle variables were included in the analyses.

2.5.3.3 Longitudinal studies with three or more time points

Three or more waves of data were considered by Angel et al. (2004), Freedman (1996), Kelman and Thomas (1990) and Shapiro and Tate (1985), although Shapiro and Tate essentially presented separate comparisons of baseline to 2.5 years follow-up and baseline to seven years follow-up.

Shapiro and Tate (1985) examined the effects of frequency of contacts with relatives on use of residential care over two follow-up periods of 30 months and seven years in a sample of 3902 Canadians aged 65 or more who were living in the community at baseline. After adjustment for a range of demographic, health and psychological variables, Shapiro and Tate (1985) demonstrated that those with more frequent contact with relatives were more likely to enter residential care over seven years of follow-up (OR 1.18; 95%CI 1.03 – 1.35). However, the effect of social networks with relatives on residential care use in the shorter-term was not significant.

The effects of social support on transitions from the community to nursing homes among 1855 New York residents aged 65 years or more were examined by Kelman and Thomas (1990). All participants were living in the community at baseline. Kelman and Thomas (1990) tabulated the transitions between community and residential care at each wave for participants over the follow-up period, and classified individuals as continuously living in the community, short-term stayers – that is, only one wave in residential care – and permanent stayers. Although four waves of data were available to Kelman and Thomas (1990), these authors classified individuals according to transitions between community and residential care across all of the study waves. The authors demonstrated that greater receipt of instrumental social support was among the variables that differentiated between community and people who had used residential care by

follow-up. However, little detail concerning the definition of social support was given, and furthermore the magnitude of this effect cannot be ascertained from the brief details of the discriminant function analyses that were presented by these authors.

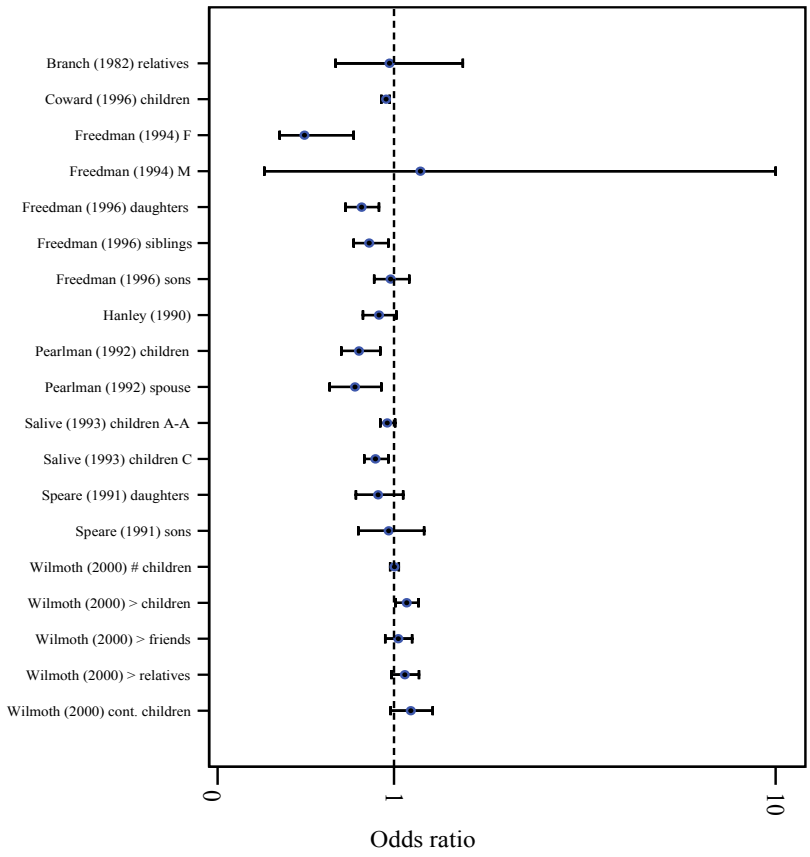
Freedman (1996) examined time to first admission to nursing home over seven years of follow-up in 2812 New Haven EPESE participants. This author controlled for a range of demographic and health variables, and based her measures of social networks with sons, daughters, and siblings on the presence or absence of these social ties. Freedman (1996) demonstrated a reduced hazard of admission to nursing home over the follow-up period for respondents with living daughters (hazard ratio (HR) 0.73; 95%CI 0.61 – 0.87), and living siblings (HR 0.79; 95%CI 0.67 – 0.95), but no effect for living sons (HR 0.93; 95%CI 0.80 – 1.09). She also reported little difference in these effects depending on whether the social networks were analysed as static or time-varying covariates in the models.

More recently, Angel et al. (2004) analysed the use of nursing homes over seven years with four waves of follow-up in a study of 3050 Hispanic people aged 65 years or more living in South-West of the US. The study was modelled on the EPESE projects. The outcome variable in this study was a combination of place of death and use of residential care. The four categories of response were i) survived irrespective of use of residential care, ii) died in residential care, iii) died without any use of residential care, and iv) died in community but used residential care before death. Social networks with children, living arrangements, and availability of emotional/instrumental support were considered by these authors. After adjusting for demographic characteristics, ADL disability and cognitive function, the authors demonstrated that participants with at least one child had roughly half the risk of dying in a nursing home compared to participants

with no children (OR 0.52; P<0.001; 95%CI not given). Participants with social support available were less likely to die in a nursing home (OR 0.54; P<0.01; 95%CI not given). However, the lack of adjustment for health status in this study suggests that these results may be confounded with participant's health.

The effects of social networks in particular on residential care utilization are summarized in Figure 2.8. As was the case for disability, the forest plot concerning social networks and use of residential care must be considered in light of several points. The first of these is that a wide range of definitions of residential care use was considered in the various studies summarized in Figure 2.8. The second point is that not all studies that analysed the effects of social networks on residential care use reported results in such a way that enabled them to be included in the figure. Thus the figure needs to be interpreted recognising that not all studies could be represented in the forest plot. Figure 2.8 suggests a protective effect of social networks on use of residential care in around half of the studies, but the wide variety of study sizes and designs makes an overall estimate of the effect from the existing research infeasible.

Figure 2.8: Forest plot of effects of social networks on use of residential care[1]



1: M=males; F=females; Salive: C=Caucasian; A-A=African American; Wilmoth: > children represents unbalanced exchange such that children give more than receive; similar for > friends and > relatives

Table 2.3: Summary of studies that examined effects of social relationships on use of residential care

Author	Country	Baseline age	n	Sample	# Time points	Follow-up length	Residential care use definition	Covariates	Effect of social relationship adjusted for covariates	Main statistical method	Comments
Angel et al. (2004)	US	65+	3050	Community	4	7 years	Combination of place of death and use of care	Demographic Health Psychological	Network with children * ↓ Support * ↓	Logistic regression	Confusing definition of residential care use
Branch and Jette (1982)	US	65+	1625	Community	2	6 years	≥1 nursing home admission	Demographic Health Psychological	Network with relatives NS	Logistic regression	
Coward et al. (1996)	US	70+	7527	Community	2	6 years	≥1 nursing home admission	Demographic Health Psychological	Network * ↓ Support * ↑	Logistic regression	Receipt of instrumental social support risk factor for admission
Freedman et al. (1994)	US	65+	2812	Community	2	3 years	≥1 nursing home admission	Demographic Health Psychological	Relatives network * ↓ [F only] Size of network NS	Logistic regression	
Freedman (1996)	US	65+	2812	Community	8	8 years	Age at admission to nursing home	Demographic Health Psychological	Sibling network * ↓ Daughter network * ↓ Sons network NS	Cox PH regression	
Greenberg and Ginn (1979)	US	60+	266	Nursing home entrants and persons who began in-home services in a 3 months period	1	-	≥1 nursing home admission	Demographic Health	Network + ↓ Receive Support * ↓	Logistic regression	Instrumental support from relatives protective

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); NS not significant; $ Community to nursing home transition (C→NH) or Nursing home to Community (NH→C); ↑ Risk factor; ↓ Protective factor

Table 2.3 (continued): Summary of studies that examined effects of social relationships on use of residential care

Author	Country	Baseline age	n	Sample	# Time points	Follow-up length	Residential care use definition	Covariates	Effect of social relationship adjusted for covariates	Main statistical method	Comments
Greene and Ondrich (1990)	US	65+	3332	Community-dwelling or probable discharge from nursing home care in ≤90 days	2	1 year (retrospec-tive)	Use per month	Demographic Health Psychological	C→NH$^{\$}$ Support NS NH→C$^{\$}$ Support + ↑	Discrete-time hazard model	More informal care hours received at baseline associated with marginally reduced odds of exit from nursing home
Hanley et al. (1990)	US	65+	18777	Community	2	2 years	Place of residence at follow-up	Demographic Health Psychological	Network NS Support NS	Logistic regression	Instrumental support measured by receipt of paid home care risk factor for admission
Kelman and Thomas (1990)	US	65+	1855	Community	4	3 years	Place of residence at each wave	Demographic Health Psychological	Support NS	Discrimin-ant function analysis	Measure of instrumental support combined paid and unpaid support
Kraus et al. (1976)	Canada	65+	334	Applicants for long-term care and community	1	-	Applicants to variety of residential care settings	Univariate analyses only	Network not known Support not known	Not stated; ? chi-square tests of association	Availability of instrumental support associated with being an applicant
Liu et al. (1991)	US	65+	5795	Community with chronic disability	2	2 years	Admission to and length of stay in nursing home	Demographic Health Psychological	Receive support * ↓	Cox PH regression	Instrumental support protective against entry

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); †NS not significant ‡ Cox proportional hazards regression; $ Community to nursing home transition (C→NH) or Nursing home to Community (NH→C); ↑ Risk factor; ↓ Protective factor

Table 2.3 (continued): Summary of studies that examined effects of social relationships on use of residential care

Author	Country	Baseline age	n	Sample	# Time points	Follow-up length	Residential care use definition	Covariates	Effect of social relationship adjusted for covariates	Main statistical method	Comments
McCoy and Edwards (1981)	US	65+	5192	Community welfare recipients	2	1 year	Resident ≥30 days in residential care during follow-up	Demographic Health	Network * ↓	Logistic regression	
Newman et al. (1990)	US	65+	5580	Community with chronic disability	2	2 years	Resident ≥90 days during follow-up	Demographic Health Psychological	Paid support * ↑ Unpaid support NS	Logistic regression	
Palmore (1976)	US	60+	207	Community	2	20 years	≥1 nursing home admission	Demographic	Network * ↓	Linear regression	Only decedents considered
Pearlman and Crown (1992)	US	65+	5273	Community	2	2 years	≥1 nursing home admission	Demographic Health Psychological Prior nursing home use	Network type * ↓ Support NS	Logistic regression	Sample have chronic disabilities; Instrumental support measured
Salive et al. (1993)	US	65+	4074	Community	2	3 years	≥1 nursing home admission	Demographic Health Psychological Prior nursing home use	Network with children * ↓ Support * ↓	Logistic regression	Emotional and instrumental support pooled measure
Shapiro and Tate (1985)	Canada	65+	3383	Community	3	2.5 and 7 year follow-up	≥1 admission to a long-term care facility	Demographic Health Psychological	Network with relatives* ↑ @ 7 year follow-up	Logistic regression	

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); †NS not significant ‡ Cox proportional hazards regression; $ Community to nursing home transition (C→NH) or Nursing home to Community (NH→C) ; ↑ Risk factor; ↓ Protective factor

Table 2.3 (continued): Summary of studies that examined effects of social relationships on use of residential care

Author	Country	Baseline age	n	Sample	# Time points	Follow-up length	Residential care use definition	Covariates	Effect of social relationship adjusted for covariates	Main statistical method	Comments
Speare et al. (1991)	US	65+	5151	Community	2	2 years	Place of residence at follow-up	Demographic Health Psychological	Network NS	Logistic regression	
Steinbach (1992)	US	65+	5151	Community	2	2 years	Not stated	Demographic Health	Network NS Engagement * ↓	Logistic regression	No measure of cognitive status included in analyses
Vicente et al. (1979)	US	55+	521	Community	2	10 years	≥1 nursing home admission	Demographic Health	Network * ↓	Discrim-inant function analysis	Measure of network based on marital status and living arrangement
Wan and Weissert (1981)	US	Mean age 75.6	1871	Community	2	3 years	≥1 nursing home admission	Demographic Health Psychological	Children network * ↓ Siblings network * ↑ Grandchildren network * ↓ Spouse network NS Other relatives network NS Friends network NS	Linear regression	
Wilmoth (2000)	US	60+	2713	Community	2	6 years	Not stated	Demographic Health	Children network + ↓ Social support from children * ↑	Event history analysis	
Wolinsky et al. (1992)	US	70+	5151	Community	2	6 years	≥1 nursing home admission	Demographic Health Psychological	Non-kin network * ↓ Kin network NS	Logistic regression	

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); †NS not significant ‡ Cox proportional hazards regression; $ Community to nursing home transition (C→NH) or Nursing home to Community (NH→C) ; ↑ Risk factor; ↓ Protective factor

2.5.4 Summary and conclusions

The reviewed literature demonstrates a lack of consistency in findings of the effects of social relationships on residential care use. This may be contributed to by the variety of definitions of both social relationships and residential care use that have been used. Differences in follow-up time, different statistical methods, different covariate adjustments and differences in populations may also partially explain the inconsistent results.

The findings concerning the effects of social networks on use of residential care were equivocal, and were dependent on the definition of social networks that was used. Some authors compared the effects of kin and non-kin social relationships (Wan and Weissert, 1981; Wolinsky et al., 1992; Freedman et al., 1994; Freedman, 1996; Wilmoth, 2000). Significant beneficial effects of networks with children (Wan and Weissert, 1981; Freedman, 1996; Wilmoth, 2000), siblings (Wan and Weissert, 1981; Freedman, 1996) and grandchildren (Wan and Weissert, 1981) were reported. In contrast, Wolinsky et al. (1992) demonstrated no significant effect of kin relationships on admission to residential care, but found a protective effect of better non-kin relationships. Thus specific social networks do appear to play some role in protecting against residential care use, but the specific social networks that are of most benefit remain unclear.

As was found for the studies of disability, a range of measures of social support were considered in the studies reviewed here. The definitions of social support were not always described, making it difficult to compare studies. The effects of instrumental social support were most commonly investigated, with most authors reporting that the receipt or availability of greater instrumental support was a risk factor for use of

residential care (e.g. Greene and Ondrich, 1990; Newman et al., 1990; Coward et al., 1996; Wilmoth, 2000). However, some authors reported instrumental support protected against residential care use (Greenberg and Ginn, 1979; Liu et al., 1991), although recall bias was possibly a factor here. Social engagement was examined far less extensively for residential care use than for disability. Since social engagement in particular is likely to be confounded with physical and psychological health status, this may well be reflected by the limited research on the relationship between social engagement and use of residential care.

The choice of the covariates that were included in the analyses reported in the studies reviewed here was not consistent. Furthermore, the set of covariates that were included in some studies was arguably inadequate. For example, many studies failed to adjust for cognitive status, yet this is a known risk factor for residential care admission. No study was identified that adjusted for a broad range of demographic, health, psychological and lifestyle covariates in the analyses.

While there is a general consensus that greater receipt of instrumental social support is a risk factor for use of residential care, the effects of the mezzo level of social relationships on residential care is less clear. Furthermore, the effects of specific social networks on use of residential care remain uncertain.

In summary, the literature review found no studies that examined changes in residential care use over a long follow-up period, used multiple waves of data, examined the effects of specific social networks with both kin and non-kin, and controlled for a broad range of covariates that encompassed demographic, health, psychological and lifestyle factors. Furthermore, the findings from studies carried out in the United States may be difficult

to translate to the tiered Australian residential care system. Finally, no literature has addressed the distinction between effects of different social networks on low-level and high-level care in Australia.

2.5.5 Social relationships and mortality

More evidence has accrued demonstrating the beneficial effects of social relationships on survival than for any other health outcome. Since the first study appeared on this topic in 1979, numerous studies have been published, with participants whose ages have covered the entire adult lifespan. Studies have most commonly investigated all-cause mortality, although some authors have also considered the effects of social relationships on cardiovascular mortality. In this review, the results relating only to the former are discussed, although it is noted that some of the reviewed articles also reported results for cause-specific mortality. Table 2.4 (pp. 104-109) presents an overview of the studies.

The seminal article in this area is that of Berkman and Syme (1979). In this study, data were available for 4725 participants in the Alameda County Study, who were aged 30 to 69 years at baseline, and deaths were tracked over nine years. Berkman and Syme's SNI (as outlined in Section 2.3.6) measured at baseline was used in this study. The SNI was based on a weighted sum of indicators of marital status, contacts with close friends and relatives, church membership, and group membership, and thus reflected a hybrid of social networks and social engagement. Berkman and Syme (1979) demonstrated that the risk of death for those in the highest quartile of the SNI versus the lowest quartile was 0.43 for males ($P<0.001$) and 0.36 for females ($P<0.001$); confidence intervals were not presented. The authors also alluded to a threshold effect, suggesting it was those who were largely bereft of contact with others who were at most risk of

death during follow-up. However, their presented analyses suggest a gradient in risk of death from those with the highest SNI to those in the lowest quartile of SNI. The analyses controlled for a broad range of health status variables and were carried out separately for genders and different age groups.

The results from this important study, however, provided little insight into which aspects of social relationships protect against death. As well, physical disability status and cognitive health status were potentially confounded with components of the SNI. Furthermore, no participants were aged more than 70 years.

Soon after the publication of Berkman and Syme's results, several studies attempted to replicate their work. For example, House et al. (1982) examined the effects of hybrid measures of social networks and social engagement on deaths over a decade of follow-up among 2754 Americans aged 35 to 69 years at baseline. Significant protective effects of group membership were found for men (OR 0.36; 95%CI not given) and church attendance for women (OR not given). For women, more time spent watching television was also associated with an increased risk of death (OR not given). House et al. (1982) presented results to support a threshold effect of social relationships for men and women, such that those lacking any ties were at greatest risk of death.

However, the same criticisms of the measures used in Berkman and Syme's work can be levelled at the variables used in this analysis. Attendance at church requires some minimum level of physical and cognitive ability. Similarly, measures of social engagement that are based on attendance at meetings of clubs or groups are confounded with disability and cognitive status. Furthermore, a narrower set of control variables

were used in House et al. (1982), and the covariates did not include cognition or disability status.

Another study modelled after the work of Berkman and Syme (1979) was published by Schoenbach et al. (1986). These authors drew data from 2059 participants aged 47 years or more at baseline in the Evans County Cardiovascular Epidemiologic Study carried out in Georgia. Twelve years of follow-up for deaths were conducted. Like Berkman and Syme (1979), they examined the effects of the SNI on mortality. After adjustment for age and cardiovascular disease risk factors, those participants with better SNI were at reduced risk of death during the follow-up period (HR 0.63; 95%CI 0.45 – 0.83). Subgroup analyses showed this effect was strongest among 60 to 80 year old Caucasian males (HR 0.32; 95%CI 0.16 – 0.63); the effect for females and younger participants was not significant. Of note, this study was the first on this topic to incorporate time-to-event in the analyses and used Cox proportional hazards models, and was one of the first studies to report an effect of social relationships on mortality for older people. These authors also reported a threshold effect among the oldest participants, such that those with the fewest social ties were at elevated risk.

Blazer (1982) reported results for an older, but smaller, cohort than the three aforementioned studies. Using data from 331 community-dwelling participants aged 65 to 93 years, Blazer (1982) analysed the effects of social networks and perceived social support on mortality over a 30 month follow-up period. Social networks were measured in two ways: i) the weighted sum of having a spouse (weight of 2) and number of children and siblings (weight of 1 per relative) and ii) the frequency of personal and phone contact with friends or relatives. A wide range of demographic, health and psychological variables were adjusted for in the analyses presented by Blazer (1982).

The analyses showed better networks (more children/siblings/spouse relative risk (RR) 0.49; 95%CI 0.28 – 0.86), more frequent contact with relatives or friends (RR 0.53; 95%CI 0.31 – 0.90) and more perceived support (RR 0.29; 95%CI 0.16 – 0.53) were significant risk factors for death during the follow-up period. Blazer (1982) reported a threshold effect for those participants with the least perceived support and poorest social networks at increased risk of death. However, this author also stated 'increased mortality was associated with a gradational decrease' in the first of the social network variables, but did not quantify this result.

Another early study was that by Zuckerman et al. (1984). In this work, the authors examined the effects of social networks on mortality over a two year follow-up period as part of a case-control study of 400 people. Study participants were aged 62 or more, had low socioeconomic status, and lived in Connecticut at baseline. Social networks were assessed through nine questions about the existence of, and contact with, confidants, friends, and children. The analyses controlled for health status and gender; other demographic and health variables were not significant in the univariate analyses. The results showed that having more children was a protective factor against mortality (OR 0.60; $P<0.05$; 95%CI not given). The effects of friends and confidants were not significant. However, Zuckerman et al. (1984) cautioned these findings may not hold in the general older population in which there is a much greater mix of socioeconomic circumstances.

In the first of a series of studies that investigated mortality and social relationships in Scandinavia, Welin et al. (1985) tracked survival in an all-male cohort. These authors followed the mortality outcomes over nine years for 989 men aged 50 years and 60 years at baseline living in Sweden. A combined measure of social networks and social

engagement in this study was termed ‘social activities’ by these authors. Their measure reflected visiting or hosting friends and relatives, attendance at organized meetings, and participation in organized sport. After controlling for age and health status, the risk of death for those in the highest quintile of social activities was less than one third that of those in the lowest quintile of social activities (OR 0.29; P<0.001; 95%CI not given). There was no evidence of a threshold of effect of social activity on mortality risk in the figures presented by Welin et al. (1985).

An additional Swedish study concerning mortality was conducted by Orth-Gomér and Johnson (1987). In this research, the effect of social networks on mortality over six years of follow-up in 17433 men and women aged 29 to 74 was considered. Orth-Gomér and Johnson (1987) developed a three-level social network scale that reflected the number of sources and degree of social contact with parents, children, siblings, friends, neighbours and co-workers. The authors showed that after controlling for age, lifestyle and health, better social networks at baseline were associated with a 25 per cent reduction in the risk of death in the subsequent six years (OR 0.75; 95%CI 0.59 – 0.94). Orth-Gomér and Johnson (1987) reported a threshold effect of social networks for young and middle-aged participants, such that there was little difference in mortality risk between the middle and upper social network categories. However, for participants aged 65 to 74 years, the differences between the three groups were more pronounced for men, and for older women those in the upper percentile band had an increased risk of mortality. These results suggest a threshold effect may not apply among older people, and that advantages from better social networks may continue to accrue.

Seeman et al. (1987) extended the analyses by Berkman and Syme (1979) and used 17 years of follow-up mortality data from the Alameda County Study. These authors

considered 4174 participants aged 38 years or more but unlike Berkman and Syme, Seeman et al. also included participants aged 70 to 94 years. In the survival analyses conducted in this article, adjustment was made for variables relating to demographic characteristics, health status, lifestyle, and depression. Seeman et al. (1987) considered marital status, social networks with friends and relatives, church membership, and group membership, and Berkman and Syme's SNI. Study participants aged 60 years or more with better social networks with friends and relatives had a reduced hazard of death over the follow-up period compared with their contemporaries with better social networks (HR 0.77; 95%CI 0.65 – 0.91). Church membership was also protective against mortality (HR 0.76; 95%CI 0.65 – 0.88). Persons aged 60 and older scoring in the highest quartile of the SNI had a mortality risk at least 30% less than that of persons in the lowest quartile (60-69: OR 0.66; 95%CI 0.48 – 0.92; ≥70: OR 0.59; 95%CI 0.44 – 0.81). However, the pooling of friends and relatives into one measure, as was done in this study, does not permit insight into which of these two types of social networks had an effect on survival.

A third study that drew participants from the Swedish population was conducted by Hanson et al. (1989), who studied five year mortality in 500 older male Swedes. In their research, Hanson et al. (1989) distinguished between social networks, social support and social engagement. A range of demographic and health variables were controlled for in the analyses. The social network variable was not predictive of mortality (HR 1.25; 95%CI 0.63 – 2.5), whereas better availability of emotional support (HR 0.40; 95%CI 0.19 – 0.83) and better social engagement (HR 0.45; 95%CI 0.23 – 0.91) were protective against death. Although these authors claimed support for a threshold of effects of social relationships on mortality, their dichotomization of social relationship variables at approximately the lowest quartile implies that the possibility of

a gradation in mortality risk according to social relationship category, rather than a threshold effect, was not examined.

The effects of social networks and social engagement on mortality over six and a half years of follow-up among 1060 Finnish residents aged 60-89 were investigated by Jylhä and Aro (1989). Unlike most other studies on this topic, the cohort in this study comprised people who lived in residential care in addition to people who lived in the community. Social networks were based on marital status, living arrangements, and proximity of children. Social engagement was measured through the frequency of participation in travel plus a range of family, cultural, religious and sporting events. The analyses were adjusted for age, perceived health, physical disability, and chronic disease. Social networks were not predictive of death during the follow-up period. There was evidence of an effect of better social engagement on decreased mortality (males: HR 0.93; 95%CI 0.87 – 0.99; females: HR 0.91; 95%CI 0.84 – 0.99). Jylhä and Aro (1989) noted that when social engagement was dichotomized, it was not a significant predictor of mortality. Thus the the substantive conclusions in the article appeared to be quite sensitive to the specification of social relationships.

The mortality outcomes of a sample of 645 French adults aged 60 years or more were tracked over four years by Grand et al. (1990). The effects of social networks and social engagement on survival were examined. These authors found a significant protective effect of greater frequency of visiting others after adjusting for a range of health variables, age and gender (HR 0.49; $P<0.05$; 95%CI not given). However, the majority of the social relationship measures had non-significant effects on mortality.

Using data from a cohort of 1752 Danish people aged 70 or more, Olsen et al. (1991) examined the relationship between two measures of social networks and mortality over 14 years of follow-up. The analyses were adjusted for age, gender and health status. Social networks in this study were measured by the frequency of contact with i) children, siblings and friends and ii) grandchildren and other relatives. These authors reported no effect of social networks with children, siblings and friends (better network HR 0.77; 95%CI 0.42 – 1.40) nor for social networks with grandchildren and other relatives (better network HR 1.13; 95%CI 0.83 – 1.53). However, the size of the referent category for both social network variables was extremely small (n=11 and n=59 respectively), and this may have impacted on the results.

In a study of 1054 Hong Kong Chinese participants aged 70 years or more, Ho (1991) studied two-year mortality in relation to social networks, social support and social engagement. After adjusting for demographic, health and cognitive variables, these three aspects of social relationships were not significant predictors of mortality, although the adjusted results were not quantified in Ho (1991). She contended that social relationships may modify the effects of other health indicators on mortality. However, the analyses presented by Ho did not directly address this hypothesis, and so this conclusion cannot be substantiated from the results presented in Ho (1991).

The first Australian study to examine the effects of social relationships on mortality was reported by Jorm et al. (1991). In a sample of 274 Tasmanians aged at least 70 years, social networks and perceived social support were assessed via the availability and adequacy of relationships with a confidant and with friends or neighbours. The Interview Schedule for Social Integration (Henderson et al., 1980) was used to collect these data. These authors reported no association between five year mortality and social

relationships. However, only univariate analyses were undertaken. Also of note is that vital status for a relatively large proportion of study participants (n=46; 17%) was unknown. This may have diluted any effects of social networks. Sensitivity analyses that could have examined this issue were not presented by the authors.

Shahtahmasebi et al. (1992) considered the effects of different network typologies (Wenger, 1989) on eight year mortality in a sample of 534 Welsh people aged at least 65 years. These authors also considered the effects of frequency of contact with family, the size of the total social network, and the non-spousal relative seen most often on mortality over the specified period. Using forward selection of variables into a Weibull model, these authors found no significant effects of the social network measures on mortality after adjustment for sociodemographic variables and self-rated health.

Vogt et al. (1992) followed a cohort of 2573 subscribers to a US health insurance fund aged 18 years or more. Mortality and first incidence of cancer, hypertension, ischaemic heart disease, and stroke were assessed over the ensuing 15 years. Vogt et al. (1992) considered the effects of network density, size and the frequency of contact with relatives, friends, and the community. Cox proportional hazards models were used to test the effects of social network variables upon the outcomes. Age, gender, socioeconomic status, smoking history, and self-rated health were included in the analyses. The three measures of better social networks were protective against all-cause mortality over the follow-up period (network density: HR 0.37; 95%CI 0.25 – 0.56; frequency of contact: HR 0.67; 95%CI 0.50 – 0.91; size: HR 0.71; 95%CI 0.56 – 0.91). For the oldest participants aged 75 or more, network density (HR 0.43; 95%CI 0.24 – 0.77) and network size (HR 0.50; 95%CI 0.26 – 1.00) exhibited significant protective effects against mortality from all causes.

Steinbach (1992) considered the effect of social networks and social engagement upon mortality over two years of follow-up in a cohort of 5151 US residents aged 70 years or more. (She separately considered admissions to residential care in the same article, as described in Section 2.6.4.) Demographic and health status were controlled for in the analyses of mortality, but cognitive status was not included in the set of covariates. Steinbach (1992) showed that better social networks with friends or relatives (talked on the telephone with friends/neighbours or relatives, got together with friends/neighbours or relatives) and social engagement over the preceding 12 months (attendance at a senior centre, church, or movies) each decreased the risk of mortality (network: OR 0.60; 95%CI 0.42 – 0.85; engagement: OR 0.59; 95%CI 0.49 – 0.70).

The effects of network size and a hybrid measure of social networks and social engagement on survival over seven years of follow-up were examined in a sample of 363 New York residents by Forster and Stoller (1992). After adjustment for indicators of health status, the authors found larger social networks were a significant risk factor for mortality in males (OR 1.09; 95%CI 1.00 – 1.17) and females (OR 1.07; 95%CI 1.00 – 1.15). For female participants, there was also a protective effect of better social networks/engagement (OR 0.93; 95%CI 0.87 – 1.00). The risk of death associated with larger social networks is contrary to the majority of literature on this topic, but these findings need to be interpreted in the context of how the size of the network was ascertained. The respondents were asked to name up to five people who provided instrumental support, and these five people were also asked to identify members of the 'helping' network. Thus in this study the size of network probably actually reflected the extent of instrumental support, and probably included paid as well as unpaid care. In addition, the analyses did not control for demographic nor psychological characteristics

of participants. A final point to be made is that the sample size was small, with around 120 deaths among participants.

A second study of mortality published by Seeman et al. (1993) compared the effects of a hybrid of social networks (with spouse and friends/relatives) and social engagement (church attendance and group membership) on mortality over five years among older participants in three sites of the EPESE studies. These authors showed that there was a protective effect of more social network/engagement on mortality for male and female participants in the New Haven EPESE (males: OR 0.42; 95%CI 0.33 – 0.74; females: OR 0.53; 95%CI 0.33 – 0.85) and female participants in the Iowa EPESE (OR: 0.56; 95%CI 0.33 – 0.95) but no significant effects were found in East Boston. Seeman et al. (1993) used these results to posit that socio-cultural differences may be important in explaining differences between findings from various studies. Furthermore, this was one of the first studies to include cognitive status along with socioeconomic status, health and lifestyle factors in the set of covariates included in the survival analyses.

Sabin (1993) considered the effects of social networks, social support, and social engagement on four year mortality among 7502 participants in the LSOA. In this work, an exploratory factor analysis of 13 social relationship items was carried out, and from this analysis five scales were derived. Sabin termed these *instrumental* (availability of help for ADL or IADL disability), *socio-expressive* (phone and personal contact with friends or relatives, church attendance, volunteer work), *someone to help* (whether lived alone and availability of someone to help with care if needed), and *kin and kin contact* (number of children and relatives and frequency of contact with relatives). His results demonstrated the socio-expressive scale and kin and kin contact scale were protective against mortality whereas instrumental support was a risk factor for mortality over the

four year follow-up period. However, there is clear overlap between the socio-expressive scale and the kin and kin contact scale. Furthermore, parameter estimates were not presented, making a quantitative comparison with other studies difficult.

Sugisawa et al. (1994) considered the effects of social networks, social support and social engagement on mortality in 2,200 Japanese adults aged 60 and over. The vital status of participants was followed for three years after baseline data collection. Social networks were measured by the average frequencies of visiting with children, relatives and friends. Social engagement was defined by organizational attendance. Perceived instrumental support and emotional support were also assessed. Sugisawa et al. (1994) found no significant effects of the frequency of contact or social support on mortality. However, there was a significant protective effect of social engagement against mortality (HR 0.68; $P<0.05$; 95%CI not given). The analyses controlled for many covariates, including sociodemographic, health status, disability and lifestyle. However, cognition and depression were not among the covariates that were included in the analyses in this study.

Subsequent to the publication of Jorm et al. (1991), two additional Australian studies that considered the effects of social relationships on mortality were published in the mid-1990s (McCallum et al., 1994; Simons et al., 1996). In the first of these, McCallum et al. (1994) was primarily interested in the effect of self-rated health on seven year mortality in 1050 participants aged 60 years or more who lived in Sydney. However, this research is included in this review as it is one of the few Australians studies that have considered the effects of social relationships on mortality *per se.* These authors considered the effects of a confidant social network and social engagement on mortality. After adjustment for a range of demographic and health

variables plus symptoms of depression, the authors found a protective effect of a better confidant network for women (HR 0.78; 95%CI 0.62 – 0.99) but not men (HR 1.05; 95%CI 0.82 – 1.36). Social engagement did not have a significant effect on mortality in McCallum et al.'s study.

Simons et al. (1996) examined the effects of social networks with friends and social engagement on mortality over five years in a sample of 2805 people aged at least 60 years who took part in the Dubbo Study of Australian Elderly. After adjustment for a broad range of demographic, lifestyle and health variables, no significant effects of social relationships on mortality were found. Although assessed, cognitive status was not included in the set of control variables used in the survival analyses.

Kawachi et al. (1996) examined the effects of social networks and social engagement on four year mortality in a cohort of 32624 US male health professionals aged 42 to 77 years. These authors calculated the Berkman-Syme SNI and found that after adjustment for demographic, lifestyle, and health variables, those in the highest SNI quartile had close to a 30 per cent reduction in the hazard of death in comparison to those in the lowest quartile (HR 0.72; 95%CI 0.52 – 1.01). Kawachi et al. also reported that having close friends and relatives (HR 0.75; 95%CI 0.56 – 0.99) and belonging to a church group (HR 0.70; 95%CI 0.59 – 0.84) were significantly protective against all-cause mortality. However, only a small proportion of the men died during the follow-up period (1.6%) and the average age of the participants was unclear. Furthermore, the participants were dentists, vets, pharmacists, optometrists, osteopaths and podiatrists, and therefore collectively the sample had high socioeconomic status.

The effects of social networks and social support on mortality over two and half years in 2829 participants in the Longitudinal Aging Study Amsterdam were reported by Penninx et al. (1997). Social network size was calculated from the number of people in the same household, children, other relatives, neighbours, work and study contacts, contacts in organizations and other contacts that were reported by study participants. Emotional support and instrumental support were calculated as the average amount of each type of support received from network members. The authors also argued their measure of loneliness reflected received social support. After controlling for demographic, health and lifestyle variables, the adjusted hazard of death for participants receiving high levels of instrumental support was almost twice that of those receiving low levels of instrumental support (HR 1.74; 95%CI 1.12 – 2.69). Conversely, the effects of high levels of emotional support were protective (HR 0.68; 95%CI 0.47 – 0.98). Participants who reported greater loneliness were also more likely to die during the follow-up period (HR 1.06; 95%CI 1.00 – 1.12). The effect of network size was not significant (HR 0.99; 95%CI 0.97 – 1.01), although this may reflect the scoring of network size (with a range from 0 – 75) as much as the true absence of an effect. Penninx et al. (1997) concluded their findings were consistent with the direct effects hypothesis.

In a study of 806 older women, Yasuda et al. (1997) examined the effects of individual variables that measured social networks and social engagement in the local neighbourhood on five year mortality. The analyses adjusted for health status and educational attainment, and were separately conducted for younger and older women. Among women aged 65 to 74 years, the women who lived alone had a lower risk of death than those who lived with a spouse (HR 0.4; 95%CI 0.2 – 0.9). Less than three children was also protective against mortality in the younger-old women (0 vs 3+

children HR 0.4; 95%CI 0.1 – 0.8; 1-2 vs 3+ children HR 0.4; 95%CI 0.2 – 0.8). Among the women aged 75 years or more, visits or telephone contact from children (HR 0.3; 95%CI 0.1 – 0.8) and friends (HR 0.5; 95%CI 0.2 – 1.0) were protective against death, as were group membership (HR 0.4; 95%CI 0.2 – 0.9) and attendance at group organizations (HR 0.4; 95%CI 0.2 – 0.8). More than a decade's residence in the neighbourhood (HR 0.4; 95%CI 0.2 – 0.8) and friendly shopkeepers (HR 0.5; 95%CI 0.3 – 0.8) were also protective against mortality in the women aged 75 years or more. The authors argued that a compensation of other network members may operate among the younger women who lived alone and had few children, first suggested by Cantor (1979). However, this does not adequately explain the lack of an effect seen in the older women. It is difficult to interpret the results from the large number of uni-dimensional measures considered in this study.

Cerhan and Wallace (1998) evaluated the association between change in SNI over a three year period and subsequent mortality over eight years of follow-up in a sample of 2575 participants aged 65 years and older in the Iowa EPESE. The results showed that after adjustment for demographic, health, and psychological variables, there was a protective effect of better social relationships for males at both baseline (HR 0.6; 95%CI 0.4 – 0.9) and follow-up (HR 0.7; 95%CI 0.5 – 0.9). Similar protective effects were observed for female participants at baseline (HR 0.8; 95%CI 0.6 – 1.1) and follow-up (HR 0.7; 95%CI 0.5 – 0.9). Cerhan and Wallace also showed that participants with a high SNI at both interview times had a lower hazard of death over the ensuing follow-up period (males HR 0.4; 95%CI 0.3 – 0.7; females HR 0.6; 95%CI 0.4 – 0.8).

The mortality outcomes in a sample of 1010 Norwegians aged 20 years or more at baseline were followed-up over 17 years by Dalgard and Haheim (1998). In this study,

social network was measured in terms of the number of close relationships with family, friends and neighbours, the frequency of contact and the quality of relationships, while social engagement was based on the number of organizational memberships, frequency of meeting attendance and the self-assessed importance of the groups or associations. After adjustment for a range of demographic, health and lifestyle variables, greater social engagement protected against death in both men (HR 0.65; 95%CI 0.50 – 0.84) and women (HR 0.80; 95%CI 0.65 – 0.99). Having more close relationships had a marginally significant protective effect for men (HR 0.93; 95%CI 0.86 – 1.01) but not women (HR 1.02; 95%CI 0.95 – 1.10). The self-reported quality of the relationships did not have a significant effect on mortality. However, given that the average age of participants was only 46.3 years at the initial interview and results for younger and older participants were not presented separately, these findings add little insight to understanding the effects of social relationships on mortality in older people.

Korten et al. (1999) published a fourth Australian study in this area. These authors examined the effects of the size of the total social networks, the existence of a confidant network, and perceived instrumental social support on mortality over three and a half years of follow-up. The study included 897 participants aged 70 years or more who were living in the community in the Canberra region at baseline. Cox proportional hazards models were fitted that controlled for gender, health and cognitive status, and the analyses showed no significant effects of any of the social network measures on mortality. Separate analyses by gender were carried out but these may have been underpowered, as there were 29 covariates considered in the statistical models, but only 110 deaths among males and 62 among females occurred during the follow-up period.

The effect of social engagement on mortality over 13 years among participants in the New Haven EPESE was examined by Glass et al. (1999). In this study, social engagement was measured from church attendance, visits to the cinema, restaurants or sporting events, day or overnight trips, playing games such as cards and bingo, and participation in social groups. After adjustment for a range of demographic, health and lifestyle variables, the participants in the highest category of social engagement were significantly less likely to die during the follow-up period than the participants in the lowest activity category (HR 0.81; 95%CI 0.74 – 0.89). It is possible that the activities considered in the measure of social engagement are correlated with both physical and cognitive status, as several correspondents concerning this article suggested (Riddoch, 2000; Molineux, 2000). Thus the measure termed social engagement may be a surrogate measure of physical health or cognitive status.

Walter-Ginzburg et al. (2002) examined the effects of social networks, social support and social engagement on deaths among 1340 Jewish people aged 75 to 94 years living in Israel. Mortality was followed for eight years among participants. An extensive number of demographic, health, lifestyle and cognitive variables were included in the analyses. The authors found that perceived emotional support, the number of living children, or engagement in solitary leisure activity were not predictive of mortality risk. Social engagement in the form of group leisure activity was significantly protectively against death (HR 0.78; 95%CI 0.63 – 0.96). Participants who lived in the community without a spouse but with a child were found to be at increased risk of death compared with those who lived alone in the community (HR 1.49; 95%CI 1.15 – 1.91).

In a recent study of mortality and twinship, Rasulo et al. (2005) examined the effects of social networks with children, other relatives and friends on mortality over six years of

follow-up among 1734 older Danish twins. The results showed there a significant risk of mortality for those with infrequent contact with friends compared to those with more frequent contact. Having a spouse and close ties with their co-twin were protective against death (ORs not presented by Rasulo et al.). There were no significant effects of networks with other relatives nor with children. The sample of study participants that was included in the analysis was restricted to currently or previously married people, with the rationale for this the high correlation between marital status and having children. Only demographic variables plus self-rated health were included as covariates in the statistical models. It was not clear from Rasulo et al. (2005) if clustering due to pairs of twins that were included in the sample was adjusted for in the statistical analysis.

Among a sample of 295 Swiss octogenarians, Guilley et al. (2005) examined five year mortality in relation to kin and non-kin networks. Their results showed that having at least one living sibling at baseline was associated with a reduced hazard of death (HR 0.56; 95%CI 0.35 – 0.89), as was having one or more close friends (HR 0.60; 95%CI 0.37 – 0.99). The number of children was not significant in univariate analyses (HR 0.96; P-value=0.883; 95%CI not given). Functional aspects of relationships with friends did not have significant effects on the mortality risk. However, a limited number of covariates – age, gender, disability and depressive symptoms – were included in the analyses by Guilley et al. (2005). In addition, the measure of close friends possibly also reflected confidants, as the wording for this question in the baseline interview was 'Outside your family, do you have a close friend (i.e. a person you love, a person about whose future you feel concern, toward whom you show solidarity?)'.

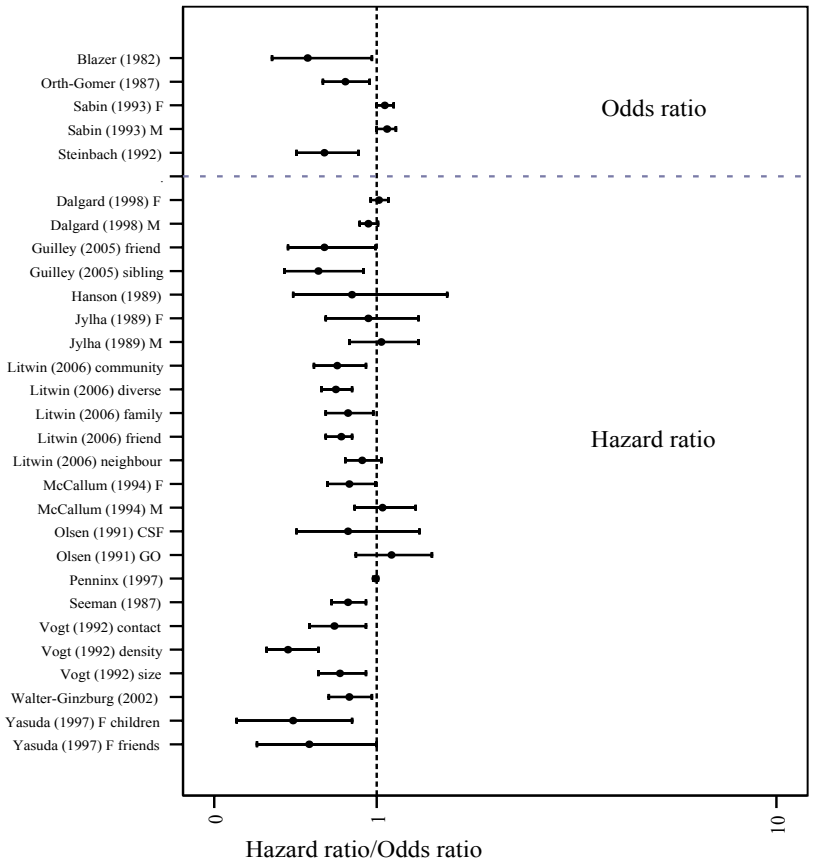
Recently, Litwin and Shiovitz-Ezra (2006) examined the effect of network typology on seven year mortality risk among more than 5000 Israelis aged 60 and older. Each participant in the study was classified from their baseline responses as one of six network typologies, namely diverse, friend focused, neighbour focused, family focused, community-clan, or restricted networks (Litwin, 2001). The results showed significantly protective effects against mortality for participants with diverse (HR 0.68; 95%CI 0.58 – 0.80), friend (HR 0.72; 95%CI 0.61 – 0.86), family (HR 0.77; 95%CI 0.61 – 0.97) and community-clan networks (HR 0.69; 95%CI 0.53 – 0.91) when compared to those with restricted networks. There was no significant effect conferred to those in neighbour networks in comparison with restricted networks, suggesting participants in these two network typologies were at the greatest risk of death during the follow-up period.

In keeping with the focus of this thesis, the size of the effects for those studies which specifically addressed the relationship between social networks and mortality are summarized in Figure 2.9. As for the analogous plots for disability and residential care use (in Figures 2.7 and 2.8), there are some limitations that must be borne in mind for Figure 2.9. Most studies have used logistic regression or Cox proportional hazards to analyse the effects of social networks on mortality, and thus odds ratios, relative risks and hazard ratios are all shown in Figure 2.9. Studies that did not report results in terms of an odds ratio, relative risk or hazard ratio are not included in the figure, and similarly studies that did not report a confidence interval are also excluded from the figure.

The combined results from the studies shown in Figure 2.9 suggest an overall protective effect of social networks on mortality, although the substantial variation in the size of the effects is clear from the figure. Table 2.4, which follows Figure 2.9, is then presented which gives an overview of the studies reviewed in Section 2.5.5. A summary of the

studies and limitations in current knowledge is then presented in Section 2.5.6, which begins on page 110.

Figure 2.9: Forest plot of effects of social networks on mortality



M=males; F=Females; CSF=children, spouse, friends; GO=grandchildren, other relatives; Blazer through Steinbach analysed using logistic regression; remainder analysed using Cox PH

Table 2.4: Summary of studies that examined effects of social relationships on mortality

Author	Country	Baseline age	Mean length of follow-up	# deaths/ total n	Sample	Covariates	Effect of social relationship adjusted for covariates	Main statistical method used	Comments
Berkman and Syme (1979)	US	30-69	9 yrs	371/4725	Community	Demographic Health Lifestyle	Network-Engagement hybrid * ↓	Binary linear regression	Seminal article
Blazer (1982)	US	65-93	2.5 yrs	50/331	Community	Demographic Health Lifestyle Psychological	Network * ↓ Support * ↓	Binary linear regression	Emotional social support protective
Cerhan and Wallace (1998)	US	65+	8 yrs	1059/2575	Community	Demographic Health Lifestyle Psychological	Network-Engagement hybrid * ↓	Cox PH	Rural population
Dalgard and Haheim (1998)	Norway	20+	17 yrs	217/1010	Community	Demographic Health Lifestyle	Network + ↓ [M only] Engagement * ↓	Cox PH regression	
Forster and Stoller (1992)	US	65+	7 yrs	Deaths not stated ~123/363	Community	Health	Network * ↑ Engagement * ↓ [F only]	Logistic regression	Multi-collinearity of control variables not addressed; Larger network risk factor; Instrumental support risk factor
Glass et al. (1999)	US	65+	13 yrs	Deaths not stated (~1743)/2812	Community	Demographic Health Lifestyle	Engagement * ↓	Cox PH regression	Correlation between social and physical activity may have affected results

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); †NS not significant ‡ Cox proportional hazards regression; ↑ risk factor; ↓ protective factor

Table 2.4 (continued): Summary of studies that examined effects of social relationships on mortality

Author	Country	Baseline age	Mean length of follow-up	# deaths/ total n	Sample	Covariates	Effect of social relationship adjusted for covariates	Main statistical method used	Comments
Grand et al. (1990)	France	60+	4 yrs	111/645	Community	Demographic Health Lifestyle	Network * ↓ Engagement NS †	Cox PH regression	Rural population
Guilley et al. (2005)	Switzerland	80-84	5 yrs	84/295	Community	Demographic Health Psychological	Network with friends * ↓ Network with siblings * ↓	Cox PH regression	Limited covariates and small sample
Hanson et al. (1989)	Sweden	68-69 year old	5 yrs	67/500M	Not stated but probably community	Demographic Health Lifestyle	Network NS Support * ↓ Engagement * ↓	Kaplan-Meier and Cox PH	Emotional support protective
Ho (1991)	Hong Kong	70+	2 yrs	89/1054	Community and residential care	Demographic Health Lifestyle Psychological	Network NS Support NS Engagement NS	Logistic regression	Receipt of instrumental social support not predictor
House et al. (1982)	US	35-69	10.5 yrs	259/2754	Community	Demographic Health	Network NS Engagement * ↓	Logistic regression	
Jorm et al. (1991)	Australia	70+	5 yrs	94/274	Community	Univariate analyses only	Support NS	Independent samples t-tests and chi-square tests of association	For 46 people vital status undetermined; Perceived availability and adequacy of emotional support assessed
Jylhä and Aro (1989)	Finland	60-89	6.5 yrs	393/1060	Community and residential care	Demographic Health	Network NS Engagement * ↓	Cox PH regression	Separate analyses for males and females

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); †NS not significant ‡ Cox proportional hazards regression; ↑ risk factor; ↓ protective factor

Table 2.4 (continued): Summary of studies that examined effects of social relationships on mortality

Author	Country	Baseline age	Mean length of follow-up	# deaths/ total n	Sample	Covariates	Effect of social relationship adjusted for covariates	Main statistical method used	Comments
Kawachi et al. (1996)	US	42-77	4 yrs	511/32624 M	Community	Demographic Health Lifestyle	Network-engagement hybrid * ↓	Cox PH regression	Also considered cause specific mortality significant trends particularly for cardiovascular disease and accident/ suicide
Korten et al. (1999)	Australia	70+	3.5 yrs	172/897	Community	Demographic Health Lifestyle Psychological	Network NS Support NS	Cox PH regression	Broad range of covariates
Litwin and Shiovitz-Ezra (2006)	Israel	60+	7 yrs	1440/5055	Community	Demographic Health	Network typology * ↓	Cox PH regression	Presence of Alzheimer's disease but not cognitive status included as covariate
McCallum et al. (1994)	Australia	60+	7 yrs	~231/1050	Community	Demographic Health Psychological	Network with confidant * ↓ [F only] Engagement NS	Cox PH regression	Primary focus on self-rated health
Olsen et al. (1991)	Denmark	70-100	15.5 yrs	1501/1752	Community	Demographic Health	Network NS	Cox PH regression	Also not related to cardiovascular mortality

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); †NS not significant ‡ Cox proportional hazards regression; ↑ risk factor; ↓ protective factor

Table 2.4 (continued): Summary of studies that examined effects of social relationships on mortality

Author	Country	Baseline age	Mean length of follow-up	# deaths/ total n	Sample	Covariates	Effect of social relationship adjusted for covariates	Main statistical method used	Comments
Orth-Gomér and Johnson (1987)	Sweden	29-74	6 yrs	841/17433	Community and residential care	Demographic Health Lifestyle	Network * ↓	Logistic regression	Cardiovascular mortality also considered; social network marginally significant effect
Penninx et al. (1997)	Netherlands	55-85	2.5 yrs	202/2829	Community	Demographic Health Lifestyle	Network NS Instrumental support * ↑ Emotional support * ↓ Engagement *	Cox PH regression	Instrumental support risk factor; emotional support protective
Rasulo et al. (2005)	Denmark	75+	6 yrs	Deaths not stated/1734	Twins; not stated if only community-dwelling	Demographic Self-rated health	Network with children NS Network with relatives NS Network with friends *	Gompertz model	Limited set of covariates; possible interaction of gender and contact with friends
Sabin (1993)	US	70+	4 yrs	1719/7502	Community	Demographic Health	Network type* Support NS Engagement * ↓	Logistic regression	? if correction for multiple comparisons should have been applied
Schoenbach et al. (1986)	US	47+	12 yrs	530/2059	Community	Demographic Health	Network-Engagement hybrid * ↓	Cox PH and logistic regression	
Seeman et al. (1987)	US	38-94	17 yrs	1219/4174	Community	Demographic Health Lifestyle Psychological	Network * ↓ Engagement * ↓ Network-Engagement hybrid * ↓	Cox PH regression	Effects not seen in younger participants

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); †NS not significant ‡ Cox proportional hazards regression; ↑ risk factor; ↓ protective factor

Table 2.4 (continued): Summary of studies that examined effects of social relationships on mortality

Author	Country	Baseline age	Mean length of follow-up	# deaths/ total n	Sample	Covariates	Effect of social relationship adjusted for covariates	Main statistical method used	Comments
Seeman et al. (1993)	US	65+	5 yrs	Deaths not stated; n NH 2812; EB 3809; I 3673	Community	Demographic Health Lifestyle Psychological	Network-Engagement hybrid * in New Haven [M&F] and Iowa F only	Kaplan-Meier and Cox PH regression	New Haven most diverse of three populations
Shahtahmasebi et al. (1992)	Wales	65+	8 yrs	264/534	Community	Demographic Health	Network type NS	Weibull model	Date of death unknown for 40 people
Simons et al. (1996)	Australia	60+	5 yrs	419/2805	Community	Demographic Health Lifestyle Psychological	Network NS Engagement NS	Cox PH regression	
Steinbach (1992)	US	70-99	2 yrs	Deaths not stated/5151	Community	Demographic Health	Network * ↓ Engagement * ↓	Logistic regression	Use of residential care other main outcome (Table 2.3)
Sugisawa et al. (1994)	Japan	60+	3 yrs	161/2200	Not stated	Demographic Health Lifestyle	Network NS Support NS Engagement * ↓	Hazard rate model assuming hazard function follows Weibull distribution	Compared Weibull with Cox proportional hazards model
Vogt et al. (1992)	US	18+	15 yrs	502/2573	Subscribers to health maintenance organization	Demographic Health Lifestyle	Network * ↓	Cox PH regression	Also predicted 5 year incidence of ischaemic heart disease

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); †NS not significant ‡ Cox proportional hazards regression; ↑ risk factor; ↓ protective factor

Table 2.4 (continued): Summary of studies that examined effects of social relationships on mortality

Author	Country	Baseline age	Mean length of follow-up	# deaths/ total n	Sample	Covariates	Effect of social relationship adjusted for covariates	Main statistical method used	Comments
Walter-Ginzburg et al. (2002)	Israel	75-94	5.3 yrs	812/1340	From National Population Register; includes community and institution	Demographic Health Lifestyle Psychological	Network NS Support NS Engagement * ↓	Cox PH regression	Comprehensive set of covariates
Welin et al. (1985)	Sweden	50 and 60	9 yrs	151/989M	Community	Demographic Health	Engagement * ↓	Logistic regression	Males only
Yasuda et al. (1997)	US	65-99	5 yrs	149/806F	Community	Demographic Health	Network * ↓ Engagement * ↓	Cox PH regression	Females only
Zuckerman et al. (1984)	US	62+	2 yrs	47/398	Public assisted housing in community	Demographic Health	Children network * ↓ Confidant network NS Friends network NS	Logistic regression	Early example of specific network investigation

* Significant (P<0.05); + Marginally significant (0.05<P<0.10); †NS not significant ‡ Cox proportional hazards regression; ↑ risk factor; ↓ protective factor

2.5.6 Summary and conclusions

There is a diverse literature in this area, and the wide range of publications reflect different cultures, disciplinary orientations, different follow-up length, different sets of covariates, a wide range of sample sizes and varied participant ages.

Better social networks were shown to protect against mortality in the majority of studies, although a sizeable number reported no effect (House et al., 1982; Hanson et al., 1989; Ho, 1991; Olsen et al., 1991; Shahtahmasebi et al., 1992; Sugisawa et al., 1994; Simons et al., 1996; Penninx et al., 1997; Korten et al., 1999). Specific social networks types were considered in only a few studies that generally supported the importance of networks with kin, although Zuckerman et al. (1984) reported significant protective effects of networks with children whereas Sabin (1993) demonstrated siblings but not children were protective against death. The recent work by Guilley et al. (2005), in which it was shown that close friends and siblings, but not children, were protective against death also supports the importance of considering different types of social networks in different cultures. The effect of different types of social networks on mortality remains an important area where additional research is needed.

In general, social support was not predictive of mortality (Ho, 1991; Jorm et al., 1991; Sabin, 1993; Sugisawa et al., 1994; Korten et al., 1999; McCallum et al., 1994; Walter-Ginzburg et al., 2002), although protective effects of emotional social support were reported by Blazer (1982), Hanson et al. (1989) and Penninx et al. (1997), and an increased hazard of death associated with instrumental support was demonstrated by Forster and Stoller (1992). As noted by Walter-Ginzburg et al. (2002), the positive effect of perceiving emotional or instrumental support and the negative effect of

needing the support possibly attenuated any protective effect of social support on the risk of mortality.

Social engagement was generally shown to protect against mortality, although several authors failed to demonstrate a significant association (Grand et al., 1990; Ho, 1991; McCallum et al., 1994; Simons et al., 1996). Studies that have followed closely the work of Berkman and Syme (1979) – that is, House et al. (1982), Schoenbach et al. (1986), Seeman et al. (1987), Seeman et al. (1993), Kawachi et al. (1996), and Cerhan and Wallace (1998) – have all shown protective effects of a social network-engagement hybrid measure (usually the SNI) on mortality. Of note, Seeman et al. (1993) found an absence of an effect in one of the communities (East Boston) that they studied. The problems that may exist in the analyses of social engagement with respect to confounding may also have contributed to difficulties in interpreting findings from studies with hybrid measures such as the SNI.

Several early studies reported a threshold of social relationships above which little advantage in terms of survival was gained (Berkman and Syme, 1979; House et al., 1982; Hanson et al., 1989). Among older people, the evidence is less clear. Blazer (1982) and Schoenbach et al. (1986) argued their studies provided evidence for a threshold effect. In contrast, Orth-Gomér and Johnson (1987) and Welin et al. (1985) found evidence of a dose-response effect leading to the conclusion of a gradient in risk. None of the studies formally tested the hypothesis of threshold effects of specific types of social networks on mortality. This appears to have been a neglected area of inquiry for several decades. More work in this area is needed to ascertain if a low level of social networks predict death or whether there is a gradient in risk of death associated with social networks.

Over time, the statistical methods used to analyse the association between mortality and social relationships have evolved, reflecting the adoption of advances in statistical methods into mainstream software packages. Of the many published studies in this area, only Sugisawa et al. (1994) presented an assessment of model fit, through a comparison of the fit of the Weibull model to that of a Cox proportional hazards model. However, other aspects of model checking, such as an assessment of the proportionality assumption in the Cox model, were not reported by any authors.

Covariates that reflect demographic, health, lifestyle and psychological domains were rarely all included in the reviewed studies. Some notable exceptions that adjusted for a comprehensive set of covariates in their analyses included Blazer (1982), Korten et al. (1999), Seeman et al. (1993) and Walter-Ginzburg et al. (2002).

Thus despite the large amount of research in this area over a sustained period, questions remain concerning the effects of social networks on mortality. Better social networks in general appear to protect against mortality, but it remains unclear if there are different effects of specific social networks on survival. There is a paucity of studies that have controlled for a broad range of covariates and examined the effects of specific social networks with both kin and non-kin on mortality. An assessment of the fit of the statistical models in these studies has been largely lacking. The evidence concerning a threshold effect of total social networks on mortality among older people is equivocal, and no studies have investigated if there are threshold effects of specific social networks.

2.6 AIMS OF THIS THESIS

Despite the call for '[f]urther research concerning the relative importance and meanings of various types of social networks…' over a decade ago (Sugisawa et al., 1994), this literature review has demonstrated that there are still important gaps in knowledge concerning the effects of social networks on health.

As this review has shown, the effects of specific social networks on disability, residential care utilization, and survival respectively have been examined by relatively few authors. For disability, the majority of studies have used two time points of observation, with a relatively short follow-up time of two to three years. Longitudinal studies with at least three time points of observation and follow-up time over the medium to longer-term were scarce. The findings by Mendes de Leon et al. (1999; 2001) represent the only research to date that has examined the effects of specific social networks on disability. No research that has considered the effects of specific social networks on Nagi disability was identified in the review.

The delivery of a health service such as residential care is highly dependent on the local context, in terms of policy and provision of places. For this reason, the translation of knowledge from studies concerning residential care use conducted in the United States to the Australian environment may be inappropriate and, worse still, misleading. This review has shown the majority of studies concerning use of residential care have used two times of observation, with a relatively short follow-up time of two or three years. Few longitudinal studies that used at least three time points of observation with longer-term follow-up have examined the impact of social networks on use of residential care, and no such studies have been reported from Australia.

Although the effects of social networks on mortality have been examined in a larger number of studies, only three studies that examined the effects of specific types of social networks on mortality were identified (Zuckerman et al. 1984; Sabin, 1993; Guilley et al. 2005). There were limitations in these three studies. Zuckerman et al. (1984) and Guilley et al. (2005) each comprised fewer than four hundred participants, while Sabin did not quantify the size of the effects in his study. As is the case for disability and residential care use, no Australian studies have been conducted that explicitly consider the effects of specific social networks on mortality.

An important point that has frequently been overlooked in the literature concerning social relationships and the three health outcomes considered in this review is the potential for correlations between covariates and measures of social relationships. The possibility of confounding of results in the reported literature for all three outcomes is ostensibly high. In many of the studies reviewed here, important covariates, such as cognitive function or the number of comorbid conditions, have been omitted from the analyses. In some instances, the studies from which data were drawn did not include these measures, but often no explanation for the choice of covariates was made.

Despite the research activity for the three health outcomes considered here, there has been little research that draws together the effects of social networks on disability, residential care use and mortality in a systematic fashion. To date, there has not been an examination of the effects of social networks on all three of the specified outcomes using data from the same cohort of older people and with a consistent period of follow-up across the outcomes.

An additional concern is the lack of a conceptual model underpinning much of the research work in this field. The underlying conceptual model of how social relationships might broadly affect health outcomes has been explicated in few publications concerning disability, use of residential care or death.

A final point to make here is that few authors have presented an assessment of the fit of the statistical models that they have used. Thus an integrated description of the effects of social networks on disability, use of residential care and mortality is currently lacking.

The starting point for this thesis was the identified need to clarify the effect of social networks across three distinct health outcomes in a single cohort of older people. Stated more completely, there was a clear need to investigate the effects of social networks on the health of older people after the effects of a wide range of covariates, covering demographic, health, psychological and lifestyle characteristics, were controlled for in the statistical analyses.

The ALSA provided an ideal dataset for these purposes. The breadth of the available data meant that it was possible to investigate the effects of specific social networks on disability, use of residential care and mortality, while taking into account the effects of many covariates. It was recognized at the outset that there were potentially significant associations between the covariates and the social networks themselves. The use of statistical techniques that incorporated these potential associations was necessary, and this point is returned to in Chapter 4.

To progress the investigation of the effects of social networks on health among the ALSA cohort, it was first necessary to develop a means for measuring social networks in this study. This was the first aim of this thesis. Aims that corresponded to the effects of social networks on each of the health outcomes of interest were also of natural interest. For each of the health outcomes, it was also important to determine if there were threshold effects for the total and specific social networks.

Thus stated explicitly, the aims of this thesis (see also Section 1.2) were to:

1. Develop a measurement model of social networks.
2. i) Examine the effects of total and specific social networks on disability and
   ii) Investigate the existence of threshold effects of total and specific social networks on disability.
3. i) Determine the effects of total and specific social networks on use of residential care and
   ii) Ascertain whether there are threshold effects of total and specific social networks on use of residential care.
4. i) Investigate the effects of total and specific social networks on survival and
   ii) Determine whether threshold effects of total and specific social networks on survival exist.

The following two chapters outline the study methods and statistical methods, respectively, before turning to a presentation of the results in Chapters 5 through 8. Chapter 9 presents general conclusions and recommendations for future research.

3 THE AUSTRALIAN LONGITUDINAL STUDY OF AGEING

3.1 STUDY PROCEDURE

ALSA is a large epidemiological study which aims to increase our understanding of how social, biomedical, behavioural, economic and environmental factors are associated with age-related changes in the health and social well-being of older persons (Andrews et al., 2002).

ALSA began in 1992, after a pilot feasibility study was carried out in 1989 and 1990 (Andrews et al., 1989). The primary sample for ALSA was randomly selected from the South Australian Electoral Roll, and was stratified by Local Government Area (LGA), gender, and five year age groups from 70–74 years through to 85 years and over. Older males aged 85 years or more were deliberately over-sampled to provide sufficient numbers of males for longitudinal follow-up. Persons identified through the Electoral Roll were defined as eligible for the study if they were resident in the Adelaide Statistical Division and were aged 70 years or more on 31 December 1992. Both community-dwelling and people living in residential care were eligible to take part in ALSA.

At the time work towards this thesis began, six waves of data had been collected from the ALSA participants. Data were collected for Wave 7 in 2003-2004 and Wave 8 took place in 2005 but are not considered further in the thesis. Waves 1 to 4 were annual interviews beginning in 1992, Wave 5 occurred in 1998, and Wave 6 was conducted in 2000-2001. Waves 1, 3, and 6 involved detailed personal interviews that covered demographic, medical, psychological, social and economic areas of participants' lives. As well, clinical assessments of participants were carried out in these waves. The clinical examination included anthropometric, psychological, physical performance,

balance, and gait measures. Both the interview and clinical assessment were carried out in the participant's usual place of residence. Waves 2, 4 and 5 each consisted of a brief telephone interview that concentrated mainly on health and lifestyle.

Ethical approval for the study was granted by the Flinders Medical Centre's Committee on Clinical Investigation, with a copy of the approval letter for Wave 1 shown in Appendix 2. The study has been re-approved at an annual ethical review by the committee (now named the Flinders Clinical Research Ethics Committee) each year since 1992. Each study participant gave written informed consent.

3.2 RESPONSE RATE

Of the original sample of 3263 persons drawn from the Electoral Roll, 2703 were eligible for inclusion in the study and 1477 (54.6%) agreed to participate. Table 3.1 provides further details concerning the response of the 3263 members of the sample list.

Table 3.1: Summary of responses at Wave 1 of ALSA

Response category	n	% of eligible
Eligible		
Interviewed	1477	54.6
Refused	1226	45.4
Total	**2703**	
Ineligible		
Deceased	210	
Translator not available	88	
Not contacted at address	189	
Out of geographical scope	37	
Other	36	
Total	**560**	

In addition to the primary sample drawn from the Electoral Roll, spouses and other members of the households of eligible persons were invited to take part in ALSA. The age requirement for spouses was 65 years or more (since wives are usually younger than their husbands), and for other household members was 70 years or more. This strategy recruited 597 spouses and 13 other household members. Thus a total of 2087 persons participated in Wave 1 of ALSA. However, because of the focus in this thesis on social networks and because of the anticipated large correlation of social networks of husbands and wives, the analyses presented in this thesis are based on the 1477 primary participants only.

Table 3.2 tracks the response of the 1477 primary ALSA participants over the six waves of the study. As expected, the retention of participants across the study diminished over time, from around 90 per cent of eligible survivors at Waves 2 and 3 to approximately 70 per cent of the eligible survivors continuing to take part in the sixth study wave.

Table 3.2 ALSA primary cohort: response over Waves 1 to 6

Wave		Interviewed	Lost to follow-up[1]	Refused[2]	Deceased
1	**n**	1477			
	%[3]	*100*			
2	**n**	1260	17	120	80
	%	*85.3*	*1.1*	*8.1*	*5.4*
	% survivors[4]	*90.2*	*1.2*	*8.6*	
3	**n**	1171	31	82	193
	%	*79.3*	*2.1*	*5.6*	*13.1*
	% survivors	*91.2*	*2.4*	*6.4*	
4	**n**	1036	35	115	291
	%	*70.1*	*2.3*	*7.8*	*19.7*
	% survivors	*87.4*	*3*	*9.7*	
5	**n**	773	67	131	506
	%	*52.3*	*4.5*	*8.9*	*34.3*
	% survivors	*79.6*	*6.9*	*13.5*	
6	**n**	500	39	176	762
	%	*33.9*	*2.6*	*11.9*	*51.6*
	% survivors	*69.9*	*5.5*	*24.6*	

1: moved out of Adelaide Statistical Division or not traceable
2: refused interview in that wave
3: % of Wave 1 participants (n=1477)
4: % of surviving participants

3.3 COVARIATES

A number of covariates were selected to encompass the physiological, psychological, social, economic and environmental domains experienced by the ALSA participants. Drawing from the studies cited in the literature review, covariates that covered demographic, health, psychological, sensory function and lifestyle aspects of participant's lives were considered. Self-reported data from the Wave 1 interview were used to define the covariates.

The specific covariates considered covered a number of domains, namely:

- *Demographic*: age, gender, marital status, household income, education, housing type;
- *Health*: self-rated health, number of morbid conditions (based on prevalent arthritis, cancer, cataracts, chronic bronchitis or emphysema, corns and bunions, diabetes, fractured hip, gout, heart attack, heart condition, hernia (including hiatus), hypertension, ingrown toenails, osteoporosis, stroke, and varicose veins), hearing ability, and visual ability;
- *Psychological*: depressive symptomatology, cognitive function; and
- *Lifestyle*: alcohol use, tobacco consumption, exercise level.

In addition, disability was included as a covariate in the analyses of use of residential care and mortality.

Household income was coded as ≤$12,000 per annum or >$12,000 per annum. Education was coded as less than or equal to 14 years of age or more than 14 years of age when the participant left full-time education, as used previously in this cohort by Finucane et al. (1997). Housing type was operationalized on the basis of whether participants were living in the community or an aged care facility at baseline. Community-dwelling was further broken down according to whether individuals were home owners, rented their residence, or had another type of community-dwelling living arrangement (such as living in their children's home).

Self-rated health was classified as excellent/very good, good, and fair/poor. The number of chronic conditions was derived from self-reported information on whether each participant had ever suffered from arthritis (rheumatoid arthritis or osteoarthritis), cancer (excluding non-melanocytic skin cancer), cataracts, chronic bronchitis or

emphysema, corns and bunions, diabetes, fractured hip, gout, heart condition, hernia (including hiatus hernia), hypertension, ingrown toenails, myocardial infarction, osteoporosis, stroke or varicose veins. Hearing difficulty and difficulty with corrected vision were based on self-report and simple dichotomies of yes or no were used in this thesis (Sanchez, 1998). In the analyses of residential care use and mortality, mobility disability (see definition in Section 3.4) was also included as a covariate.

Symptoms of depression were assessed using the 20-item Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977), with scores of 17 or more out of a possible 60 suggesting depressive symptomatology. Cognitive function was assessed using a subset of items from the Mini-Mental State Examination (Folstein et al., 1975). Possible scores ranged from zero to 21, with scores of 16 or below indicative of cognitive impairment (Luszcz et al., 1997).

A series of questions about smoking in the Wave 1 interview led to participants being classified as current, former or never smokers. Alcohol consumption was assessed by the 10-item AUDIT scale (Barbor et al., 1992), and participants were classified as having a hazardous drinking problem if their score was at least eight out of a maximum possible of 40. Questions in the Wave 1 interview that related to exercise undertaken in the previous fortnight enabled participants to be classified as exercisers or sedentary (Finucane et al., 1997).

Summary statistics for these covariates at Wave 1 are presented in Table 3.3. The average age at selection was 79.8 years (SD=6.9), and close to two-thirds of the sample were male. More than half of the participants had left school before the age of 15 years, and approximately half of the sample was married or partnered. Participants most

commonly had one morbid condition, and 15 per cent of participants showed some signs of cognitive deficits. More than half of the participants were former or current smokers, and almost half of the participants were sedentary.

Table 3.3: Summary of covariates at Wave 1

Variable	Classification	n	(%)
Age group	70-74	379	(25.7%)
	75-79	352	(23.8%)
	80-84	341	(23.1%)
	85+	405	(27.4%)
Gender	Male	928	(62.8%)
	Female	549	(32.2%)
Education	Left school >14 yrs	633	(42.9%)
	Left school ≤14 yrs	830	(56.2%)
	Missing	14	(0.9%)
Marital status	Married/de facto	771	(52.2%)
	Widowed	586	(40.0%)
	Single	120	(8.1%)
Household income	>$AUD12,000	779	(52.7%)
	≤$AUD12,000	590	(39.9%)
	Missing	108	(7.3%)
Home ownership	Owns home	1038	(71.0%)
	Renting	242	(16.4%)
	Other	50	(3.4%)
	In residential care	137	(9.3%)
Number of chronic conditions	0	264	(17.9%)
	1	494	(33.4%)
	2	421	(28.5%)
	3+	298	(20.2%)
Self-rated health	Excellent/very good	563	(38.1%)
	Good	440	(29.8%)
	Fair/poor	469	(31.8%)
	Missing	5	(0.3%)
Hearing difficulty	No	726	(49.2%)
	Yes	746	(50.5%)
	Missing	5	(0.3%)

(continued)

Table 3.3 (continued): Summary of covariates at Wave 1

Variable	Classification	n	(%)
Difficulty with (corrected) vision (Sanchez, 1997)	No	1035	(70.1%)
	Yes	375	(25.4%)
	Missing	67	(4.5%)
Depressive symptoms (CES-D; Radloff, 1977)	<17/60	1181	(80.0%)
	≥17/60	219	(14.8%)
	Missing	77	(5.2%)
Cognitive function (Folstein et al., 1975, Luszcz et al., 1997)	>16/21	1221	(82.7%)
	≤16/21	219	(14.8%)
	Missing	37	(2.5%)
Alcohol consumption (AUDIT) (Barbor et al., 1992)	<8/10	1401	(94.9%)
	≥8/10	65	(4.4%)
	Missing	11	(0.7%)
Exercise status (Finucane et al., 1997)	Exerciser	794	(53.8%)
	Sedentary	663	(44.9%)
	Missing	20	(1.4%)
Smoking status	Never	661	(44.8%)
	Former	667	(45.2%)
	Current	123	(8.3%)
	Missing	16	(1.1%)

Comparisons of the ALSA sample to the older Australian population are limited as many reports by government agencies combine results for all persons aged 65 years or more. In addition, the timing of some of the relevant surveys was discordant with the commencement of ALSA in 1992. For example, the 1995 National Health Survey (Australian Bureau of Statistics, 1995) provides some basis for comparison of the ALSA sample to the older Australian population in terms of morbidity and lifestyle, as do some of the summaries reported in "Older Australia at a Glance" (Australian Institute of Health and Welfare, 1997). However, older age was defined as 65 years or more in the National Health Survey, while various lower ages ranging from 55 to 70

years were used to define older age in "Older Australia at a Glance". Table 3.4 presents a comparison of some key indicators of the ALSA sample versus national studies that were conducted at a similar time. This shows that the ALSA sample had a higher proportion of men (due to deliberate oversampling), were similar in terms of self-rated health and smoking profile but possibly more sedentary than the older Australian population in the early 1990s.

Table 3.4: Comparison of ALSA sample to national studies conducted at a similar time

Variable	ALSA	National Study
Gender *% male*[1]	62.8	40.5
Home owners *% yes*[2]	71.0	76.8
Self-rated health *% excellent/very good*[3]	38.1	32.3
Self-rated health *% good*	29.8	26.7
Self-rated health *% fair/poor*	31.8	40.9
Smoking status *% current smokers*[3]	8.4	50.5
Smoking status *% former smokers*[3]	45.2	38.3
Smoking status *% never smokers*[3]	46.3	11.2
Exercise status % sedentary	44.9	25.7

1: National study from Estimated Resident Population, 1992 (Australian Bureau of Statistics, 1993)
2: National study from Older Australia at a Glance (Australian Institute of Health and Welfare, 1995) population aged 60 years or more
3: National study from 1995 National Health Survey (Australian Institute of Health and Welfare, 1995) population aged 65 years or more

3.4 SOCIAL NETWORKS

Chapter 5 of this thesis describes in detail the derivation of the social network variables. Thus the measures of social networks that were derived and used in this thesis are only outlined briefly in the present section.

Four specific social network types were considered, and these corresponded to networks with children, relatives, friends and confidants, following from work conducted by Glass et al. (1997). A measurement model was fit using confirmatory factor analysis (CFA), and a process of model refinement was then used to derive a final measurement model.

A variable reflecting networks with children was derived from four observed variables that measured the number of children, the proximity of where children lived relative to their parents, the frequency of personal contact, and the frequency of telephone contact. Social networks with (other) relatives was measured from the number of other relatives and the frequency of telephone and personal contact with relatives. Similarly, a friend network variable was based on the number of close friends and the frequency of telephone and personal contact with friends. A confidant network variable was also calculated. In the ALSA, there were fewer questions concerning confidants than there were questions for the other types of social networks. In particular, there was no question that reflected frequency of contact with a confidant. Therefore the confidant network was calculated from three variables that measured the existence of a primary confidant, a second confidant, and whether a participant's spouse was their confidant. A total social network variable was also calculated from the four specific social network variables.

How changes in social networks over time affect health is of considerable interest. However, data that would allow such changes to be calculated were not available in ALSA. A much briefer set of questions concerning social networks was used in subsequent waves of ALSA, precluding the calculation of changes in the specific and total social network variables. Thus only Wave 1 data were used in the derivation of the social network variables, and changes in social networks were not considered in this thesis.

3.5 DEFINITION OF DISABILITY

Two complementary measures of self-reported disability were considered, namely mobility disability (Rosow and Breslau, 1966) and Nagi disability (Nagi, 1976). Participants were defined as having no mobility disability if they reported they were able to walk up and down a flight of stairs and walk half a mile without help. If either or both of these activities could not be completed, they were classified as having a mobility disability. Other studies have used a similar dichotomous measure of mobility (e.g. Mendes de Leon et al., 1999; 2001; 2003).

The second disability measure was derived from questions developed by Nagi (1976). Participants reported their level of difficulty in performing five tasks (pushing or pulling large objects, stooping or crouching or kneeling, lifting or carrying 10 pounds, reaching or extending arms, and writing or handling small objects). There were five response categories for each task, namely 'no difficulty', 'a little difficulty', 'some difficulty', 'a lot of difficulty' or 'just unable to do it'. Participants were defined as having no Nagi disability if they reported no more than a little difficulty for all five Nagi questions. Participants who reported at least some difficulty for at least one of the five questions

were classified as having a Nagi disability. The same definition for Nagi disability was used by Beckett et al. (1996).

For Waves 1 through 6, participants could have missing values on one or more of the component disability questions. Participants with missing values for one or both of the mobility questions at any wave were coded as missing for that wave, unless one of the non-missing items indicated mobility disability. In this case, participants were coded as having a mobility disability (Mendes de Leon et al., 1999). Similarly, the response for participants with missing values for at least one of the five Nagi tasks within a wave was coded as missing for that wave, unless a non-missing response to one of the tasks indicated disability. In this case, participants were classified as having a Nagi disability. This was done to minimize the number of missing observations in the disability outcome variables.

3.6 DEFINITION OF USE OF RESIDENTIAL CARE

Participant's place of residence was recorded by the interviewer at each wave. At each interview, participants were classified as living in the community, low-level residential care, or high-level residential care (nursing home). The starting date of a stay in residential care was not available.

As described in Section 2.5.3, low-level residential care (also referred to colloquially as 'hostels') provides help and housing to older people who do not need continual, high level access to nursing care but have physical, medical, psychological or social care needs that cannot be met through living in the community. High-level residential care is synonymous with nursing home care in the Australian setting.

For the purposes of this thesis, community-dwelling was defined as living in a house, home unit, granny flat, bedsitter's unit, semi-detached home or retirement village. At each wave, participants were classified as living in low-level residential care if they were interviewed in a hostel or other form of residential care that provided assistance with basic ADLs but did not provide around-the-clock nursing care. Participants were defined as being in high-level residential care if they were resident in a nursing home or private rest home at the time of interview. For Waves 2 through 6, participants were classified as missing if they refused an interview or were untraceable.

3.7 DEFINITION OF MORTALITY

Survival status was ascertained by searches of the database of official death certificates conducted by the Epidemiology Branch of the Department of Health in South Australia, and deaths were confirmed by the South Australian Births, Deaths and Marriages Bureau. Full name, date of birth, gender and last known address of ALSA participants were used in the data linkage with the database of death certificates. If no direct match was made, the Electoral Roll was checked for errors in birth dates, changes or errors in recorded name, and changes or errors in recorded address. The few participants who died interstate or overseas could not be identified through this method, as the South Australian deaths index only included deaths that occurred in South Australia. Informants nominated by ALSA participants at Wave 1 were contacted if participants could not be located at Waves 2 through 6. The date of death supplied by informants was used if the study team were notified by an informant that a participant had died outside of South Australia. These methods of death ascertainment for ALSA participants have been validated previously (Anstey et al., 2001).

The response variable considered in the analyses of the effects of social networks on mortality was the number of days to death from Wave 1 interview for decedents and 3653 days for participants who survived 10 years after their Wave 1 interview.

3.8 SUMMARY

The wealth of data available from the ALSA has not been fully described in this chapter; indeed, this task is beyond the scope of this thesis. Suffice it to say, ALSA presented a unique opportunity to examine the effects of social networks on three health outcomes – disability, residential care use, and mortality – in an older Australian cohort. The next chapter describes the statistical methods that were used in this thesis, before the study results are presented in Chapters 5 through 8.

4 STATISTICAL METHODS

4.1 INTRODUCTION

The aims of this thesis are to derive measures of social networks and then investigate their effects on disability, use of residential care and survival, while taking into account the effects of a wide range of covariates. To address these aims, various statistical techniques have been used and are reviewed in this chapter.

The validation of the social networks measures used CFA. Broadly speaking, CFA tests a proposed measurement model that describes the relationships between observed and latent variables. In the present case, the latent variables are the social networks. A general review of CFA and the approach used in the CFA in this thesis is presented in Section 4.2.

The effects of the derived social network measures on the health outcomes were then considered. Transition models, as described by Diggle et al. (2002), were used in the analyses for disability and use of residential care. The formulation of transition models and the framework within which the estimates of the effects of social networks were derived are presented in Section 4.3.

Participants' survival was also hypothesised to be a function of social networks and other covariates. Cox proportional hazards models have commonly been used in the analysis of survival data, and these models are reviewed in Section 4.4. Contemporary methods of the assessment of model fit in survival analysis are also described in Section 4.4.

As noted in Section 2.6, social networks themselves are likely to be related to an individual's characteristics such as socioeconomic status, gender, health, and lifestyle. For example, it is possible that ALSA participants with better social networks also had better education. It was therefore necessary to adjust for such covariates to avoid bias in the parameter estimates of the effects of social networks. In this thesis, propensity score adjustment was used to adjust for covariates in the context of logistic regressions and Cox proportional hazards models, and the method is reviewed in Section 4.5.

Finally, the method through which the existence of threshold effects of social networks was formally tested is explained in Section 4.6.

4.2 CONFIRMATORY FACTOR ANALYSIS

CFA is a particular form of covariance structure modelling that is primarily used to evaluate measurement models that are hypothesised to describe the relationship between observed variables and unobserved, or latent, variables (Kline, 1998). In the sense that CFA is concerned with the analysis of covariance matrices, it is related to exploratory factor analysis and path analysis. Each of these techniques can be viewed as special cases of structural equation modelling, a technique that has gained much popularity over the past few decades, particularly in education, economics, and the social sciences (Loehlin, 1998).

Despite the widespread use of structural equation models in these disciplines, their use has not been embraced by the statistical community. Indeed, the application of structural equation models has been criticized by some renowned statisticians for many years (e.g. Freedman, 1987; Freedman, 2004; Holland, 1986). Two main problems exist in much of the applied literature that uses structural equation models: i) in general,

little attention has been paid to underlying assumptions and ii) results from the fit of a structural equation model are frequently cited to have established causality (e.g. Kenny, 1979). The assessment of fit of structural equation models is also contentious, and many of the summary indices that have been proposed to measure goodness of fit are problematic.

The notation and specification of a measurement model using CFA is introduced in Section 4.2.1. An overview and discussion of some of the fit indices and other methods of assessment of fit are then presented in Section 4.2.2 and subsections thereof. The general criticisms of structural equation modelling and their relevance to this work are discussed in Section 4.2.3.

4.2.1 Specification of a measurement model

CFA compares the variance-covariance matrix from a hypothesised model involving latent variables to a sample variance-covariance matrix (Loehlin, 1998; Mueller, 1996). The hypothesised model in CFA is a measurement model which specifies how the latent variables are measured as a function of the observed variables (Jöreskog and Sörbom, 1981). Thus CFA is a technique for testing a measurement model that describes the relationships between the observed variables and the latent variables. It is based on the premise that the observed variables are 'imperfect indicators' of the latent variables (Mueller, 1996 p. 64). A CFA model specifies each observed variable to be linearly dependent on one or more latent variables and a measurement error term.

The general form of a CFA model is $X_i = \sum_{j=1}^{q} \lambda_{ij} \xi_j + \delta_i$, where the observed variables are denoted by X_i for i=1, …, p, the latent variables are denoted by ξ_j for j=1, …, q, the

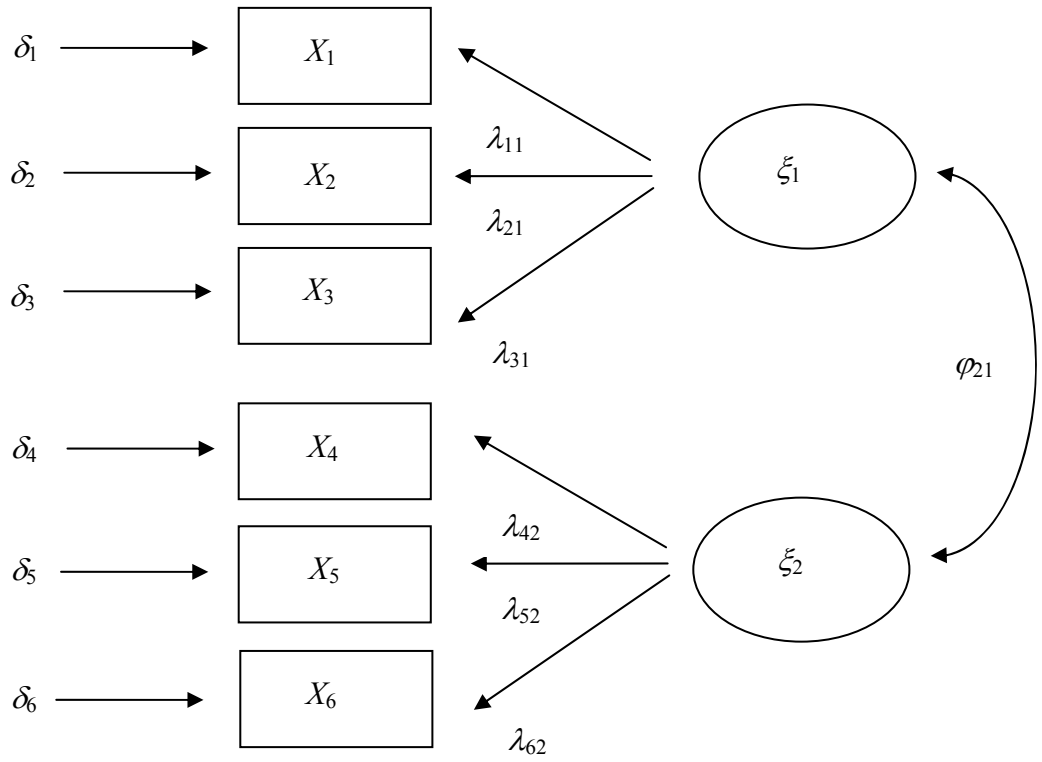
structural coefficients for the linear relationship between each X_i and ξ_j are represented by λ_{ij} and the measurement errors associated with X_i are denoted by δ_i. The ξ_j represent the theoretical 'constructs' that are under examination, while the X_i terms are the manifest variables that are expressed as a function of the latent variables. The λ_{ij} are commonly referred to as factor loadings, and can be interpreted as unstandardized regression coefficients that estimate the direct effects of ξ_j on X_i. Standardized estimates of λ_{ij}, denoted here as λ_{ij}^{S} such that $|\lambda_{ij}^{S}| \leq 1$, can be derived by standardizing the X_i, through subtracting the mean and dividing by the standard deviation, and conducting the factor analysis on the standardized variables. The λ_{ij}^{S} are useful to compare the relative importance of observed variables as indicators of their associated latent variables. Thus a standardized coefficient has the same interpretation as a standardized regression coefficient. The X_i are assumed to be measured as deviations from their means – that is, $E(X_i) = 0$ – and are usually assumed to be drawn from a multivariate Normal distribution. If an X_i is specified to load on a single latent variable, then λ_{ij}^{S} is a correlation, and the square of this coefficient is the proportion of variance of X_i explained by ξ_j (Kline, 1998) – i.e. a coefficient of determination.

A CFA model can also be expressed in matrix form as $\mathbf{X} = \Lambda_{\mathbf{X}}\xi + \delta$. In addition to the $\Lambda_{\mathbf{X}}$ matrix, variance-covariance matrices pertaining to the latent and observed variables are needed to specify a particular CFA model (Mueller, 1996). Denote the variance-covariance matrix of the latent variables ξ by $\text{Var}(\xi) = \Phi$ and the variance-covariance matrix of δ by $\text{Var}(\delta) = \Psi$. In exploratory factor analysis, Ψ is a diagonal matrix. In contrast, associations among measurement error terms may be specified in CFA – that is, the off-diagonal terms in Ψ may be non-zero.

Finally, let the population variance-covariance matrix of the observed variables be Var($\mathbf{X}$) = Σ. Under the assumption ξ and δ are uncorrelated, Σ can be written as $\Lambda_{\mathbf{X}}\Phi\Lambda_{\mathbf{X}}' + \Psi$ (e.g. Kline, 1998; Mueller, 1996; Steiger, 2001). In other words, when a particular model is hypothesised, Σ can be expressed as a function of the model-implied structural coefficient matrix and the variance-covariance matrices for the latent variables and measurement errors. $\Sigma(\theta)$ is traditionally used to denote this model-implied variance-covariance matrix, where θ is a vector that contains the parameters in $\Lambda_{\mathbf{X}}$, Φ and Ψ. In this light, a CFA model can be seen to postulate a certain parametric form $\Sigma = \Sigma(\theta)$.

Figure 4.1 presents a path diagram that shows this notation on a hypothesised measurement model with six observed variables and two latent variables. Path diagrams typically use four geometric symbols to convey particular meanings, and this convention is followed here. An ellipse or circle is used to represent latent variables, while rectangles or squares represent observed variables. A single-headed arrow represents the effect of one variable on another, and a double-headed arrow represents the covariance between a pair of variables (e.g. Byrne, 2005).

Figure 4.1: A notated confirmatory factor analysis model



In equation form, this model is represented by:

$X_1 = \lambda_{11}\xi_1 + \delta_1$

$X_2 = \lambda_{21}\xi_1 + \delta_2$

$X_3 = \lambda_{31}\xi_1 + \delta_3$

$X_4 = \lambda_{42}\xi_2 + \delta_4$

$X_5 = \lambda_{52}\xi_2 + \delta_5$ and

$X_6 = \lambda_{62}\xi_2 + \delta_6$.

For the example in Figure 4.1, Λ_X is given by $\begin{bmatrix} \lambda_{11} & 0 \\ \lambda_{21} & 0 \\ \lambda_{31} & 0 \\ 0 & \lambda_{42} \\ 0 & \lambda_{52} \\ 0 & \lambda_{62} \end{bmatrix}$ and the variance-covariance matrix of the latent variables has the form $\Phi = \begin{bmatrix} \varphi_{11} & \\ \varphi_{21} & \varphi_{22} \end{bmatrix}$. In this case Ψ is the 6 x 6 diagonal matrix

$$\begin{bmatrix} \psi_{11} & & & & & \\ 0 & \psi_{22} & & & & \\ 0 & 0 & \psi_{33} & & & \\ 0 & 0 & 0 & \psi_{44} & & \\ 0 & 0 & 0 & 0 & \psi_{55} & \\ 0 & 0 & 0 & 0 & 0 & \psi_{66} \end{bmatrix}.$$

If a correlation between the error terms for, say, X_3 and X_6 was to be included in the model, then ψ_{63} would be non-zero.

4.2.2 Fitting a measurement model

In practice, the elements of Σ are unknown, and can be estimated by the sample variance-covariance matrix **S**. Assume that a suitable discrepancy function between **S** and Σ(θ) is given by $F[\mathbf{S}, \Sigma(\theta)]$. Then parameter estimates of the model-implied matrices, denoted by $\hat{\theta}$, are derived such that $\hat{\theta} = \arg\min_{\theta} F[\mathbf{S}, \Sigma(\theta)]$.

When multivariate Normality is assumed, various discrepancy functions can be used, corresponding to maximum likelihood, generalized least squares, or unweighted least squares. Maximum likelihood is the most commonly used estimation method, and the associated discrepancy function is $F_{ML}[\mathbf{S}, \Sigma(\theta)] = \log|\Sigma(\theta)| - \log|\mathbf{S}| + \text{tr}[\mathbf{S}\Sigma^{-1}(\theta)] - p$. If the assumption of multivariate Normality is not tenable, then alternative estimation methods such as the asymptotic distribution free method (Browne, 1984), categorical variable methodology (Muthén, 1984) or elliptical distribution methods (Bollen, 1989) have been proposed. However, these methods have not been rigorously explored and can be computationally demanding (West et al., 1995).

Several authors have investigated the effects of non-Normality on the performance of various estimation methods, including maximum likelihood and the asymptotic distribution free method (Chou et al., 1991; Curran et al., 1996). The point at which

multivariate non-Normality becomes a problem remains unclear, although simulations carried out by Curran et al. (1996) with a sample size equal to 1000 suggested univariate skewness greater than two and kurtosis greater than seven were problematic. The results of Curran et al. (1996) suggested that maximum likelihood estimates of parameters had little bias even with severely non-Normal data, but that some caution should be used in the interpretation of model fit.

4.2.3 Assessment of fit

A variety of methods have been proposed to assess the fit of $\Sigma(\hat{\theta})$ to **S** (Marsh et al., 1988), but there is contention about the different methods. None is wholly satisfactory, and the substantive results from the different methods of fit assessment can often disagree. Broadly, the methods fall into five main classes, and these are: i) a test of H_0: $\Sigma = \Sigma(\theta)$, ii) fit indices that compare the amount of variance-covariance information that can be accounted for by the hypothesised model compared with some baseline model, iii) the Root Mean Square Error of Approximation, iv) modification indices, cross-validation approaches and standardized residuals and v) measures of reliability. Each of these methods is now described and commented on.

4.2.3.1 Test of $\mathbf{H_0}$**:** $\Sigma = \Sigma(\theta)$

Under H_0: $\Sigma = \Sigma(\theta)$ and under the assumption of multivariate Normality, $(n-1)F_{ML}[\mathbf{S}, \Sigma(\hat{\theta})]$ is asymptotically distributed as $\chi^2_{p(p+1)/2 - c}$ where n is the sample size, $p(p+1)/2$ is the number of unique elements in Σ and c is the number of parameters to be estimated. Therefore if H_0: $\Sigma = \Sigma(\theta)$ holds in the population and the observed variables are drawn from a multivariate Normal distribution, then $(n-1)F_{ML}[\mathbf{S}, \Sigma(\hat{\theta})]$ can

be compared to a χ^2 statistic on $p(p+1)/2 - c$ degrees of freedom in an assessment of model fit.

However, the χ^2 test is problematic on several levels, and these issues have been raised by a number of authors. The asymptotic null distribution of $(n-1)F_{ML}[\mathbf{S}, \Sigma(\hat{\theta})]$ requires that the observed variables are multivariate Normal and the sample size is 'large enough' (Mueller, 1996 p. 83), although it is unclear what 'large enough' means. Kline (1998, p. 12) suggests that sample sizes in excess of 200 observations are large, but does not relate this size of sample to the number of degrees of freedom or the number of observed and latent variables under consideration. In a large sample, values of the χ^2 test may be large (and thus statistically significant) even when the discrepancy between $\mathbf{S}$ and $\Sigma(\hat{\theta})$ is quite small in 'meaningful' terms (e.g. Mueller, 1996 p. 83; Tanaka, 1993). This argument is dutifully given in almost every book concerning CFA and structural equation models. Conversely, in a small sample the test statistic may also be small and not statistically significant due to a lack of statistical power (Tanaka, 1993). The latter point is made far less frequently in the structural equation modelling literature. The same comments can of course be made with equal validity about any hypothesis test and it is not clear why hypothesis testing is often dismissed from CFA but not elsewhere.

Perhaps more relevant to an interpretation of the magnitude and significance of the χ^2 statistic is the actual plausibility of the null hypothesis. While Box's 'all models are wrong' dictum applies equally well in the structural equation modelling paradigm, it might be better expressed as 'all models are simplifications of an underlying process' (Tanaka, 1993). In this light, it may be argued that a formal test H_0: $\Sigma = \Sigma(\theta)$ is of

limited interest and that the objective of fitting a model should be to determine whether the model provides a useful simplification.

An alternative use of the χ^2 statistic was suggested by Wheaton et al. (1977; cited in Arbuckle, 2005). These authors proposed that a small ratio of the model χ^2 to the model degrees of freedom (χ^2/df) was indicative of good model fit. Many others have reiterated this idea (e.g. Jöreskog and Sörbom, 1993). For large samples, Marsh et al. (1988) proposed that a ratio of less than five is suggestive of good model fit; others have stated that 'a frequent suggestion is that this ratio be less than three' (Kline, 1998, p. 128) or should be less than two to indicate an 'adequate' fit (Byrne, 2000).

However, since the degrees of freedom for the χ^2 statistic is given by $p(p+1)/2-c$ and is therefore independent of n, the ratio does not overcome the argument that large sample sizes lead to a large χ^2 statistic. Furthermore, there appears to be no justification for any of the proposed acceptable upper values for the ratio. If the null hypothesis is true and the assumption of multivariate Normality holds then the expected ratio is close to one, since the χ^2 is asymptotically distributed with a mean equal to its degrees of freedom. When the null hypothesis is not true or the assumption of multivariate Normality is violated, it is not clear how this ratio would behave. For these reasons, the ratio is generally regarded as a poor measure of model fit and its use has been discouraged by some prominent authors in the field of structural equation modelling (e.g. Steiger, 2000). Despite this, it is still reported in virtually every applied article concerning structural equation models or CFA.

In summary, the χ^2 statistic and the χ^2/df ratio are frequently used and appear in the majority of applications of CFA. However, their utility as measures of model fit is poor. The test of H_0: $\Sigma = \Sigma(\theta)$ may be of limited interest, although the change in the χ^2 statistic can provide a useful comparison of two nested models. The χ^2/df ratio does not overcome the oft-cited rationale that the size of the χ^2 statistic depends on the sample size, and so as a test of good or bad model fit the χ^2/df ratio is not useful. Thus for the purpose of model fit assessment in this thesis, other methods were explored, some of which are now described.

4.2.3.2 Fit indices

As an alternative to the χ^2 statistic and the χ^2/df ratio, a range of fit indices have been proposed that de-emphasise hypothesis testing. These fit indices attempt to quantify something analogous to an R^2 statistic from a linear model (Hu and Bentler, 1995; Tanaka, 1993 p. 19), in that the range of possible values of the fit indices are bounded between zero and one with larger values indicative of better fit. More than thirty fit indices were compared by Marsh et al. (1988), and additional indices been developed since then (as discussed in Hu and Bentler, 1999).

Fit indices are classified as either absolute fit indices or incremental fit indices (Hu and Bentler, 1999). An absolute fit index is used to assess how well a particular model reproduces the sample variance-covariance matrix, while an incremental fit index compares the fit obtained from a hypothesised model to a nested, more restricted baseline model (Hu and Bentler, 1999). A few of the more commonly used fit indices are described here, with the intent of this section being to highlight in broad terms the differences between the indices.

4.2.3.2.1 Absolute fit indices

The most commonly reported absolute fit indices are the Goodness of Fit Index (GFI; Jöreskog and Sörbom, 1981) and the adjusted GFI (AGFI). The GFI is given by $1 - (F[\mathbf{S}, \Sigma(\hat{\theta})] / F[\mathbf{S}, \Sigma(0)])$ where $F[\mathbf{S}, \Sigma(0)]$ corresponds to the discrepancy function evaluated when all parameters in $\Lambda_{\mathbf{X}}$, Φ and $\mathbf{\Psi}$ are fixed and thus no parameters require estimation. The GFI is interpreted as how much better the hypothesised model fits compared to the null model (Jöreskog and Sörbom, 1993). If $F[\mathbf{S}, \Sigma(\hat{\theta})] = F[\mathbf{S}, \Sigma(0)]$, then the GFI is zero, since the fitted model is the null model. On the other hand, if there are no constraints placed on the estimation of $\Sigma(\theta)$, then trivially $\mathbf{S} = \Sigma(\hat{\theta})$ so that $F[\mathbf{S}, \Sigma(\hat{\theta})] = 0$, and thus the GFI is one.

An adjusted GFI (AGFI) that 'shrinks' the GFI for the model degrees of freedom was proposed by Jöreskog and Sörbom (1981). The AGFI can be thought of in relation to the GFI in the same way that an adjusted R^2 is related to an R^2 value – that is, the AGFI takes into account the model complexity and 'down-weights' the value of GFI (Mueller, 1996 p. 85). It is estimated as $1 - \frac{p(p+1)/2}{p(p+1)/2 - c}$ [1-GFI]. However, like an adjusted R^2, the AGFI can lie outside of the range zero to one.

Some authors have contended that in general, values of the GFI/AGFI greater than 0.90 indicate adequate fit (e.g. Bentler and Bonnett, 1980), although there is no obvious reason for this value as opposed to say 0.85 or 0.95. The latter value was suggested by Hu and Bentler (1999), and was based on the results of a simulation study. In practical terms, the values of the GFI and AGFI are often close to one, giving little insight into the fit of the model. For example, Glass et al. (1997) reported for their initial model

GFI=0.983 and AGFI=0.975, yet concluded these indices represented a 'less than favourable fit'. For the final model of Glass et al. (1997), the GFI and AGFI were 0.996 and 0.994 respectively. These latter indices were interpreted to represent 'an improved and suitable fit to the data'. The example demonstrates that the GFI and AGFI values are large in both models and that based on these indices alone, there is little to discriminate between the two models.

The GFI/AGFI uses the observed data as its comparison point and not a relative comparison to another model (Tanaka, 1993 p. 26). The absolute fit indices therefore do not lie within the usual model testing framework and provide no meaningful insight into the fit of alternative models. Thus this method of fit assessment appears poor and was not pursued any further in this thesis.

4.2.3.2.2 Incremental fit indices

As an alternative to absolute fit indices, incremental fit indices that compare nested models have been proposed. One widely used incremental fit index is the Comparative Fit Index (CFI; Bentler, 1990). The CFI compares the fit of the hypothesised model to a sub-model (usually the independence model). The independence model is a restricted model in which no latent variables are hypothesised to underlie the observed variables (i.e. $\Lambda_{\mathbf{X}} = \mathbf{I}$), the observed variables are assumed to be measured without error (i.e. $\boldsymbol{\Psi} = \mathbf{0}$), and the $\mathbf{X}$ are specified to be independent, so that Φ is a diagonal matrix.

Let F_i be the value of the discrepancy function $F[\mathbf{S}, \Sigma(\hat{\theta})]$ for the independence model with associated residual degrees of freedom df_i. Furthermore, let F_h and df_h be the analogous values for the hypothesised model. Then the CFI is given by

$$CFI = 1 - \frac{\max(0, (n\text{-}1)F_h - df_h)}{\max(0, (n\text{-}1)F_h - df_h, (n\text{-}1)F_i - df_i)}$$

and is bounded by zero and one. The CFI is interpreted as the relative improvement in fit of the hypothesised model compared to the independence model. If the independence model is a good fit, then only a small improvement in fit may be achieved through fitting the hypothesised model. In this case, the quantity $\frac{\max(0, (n\text{-}1)F_h - df_h)}{\max(0, (n\text{-}1)F_h - df_h, (n\text{-}1)F_i - df_i)}$ will be close to 1, and thus the CFI $\rightarrow$0. Thus a small CFI does not necessarily reflect a poorly fitting model.

As is the case for the GFI and AGFI, rules of thumb concerning cut-off criteria have been proposed for the CFI. Values exceeding 0.90 (e.g. Bentler and Bonnett, 1980; Kline, 1998, p. 131) or 0.95 (Hu and Bentler, 1999) have been suggested to indicate a well-fitting hypothesised model in comparison to the independence model. However, these cut-off criteria are based on the results of limited simulation studies and their rationale is rather inadequate, given that the hypothesised model may be close to the independence model. Nonetheless, the CFI is preferable to the GFI/AGFI since the fit of alternative models are compared within a model testing framework.

4.2.3.3 Root Mean Square Error of Approximation

A different approach to evaluating the fit of a model was proposed by Steiger and Lind (1980, cited in Steiger, 1990). The error of approximation refers to the lack of fit of the model to the population variance-covariance matrix Σ - that is, the true lack of fit of the model in the population. An estimate of the error of approximation ω is given by

$$\hat{\omega} = \max(0, F[\mathbf{S}, \Sigma(\hat{\theta})] - \frac{df_h}{n})$$

(Browne and Cudeck, 1993 p. 143).

As parameters are added to the model, $F[\mathbf{S}, \Sigma(\hat{\theta})]$ will generally decrease while $\frac{df_h}{n}$ will generally increase, and thus using $\hat{\omega}$ as a measure of lack of model fit could lead to a complex model that is far from parsimonious (Browne and Cudeck, 1993 p. 144). To overcome this, Steiger and Lind (1980, cited in Steiger, 1990) proposed the root mean square error of approximation (RMSEA) ε, which is estimated as $\hat{\varepsilon} = \sqrt{\hat{\omega} / df_h}$.

$\hat{\varepsilon}$ provides a measure of the lack of fit of the model per degree of freedom. Steiger and Lind (1980) advocated that a 90% confidence interval is reported along with $\hat{\varepsilon}$, although the reason for this width of confidence interval was not clear from their original work.

Browne and Cudeck (1993) demonstrated the relationship between the RMSEA and a test of H_0: $\Sigma = \Sigma(\theta)$, showing that the latter can alternatively be expressed as H_0: $\varepsilon = 0$. These authors suggested replacing the 'point' null hypothesis with an interval null hypothesis of *close* fit, namely H_0: $\varepsilon \leq 0.05$. The rationale for the value of 0.05 was not given and appears to have been based on a simulation study conducted by these authors, although this was not explicitly stated in their 1993 article. The null hypothesis of close fit was proposed as more realistic than the null hypothesis of exact fit. Browne and Cudeck suggested that from a practical stance, $\hat{\varepsilon}$ values below 0.08 indicate a model that does not demonstrate substantial lack of fit, although the reason for 0.08 was again not provided by Browne and Cudeck. Thus $\hat{\varepsilon}$ can be useful in an assessment of model fit, especially in a test of a null hypothesis of close fit. How the suggested cut-off values for ε were derived is not clear, and thus such cut-offs should be used judiciously. A comparison of $\hat{\varepsilon}$ between alternative models may be more useful as a means of

comparing the fit of different models, with smaller values desirable and an indication of better fit.

4.2.3.4 Model modification

The methods of assessing the fit of a model described so far all assess the 'goodness' or 'badness' of fit of the overall model. An alternative way of considering the fit of a model is through the use of Lagrange multipliers, to determine particular areas for which significant improvements in model fit may be possible. Univariate Lagrange multipliers are usually referred to as modification indices in structural equation modelling (Jöreskog and Sörbom, 1984). For each constrained parameter in the hypothesised model, the associated modification index is an estimate of the change in $(n-1)F[\mathbf{S}, \Sigma(\hat{\theta})]$ if the parameter was instead estimated without constraint. Each modification index follows a χ_1^2 distribution asymptotically. Some authors have suggested the modification index needs to be interpreted in conjunction with the size of the change in the associated parameter estimate (e.g. Kaplan, 1990; Saris et al., 1987), so that parameter estimates that are close to zero with a statistically significant modification index are not automatically included in a re-specified model.

The same criticisms that are made of forwards and stepwise multiple regression procedures can be made of the use of modification indices if they are used to inform model modification without consideration of the substantive meaning of the proposed change to the model. A forwards or stepwise selection strategy in a conventional linear regression analysis can build on an initial model in such a way that certain combinations of variables may never be included in the model, and so it is possible that the best-fitting model is never actually fit to the data. Therefore the final model arrived at through such a model selection strategy may not be the best-fitting model. Similarly, in

CFA an initial measurement model may have been badly mis-specified and be a poor representation of the variance-covariance structure. Modifications to this initial model may be statistically significant, since changing one parameter in a poorly specified model may have a large effect on $\Sigma(\hat{\theta})$, but the change in parameter estimates stems from the underlying poor fit of the initial model and better fitting initial models may have led to a different final model that had a superior fit.

In addition, the line of reasoning for ignoring 'negligible' parameter estimates seems to be consistent with the argument that many authors have put forward for ignoring a significant χ^2 statistic resulting from a test of H_0: $\Sigma = \Sigma(\theta)$ – that is, the size of the sample leads to 'too much power' (Steiger, 1990) . A large modification index is, *prima facie*, at least worthy of further investigation irrespective of the expected parameter change, which is itself scale dependent. In essence, modification indices are a series of tests of hypothesised models. However, there has been little discussion in the CFA literature as to how multiple tests of a series of models affect the Type I error rate. Furthermore, it remains unclear as to how large a modification index should be to warrant further attention.

Although Steiger (1990) dismisses modification indices entirely (and advocates his RMSEA instead), inspection of modification indices is one plausible means of identifying potential lack of fit, and can be a useful adjunct to other measures of fit. A conservative approach would be to consider only very large modification indices as indicative of parameters that may need to be included in a modified model.

Other authors have proposed cross-validation to protect against fitting a model with poor predictive validity that is strongly influenced by a small number of observations in

the sample. Two ways to cross-validate structural equations models have been suggested in the literature (Browne and Cudeck, 1993). In the classical approach, the sample is randomly split into calibration and validation sub-samples. The distance between the validation sample variance-covariance matrix and the hypothesised variance-covariance matrix that is fit to the calibration sample is calculated, and distances close to zero suggest close agreement between the results in the two samples, and thus a model that has predictive validity.

An alternative method was proposed by Browne and Cudeck (1989) who suggested a measure of the expected distance between validation and calibration samples based on a single sample estimate. The Expected Cross Validation Index (ECVI) is a measure of the expected overall discrepancy across all possible calibration samples (Browne and Cudeck, 1993, p. 150). It is calculated as $F[\mathbf{S}, \Sigma(\hat{\theta})] + \frac{2c}{n - 1}$. Values close to zero are desirable, although considerations of parsimony must also be made in interpretation of the ECVI (Mueller, 1996 p. 105). The ECVI is most useful when comparing alternative measurement models to assess which has superior fit.

Inspection of the residual matrix $\mathbf{S} - \Sigma(\hat{\theta})$ for a given $\hat{\theta}$ is yet another way of detecting lack of fit (Kline, 1998), although this simple approach does not appear to be advocated often in the structural equation modelling literature. Again drawing a parallel with linear regression, model mis-fit may be indicated by large standardized residuals. Standardized residuals that are greater than 1.96 or 2.58 in absolute value (corresponding to the critical values for a standard Normal deviate at $\alpha = 0.05$ and $\alpha = 0.01$ respectively) indicate covariances that are not adequately described by the hypothesised model. Jöreskog (1993) discussed the use of standardized residuals in the

assessment of model fit, and argued that standardized residuals greater than 2.58 in absolute value were large residuals and indicated poor fit of the hypothesised model. However, the use of standardized residuals to augment other assessment of model fit has received little attention in the applied literature.

In summary, modification indices and standardized residuals may be used to identify parameters that may be estimated in a revised model in the pursuit of improved model fit. However, the use of modification indices in a forwards or stepwise model selection strategy for a better fitting model is eschewed. The ECVI may also provide useful additional information in deciding between alternative measurement models.

Thus a strategy that uses modification indices prudently and jointly considers information from the ECVI and matrix of standardized residuals can be a useful addition to the assessment of model fit, but should not be used in isolation from other methods of fit assessment.

4.2.3.5 Reliability measures

The final measures considered in the assessment of fit of CFA models in this thesis are measures of reliability. Different methods have been proposed to assess the reliability of the observed and latent variables respectively. The simple approach that is used for the reliability of observed variables is to compare the squared standardized factor loadings $[\lambda_{ij}^{s}]^2$. The $[\lambda_{ij}^{s}]^2$ are coefficients of determination, and a comparison of these terms can be used to assess the relative importance of each observed variable as an indicator of the associated latent variable.

Fornell and Larcker (1981) presented a composite reliability measure for each latent variable given by

$$\rho(\xi_j) = \frac{\left[\sum_{i=1}^{p} \lambda_{ij}^{s}\right]^2}{\left[\sum_{i=1}^{p} \lambda_{ij}^{s}\right]^2 + \sum_{i=1}^{p}(1 - \lambda_{ij}^{s\,2})}$$

that is constrained to lie between zero and one. Larger values are interpreted to indicate greater internal consistency of the observed variables within a latent variable.

Despite almost 1300 citations to this work by late 2006 (Web of Science, 2007), the justification for this statistic is not made in the original article by Fornell and Larcker (1981). Values of $\rho(\xi_j)$ greater than 0.7 are generally interpreted to represent good internal consistency and a reliable latent variable (e.g. Glass et al., 1997). However, the value of 0.7 appears to have been borrowed from common recommendations concerning Cronbach's α (Cronbach, 1951) that α values of 0.7 to 0.8 are satisfactory (e.g. Bland and Altman, 1997).

Thus the standardized factor loadings provide the basis for assessing the reliability of individual observed variables and latent variables in the measurement model. Despite its weak justification in the original article, $\rho(\xi_j)$ is very widely used and gives a summary statistic that is intuitively similar to Cronbach's α. The coefficients of determination, together with the index of composite reliability, are used in this thesis to augment the assessment of model fit obtained via fit indices and the model modification methods.

4.2.3.6 Summary

There is a large choice of methods for fit assessment, although many of the methods do not appear to have a strong theoretical basis. There has been ongoing debate in the structural equation modelling literature about the best single method or combination of methods of fit assessment, with little resolution (Hu and Bentler, 1999). There are identified problems with the majority of the methods of fit assessment, and many authors have cautioned against the sole reliance on one index or other measure of fit (e.g. Byrne, 2005; Kline, 1998; Mueller, 1996 p. 125; Steiger, 1990). A decision concerning a given model that is based on a combination of methods is generally regarded as superior to a single method in the assessment of fit, and offers some protection against the problems with a particular index or method.

Thus despite the large amount of research activity in this area by proponents of structural equation modelling, a unified presentation that is theoretically grounded and complements standard statistical texts concerning multivariate statistical methods does not appear to have been published to date. While many different measures of model fit have been proposed, guidance in their use for the practitioner fitting such models is lacking. The philosophy adopted in this thesis has been to fit an initial measurement model based on a previously validated model, and then refine this measurement model in pursuit of a better fit. Thus CFA was used in the spirit of validation in this thesis.

4.2.4 Causality and assumptions underlying structural equation models

As mentioned briefly in the introductory paragraph of Section 4.2, structural equation models (or more precisely, users of structural equation models) have been criticized for two main reasons. The first criticism is of the notion that structural equation models allow one to infer causality (Freedman, 1999; Freedman, 1987; Freedman, 2004).

Second, and also of considerable concern, is that the majority of articles in the applied literature do not state the stochastic assumptions underlying the models that are fit (Freedman, 1987; Freedman, 1991; Freedman, 2004).

With reference to the notion of drawing causal inference from structural equation models, Ling (1983) wrote a scathing review of the book "Correlation and causation" (Kenny, 1979). A quotation from Ling (1983) nicely summarizes the causal argument drawn from structural equation models:

> *"A researcher believes that malaria may be caused directly by one's exposure to swampy air ('mal air' or bad air). Having specified his [sic] causal assumption by a path diagram, he finds a significant correlation between the incidence of malaria (Y) and the swampiness index (X) of numerous locations sampled in the study. Ergo, the researcher concludes that 'mal air' is the direct cause of malaria."*

Steiger (2001) is equally unmoved:

> *"Although the phrase 'correlation is not causation' is still recited in virtually every introductory course in social sciences statistics, an entire generation of psychologists has vague notions that this principle is somehow suspended in SEM".*

Freedman (1987) published a broad critique of structural equation models. The majority of his criticisms concerning causal inference were directed at linked sets of structural equations in which the latent variables in a measurement model (developed using confirmatory factor analysis) were subsequently used as explanatory variables in a second equation. These structural equation models are commonly, but incorrectly,

interpreted as establishing causality. By analogy to multiple linear regression, causal inference is not made from fitting a linear regression model; structural equation models should be interpreted in a similar fashion. There are, however, thousands of applied articles that fit structural equation models and then claim a causal relationship as established based on the results of the model fit.

The second criticism made originally by Freedman (1987) and reiterated more recently by Freedman (2004) and Steiger (2001) is that the assumptions on which structural equation models rest are rarely made explicit in published applications. It is possible that some users of such techniques are not aware of the assumptions they are making or alternatively that users misunderstand the assumptions, either in part or entirely. Assumptions need to be explicitly stated in CFA and in structural equation models.

A final related point worth making here is that of the 'naming fallacy', whereby the assignment of a label to a latent variable is somehow taken to imply that the underlying construct actually exists, and then based on the assumption of existence, that it is understood and/or correctly named (Kline, 1998). For example, observed variables that measure an individual's height, weight, and shoe size are unlikely to be indicators of a latent variable 'socioeconomic status'. While this is an obvious point, and relates to the face validity of CFA, it appears to have been often overlooked in the applied literature.

4.2.5 Application of confirmatory factor analysis in this thesis

As described in detail in Chapter 5, the measurement model developed by Glass et al. (1997) was the starting point in the development of measures of social networks for the ALSA sample. The purpose of the CFA in the present thesis was to determine whether the model developed by Glass et al. (1997) using CFA provides an adequate description

of the social networks of ALSA participants. Thirteen variables concerning social networks of ALSA participants were available for the CFA, as outlined in Section 3.4.

A measurement model based on Glass et al. (1997) was specified and fit. The fit of the model was assessed using a selection of the above indices and approaches. The recent suggestion that the CFI, RMSEA and modification indices be jointly considered in the assessment of model fit (Byrne, 2005) has been adopted in this thesis. This was augmented by examination of the standardized residuals, the ECVI, and coefficients of determination. The change in the χ^2 statistic was used in the comparison of nested models, to formally test whether the inclusion of additional parameters in the model made a significant difference to the model fit. Based on the results of the initial model fit, the model was modified and a new model was fit. The fit indices, standardized residuals, ECVI and coefficients of determination for the modified model were then calculated. Alternative model specifications with different numbers of latent variables were also used to assess the fit of the modified model in the broader context.

Once a model with adequate fit was finalized, summary measures of social networks were derived based on the observed variables in the measurement model. Liang et al. (1990) have suggested that when a composite variable is created from observed variables with similar variances and factor loadings, the difference between simple averaging of the observed variables and differential weighting by λ_{ij} is very small in terms of reliability. On this basis, Glass et al. (1997) calculated the specific social network variables in their study by summing the standardized observed variables that made up each latent variable. However, this led to variables with different scales. For this reason, averaging of the standardized observed variables in each latent variable was carried out in this thesis. The means of the standardized observed variables were

calculated as there were four observed variables for the children social network variable and three observed variables in each of the latent variables corresponding to relatives, friends and confidants.

Alternatively, the conditional expectation of ξ given **X**, i.e. $\boldsymbol{E}(\xi \mid \mathbf{X}) = \mathbf{X}(\Lambda_{\mathbf{X}}\Phi\Lambda_{\mathbf{X}}'+ \Psi)^{-1}\Lambda_{\mathbf{X}}\Phi$, can be calculated and this also been done in this thesis. The results for the three alternative methods of calculating each specific network variable (i.e. averaging, differential weighting and conditional expectation) were compared. The total of all of the standardized observed variables used in the CFA was also calculated. Thus specific and total social network variables were derived based on the results of the CFA.

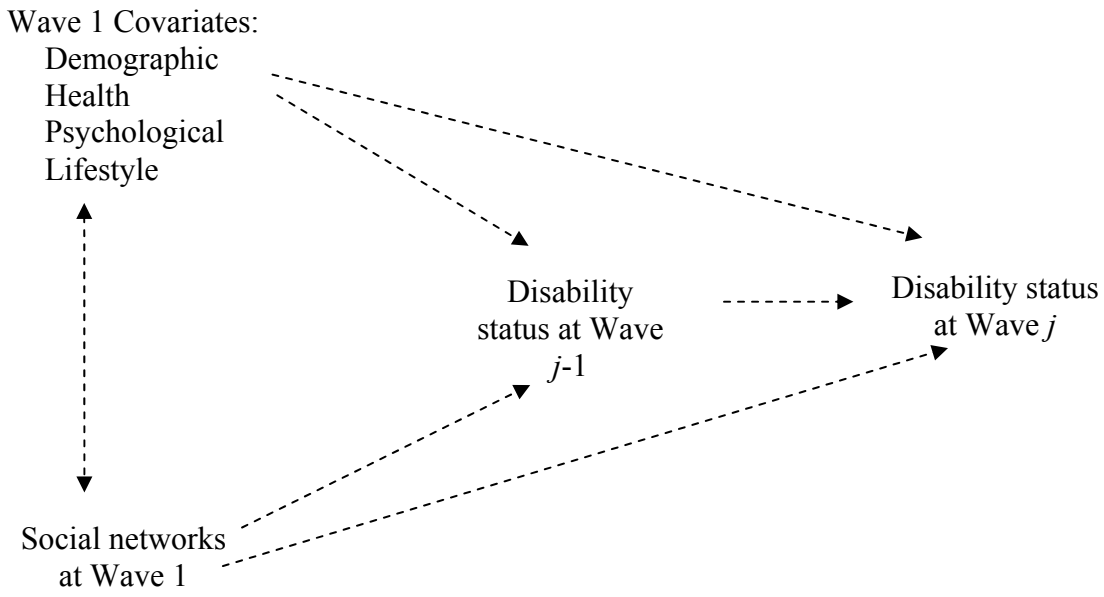
To allow a more ready interpretation of the total and specific social network variables, each of the derived social network variables was also classified according to the tertiles of its distribution. Both the continuous and categorized versions of the social network variables are used in this thesis.

The CFA was a way to check that the social network variables represented a reasonable simplification of the full set of variables from which they were derived. In the present application of CFA, care has been taken to avoid over-interpretation of the CFA results. The technique was used only in the verification of measures of social networks. Furthermore, the assumptions made in carrying out the CFA are explicitly stated in Chapter 5, and some evidence concerning the robustness of the technique is presented. An approach of validation is taken in the present thesis. No attempt is made to infer causality from the results. Thus the major conclusions in the thesis are not solely predicated on the validity of the CFA.

4.3 ANALYSIS OF TRANSITION DATA

The goal of the analyses of disability was to assess how social networks influenced disability over the six waves of the study, while adjusting for the effects of a range of other covariates. Similarly, the goal of the analyses of residential care use was to assess the effects of social networks on the use of residential care over the study waves, also taking into account the effects of other covariates. For both outcomes, the health state at a particular wave was also hypothesised to be a function of a participant's health state at the previous wave. For example, disability status at Wave 3 was hypothesised to be affected by disability status at Wave 2, social networks and other covariates. Figure 4.2 illustrates the conceptual model of interest. Note that dashed lines are used here only to reinforce that Figure 4.2 is not a path diagram but rather a proposed map of the relationships between disability and the explanatory variables.

Figure 4.2: Conceptual model for effect of social networks on disability



A class of models suitable for the analysis of response variables that are a function of previous responses and other covariates are transition models (Diggle et al., 2002). In

this section, transition models for the analysis of categorical response variables such as disability and use of residential care are described. Specific reference to the example of the analysis of disability is made in introducing the notation. Estimation methods that recognize the correlation between repeated observations from an individual are also discussed.

First, consider a binary response variable – for example, no disability or disability, for now ignoring the category of death. Following the notation of Diggle et al. (2002), let the response status of individual i at time j be represented by y_{ij}, with j=1, …, 6 used here to denote the study waves and y_{ij} taking the values of 0 (i.e. no disability) or 1 (disability). Previously, Mendes De Leon et al. (1999) fitted separate logistic regression models, of the form logit $\Pr(Y_{ij} = 1 \mid Y_{ij\text{-}1} = 0) = \boldsymbol{x}_{ij}'\beta_0$ and logit $\Pr(Y_{ij} = 0 \mid Y_{ij\text{-}1} = 1) = \boldsymbol{x}_{ij}'\beta_1$ where the explanatory variables are denoted by $\boldsymbol{x}_{ij}'$ and β_0 and β_1 are vectors of regression coefficients that may differ depending on the previous value of $y_{ij\text{-}1}$. In this thesis, the explanatory variables comprise the social network variables and the other covariates, the latter summarized using propensity scores (Section 4.5). These equations partially describe a first order Markov chain regression model that is characterized by the first order transition matrix $\begin{bmatrix} \pi_{00} & \pi_{01} \\ \pi_{10} & \pi_{11} \end{bmatrix}$ where $\pi_{ab} = \Pr(Y_{ij} = b \mid Y_{ij\text{-}1} = a)$ and is dependent on the explanatory variables. Diggle et al. (2002) described how the separate logistic regressions could be expressed more succinctly in one model as

$$\text{logit } \Pr(Y_{ij} = 1 \mid Y_{ij\text{-}1} = y_{ij\text{-}1}) = \boldsymbol{x}_{ij}'\beta_0 + y_{ij\text{-}1}\boldsymbol{x}_{ij}'\alpha \qquad (4.1)$$

An additional advantage of the Diggle et al. approach is that the fit of nested models can be formally tested against each other.

To fit such a model to the data – that is, to estimate $\beta = \begin{pmatrix} \alpha \\ \beta_0 \end{pmatrix}$ – maximum likelihood estimation can be used. Under maximum likelihood, the asymptotic variance-covariance matrix of the parameter estimates is given by the inverse of the Fisher information matrix $\text{Var}(\hat{\beta}) = (\text{T}' \hat{\text{V}}\, \text{T})^{-1}$ where T is the model matrix and $\hat{\text{V}}$ is a diagonal matrix with entries corresponding to the binomial variances $\hat{\pi}_{ij}(1-\hat{\pi}_{ij})$ estimated using $\hat{\beta}$.

While maximum likelihood gives consistent estimates of β, the standard errors associated with $\hat{\beta}$ may be too small if there is dependence between times of repeated measurement. One method that gives a 'robust' variance estimate is the sandwich variance estimator, proposed by Huber (1967) and White (1982). The robust variance estimate is consistent under misspecification of the dependence structure between repeated observations on the same individual. It is calculated as

$$\text{Var}_{rob}(\hat{\beta}) = (\text{T}' \hat{\text{V}}\, \text{T})^{-1} \left[\sum_{i=1}^{n} (\text{T}_i' (\boldsymbol{y}_i - \hat{\pi}_i) (\boldsymbol{y}_i - \hat{\pi}_i)' \text{T}_i) \right] (\text{T}' \hat{\text{V}}\, \text{T})^{-1},$$

where T_i, $\boldsymbol{y}_i$, and $\hat{\pi}_i$ are the components of T, $\boldsymbol{y}$ and $\hat{\pi}$ for the i^{th} individual. Using the robust estimate of the variance ensures that the standard error estimate is consistent.

If death is also included as a possible category of response, the disability response variable then takes an ordinal form (with categories of say 0 = no disability, 1 = disability and 2 = dead). Turning to transition models for ordered categorical data, let the response variable y_{ij} now take values a, b = 0, 1 and 2. The first order transition matrix is defined by $\Pr(Y_{ij} = b \mid Y_{ij-1} = a)$ for a, b = 0, 1 and 2. For the specific example of death, trivially $\pi_{20} = \pi_{21} = 0$ and $\pi_{22} = 1$ but this restriction will not hold in general.

As described for the binary case, a separate model for logit $\Pr(Y_{ij} = b \mid Y_{ij\text{-}1} = a)$ could be fit for each a and b.

Alternatively, a proportional odds model could be fit as a more parsimonious way of expressing these models (McCullagh, 1980). In the proportional odds model, the cumulative probabilities $\Pr(Y_{ij} \leq b \mid Y_{ij\text{-}1} = a)$ are considered rather than the cell probabilities from the first order transition matrix. Given the cumulative probabilities, the cell probabilities can be derived since $\Pr(Y_{ij} \leq b) = \Pr(Y_{ij} \leq b - 1) + \Pr(Y_{ij} = b)$. Unlike the binary case in which a single logit is modelled, several cumulative logits are modelled in the proportional odds model. In the case of a response variable with levels 0, 1 and 2 (for example, no disability, disability and dead), two logits corresponding to levels 0 and 1 versus level 2 and level 0 versus levels 1 and 2 would be fit.

Expressed formally, consider a vector of response variables defined by $Y^*_{ij} = (Y^*_{ij;0}, Y^*_{ij;1})$ such that

$$Y^*_{ij;0} = \begin{cases} 1 \text{ if } Y_{ij} = 0 \\ 0 \text{ otherwise} \end{cases} \text{ and}$$

$$Y^*_{ij;1} = \begin{cases} 1 \text{ if } Y_{ij} \leq 1 \\ 0 \text{ otherwise} \end{cases} \qquad (4.2)$$

(Clayton 1992, cited in Diggle et al., 2002). The proportional odds model here is a logistic regression for $Y^*_{ij;0}$ and $Y^*_{ij;1}$, since

$$\log \frac{\Pr(Y_{ij} \leq 0)}{\Pr(Y_{ij} > 0)} = \text{logit } \Pr(Y^*_{ij;0} = 1) = \theta_0 + \boldsymbol{x}_{ij}'\beta$$

and

$$\log \frac{\Pr(Y_{ij} \leq 1)}{\Pr(Y_{ij} > 1)} = \text{logit } \Pr(Y^*_{ij;1} = 1) = \theta_1 + \boldsymbol{x}_{ij}'\beta$$

where θ_b is the intercept for level b = 0, 1 of the response variable, the covariates $\boldsymbol{x}_{ij}$ are assumed to have the same effect on $Y^*_{ij;0}$ and $Y^*_{ij;1}$, and β is a vector of regression coefficients.

If the covariates are instead allowed to have differing effects on Y_{ij} for each previous state of Y_{ij}, then the cumulative probabilities $\Pr(Y_{ij} \leq b \mid Y_{ij\text{-}1} = a)$ can be modelled as

$$\text{logit } \Pr(Y_{ij} \leq b \mid Y_{ij\text{-}1} = a) = \theta_{ab} + \boldsymbol{x}_{ij}'\beta_a \text{ for } a = 0, 1 \text{ and } 2. \tag{4.3}$$

However, as in the binary case, this can be rewritten in a single equation for the specific application of a transition model as

$$\text{logit } \Pr(Y_{ij} \leq b \mid \boldsymbol{Y}^*_{ij\text{-}1} = \boldsymbol{y}^*_{ij\text{-}1}) = \theta_b + \sum_{k=0}^{1} \alpha_{kb} y^*_{ij\text{-}1;k} + \boldsymbol{x}_{ij}'\beta + \boldsymbol{x}_{ij}' \sum_{k=0}^{1} \gamma_k y^*_{ij\text{-}1;k} \tag{4.4}$$

where $\gamma_0 = \beta_0 - \beta_1$ and $\gamma_1 = \beta_1 - \beta_2$ and α represents the regression coefficients for the $y_{ij\text{-}1}$ terms (Diggle et al., 2002). This formulation of the model allows a test of whether or not the effect of $\boldsymbol{x}_{ij}$ on Y_{ij} is the same for adjacent categories of $Y_{ij\text{-}1}$ through a test of $\gamma_k = 0$. As is the case with a binary response variable, the sandwich estimate of the variance-covariance matrix for the parameter estimates can be used to derive robust standard errors.

If the assumption of proportional odds is not met reasonably well, then alternative models such as a multinomial logistic regression model must be explored. One method for assessing the assumption of proportional odds was proposed by Fu (1998), in an extension of earlier work by Brant (1990). The idea underlying Fu's method was to compare the fit of the proportional odds model to what Fu termed a generalized ordered logit model. In the generalized ordered logit model, a separate set of β coefficients (say

β_b) are estimated for each of the points at which the dependent variable can be dichotomized – that is, the model fit is

$$\text{logit Pr}(Y_{ij} \leq b \mid \boldsymbol{Y}^{*}_{ij\text{-}1} = \boldsymbol{y}^{*}_{ij\text{-}1}) = \theta_b + \sum_{k=0}^{1} \alpha_{kb} y^{*}_{ij\text{-}1;k} + \boldsymbol{x}_{ij}'\beta_b + \boldsymbol{x}_{ij}' \sum_{k=0}^{1} \gamma_k y^{*}_{ij\text{-}1;k} .$$

A test of the fit of the proportional odds model versus the generalized ordered logit model is a test of H_0: $\beta_b = \beta$. This can be achieved through the calculation of the change in the log-likelihood between the two models, which asymptotically follows a chi-square distribution. Rejection of this null hypothesis indicates a violation of the assumption of proportional odds.

If the proportional odds model is rejected, then the generalized ordered logit model could be fit. However, this model is not constrained in such a way that the cumulative probabilities are naturally ordered. Therefore, a better alternative is to model the transition probabilities $\Pr(Y_{ij} = b \mid Y_{ij\text{-}1} = a)$ using a multinomial logistic regression model (Diggle et al., 2002 p. 201). Such a model may be formulated as

$$\text{logit Pr}(Y_{ij} = b \mid Y_{ij\text{-}1} = y_{ij\text{-}1}) = \boldsymbol{x}_{ij}'\beta_b + y_{ij\text{-}1}\boldsymbol{x}_{ij}'\alpha_b \qquad (4.5)$$

A saturated model of the transition matrix may be obtained by fitting a separate regression for each of the values of $Y_{ij\text{-}1}$. As before, the sandwich estimate of the variance-covariance matrix can be used to derive robust standard errors and 95% confidence intervals for the parameter estimates.

4.3.1 Application of transition models in this thesis

The transition models as described in the previous section were used in separate analyses of disability and residential care use to ascertain the effects of social networks on these two distinct outcomes.

Disability was analysed in two different ways in this thesis. First, the disability response variables that were considered excluded death as a response category, and binary logistic regression transition models were fit. All available data until the time of death were included in the analysis for individuals who died, so that if an individual was alive at Wave 2 and dead at Wave 3, their disability status at Waves 1 and 2 would be included in the binary logistic regression analyses. In the second approach, the disability variables were considered as ordered categorical variables, in which death was included as a possible response category. Thus for an individual alive and disabled at Waves 1 and 2 and dead at Wave 3, their response would be included in the model as disabled at Wave 1, disabled at Wave 2, and dead at Wave 3. In these analyses, proportional odds models were initially fit to the data, and an assessment of the proportional odds assumption was made using Fu's (1998) method. Multinomial logistic regression models were fit if the assumption of proportional odds was violated. Irrespective of whether disability was considered as a binary response variable or ordered categorical variable, the sandwich variance estimator $\text{Var}_{rob}(\hat{\beta})$ was used to calculate standard errors for the parameter estimates that arose from all models.

In the analyses of residential care use, there were many possible responses for use of residential care over the course of the ALSA study. Therefore the analyses of residential care use considered the response variable as an ordered categorical variable. As for disability, separate analyses were carried out when death was excluded and then included as a possible response category. The assumption of proportional odds was again checked for the models fitted in the analyses of residential care use using the method of Fu (1998), and $\text{Var}_{rob}(\hat{\beta})$ was used in the calculation of standard errors for the parameter estimates.

The explanatory variables in the fitted models included propensity scores (that summarized demographic, health, psychological and lifestyle variables for the ALSA participants, as described later in Section 4.5), age group, gender, the social network variables, and key two-way interactions. The fit of the initial and nested models were compared through the calculation of likelihood ratio (LR) tests (i.e. -2 x change in log-likelihood between initial and nested model). A separate model-fitting regime was undertaken for each of the children, relatives, friends and confidants social network variables, as well as for the total social network variable, and separate analyses were undertaken for the continuous and categorized social network variables.

4.4 SURVIVAL ANALYSIS

Analyses were performed to assess how social networks influenced survival over the decade following ALSA participants' initial interview, while adjusting for the effects of other covariates. In this section, a brief overview of the methods used in the analysis of survival data in this thesis is given, along with the methods used in the assessment of model fit.

The Cox proportional hazards model (Cox, 1972) is the most commonly used model in the assessment of the effects of explanatory variables upon a censored response variable such as survival (Therneau and Grambsch, 2000 p. 39). The Cox proportional hazards model specifies the hazard function $h_i(t;\boldsymbol{x}_i)$ for an individual i with a vector of p explanatory variables $\boldsymbol{x}_i'=(x_{1i}, x_{2i}, \ldots, x_{pi})$ as $h_i(t;\boldsymbol{x}_i) = h_0(t)e^{\boldsymbol{x}_i'\beta}$. Here, the baseline hazard function $h_0(t)$ is an unspecified non-negative function of time that represents the hazard function for an individual with covariate values all equal to zero, and β is a p x 1 vector of regression coefficients. The hazard ratio for persons i and k is given by

$$\frac{h_i(t;\boldsymbol{x}_i)}{h_k(t;\boldsymbol{x}_k)} = \frac{h_0(t)\, e^{\boldsymbol{x}_i'\beta}}{h_0(t)\, e^{\boldsymbol{x}_k'\beta}} = e^{(\boldsymbol{x}_i - \boldsymbol{x}_k)'\beta}. \tag{4.6}$$

Further define the cumulative hazard as $H(t) = \int_0^t h(s)ds$. This model assumes that the explanatory variables are time-invariant, although variables that are time-varying can also be incorporated in the model (Therneau and Grambsch, 2000, p. 130). In this thesis, only time-invariant explanatory variables were considered and therefore time-varying explanatory variables are not discussed any further.

In the case of a single binary explanatory variable, the ratio of the hazard functions given in (4.6) reduces to e^{β} and is thus constant with respect to time, so the two hazard functions are assumed to be proportional. In general, validation of proportionality is needed as part of the model fitting process, and some of the methods that have been proposed for this are described later in this section.

In recent years, the theoretical basis for the Cox proportional hazards model has been connected to the study of counting processes and Martingale theory, which has led to extensions to the original model (Fleming and Harrington, 1991; Therneau and Grambsch, 2000), and contributed to the development of methods for checking model fit. Using a counting process formulation, the Nelson-Aalen cumulative hazard estimator can be derived (Aalen, 1978; Nelson, 1969). This estimator can be used in the Cox proportional hazards model and in the methods for the assessment of model fit, as follows.

Following the notation of Therneau and Grambsch, let $Y_i(t)$ be an indicator function such that $Y_i(t) = 1$ from time zero until event occurrence or censoring, and $Y_i(t) = 0$

thereafter. Let $N_i(t)$ be another indicator variable such that $N_i(t)$ is zero until the moment of the event occurrence and 1 thereafter. $N_i(t) = 0$ for all t if the observation for unit i is censored. In other words, if an event occurs at time s, then

$$Y_i(t) = \begin{cases} 1 & t \le s \\ 0 & t > s \end{cases}$$

and

$$N_i(t) = \begin{cases} 0 & t < s \\ 1 & t \ge s. \end{cases}$$

Further define the aggregated processes $\bar{Y}(t) = \Sigma_i Y_i(t)$ and $\bar{N}(t) = \Sigma_i N_i(t)$, such that $\bar{Y}(t)$ is the total number of units at risk of event occurrence at time t, and $\bar{N}(t)$ is the total number of events up to and including time t. Now consider the difference in the cumulative hazard function over a brief interval of time, say $(s, s+u]$. The difference $H(s+u) - H(s) \approx h(s)u = \Pr(\text{event in } (s, s+u] \mid \text{at risk at } s)$ can be estimated as $\bar{N}(s+u) - \bar{N}(s) / \bar{Y}(s)$ – that is, by the number of events occurring in $(s, s+u]$ divided by the number of individuals at risk at s. Summing these terms over subintervals $(0, t]$ that are sufficiently small so that they contain at most one event time gives the Nelson-Aalen cumulative hazard estimator $\hat{H}(t) = \int_0^t \frac{d\bar{N}(s)}{\bar{Y}(s)}$. The notation $d\bar{N}(s)$ is used in principle to accommodate both discrete and continuous components of the counting process, such that $d\bar{N}(s) = \Delta\bar{N}(s) + n(s)ds$. The discrete component, $\Delta\bar{N}(s)$, represents the number of events occurring precisely at s and is given by $\bar{N}(s) - \bar{N}(s–)$ while the continuous component of the equation is given by $n(s)ds$. Because counting processes are pure jump processes, the continuous part is not necessary so $d\bar{N}(s) = \Delta\bar{N}(s)$. The Kaplan-Meier estimate of the survival function is calculated as $\hat{S}(t) = \prod_{j;t_j \le t} [1 - d\hat{H}(t_j)]$ where t_j is the time of the j^{th} event and $d\hat{H}(t_j)$ is the increment in the Nelson-Aalen cumulative hazard estimator at the j^{th} event, given by $d\bar{N}(t_j) / \bar{Y}(t_j)$.

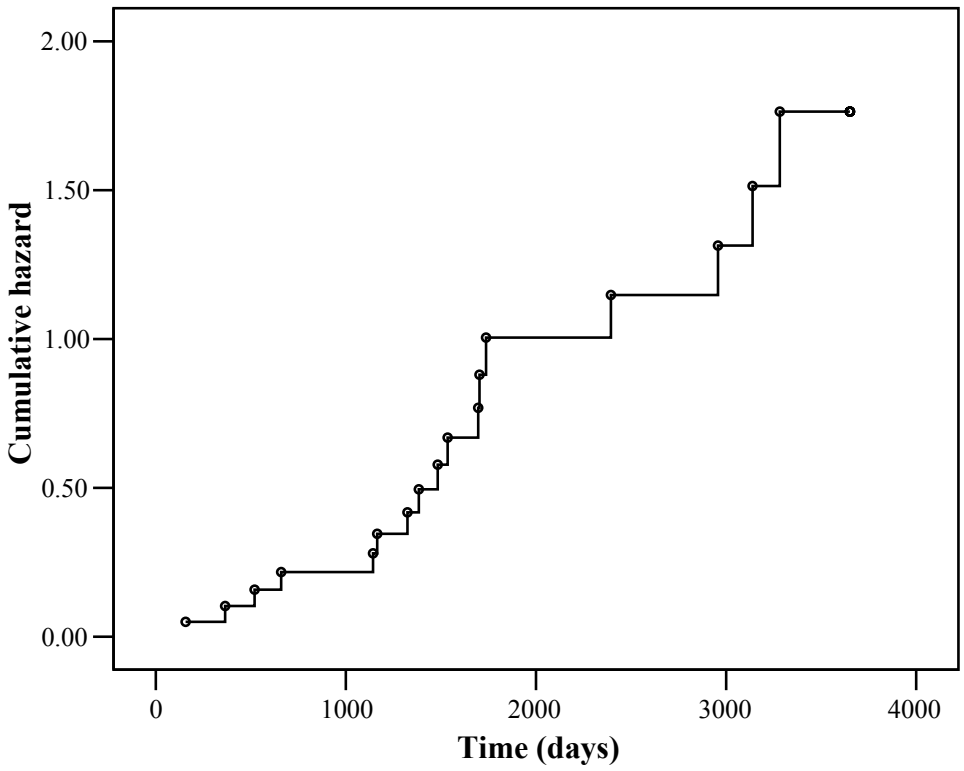
To illustrate the calculation of the Nelson-Aalen cumulative hazard estimator for the no-covariate case, consider the 10-year follow-up of survival status of the first 20 ALSA participants, ordered according to survival time in days. Seventeen of these 20 participants died over the decade following their initial interview. Observation of the vital status of the three surviving participants was censored on the tenth anniversary of their initial interview (that is, their censored survival time was 3653 days). In Table 4.1, the survival times for the 17 deceased participants are shown. The Nelson-Aalen cumulative hazard estimate at, say, day 730 is 1/20 + 1/19 + 1/18 + 1/17= 0.217, as there were four deaths (on days 157, 365, 520, and 660) by the second anniversary of the initial interview for this sample of 20 participants.

Table 4.1: Nelson-Aalen cumulative hazard estimator for the first 20 ALSA participants

Death no.	Days survival	$\bar{Y}(t)$	$\hat{H}(t)$	Death no.	Days survival	$\bar{Y}(t)$	$\hat{H}(t)$
1	157	20	0.050	10	1535	11	0.669
2	365	19	0.103	11	1696	10	0.769
3	520	18	0.158	12	1703	9	0.880
4	660	17	0.217	13	1737	8	1.005
5	1143	16	0.280	14	2394	7	1.148
6	1165	15	0.346	15	2957	6	1.314
7	1324	14	0.418	16	3139	5	1.514
8	1384	13	0.495	17	3283	4	1.764
9	1483	12	0.578				

As reviewed by Therneau and Grambsch (2000), there are two ways in which to interpret the Nelson-Aalen cumulative hazard estimator. The first interpretation is that $\hat{H}(t)$ is an estimate of the average number of failures in $(0, t]$ for a unit 'perpetually at risk'. The second way to interpret $\hat{H}(t)$ is that the slope of a plot of $\hat{H}(t)$ versus time provides an estimate of $h(t)$. For the cohort of 20 participants, the plot of $\hat{H}(t)$ versus time is presented in Figure 4.3.

Figure 4.3: Nelson-Aalen cumulative hazard estimator for the first 20 ALSA participants



The counting process formulation of a Cox proportional hazards model is useful in the derivation of several different kinds of residuals (Therneau and Grambsch, 2000, p. 79). In this thesis, Schoenfeld residuals were used in the assessment of fit. Schoenfeld residuals for the Cox proportional hazards model are partial residuals that are calculated at event times. For a given covariate, there is one Schoenfeld residual for each observed event (that is, each death) rather than for each individual unit of observation (Schoenfeld, 1982). Let $\bar{x}(\beta, s)$ be the weighted mean of the covariates $\boldsymbol{x}_i$ over those observations still at risk at time s – that is, $\bar{x}(\beta, s) = \dfrac{\sum Y_i(s)R_i(s)\boldsymbol{x}_i}{\sum Y_i(s)R_i(s)}$ where the risk score $R_i(s)$ is given by $e^{\boldsymbol{x}_i'\beta}$. Then the Schoenfeld residual estimated at the j^{th} event time is defined as

$$\hat{r}_j = \int_{t_{j-1}}^{t_j} \sum_i [\boldsymbol{x}_i - \bar{x}(\hat{\beta}, s)]\, dN_i(s) \tag{4.7}$$

where $\hat{\beta}$ is the maximum partial likelihood estimate of β under the proportional hazards model (Cox, 1972) and $\bar{x}(\hat{\beta}, s)$ is the value of $\bar{x}(\beta, s)$ at $\hat{\beta}$. When there are no tied event times, this simplifies to

$$\hat{r}_j = \boldsymbol{x}_{(j)} - \bar{x}(\hat{\beta}, t_j) \tag{4.8}$$

where $\boldsymbol{x}_{(j)}$ is the covariate vector of the individual experiencing the j^{th} event (Therneau and Grambsch, 2000).

Grambsch and Therneau (1994) proposed a scaled Schoenfeld residual that is standardized according to the inverse of the variance $V(\beta, s)$ of the covariates at time s. $V(\beta, s)$ is estimated by $V(\hat{\beta}, s)$ as $\dfrac{\sum Y_i(s)R_i(s)[\boldsymbol{x}_i - \bar{x}(\hat{\beta}, s)]'\,[\boldsymbol{x}_i - \bar{x}(\hat{\beta}, s)]}{\sum Y_i(s)R_i(s)}$. Then the scaled Schoenfeld residual $\hat{r}_j^*$ is calculated as

$$\hat{r}_j^* = V^{-1}(\hat{\beta}, s)\,\hat{r}_j. \tag{4.9}$$

As reviewed by Hess (1995), many numerical goodness-of-fit tests and some graphical tests of the proportional hazards assumption in the general Cox proportional hazards model have been proposed. While none of the numerical goodness-of-fit tests have been widely implemented (Hess, 1995), a graphical method proposed by Grambsch and Therneau (1994) based on scaled Schoenfeld residuals has become increasingly popular, especially following its implementation in some standard statistical software packages. Grambsch and Therneau (1994) proposed that if the assumption of proportional hazards holds, then a plot of $\hat{r}_j^*$ versus t_j or some function of time $g(t_j)$ centres around $\hat{\beta}$. A line

can be fit to the plot, and then a test of the null hypothesis that the slope of the line is zero can be conducted. A non-zero slope is indicative of a departure from proportional hazards (Grambsch and Therneau, 1994; Therneau and Grambsch, 2000).

If there is significant non-proportionality, then several different approaches summarized by Therneau and Grambsch (2000, pp. 145-147) are available. The simplest of these approaches is to incorporate the covariates with non-proportional effects into the model as stratification factors, not regressors. A second approach is to partition the time axis, since the proportional hazards assumption may hold over short time periods, but not over the entire study. A third alternative is to model the non-proportionality through the use of time-dependent covariates, although Therneau and Grambsch (2000, p. 147) noted that this approach had to be used cautiously. The final suggestion of these authors was to use a different model, such as an accelerated failure time or additive hazards model.

4.4.1 Application of survival analysis in this thesis

In the analysis of survival of the ALSA participants over the decade following their initial interview, a combination of the methods described above was used.

The response variable was the number of days to death from Wave 1 interview for decedents and 3653 days for participants who survived 10 years after their initial interview. The cumulative hazard of death over time was compared graphically for each social network type using the Nelson-Aalen cumulative hazard estimator. For each type of specific social network as well as for the total social network variable, a separate Cox proportional hazards model was fit to the data, that included the propensity scores, age group, gender and the social network variable. Simpler nested models that omitted

the social network variable were then fit and compared to the initial model through LR tests.

The assumption of proportional hazards was assessed by regressing the scaled Schoenfeld residuals against the log of time and testing for zero slope. A non-zero slope provided evidence against proportional hazards (Grambsch and Therneau, 1994). If significant non-proportionality was detected for a covariate, then the covariate(s) with non-proportional effects were included in the model as stratification factors instead of explanatory variables.

4.5 PROPENSITY SCORE ADJUSTMENT

In randomized controlled trials, group assignment is, by definition, randomized and so the differences in observed covariates between treatment groups are due solely to chance. However, in observational studies such as ALSA, there is no manipulation of 'treatment' assignment, and so there is the potential for large differences between observed covariates in the different treatment groups. Ignoring these differences could potentially lead to biased estimates of treatment effects. Traditional methods of adjusting for observed covariates in analyses, such as matching or stratification, may be difficult to use if there are a large number of covariates. Regression adjustment can also be problematic. Missing values in one or more covariates for an individual *i* will result in all data for that individual being dropped from a regression analysis unless estimation of the missing covariate values is carried out. Another problem in regression adjustment lies in finding and fitting an appropriate functional form for each covariate.

Propensity scores have been proposed as an alternative method to adjust for a set of covariates (Joffe and Rosenbaum, 1999; Rosenbaum and Rubin, 1983). Most

applications of propensity scores to date have involved simple cross-sectional studies with binary treatments. In this section, propensity scores and some of their properties are first described for a binary treatment. More recent work by Joffe and Rosenbaum (1999) that extends the propensity score to treatments with multiple categories is then outlined.

Let Z_i be an indicator variable of assignment to a treatment for individual i, such that $Z_i = \begin{cases} 1 & \text{if treated} \\ 0 & \text{if control} \end{cases}$. The propensity score $p(\boldsymbol{x}_i)$ is defined as the conditional probability of assignment to the treatment given a vector of observed covariates $\boldsymbol{x}_i$ – that is, $p(\boldsymbol{x}_i) = \Pr(Z_i = 1 | \boldsymbol{X}_i = \boldsymbol{x}_i)$. Thus $p(\boldsymbol{x}_i)$ is the probability that an individual would have been treated based solely on the individual's covariate information (D'Agostino, 1998).

As summarized by D'Agostino (1998), two key results justify the use of propensity scores as an adjustment method that reduces bias, and these will be elaborated on in the following paragraphs. First, propensity scores 'balance' the observed covariates. Second, if it is sufficient to adjust for the covariates, then it is sufficient to adjust for their propensity score $p(\boldsymbol{x}_i)$; this property relates to *strongly ignorable* treatment assignment (Rosenbaum and Rubin, 1983).

Returning to the first of these points, Rosenbaum and Rubin (1983) defined a balancing score $b(\boldsymbol{x}_i)$ as a function of the observed covariates such that the conditional distribution of $\boldsymbol{X}_i$ given $b(\boldsymbol{x}_i)$ is the same for individuals, irrespective of whether they receive treatment or control - that is, Z_i and $\boldsymbol{X}_i$ are conditionally independent given $b(\boldsymbol{x}_i)$. The propensity score $p(\boldsymbol{x}_i)$ is the simplest, or coarsest, balancing score, and the covariates $\boldsymbol{x}_i$ themselves are the most complex, or finest, balancing score. Every balancing score

contains information in $p(\boldsymbol{x}_i)$ and some additional information from $\boldsymbol{X}_i$. In practice, it may be useful to adjust for the $p(\boldsymbol{x}_i)$ and for other functions of $\boldsymbol{x}_i$ as well (Rosenbaum and Rubin, 1983). For example, it may be useful to obtain estimates of the average treatment effect in a subgroup defined by components of $\boldsymbol{x}_i$, such as younger or older participants.

Second, treatment assignment is considered *strongly ignorable* if the treatment assignment Z_i and the response Y_i are conditionally independent given the covariates $\boldsymbol{X}_i$. Rosenbaum and Rubin (1983) proved that if treatment assignment is strongly ignorable given $\boldsymbol{X}_i$, then it is also strongly ignorable given any balancing score $b(\boldsymbol{x}_i)$.

In practice the systematic bias that can arise from imbalances in covariates cannot be distinguished from the random error that arises by chance in an estimated propensity score (Joffe and Rosenbaum, 1999; Rosenbaum and Rubin, 1983). True propensity scores remove systematic bias only. Thus adjustment for an estimated propensity score removes both random variation and systematic bias, and so estimated propensity scores tend to be superior to true propensity scores in removing bias (Rosenbaum, 1987).

More recently, propensity score methods have been extended beyond binary treatment regimes. Joffe and Rosenbaum (1999) described propensity scores with ordered doses of treatment, and Imai and Van Dyk (2004) outlined the estimation of propensity scores with arbitrary treatment regimes, encompassing ordinal, categorical, continuous, semi-continuous or multi-factored treatments. The extensions are not trivial, since for non-binary treatments the expected dose given the covariates $\boldsymbol{X}_i$ need not fully describe the distribution of doses.

However, Joffe and Rosenbaum (1999) demonstrated that if the distribution of treatment doses given $\boldsymbol{X}_i$ is accurately described by McCullagh's proportional odds model, then stratifying on $b(\boldsymbol{x}_i) = \boldsymbol{x}_i'\beta$, where β is a p x 1 vector of parameters, will balance $\boldsymbol{X}_i$ across several dose groups. More generally, it is possible that the distribution of doses Z given a large number of covariates may depend on the covariates through only a small number of linear functions of $\boldsymbol{X}$, say $\boldsymbol{X}\mathbf{G}$ for some matrix $\mathbf{G}$. Then $\boldsymbol{X}\mathbf{G}$ is a balancing score and controlling for the several variables in $\boldsymbol{X}\mathbf{G}$ will 'tend to balance the … variables in $\boldsymbol{X}$' (Joffe and Rosenbaum, 1999). For example, if a multinomial logistic regression model was adequate to describe $\Pr(Z_i = z \mid \boldsymbol{X}_i = \boldsymbol{x}_i)$ for some $z = 0, 1, \ldots, c$, then $\boldsymbol{X}\mathbf{G}$ would be an n x c matrix in which the first column defined a propensity score for level 1 of the treatment dose, the second column a propensity score for level 2 of the treatment dose, and so on up to level c of the treatment dose. Because of the linear dependence of the c^{th} propensity score on the first c-1 propensity scores, analyses would adjust for the first c-1 propensity scores only.

In practice, propensity scores are used in adjustment in three main ways, and these are the same as for the more familiar, direct covariate adjustment. The main methods of adjusting for propensity scores are via matching, stratification, and regression adjustment. The idea underlying all three methods is that if the units of observation with the same (or nearly the same) propensity scores are grouped together, then the treated and control units in these groups will have the same (or nearly the same) distributions of $\boldsymbol{X}_i$.

The method most commonly used in applications is stratification and this was used in the present study. To carry out stratification in practice, units are grouped into strata determined by the propensity scores. Quintiles are usually used for this purpose, based

on a result by Cochran (1968) who demonstrated that stratification with five strata is sufficient to remove at least 90 per cent of the bias due to the stratifying variable (cited in Rosenbaum and Rubin, 1983). In a stratum that is perfectly homogenous in a balancing score, treated and control units have the same distribution of $\boldsymbol{X}_i$ (Rosenbaum and Rubin, 1983). Thus the effectiveness of an estimated propensity score in 'balancing' the covariates within each strata (i.e. the balance status of a propensity score) can be assessed by comparing the treatment and control groups for each covariate in each strata. To estimate treatment effects, either direct standardization methods such as the combination of stratum-specific means or inclusion of the propensity score strata in a regression model can be used.

4.5.1 Application of propensity scores in this thesis

As described in Section 3.3, a core set of 15 demographic, health, psychological and lifestyle covariates were investigated in the analyses of the effects of social networks on disability, residential care use and survival. To recap briefly, these included age group, gender, marital status, income, home ownership, education, the number of chronic morbid conditions, self-rated health, hearing difficulty, vision difficulty, symptoms of depression, cognitive status, smoking status, alcohol use, and exercise habits. In addition, when the effects of social networks on residential care use and survival were considered, mobility disability was included in the set of covariates that were used to calculate propensity scores.

A subset of important covariates may be included in a fitted model twice, in that they may be used in the derivation of the propensity scores and also included in their original form as covariates in the fitted model (D'Agostino, 1998). This has benefits in terms of reducing the residual variance in the fitted model compared with propensity score

adjustment alone. In this thesis, age group and gender were included in both the propensity score model and as 'stand-alone' covariates in the fitted logistic regression models.

An additional advantage with propensity score adjustment is that a propensity score can be estimated using all of the data available from the covariates. A pragmatic approach was adopted in this thesis so that if for a given participant an observation was missing for at least one of the covariates, a propensity score was estimated using the subset of covariates with complete data for that participant. In this way, a propensity score was estimated for every participant, not only those participants with complete data for all covariates, and a propensity score was estimated for each pattern of missing covariate observations. Thus for every participant, the propensity scores were estimated using the maximum covariate information available for that participant.

Proportional odds regression models of the social network categories on the covariates were initially fit to derive propensity scores. Two sets of covariates were considered – with and without mobility disability, since propensity scores that included mobility disability in the set of covariates were not sensible in the analyses of disability. The fit of the proportional odds model was assessed for each of the categorized network variables, and a multinomial logistic regression model was alternatively used in the derivation of propensity scores if the fit of the proportional odds model was inadequate.

Complete covariate data were available for 1249 participants when mobility disability was excluded from the set of covariates and 1243 participants when disability was included in the set of covariates. For each participant with incomplete data for at least one covariate (n=228 or n=234), propensity scores were derived using the subset of the

covariates for which complete data were available for that participant. For example, if a participant had missing observations for the income and exercise habits variables, then a logistic regression model that excluded these two variables would be fit to all of the available data, and the fitted values resulting from that model fit used to derive a propensity score for that participant. Thus for every participant, a propensity score was estimated using the maximum covariate information available for that participant. In this way, propensity scores were estimated for each of the participants, and the adjusted analyses therefore included all 1477 participants and not only those with complete data for all 15 covariates and mobility disability. The propensity scores were classified into strata based on all 1477 observations. Five strata of propensity scores, classified according to the quintiles, were created when the fit of the proportional odds model was adequate. When the fit of the proportional odds model was inadequate and a multinomial logistic model was instead used, nine strata that were based on the joint distribution of the first two propensity scores were derived. This was because if 25 strata were used (i.e. five for each of the first two propensity scores), then the fitted models did not converge for the analyses of residential care use and disability.

Balance was assessed in each of the strata by chi-square tests of association of each covariate with each of the categorized social network variables (Imai and van Dyk, 2004). When the mobility disability variable was excluded from the estimation of propensity scores, 17 out of 455 comparisons for balance status of the covariates (4%) were statistically significant at $P<0.05$. When mobility disability was included in the propensity score estimation, 25 out of 484 comparisons for balance status of the covariates (5%) were statistically significant at $P<0.05$. These results indicated that the propensity score method produced balance in the observed covariates similar to that which would be expected by randomization of these covariates across the social

network categories. On this basis, it was determined that the propensity scores provided an adequate adjustment for the set of covariates in this study.

4.6 EXISTENCE OF THRESHOLD EFFECTS

The existence of threshold effects at both of the tertile cutpoints was investigated using linear splines. For each continuous social network variable, say s_i, a linear spline consisting of three variables $s_i^*(l)$, l=1, 2, 3 with knots at the tertile cutpoints c_1 and c_2 of s_i was constructed. The $s_i^*(l)$ were calculated such that

$$s_i^*(1) = s_i$$

$$s_i^*(2) = \begin{cases} 0 & \text{if } s_i \le c_1 \text{ and} \\ s_i - c_1 & \text{if } s_i > c_1 \end{cases}$$

$$s_i^*(3) = \begin{cases} 0 & \text{if } s_i \le c_2 \text{ and} \\ s_i - c_2 & \text{if } s_i > c_2 \end{cases} .$$

A series of nested models beginning with the final fitted model for each social network variable was then fit. The variables $s_i^*(2)$ or $s_i^*(3)$ were then added to the final fitted model, to test whether a bent-line regression model provided a superior fit to the final fitted model. The coefficients for $s_i^*(2)$ and $s_i^*(3)$ represented the change in the slope from the preceding percentile band. Likelihood ratio tests were then carried out to test the significance of including $s_i^*(2)$ or $s_i^*(3)$ in the model. In this way, the existence of non-linearity, for which threshold effects are a subset, at each of the 33⅓ and 66⅔rd percentile cutpoints was tested.

4.7 SOFTWARE

Several statistical packages were used in the analyses. For the CFA, PROC CALIS in SAS (SAS Institute Inc., 1998) and AMOS version 6.0 (Arbuckle, 2005) were used. Stata versions 8 and 9 (Statacorp, 2003; 2005) and SPSS for Windows versions 10, 11, 12 and 14 were used in the remaining analyses (SPSS Inc., 1999; 2002; 2004; 2005).

4.8 CONCLUSION

The statistical methods selected for use in this thesis encompassed a range of techniques that were appropriate for use with complex, longitudinal data. They were selected to both complement the extant literature in this area and also to ensure a rigorous approach was adopted in the analysis of the effects of social networks on key health outcomes.

The subsequent chapters present the results of these analyses, and expand on the statistical methods used within the context of the analyses of the ALSA data.

5 DERIVATION OF SOCIAL NETWORKS MEASURES

5.1 INTRODUCTION

In this chapter, the derivation of the social network variables that have been used subsequently in the analyses of the health outcomes is described. The measurement model developed by Glass et al. (1997) using data from the EPESE study is first described in Section 5.2. The observed social network variables that were available in the ALSA data set are then described in Section 5.3, and the fit of an initial measurement model to the ALSA data based on Glass et al. (1997) is presented. A modified model of social networks is then presented, and the fit of this modified model is summarized in Section 5.4. In order to place the modified model within the wider context of a range of possible models, the fit of the initial and modified models is compared with the fit of an alternative model, and this is described in Section 5.5. Additional measures of model fit are presented in Section 5.6. The way in which the CFA results have then been used to operationally define the social network variables is described in Section 5.7.

5.2 GLASS' INITIAL MEASUREMENT MODEL

Glass et al. (1997) proposed a multidimensional model of social networks that was based on data from the New Haven EPESE. These authors developed a measurement model that reflected social networks with children, relatives, friends, and confidants as well as total social networks for each study participant (as described in Section 2.3.1). The first aim of the present thesis was to develop a measurement model for social networks that could be used in an examination of the effects of social networks on health. To this end, the same specific social networks derived by Glass et al. were examined using data drawn from participants in the ALSA.

Table 5.1 shows the variables that were drawn from the EPESE study and used in the final CFA by Glass et al. (1997). As this table shows, there were five observed variables that corresponded to the latent variable for children, whereas the latent variables for relatives, friends and confidants social networks each corresponded to three observed variables. The initial measurement model fit by Glass et al. also included a latent variable that corresponded to networks with neighbours, but this latent variable was not supported by the model fit in Glass et al. and so is not discussed any further here.

Figure 5.1 presents the final measurement model fitted by Glass et al., including the five correlated error terms that were included in the model. These terms were included on the basis of modification indices for Glass' initial model that suggested where improvements in model fit may be possible. The test of H_0: $\Sigma = \Sigma(\theta)$ for this model had an associated χ^2 statistic of 149.4 on 68 df. The CFI was not presented by Glass et al., although they did report the GFI was 0.996 and AGFI was 0.994 for this model. These authors also noted that the modification indices suggested an improvement in model fit was possible through allowing the observed variable for number of children to load onto multiple latent variables, specifically those for children, other relatives, and friends. However, Glass et al. decided to allow each observed variable to load onto one latent variable only, so as to 'combine indicators into meaningful subscales in the most parsimonious manner'.

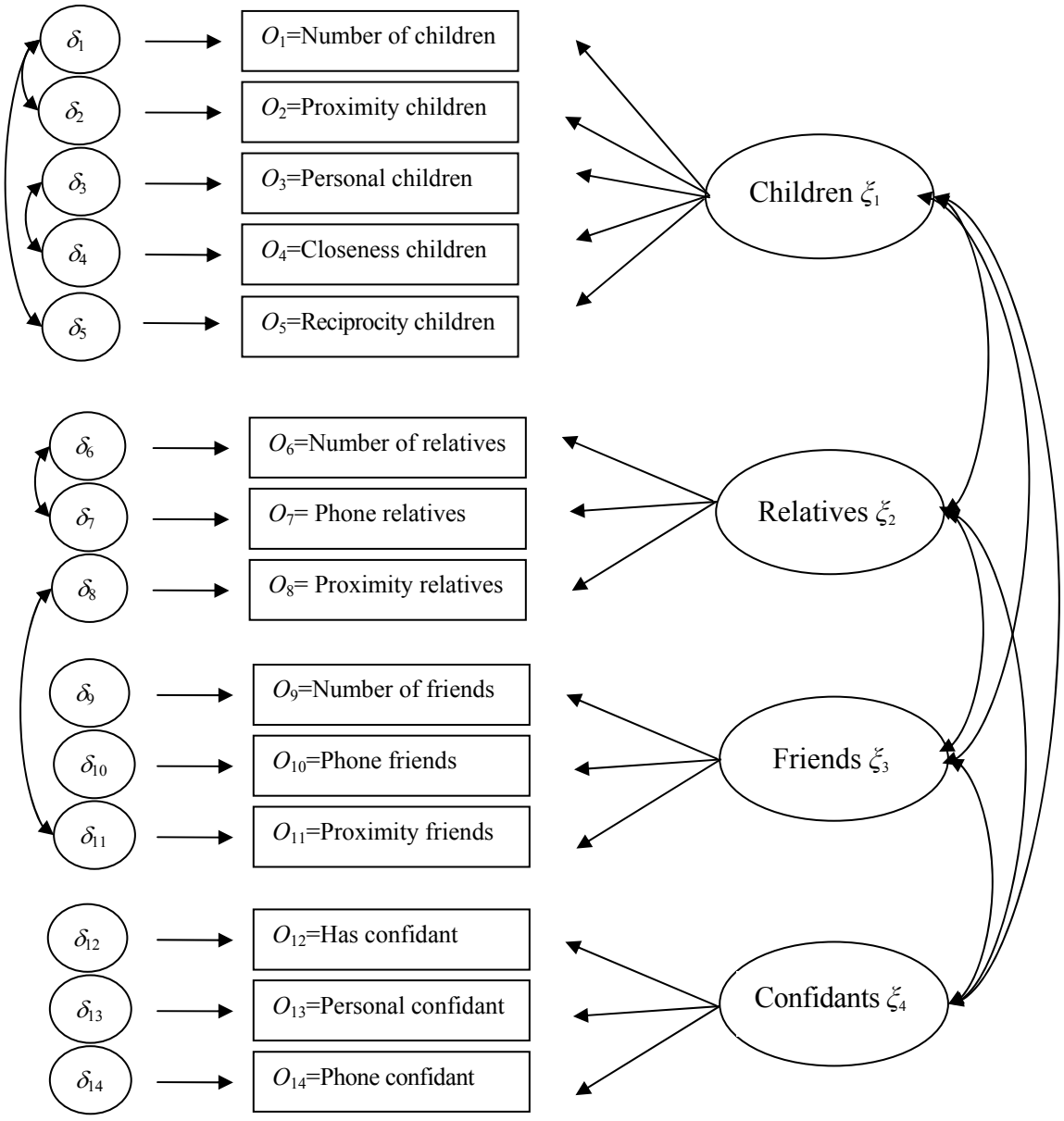
Following the derivation of the measurement model, Glass et al. calculated four social network variables by summing the observed variables that comprised a latent variable in the CFA model, thereby giving four specific social network variables. A total social network variable was then derived by Glass et al. through summing the four social

network variables. Thus the role of CFA in the research by Glass et al. was to confirm the groupings of the observed variables to the four types of social networks.

Table 5.1: Latent and observed variables in Glass et al. (1997)

Latent variable/observed variable	EPESE question
Children network	
O_1 No. living children	How many children, if any, have you had (including adopted)? How many are presently living?
O_2 Proximity of children	How many of your children live within several blocks or within the same metropolitan area of your house or apartment?
O_3 Personal contact with children	How many of your children do you see at least once a week or at least once a month?
O_4 Closeness with children	How many of your children do you feel very close to?
O_5 Reciprocity with children	Reciprocity with children: Number of items in which participant helps children divided by number of items in which participant received help from children.
Relatives network	
O_6 No. relatives seen monthly	Apart from your children, how many other relatives do you have that you feel close to? (People you feel at ease with, can talk to about private matters, or can call on for help?) How many of those relatives do you see at least once a month?
O_7 Phone contact with relatives	How many of those relatives do you correspond with whether by letter or by telephone a few times a year at least?
O_8 Proximity of relatives	How many of those relatives live in the [study city] or its suburbs?
Friends network	
O_9 No. close friends seen monthly	In general, how many close friends do you have? (People you feel at ease with, can talk to about private matters, and can call on for help?) How many of those friends do you see at least once a month?
O_{10} Phone contact with friends	How many of those friends do you correspond with either by letter or by telephone a few times a year at least?
O_{11} Proximity of friends	How many of those friends live in the [study city] or its suburbs?
Confidant network	
O_{12} Existence of confidant	Is there one special person you know that you feel very close and intimate with – someone you share confidences and feeling with, someone you feel you can depend on?
O_{13} Personal contact with confidant	How often do you get together with this person?
O_{14} Phone contact with confidant	How often do you talk on the telephone with him/her?

Figure 5.1: Final measurement model of Glass et al. (1997)



5.3 ALSA INITIAL MEASUREMENT MODEL

CFA was used to build a measurement model that described the specific social networks of ALSA participants. This built on from the work of Glass et al., although slightly different observed variables for ALSA were available on which to base the measures of social networks. Four observed variables that measured the number, proximity and frequency of phone and personal contact with children were hypothesised to be manifest variables of the underlying social network with children. The number of other relatives

and frequency of phone and personal contact with other relatives were hypothesised as indicators of the relatives social network. An analogous latent variable corresponding to friends social networks based on three observed variables was also hypothesised. Finally, a confidant social network was hypothesised. In the ALSA, there were fewer questions in the Wave 1 interview concerning confidants than there were for the other three network types. There were no confidant questions that concerned frequency of contact. Therefore the confidant network variables reflected the existence of confidants and whether a participant's confidant was their spouse.

Table 5.2 shows the questions used in the Wave 1 ALSA interview that were the basis for the observed variables used in the CFA. Descriptive statistics for the observed variables are also presented in Table 5.2.

Table 5.2: Components of social networks for children, other relatives, friends and confidants

Observed variable (X_i)	Latent variable (ξ_j)	Derived from ALSA question(s)	Coding	Mean (SD) of X_i	Frequency (%) of X_i
X_1 No. living children	ξ_1 Children	How many sons do or did you have? How many sons are still alive? How many daughters do or did you have? How many daughters are still alive?	0, 1, 2, 3, 4, ≥5	2.26 (1.44)	N/A
X_2 Proximity of children	ξ_1 Children	How many sons live within one hour's travel? How many daughters live within one hour's travel?	Proportion calculated out of number of children	0.61 (0.39)	N/A
X_3 Personal contact with children	ξ_1 Children	Think of your children and/or children-in-law who do not live with you. In the past twelve months, how often did you have personal contact with at least one of them?	Never (0)[2] Less than monthly (1) Less than weekly (2) Weekly (3) More than weekly (4)	2.63 (1.46)	17% 8% 14% 22% 39%
X_4 Phone contact with children	ξ_1 Children	Again thinking of your children and/or children-in-law who do not live with you. In the past twelve months, how often did you have phone contact with at least one of them?	Never (0) Less than monthly (1) Less than weekly (2) Weekly (3) More than weekly (4)	2.75 (1.53)	19% 4% 9% 20% 48%
X_5 No. other supportive relatives	ξ_2 Relatives	[Apart from any child or children, children-in-law or grandchildren][1] How many relatives do you have that you feel close to – that is, people you feel at ease with and talk to about private matters or can call on for help?	0, 1, 2, 3, 4, ≥5	1.27 (1.66)	N/A

(continued)

1: Text in square brackets used to denote discretionary dialogue spoken by interviewer if the participant had children, or responded affirmatively to the existence of a confidant
2: Coding of variables used in CFA; participants with no children coded as never for phone and personal contact with children

Table 5.2 (continued): Components of social networks for children, other relatives, friends and confidants

Observed variable (X_i)	Latent variable (ξ_j)	Derived from ALSA question(s)	Coding	Mean (SD) of X_i	Frequency (%) of X_i
X_6 Personal contact with relatives	ξ_2 Relatives	About how often do you spend some time with family or relatives who do not live with you?	Never (0) Less than monthly (1) Less than weekly (2) Weekly (3) More than weekly (4)	1.42 (1.26)	30% 30% 19% 13% 8%
X_7 Phone contact with relatives	ξ_2 Relatives	About how often do talk with family or other relatives on the telephone?	Never (0) Less than monthly (1) Less than weekly (2) Weekly (3) More than weekly (4)	1.70 (1.32)	23% 26% 22% 16% 13%
X_8 No. close friends	ξ_3 Friends	How many close friends do you have – that is, people you feel at ease with and talk to about private matters or can call on for help?	0,1,2,3,4, ≥5	2.23 (2.01)	N/A
X_9 Personal contact with friends	ξ_3 Friends	About how often do you spend some time with friends who do not live with you, that is you go to see them or they come to visit you or you go out to do things together?	Never (0) Less than monthly (1) Less than weekly (2) Weekly (3) More than weekly (4)	2.57 (1.34)	11% 13% 22% 20% 35%
X_{10} Phone contact with friends	ξ_3 Friends	About how often do you talk with friends on the telephone?	Never (0) Less than monthly (1) Less than weekly (2) Weekly (3) More than weekly (4)	2.41 (1.43)	16% 12% 22% 17% 33%

(continued)

1: Text in square brackets used to denote discretionary dialogue spoken by interviewer if the participant had children, or responded affirmatively to the existence of a confidant
2: Coding of variables used in CFA; participants with no children coded as never for phone and personal contact with children

Table 5.2 (continued): Components of social networks for children, other relatives, friends and confidants

Observed variable (X_i)	Latent variable (ξ_j)	Derived from ALSA question(s)	Coding	Mean (SD) of X_i	Frequency (%) of X_i
X_{11} Existence of confidant	ξ_4 Confidant	From all the people you know, including your spouse, relatives and friends, is there any one special person that you feel very close and intimate with – someone you share confidences and feelings with, someone you feel you can depend on?	No (0) Yes (1)	0.89 (0.32)	11% 89%
X_{12} Existence of another confidant	ξ_4 Confidant	Again, from all the people you know, is there any other special person that you feel very close and intimate with, someone else you share confidences and feelings with, someone else you feel you can depend on?	No (0) Yes (1)	0.66 (0.47)	34% 66%
X_{13} Confidant is spouse?	ξ_4 Confidant	[Yes to confidant][1] What is their relationship to you? [Yes to another confidant][1] What is their relationship to you?	None or not spouse (0) Spouse (1)	0.39 (0.49)	61% 39%

1: Text in square brackets used to denote discretionary dialogue spoken by interviewer if the participant had children, or responded affirmatively to the existence of a confidant
2: Coding of variables used in CFA; participants with no children coded as never for phone and personal contact with children

Thirteen observed variables were available for use in the CFA for the ALSA participants, in contrast to the fourteen variables used in the Glass et al. derivation. The correlation matrix for the thirteen observed variables is shown in Table 5.3. As this matrix demonstrates, the correlations were greatest among the observed variables hypothesised to load on to the same latent variables. There were two correlations greater than 0.20 between observed variables that were not hypothesised to load onto the same latent variables. Specifically, there was a correlation of 0.30 between X_7 (phone contact with relatives) and X_{10} (phone contact with friends), and a correlation of 0.21 between X_5 (number of relatives) and X_8 (number of friends). This suggested that a measurement model with a single latent variable for relatives and friends may also warrant investigation.

Taken together, the results from Glass et al and the observed correlation matrix for the ALSA data suggest that the four latent variable model was a plausible starting point for the CFA. As described in Section 4.2.5, the purpose of the CFA in this thesis was to validate the specific network structure for the ALSA data, and to check that averaging of the standardized observed variables in each latent variable was an adequate summary of the network variables.

Table 5.3: Correlation matrix for thirteen observed ALSA variables

	X_1	X_2	X_3	X_4	X_5	X_6	X_7	X_8	X_9	X_{10}	X_{11}	X_{12}	X_{13}
X_1	1.00												
X_2	0.31	1.00											
X_3	0.53	0.66	1.00										
X_4	0.49	0.49	0.69	1.000									
X_5	-0.09	-0.09	-0.04	-0.05	1.00								
X_6	-0.07	-0.07	-0.04	-0.05	0.38	1.00							
X_7	-0.08	-0.05	-0.02	0.08	0.43	0.61	1.00						
X_8	-0.01	-0.03	0.02	0.08	0.21	0.07	0.12	1.00					
X_9	0.01	-0.04	-0.02	0.06	0.05	0.11	0.15	0.35	1.00				
X_{10}	-0.06	-0.05	0.01	0.17	0.15	0.12	0.30	0.39	0.53	1.00			
X_{11}	0.03	0.03	0.06	0.07	0.09	0.01	0.04	0.11	0.06	0.08	1.00		
X_{12}	0.05	0.08	0.12	0.15	0.16	0.06	0.16	0.20	0.10	0.14	0.51	1.00	
X_{13}	0.14	0.08	0.11	0.15	-0.02	-0.05	-0.02	0.034	-0.04	-0.06	0.29	0.21	1.00

As already noted, there were some differences in the way the questions were worded in the EPESE and ALSA studies, and Table 5.4 shows the correspondence between the questions in the two studies.

EPESE and ALSA differed in how questions concerning frequency of contact with a particular type of network were framed. For example, in the EPESE interview schedule, participants were asked 'How many of your children do you see at least once a week or at least once a month?' In ALSA, participants were asked: 'Think of your children and/or children-in-law who do not live with you. In the past twelve months, how often did you have personal contact with at least one of them?' with possible responses on a five point scale ranging from *never* to *more than weekly*.

Table 5.4: Correspondence between ALSA and EPESE questions concerning social networks

Observed variable (X_i)	Latent variable ξ_j	Derived from ALSA question(s)	Corresponding EPESE questions
X_1 No. living children	ξ_1 Children	How many sons do or did you have? How many sons are still alive? How many daughters do or did you have? How many daughters are still alive?	How many children, if any, have you had (including adopted)? How many are presently living?
X_2 Proximity of children	ξ_1 Children	How many sons live within one hour's travel? How many daughters live within one hour's travel?	How many of your children live within several blocks or within the same metropolitan area of your house or apartment?
X_3 Personal contact with children	ξ_1 Children	Think of your children and/or children-in-law who do not live with you. In the past twelve months, how often did you have personal contact with at least one of them?	How many of your children do you see at least once a week or at least once a month?
X_4 Phone contact with children	ξ_1 Children	Again thinking of your children and/or children-in-law who do not live with you. In the past twelve months, how often did you have phone contact with at least one of them?	*No analogous question in EPESE*
		No analogous question in ALSA	How many of your children do you feel very close to?
		No analogous question in ALSA	Reciprocity with children: Number of items in which EPESE participant helps children divided by number of items in which participant received help from children.
X_5 No. other supportive relatives	ξ_2 Relatives	[Apart from any child or children, children-in-law or grandchildren] How many relatives do you have that you feel close to – that is, people you feel at ease with and talk to about private matters or can call on for help?	Apart from your children, how many other relatives do you have that you feel close to? (People you feel at ease with, can talk to about private matters, or can call on for help?) Not included in final Glass model as highly correlated with number of relatives seen ≥ monthly.

(continued)

Table 5.4 (continued): Correspondence between ALSA and EPESE questions concerning social networks

Observed variable (X_i)	Latent variable (ξ_j)	Derived from ALSA question(s)	Corresponding EPESE questions
X_6 Personal contact with relatives	ξ_2 Relatives	About how often do you spend some time with family or relatives who do not live with you?	How many of those relatives do you see at least once a month?
X_7 Phone contact with relatives	ξ_2 Relatives	About how often do talk with family or other relatives on the telephone?	How many of those relatives do you correspond with whether by letter or by telephone a few times a year at least?
		No analogous question in ALSA	How many of those relatives live in the [study city] or its suburbs?
X_8 No. close friends	ξ_3 Friends	How many close friends do you have – that is, people you feel at ease with and talk to about private matters or can call on for help?	In general, how many close friends do you have? (People you feel at ease with, can talk to about private matters, and can call on for help?)
X_9 Personal contact with friends	ξ_3 Friends	About how often do you spend some time with friends who do not live with you, that is you go to see them or they come to visit you or you go out to do things together?	How many of those friends do you see at least once a month?
X_{10} Phone contact with friends	ξ_3 Friends	About how often do you talk with friends on the telephone?	How many of those friends do you correspond with either by letter or by telephone a few times a year at least?
		No analogous question in ALSA	How many of those friends live in the [study city] or its suburbs?
X_{11} Existence of confidant	ξ_4 Confidant	From all the people you know, including your spouse, relatives and friends, is there any one special person that you feel very close and intimate with – someone you share confidences and feelings with, someone you feel you can depend on?	Is there one special person you know that you feel very close and intimate with – someone you share confidences and feeling with, someone you feel you can depend on?

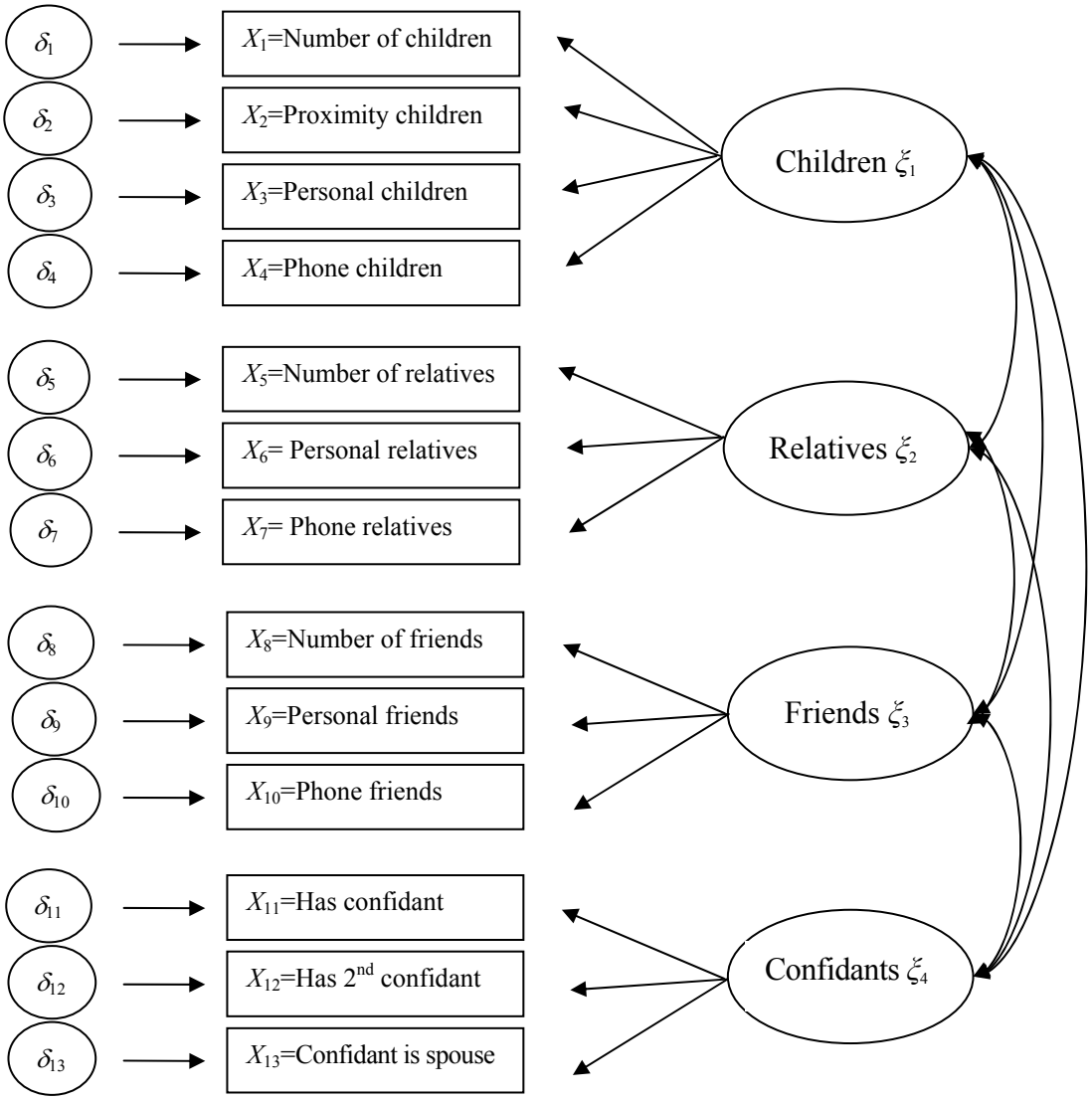
(continued)

Table 5.4 (continued): Correspondence between ALSA and EPESE questions concerning social networks

Observed variable (X_i)	Latent variable (ξ_j)	Derived from ALSA question(s)	Corresponding EPESE questions
X_{12} Existence of another confidant	ξ_4 Confidant	Again, from all the people you know, is there any other special person that you feel very close and intimate with, someone else you share confidences and feelings with, someone else you feel you can depend on?	*No analogous question in EPESE*
X_{13} Confidant is spouse?	ξ_4 Confidant	[Yes to confidant] What is their relationship to you? [Yes to another confidant] What is their relationship to you?	*No analogous question in EPESE*
		No analogous question in ALSA	How often do you get together with this person?
		No analogous question in ALSA	How often do you talk on the telephone with him/her?

Figure 5.2 illustrates the initial measurement model that was fit to the ALSA data. Maximum likelihood was used to derive parameter estimates for the initial measurement model. Research by Curran et al. (1996) has suggested that parameter estimation by maximum likelihood will perform reasonably well even in the presence of marked non-Normality.

Figure 5.2: Initial measurement model of specific social networks for ALSA



The test of H_0: $\Sigma = \Sigma(\theta)$ had an associated χ^2 statistic of 479.0 on 59 degrees of freedom, clearly significant at $P<0.001$. The CFI was 0.92, and the RMSEA was 0.07 (90% CI 0.07 – 0.08). While the CFI suggests the model fits moderately well compared to the independence model, the RMSEA is larger than desirable, and the test of H_0: $\Sigma = \Sigma(\theta)$ suggests significant model mis-fit. Therefore, modification of this initial measurement model was investigated.

5.4 MODIFIED MEASUREMENT MODEL

Table 5.5 displays the lower triangular matrix of standardized residuals for the initial measurement model. Sixteen standardized residuals were greater than 2.58 in absolute value. The term δ_4 (phone contact with children) was involved in five of these large standardized residuals, while δ_{11} and δ_{12} were each associated with four large standardized residuals.

Modification indices for several covariance terms in the initial model were large. Specifically, the modification index for the covariance between δ_4 and δ_7 (phone contact with relatives) was 39.0, δ_4 and δ_{10} (phone contact with friends) was 54.2, and δ_7 and δ_{10} was 40.7. The modification index that corresponded to the covariance between δ_5 (number of relatives) and δ_8 (number of friends) was 45.8. No other modification indices were greater than 30. Thus taken together, the standardized residuals and modification indices suggested that additional covariance terms that involved the error terms for phone contact (with children, relatives or friends) and the number of relatives and number of friends may lead to an improved model fit. δ_{11} and δ_{12} were associated with large standardized residuals but not modification indices, and therefore no additional covariance terms involving δ_{11} and δ_{12} were included in the modified model.

Table 5.5: Matrix of standardized residuals for initial measurement model[1]

	δ_1	δ_2	δ_3	δ_4	δ_5	δ_6	δ_7	δ_8	δ_9	δ_{10}	δ_{11}	δ_{12}	δ_{13}
δ_1	0.00												
δ_2	**-2.77**	0.00											
δ_3	-0.06	0.25	0.00										
δ_4	2.56	-0.35	-0.16	0.00									
δ_5	**-2.93**	**-2.87**	-0.70	-1.14	0.00								
δ_6	-1.93	-1.80	-0.67	-1.27	0.84	0.00							
δ_7	-2.15	-1.00	0.48	**3.73**	-0.44	0.06	0.00						
δ_8	-0.40	-1.13	0.29	**2.72**	**4.74**	-1.87	-0.81	0.00					
δ_9	0.01	-1.68	-0.96	2.00	-2.23	-1.59	-1.64	0.83	0.00				
δ_{10}	-2.55	-2.09	-0.18	**5.97**	0.60	-2.30	2.33	-0.57	0.08	0.00			
δ_{11}	-1.20	1.78	2.20	**4.04**	-1.46	**-2.99**	-2.21	0.14	**-3.21**	**-4.21**	0.00		
δ_{12}	-0.32	0.30	0.57	**2.77**	**3.96**	-0.34	**2.62**	**4.97**	0.41	1.26	-1.31	0.00	
δ_{13}	**4.10**	-1.79	-1.49	-0.34	1.45	-2.12	-1.65	1.46	-1.24	-1.11	1.51	-0.06	0.00

1: Bold values indicate residuals >2.58 in absolute value

Terms corresponding to the four specific covariances associated with the large modification indices in the initial model were included in a modified measurement model, illustrated in Figure 5.3. For the modified model, the test of H_0: $\Sigma = \Sigma(\theta)$ had an associated χ^2 statistic of 294.5 on 55 degrees of freedom, again statistically significant at P<0.001. The change from the initial model was 184.5 on 4 degrees of freedom, also statistically significant at P<0.001. The CFI was 0.95, and the RMSEA was 0.05 (90% CI 0.05 – 0.06). Thus the measures of model fit suggested a superior fit of the modified measurement model in comparison to the initial measurement model.

Figure 5.3: Modified measurement model of specific social networks

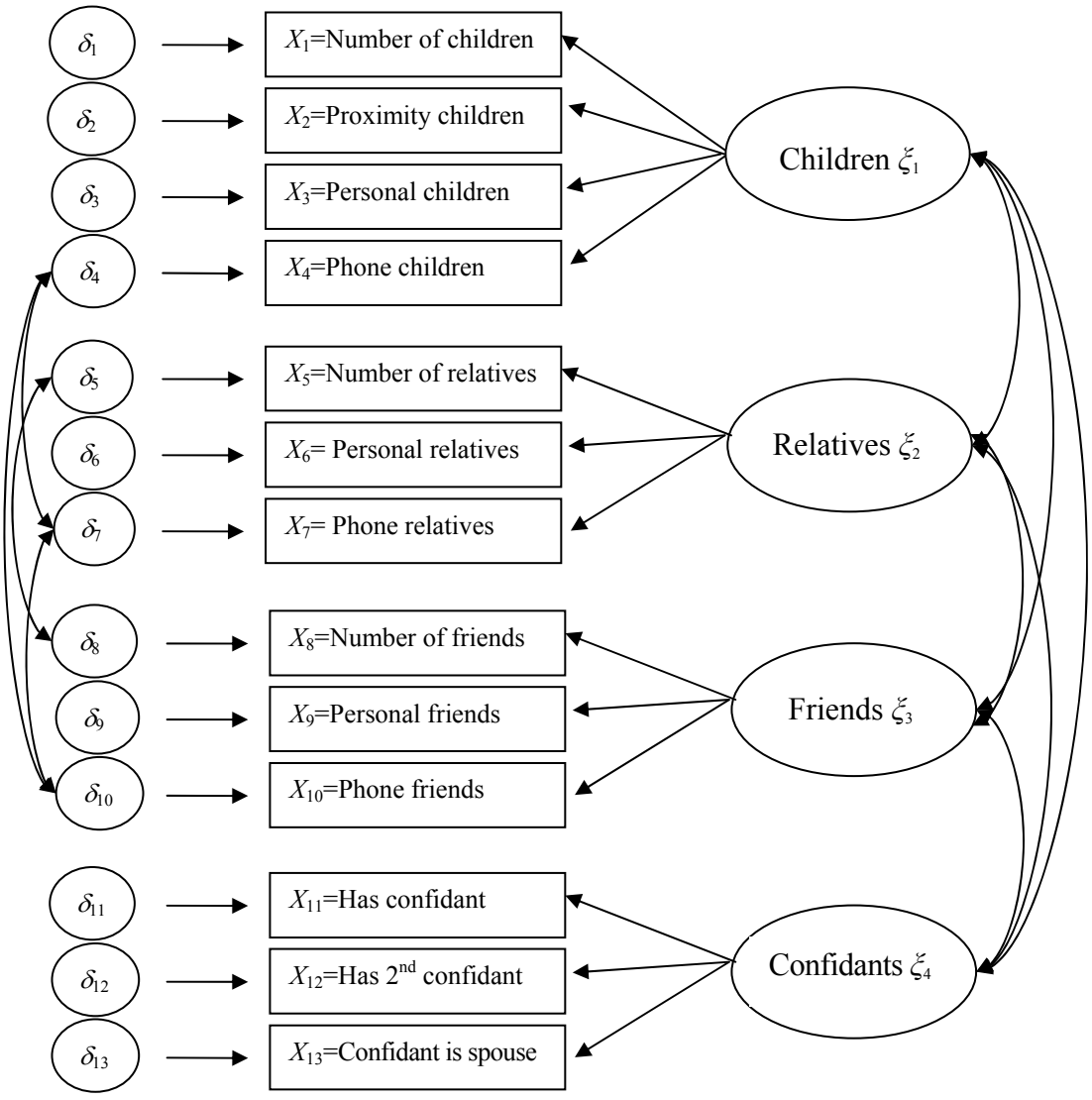


Table 5.6 displays the lower triangular matrix of standardized residuals for the modified measurement model. Thirteen standardized residuals were greater than 2.58 in absolute value. While there were still some large modification indices for this modified model, the maximum modification index in the revised model was 17.1, considerably less than the larger modification indices in the initial measurement model.

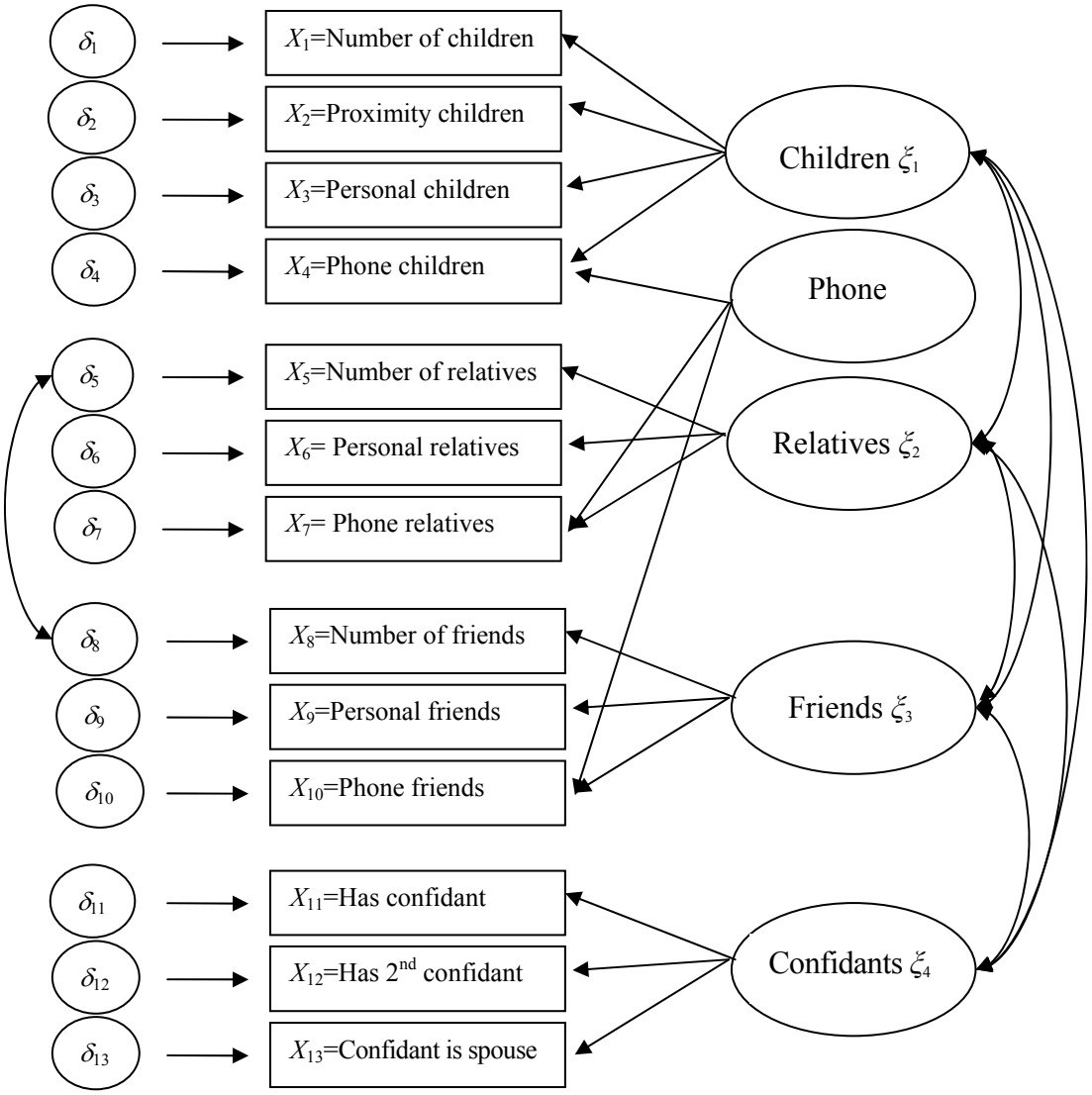
An equivalent formulation of the modified measurement model is presented in Figure 5.4. Instead of a measurement model with covariance terms between the errors for the phone items, a fifth latent variable corresponding to phone contact was included. Since this is an equivalent form of the modified measurement model, the measures of fit for the model in Figure 5.4 are identical to those for the model in Figure 5.3. The alternative formulation is included here as it provides a simpler conceptualization of the modified measurement model.

Table 5.6: Matrix of standardized residuals for modified measurement model[1]

	δ_1	δ_2	δ_3	δ_4	δ_5	δ_6	δ_7	δ_8	δ_9	δ_{10}	δ_{11}	δ_{12}	δ_{13}
δ_1	0.00												
δ_2	**-3.02**	0.00											
δ_3	-0.11	0.37	0.00										
δ_4	2.30	-0.51	-0.01	0.15									
δ_5	**-2.70**	**-2.58**	-0.33	-0.84	-0.01								
δ_6	-1.57	-1.37	-0.10	-0.81	-0.04	0.00							
δ_7	-1.82	-0.61	1.00	0.30	0.18	0.01	0.13						
δ_8	-0.23	-0.93	0.57	**2.95**	0.31	-0.97	0.76	0.10					
δ_9	0.23	-1.41	-0.59	2.30	-1.42	-0.69	0.08	-0.03	0.00				
δ_{10}	-2.31	-1.79	0.28	1.65	2.26	-0.32	0.96	0.75	0.75	0.70			
δ_{11}	-1.27	-1.86	-1.51	-0.40	1.64	-1.97	-1.15	1.60	-1.24	-0.51	0.00		
δ_{12}	-0.32	0.32	0.66	**2.80**	**4.19**	-0.11	**3.21**	**5.19**	0.50	1.98	-0.06	0.00	
δ_{13}	**4.07**	1.76	2.20	**4.03**	-1.37	**-2.91**	-1.97	0.22	**-3.20**	**-3.95**	1.30	-1.25	0.00

1: Bold values indicate residuals ≥2.58 in absolute value

Figure 5.4: Equivalent measurement model of specific social networks with phone latent variable



To further place the standardized residuals in context, the fitted correlations from the modified measurement model and observed correlations are displayed in Table 5.7. As this matrix shows, the differences between the fitted and observed correlations were generally modest.

Table 5.7: Matrix of observed and fitted correlations from modified measurement model. Fitted correlations shown beneath in italics. Bold values correspond to large standardized residuals[1].

	X_1	X_2	X_3	X_4	X_5	X_6	X_7	X_8	X_9	X_{10}	X_{11}	X_{12}
X_2	**0.31**											
	0.39											
X_3	0.53	0.66										
	0.54	*0.65*										
X_4	0.49	0.49	0.69									
	0.42	*0.51*	*0.70*									
X_5	**-0.09**	**-0.09**	-0.04	-0.04								
	-0.02	***-0.02***	*-0.03*	*-0.02*								
X_6	-0.07	-0.07	-0.04	-0.05	0.38							
	-0.03	*-0.03*	*-0.04*	*-0.03*	*0.38*							
X_7	-0.08	-0.05	-0.02	0.08	0.43	0.61						
	-0.03	*-0.03*	*-0.05*	*0.07*	*0.43*	*0.61*						
X_8	-0.01	-0.03	0.01	**0.08**	0.21	0.07	0.12					
	0.00	*0.00*	*0.00*	***0.00***	*0.21*	*0.09*	*0.10*					
X_9	0.01	-0.04	-0.02	0.06	0.05	0.11	0.15	0.35				
	0.00	*0.00*	*0.00*	*0.00*	*0.09*	*0.13*	*0.15*	*0.36*				
X_{10}	-0.06	-0.05	0.01	0.17	0.15	0.12	0.30	0.38	0.53			
	0.00	*0.00*	*0.00*	*0.13*	*0.09*	*0.14*	*0.27*	*0.37*	*0.51*			
X_{11}	0.03	0.03	0.06	0.07	0.09	0.01	0.04	0.11	0.05	0.08		
	0.06	*0.08*	*0.10*	*0.08*	*0.05*	*0.07*	*0.07*	*0.06*	*0.09*	*0.09*		
X_{12}	0.05	0.08	0.12	**0.15**	**0.16**	0.06	**0.16**	**0.20**	0.10	0.14	0.51	
	0.06	*0.07*	*0.10*	***0.08***	***0.04***	*0.06*	***0.07***	***0.06***	*0.08*	*0.09*	*0.51*	
X_{13}	**0.14**	0.08	0.11	**0.15**	-0.01	**-0.05**	-0.02	0.04	**-0.04**	**-0.06**	0.29	0.21
	0.03	*0.04*	*0.05*	***0.04***	*0.02*	***0.03***	*0.04*	*0.03*	***0.04***	***0.04***	*0.26*	*0.25*

1: Standardized residuals ≥2.58 in absolute value corresponding to Table 5.6

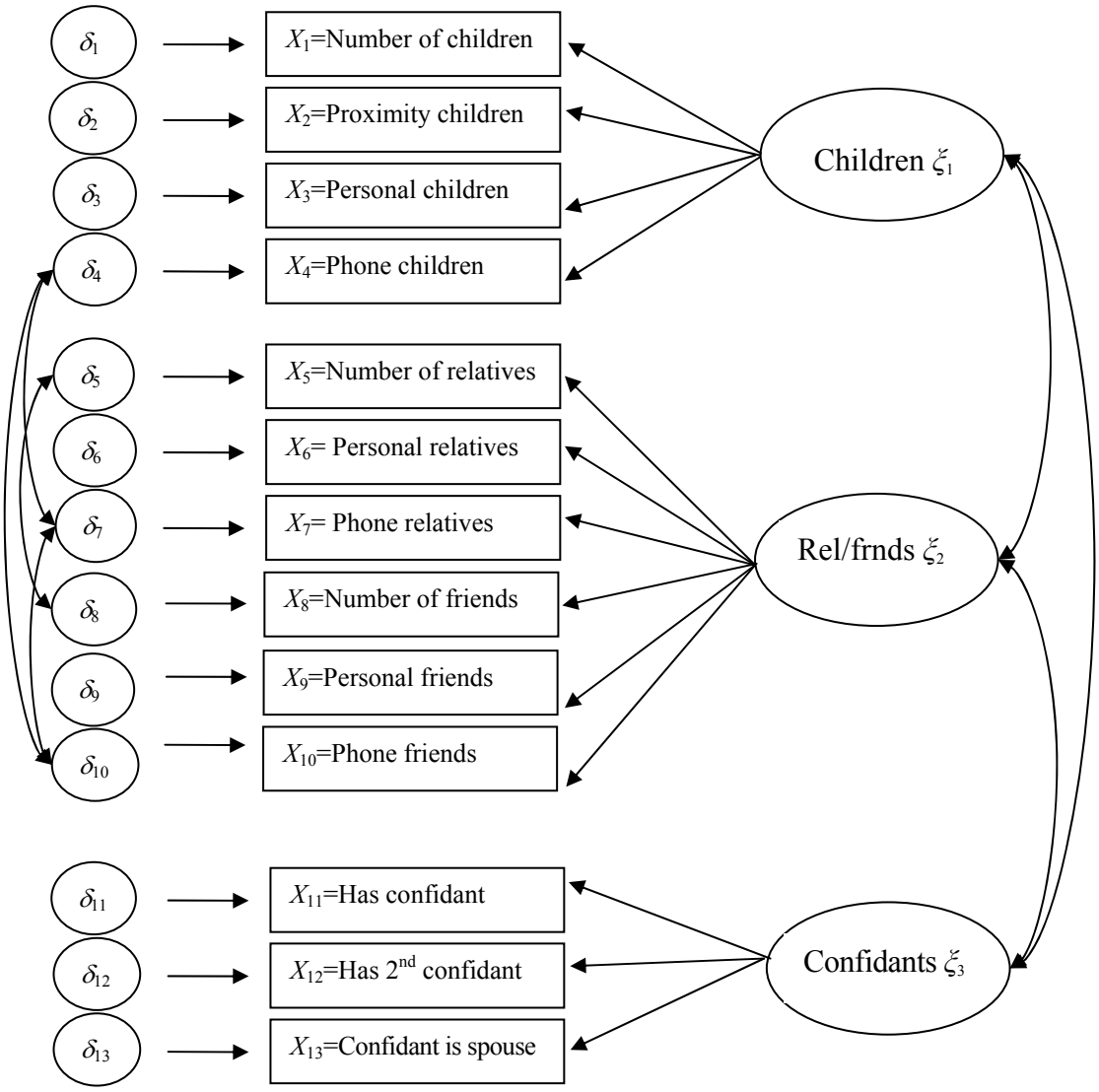
In summary, there was a significant change in the χ^2 statistic between the initial and modified measurement models, representing significant improvement in model fit of the modified model relative to the initial model. The other goodness of fit indices and the RMSEA also indicated the modified model was a better fit than the initial model. The discrepancy between the observed and fitted correlations did not show any obvious areas of model mis-fit. Thus the modified model offered a reasonable fit to the data.

5.5 COMPARISON WITH ALTERNATIVE MEASUREMENT MODEL

Although the results for the modified measurement model do not suggest a departure from the four factor model, there was an observed correlation of 0.30 between X_7 (phone contact with relatives) and X_{10} (phone contact with friends), and a correlation of 0.21 between X_5 (number of relatives) and X_8 (number of friends). Furthermore, three of the additional covariance terms in the modified model involve the relatives and friends latent variables. This suggests a simpler model with an amalgamated factor for friends and relatives may be satisfactory. Therefore, a model with three latent variables (children, relatives/friends, and confidants) and the same covariance structure as in the modified measurement model was fit. The path diagram for this simpler model is displayed in Figure 5.5.

For the model represented in Figure 5.5, the test of H_0: $\Sigma = \Sigma(\theta)$ had an associated χ^2 statistic of 976.5 on 58 degrees of freedom. The CFI was 0.82, and the RMSEA was 0.11, with associated 90% CI spanning 0.10 to 0.12. The lower triangular matrix of standardized residuals, shown in Table 5.8, indicates that 18 of 91 standardized residuals were greater than 2.58 in absolute value. There were particularly large standardized residuals, in excess of 12.0, for the covariances between δ_8 and δ_9, δ_9 and δ_{10} and δ_8 and δ_{10}.

Figure 5.5: Measurement model for three latent variables



Thus a simplification of the social networks model from four latent variables to three latent variables was not supported by the fit of the latter model. All of the measures of fit demonstrated a poorer fit of the three latent variable model than the modified measurement model illustrated in Figure 5.3.

Table 5.8: Matrix of standardized residuals for measurement model with three latent variables[1]

	δ_1	δ_2	δ_3	δ_4	δ_5	δ_6	δ_7	δ_8	δ_9	δ_{10}	δ_{11}	δ_{12}	δ_{13}
δ_1	0.00												
δ_2	**-3.01**	0.00											
δ_3	-0.10	0.36	0.00										
δ_4	2.36	-0.46	0.05	0.23									
δ_5	**-2.76**	**-2.65**	-0.42	-0.92	0.00								
δ_6	-1.68	-1.51	-0.29	-0.96	0.42	0.00							
δ_7	-1.89	-0.69	0.88	0.36	-0.24	0.22	0.00						
δ_8	-0.06	-0.72	0.86	**3.18**	0.00	-1.93	-0.64	0.00					
δ_9	0.43	-1.18	-0.26	2.55	-1.76	-0.97	-0.60	**12.02**	0.00				
δ_{10}	-1.97	-1.38	0.79	0.25	-0.06	**-3.21**	0.00	**12.50**	**17.59**	0.00			
δ_{11}	-1.24	-1.82	-1.46	-0.35	1.47	-2.10	-1.48	**3.35**	1.34	1.79	0.00		
δ_{12}	-0.19	0.47	0.86	**2.98**	**4.11**	-0.13	**3.01**	**6.89**	3.01	**4.20**	0.01	0.00	
δ_{13}	**4.09**	1.78	2.22	**4.06**	-1.44	**-2.98**	-2.12	1.06	-1.96	**-2.81**	0.91	-1.22	0.00

1: Bold values indicate residuals ≥2.58 in absolute value

The results for the different fitted models are summarized in Table 5.9. The ECVI and associated 90%CI for each of the models are also presented in Table 5.9, to aid in the comparison of the alternative measurement models. The fit of the modified measurement model appears satisfactory when compared with the initial measurement model and the three latent variable model.

Table 5.9: Summary of assessment of fit of alternative measurement models

	Three latent variables	**Initial four latent variables**	**Modified four latent variables**
H_0: $\Sigma = \Sigma(\theta)$	976.5 (58 df)	479.0 (59 df)	294.5 (55 df)
CFI	0.82	0.92	0.95
RMSEA (90%CI)	0.11 (0.10-0.12)	0.07 (0.07-0.08)	0.05 (0.05-0.06)
ECVI (90%CI)	0.75 (0.68-0.82)	0.39 (0.34-0.44)	0.26 (0.23-0.30)
# residuals≥2.58[1]	18	16	13

1: Number of standardized residuals ≥2.58

5.6 ADDITIONAL ASSESSMENT OF FIT OF MODIFIED MODEL

The factor loadings (both unstandardized and standardized) for the modified measurement model are presented in Table 5.10. The results suggest the coefficients of determination of the individual observed variables were moderate, with the reliability of the 'confidant is spouse' item in the confidants social network poor. The λ_{ij}^{s} values for the children, relatives, friends and confidants latent variables were, however, comparable in size to those reported by Glass et al., where the λ_{ij}^{s} ranged from 0.31 to 0.99. The composite reliabilities of the latent variables in the present study were 0.83 (for children social network), 0.74 (relatives social network), 0.69 (friends social network) and 0.63 (for confidant social network). The analogous figures reported by Glass et al. were 0.88 (children social network), 0.67 (relatives social network), 0.80 (friends social network) and 0.97 (confidant social network).

Table 5.10: Factor loadings and reliabilities for modified four latent variable measurement model

	λ_{ij}	se(λ_{ij})	λ^{s}_{ij}	Reliability $[\lambda^{s}_{ij}]^2$	Composite reliability
Children network					0.83
Number of children	0.73	0.04	0.57	0.32	
Proximity children	0.24	0.01	0.69	0.46	
Contact children	1.22	0.04	0.94	0.88	
Phone children	1.00	-	0.74	0.56	
Relatives network					0.74
Number of other relatives	0.78	0.05	0.52	0.27	
Contact relatives	0.86	0.05	0.74	0.54	
Phone relatives	1.00	-	0.83	0.70	
Friends network					0.69
Number of close friends	0.98	0.07	0.51	0.25	
Contact friends	0.92	0.06	0.70	0.51	
Phone friends	1.00	-	0.73	0.55	
Confidant network					0.63
Existence of confidant	1.34	0.14	0.73	0.51	
Existence of 2nd confidant	1.90	0.19	0.70	0.52	
Spouse is a confidant	1.00	-	0.35	0.12	

5.7 DERIVATION OF SOCIAL NETWORKS VARIABLES

Glass et al. calculated their specific network variables by summing the observed variables that made up each latent variable, arguing that their social network variables were essentially identical whether or not the λ_{ij} were used as weights. These authors appealed to work by Liang et al. (1990), who suggested that when a composite variable is created from variables with similar variances and factor loadings, then the difference in terms of reliability that arises from equal weighting versus differential weighting by λ_{ij} is very small. However, summing of the observed variables by Glass et al. resulted

in specific social network variables with different ranges, and these specific social network variables could not be compared easily with each other.

In this thesis, social network variables were estimated using three different methods, namely averaging of the standardized observed variables, differential weighting and the calculation of $\boldsymbol{E}(\xi \mid \mathbf{X})$, as described in Section 4.2.5. Averaging was carried out in preference to summing in the present study because there were four observed variables for the children social network and three observed variables for the relatives, friends and confidants social networks.

The pairwise correlations between the variables that arose from the three alternative methods of calculating each specific network variable for the ALSA data – that is, averaging, differential weighting, or $\boldsymbol{E}(\xi \mid \mathbf{X})$ – are displayed in Table 5.11. The minimum value of the resulting correlations was 0.947, demonstrating that there was little difference between the alternative methods of derivation. On this basis, the average of the standardized observed variables was used to calculate the four specific network variables for the ALSA data because it was the simplest method of derivation.

Table 5.11: Pairwise correlations between three alternative methods of derivation of network variables

	Averaging & Differential	**Averaging & $\boldsymbol{E}(\xi \mid \mathbf{X})$**	**Differential & $\boldsymbol{E}(\xi \mid \mathbf{X})$**
Children	0.975	0.957	0.958
Relatives	0.999	0.965	0.963
Friends	0.993	0.975	0.947
Confidants	0.980	0.954	0.960

A total social network variable was also calculated as the sum of the four specific social network variables. Each of the social network variables was also categorized according to its tertiles, resulting in variables with categories of lower, mid and upper for each social network type and the total social network variable. As described in Section 4.2.5, results for both the continuous and categorized versions of the social network variables and their effects on three health outcomes are presented in this thesis.

Table 5.12 presents summary statistics for the continuous versions of the social network variables overall, and broken down by age group and gender. The table shows that females had higher specific social network scores than males for relatives, friends and confidants social networks, but there was no significant difference between genders for children social networks nor the total social network variable. There were also statistically significant age effects, such that younger participants had higher social network scores for all four specific social networks and total social networks in comparison to participants who were older.

Table 5.12: Summary of social networks by age group and gender. Shown are means (standard deviations).

Characteristic	Children	Relatives	Friends	Confidants	Total
Overall	0.003 (0.796)	0.004 (0.795)	0.008 (0.774)	0.010 (0.736)	0.024 (1.736)
Gender					
Male	0.020 (0.757)	-0.059 (0.775)	-0.052 (0.776)	0.086 (0.770)	-0.005 (1.746)
Female	-0.027 (0.858)	0.108 (0.818)	0.108 (0.760)	-0.117 (0.654)	0.072 (1.719)
P-value[1]	0.269	<0.001	<0.001	<0.001	0.411
Age group					
70-74	0.117 (0.738)	0.084 (0.792)	0.184 (0.760)	0.111 (0.766)	0.495 (1.667)
75-79	0.089 (0.748)	0.038 (0.758)	0.071 (0.744)	0.053 (0.768)	0.252 (1.737)
80-84	-0.043 (0.820)	0.033 (0.845)	0.055 (0.751)	-0.005 (0.713)	0.040 (1.704)
85+	-0.141 (0.845)	-0.126 (0.774)	-0.252 (0.767)	-0.108 (0.680)	-0.628 (1.633)
P-value[2]	<0.001	<0.001	<0.001	<0.001	<0.001

1: P-value based on independent samples t-test
2: P-value based on one-way analysis of variance

5.8 SUMMARY

The purpose of the CFA in this thesis was to assess the applicability of Glass' model in the derivation of suitable social network scores for the ALSA data. The results of the CFA demonstrated that the four specific social network types of children, relatives, friends and confidants proposed by Glass et al. were tenable. Summary network scores for each of the social network types were derived by averaging the responses to the standardized observed variables in each of the latent variables. A total social network variable was also derived by summing together the four specific social network variables.

The effects of the specific and total social network variables on disability, residential care use and mortality were then investigated. These results are presented in the next three chapters.

6 THE EFFECTS OF SOCIAL NETWORKS ON DISABILITY

6.1 INTRODUCTION

This chapter describes the transitions between states of disability among ALSA participants, and the effects of social networks on these transitions. Two variables that measure disability are considered in this chapter, namely mobility disability and Nagi disability. Results are first presented in Section 6.2 for transitions in mobility disability status and transitions in Nagi disability status between each of the study waves, both overall and for each category of the total social networks variable. The main results concerning the effects of the specific and total social network variables on each of mobility disability and Nagi disability are presented in Section 6.3. The disability response variables were classified in two alternative ways, and the analyses used binary logistic regression (Section 6.3.1) or proportional odds and multinomial logistic regression (Section 6.3.2) depending on the classification. An investigation of the existence of threshold effects of total and specific social networks was also of interest, and to this end an additional set of analyses that examined such effects was conducted. These analyses are presented in Section 6.3.3. The chapter concludes with a discussion of the findings in Section 6.4.

6.2 TRANSITIONS IN DISABILITY STATUS

Transitions in mobility disability and Nagi disability occurred between each wave. Table 6.1 presents the observed proportion of participants who made transitions between the different states of mobility disability over Waves 1 to 6, classified according to the tertiles of the total social network variable as well as overall.

This table shows that, for example, the observed proportion of transitions from a state of no disability at Wave 2 to disability at Wave 3 was 0.17 for participants in the upper category of total social networks. Overall, the proportion continuing in a state of no disability from Wave 2 to Wave 3 was 0.79. The proportion of transitions from no mobility disability at one wave to disability at the subsequent wave appeared greatest for those in the lower category of total social networks, in which the proportions ranged from 0.15 to 0.31.

Conversely, the observed proportion of transitions from mobility disability to no disability at the next wave ranged from 0.07 to 0.34 for those participants in the upper category of total social networks, compared to a range of 0.04 to 0.16 for participants in the lower category of total social networks. In general, the proportion of participants who recovered from disability diminished over time, and this appeared to be the case regardless of social network category.

Table 6.2 shows the observed proportions of transitions between states of Nagi disability for the three total social network categories as well as overall. In general, the proportion of a transition to and from states of Nagi disability was greater than the corresponding proportions observed for mobility disability. The observed proportion of participants developing Nagi disability was also generally highest among those who were in the lower category of total social networks. Overall, the proportion of transitions from a state of no Nagi disability to disability at the following wave was between 0.24 and 0.43. For those in the lower category of total social networks, the proportion of transitions from a state of no disability to disability was 0.33 between Waves 1 and 2 and 0.51 between Waves 5 and 6. For participants in the upper total social network category, the proportion of participants who made a transition from no

disability to disability between Waves 1 and 2 was 0.23, while between Waves 5 and 6 the proportion was 0.39. The proportion of participants with recovery from Nagi disability to no disability at the subsequent wave was between 0.06 and 0.22 overall. The proportion of people who recovered appeared greater among the participants in the upper or mid total network categories than participants in the lower total social network category. As was observed for mobility disability, the proportion of the sample that recovered from Nagi disability decreased with each study wave.

Table 6.1: Transitions from each mobility state over six waves of ALSA for lower, mid and upper total social network categories and overall

Mobility status at Wave *j*-1	Wave *j*	Mobility status at Wave *j*			Total n
		No disability	Disability	Deceased	
No disability					
Lower	2	0.71	0.25	0.04	248
	3	0.81	0.15	0.04	203
	4	0.79	0.17	0.04	178
	5	0.53	0.31	0.16	144
	6	0.59	0.28	0.13	64
Mid	2	0.79	0.19	0.02	279
	3	0.79	0.19	0.03	242
	4	0.83	0.14	0.03	206
	5	0.70	0.18	0.12	178
	6	0.58	0.29	0.13	118
Upper	2	0.83	0.14	0.03	328
	3	0.79	0.17	0.04	299
	4	0.79	0.17	0.05	257
	5	0.77	0.15	0.08	210
	6	0.67	0.19	0.14	154
Overall	2	0.78	0.19	0.03	855
	3	0.79	0.17	0.04	744
	4	0.80	0.16	0.04	641
	5	0.68	0.20	0.12	532
	6	0.63	0.24	0.13	336
Disability					
Lower	2	0.16	0.68	0.15	182
	3	0.16	0.67	0.17	178
	4	0.14	0.67	0.19	150
	5	0.04	0.49	0.47	118
	6	0.06	0.34	0.60	97
Mid	2	0.24	0.68	0.08	154
	3	0.12	0.72	0.17	151
	4	0.14	0.70	0.16	150
	5	0.11	0.62	0.28	123
	6	0.10	0.48	0.42	96
Upper	2	0.34	0.58	0.08	122
	3	0.23	0.65	0.12	115
	4	0.17	0.72	0.11	124
	5	0.19	0.55	0.26	123
	6	0.07	0.56	0.38	88
Overall	2	0.24	0.65	0.11	458
	3	0.16	0.68	0.16	444
	4	0.15	0.69	0.16	424
	5	0.11	0.55	0.33	364
	6	0.08	0.46	0.47	281

Table 6.2: Transitions from each Nagi state over six waves of ALSA for lower, mid and upper total social network categories and overall

Nagi status at Wave *j*-1	Wave *j*	Nagi status at Wave *j*: No disability	Disability	Deceased	Total n
No disability					
Lower	2	0.60	0.33	0.07	156
	3	0.65	0.30	0.05	140
	4	0.66	0.31	0.03	116
	5	0.53	0.30	0.16	92
	6	0.39	0.51	0.10	49
Mid	2	0.73	0.24	0.02	166
	3	0.57	0.40	0.03	173
	4	0.74	0.24	0.02	122
	5	0.58	0.30	0.12	130
	6	0.44	0.46	0.10	81
Upper	2	0.74	0.23	0.03	213
	3	0.62	0.35	0.03	209
	4	0.77	0.19	0.03	151
	5	0.75	0.18	0.07	157
	6	0.46	0.39	0.15	123
Overall	2	0.70	0.27	0.04	535
	3	0.61	0.35	0.03	522
	4	0.73	0.24	0.03	389
	5	0.64	0.25	0.11	379
	6	0.44	0.43	0.12	253
Disability					
Lower	2	0.18	0.72	0.09	276
	3	0.12	0.75	0.13	245
	4	0.11	0.74	0.15	219
	5	0.07	0.56	0.37	175
	6	0.05	0.42	0.52	111
Mid	2	0.22	0.72	0.06	270
	3	0.09	0.79	0.12	225
	4	0.22	0.67	0.11	239
	5	0.12	0.66	0.22	178
	6	0.06	0.59	0.36	135
Upper	2	0.27	0.67	0.06	241
	3	0.14	0.77	0.09	206
	4	0.19	0.73	0.09	231
	5	0.17	0.62	0.21	176
	6	0.06	0.62	0.32	117
Overall	2	0.22	0.71	0.07	787
	3	0.12	0.77	0.12	676
	4	0.17	0.71	0.12	689
	5	0.12	0.61	0.26	529
	6	0.06	0.55	0.39	363

6.3 THE EFFECTS OF SOCIAL NETWORKS ON DISABILITY

6.3.1 Binary logistic regression model

In the investigation of the effects of social networks on disability, a series of nested binary logistic regression models were fit. The initial model considered was

$$\text{logit Pr}(Y_{ij} = 1 \mid Y_{ij\text{-}1} = y_{ij\text{-}1}) = \boldsymbol{x}_{ij}'\beta_0 + y_{ij\text{-}1}\boldsymbol{x}_{ij}'\alpha,$$

as described in Equation (4.1). In the present application, the $\boldsymbol{x}_{ij}$ consisted of the propensity score strata, age group at study entry (classified as 70-74, 75-79, 80-84 and ≥85 years), gender, Wave (coded with dummy variables as 2, 3, 4, 5 and 6), and the social network variable. The $y_{ij\text{-}1}$ term corresponded to either mobility disability status at Wave j-1 or Nagi disability status at Wave j-1, depending on which of the disability response variables was being analysed. Only states of no disability or disability were considered in the binary logistic regression models. Deaths were excluded along with missing responses.

Two way interactions between $y_{ij\text{-}1}$ and Wave, $y_{ij\text{-}1}$ and network, and $y_{ij\text{-}1}$ and the propensity scores were included in the initial model for each network variable, as was the two way interaction between Wave and network and the two way interaction between Wave and propensity score. While an interaction between the propensity score and the network variable could arise (plausibly due to interactions between individual covariates and the network variable), such interaction terms were not included in the initial model. This was because it was difficult to translate such an interaction into meaningful terms.

Backwards elimination was then applied to obtain a parsimonious model. The model fitting process for mobility disability and each network variable demonstrated that the initial model was not significantly different from a model which consisted of the social

network variable, $y_{ij\text{-}1}$, Wave, propensity score, age group, gender, $y_{ij\text{-}1}$ x Wave and $y_{ij\text{-}1}$ x propensity score, and this held for the categorized and continuous forms of the social network variable.

In modelling Nagi disability status, the final fitted model for the children, friends and confidants social network consisted of consisted of the social network variable, Wave, propensity score, age group, gender, $y_{ij\text{-}1}$, $y_{ij\text{-}1}$ x Wave and $y_{ij\text{-}1}$ x propensity score. For the relatives and total social network variables, the Wave x propensity score interaction was also statistically significant and was retained in the model. For the continuous version of the total social network variable, the difference between the initial model and sub-model which included the interactions between $y_{ij\text{-}1}$ x propensity score, $y_{ij\text{-}1}$ x Wave, and Wave x propensity score included was significant (P=0.031). This suggested that the inclusion of the interaction terms Wave x network and $y_{ij\text{-}1}$ x network may have improved the fit of this particular model. However, based on the overall results from fitting the models to the disability variables, a decision was made to enter no further terms for this specific model.

Table 6.3: Comparison of initial and final fitted model for mobility disability and Nagi disability status

		Mobility				Nagi			
Network variable		**Initial LL[1]**	**Final LL[2]**	**LR test**	**P-value**	**Initial**	**Final**	**LR test**	**P-value**
Categorical	Children	-2067.75	-2083.77	32.04	0.192	-2273.85	-2283.12	18.52	0.856
	df	49	23	26		49	23	26	
	Relatives	-2060.99	-2076.65	31.32	0.216	-2251.93	-2260.25[3]	16.63	0.083
	df	49	23	26		49	39	10	
	Friends	-2064.72	-2076.81	24.19	0.565	-2253.16	-2267.15	27.99	0.359
	df	49	23	26		49	23	26	
	Confidants	-2047.70	-2072.34	49.27	0.205	-2254.73	-2269.48	29.51	0.927
	df	73	31	42		73	31	42	
	Total	-2064.34	-2081.87	35.06	0.110	-2247.90	-2257.00[3]	18.21	0.052
	df	49	23	26		49	39	10	
Continuous	Children	-2073.90	-2083.74	19.69	0.541	-2276.25	-2283.38	14.27	0.858
	df	43	22	21		43	22	21	
	Relatives	-2064.49	-2078.20	27.43	0.157	-2258.34	-2262.56[3]	8.42	0.134
	df	43	22	21		43	38	5	
	Friends	-2066.59	-2076.63	20.09	0.516	-2258.77	-2267.40	17.26	0.695
	df	43	22	21		43	22	21	
	Confidants	-2054.27	-2073.12	37.70	0.437	-2253.20	-2269.30	32.20	0.693
	df	67	30	37		67	30	37	
	Total	-2070.63	-2082.34	23.44	0.321	-2250.30	-2256.43[3]	12.26	0.031
	df	43	22	21		43	38	5	

1: Initial log-likelihood for model including age group, gender, $y_{ij\text{-}1}$, Wave, propensity score, network variable, $y_{ij\text{-}1}$ x Wave, $y_{ij\text{-}1}$ x propensity score, Wave x propensity score, Wave x network, $y_{ij\text{-}1}$ x network.

2: Final model included age group, gender, $y_{ij\text{-}1}$, Wave, propensity score, network variable, $y_{ij\text{-}1}$ x Wave, and $y_{ij\text{-}1}$ x propensity score.

3: Final model included age group, gender, $y_{ij\text{-}1}$, Wave, propensity score, network variable, $y_{ij\text{-}1}$ x Wave, $y_{ij\text{-}1}$ x propensity score, and Wave x propensity score.

The final fitted models that were determined through this process controlled for the effects of other variables on disability status, through the inclusion of the propensity scores, age group, gender, Wave, prior disability status and key interactions in the models, apart from the social network variables. Each of these final fitted models was compared to a nested model in which the social network variable was excluded, so that the effect of each social network variable on mobility disability and Nagi disability could be ascertained using LR tests.

The effects of social networks in the binary logistic regression analyses for mobility disability and Nagi disability are presented in Tables 6.4 and 6.5. In Table 6.4, the results for the categorization of each social network variable are given. In Table 6.5, the effect of each social network variable when considered as a continuous variable is presented.

As Table 6.4 demonstrates, there was a significant protective effect of the relatives network for mobility disability. Participants in the upper relatives social network category compared to the lower category were 23 per cent less likely to make a transition to a state of mobility disability over the course of the follow-up period (OR 0.77; 95%CI 0.62 – 0.96), compared to an individual with the same age group, gender, propensity score stratum. The effects of networks with children, friends and confidants were not statistically significant in the binary analyses of mobility disability. In addition, the effect of total social networks was not statistically significant.

Networks with relatives also had a significant effect on Nagi disability (Table 6.4). For the upper category of relatives social networks compared to the lower category, the odds ratio was 0.76 (95%CI 0.62 – 0.93). Similar to the results for mobility disability, there

was no effect of networks with children, friends, or confidants upon Nagi disability in any of the binary analyses. The effect of total social networks on Nagi disability was also non-significant.

Table 6.4: Effects of categorized social network variables on transitions to mobility disability and Nagi disability

		Mobility disability[1]			Nagi disability[1]		
Network	**Category**	**OR**	**95%CI**	**P-value[2]**	**OR**	**95%CI**	**P-value**
Children	0 – 33⅓rd	1.00		0.865	1.00		0.765
	33⅓ – 66⅔rd	1.04	0.86 – 1.27		0.93	0.77 – 1.13	
	66⅔ – 100th	1.05	0.86 – 1.28		0.96	0.80 – 1.16	
Relatives	0 – 33⅓rd	1.00		0.021	1.00		0.007
	33⅓ – 66⅔rd	0.96	0.79 – 1.17		0.97	0.80 – 1.16	
	66⅔ – 100th	0.77	0.62 – 0.96		0.76	0.62 – 0.93	
Friends	0 – 33⅓rd	1.00		0.276	1.00		0.153
	33⅓ – 66⅔rd	1.00	0.82 – 1.22		1.03	0.85 – 1.25	
	66⅔ – 100th	0.88	0.70 – 1.08		0.88	0.73 – 1.06	
Confidants	0 – 33⅓rd	1.00		0.438	1.00		0.837
	33⅓ – 66⅔rd	0.89	0.73 – 1.08		0.98	0.81 – 1.19	
	66⅔ – 100th	0.91	0.74 – 1.12		0.95	0.79 – 1.14	
Total	0 – 33⅓rd	1.00		0.226	1.00		0.207
	33⅓ – 66⅔rd	0.96	0.79 – 1.18		0.95	0.79 – 1.16	
	66⅔ – 100th	0.85	0.69 – 1.04		0.85	0.70 – 1.03	

1: 4455 observations from 1298 participants for mobility disability and 4501 observations from 1302 participants for Nagi disability
2: P-value based on change in -2 x log-likelihood

The findings from the analyses that included the social network variables as continuous variables were largely consistent with the results for the categorized variables (Table 6.5). For social networks with relatives, there were significant protective effects for mobility disability (OR 0.90; 95%CI 0.81 – 1.00) and Nagi disability (OR 0.89; 95%CI

0.81 – 0.98). However, these analyses showed that the effect of total social networks was significant for Nagi disability (OR 0.95; 95%CI 0.91 – 1.00) but not for mobility disability. Since the total social networks variable was derived by summing the four specific social network variables, this result appears largely due to the inclusion of the relatives social network in the derivation of the total social network variable. The analyses also suggested marginally significant effects of the friends social networks on disability for both mobility disability (OR 0.91; 95%CI 0.82 – 1.02) and Nagi disability (OR 0.91; 95%CI 0.82 – 1.01).

Table 6.5: Effects of continuous social network variables on transitions to mobility disability and Nagi disability

	Mobility[1]			Nagi[1]		
Network	**OR**	**95%CI**	**P-value[2]**	**OR**	**95%CI**	**P-value**
Children	1.03	0.93 – 1.15	0.558	1.00	0.90 – 1.10	0.927
Relatives	0.90	0.81 – 1.00	0.032	0.89	0.81 – 0.98	0.020
Friends	0.91	0.82 – 1.02	0.086	0.91	0.82 – 1.01	0.071
Confidants	0.98	0.88 – 1.11	0.781	0.96	0.86 – 1.06	0.396
Total	0.96	0.92 – 1.01	0.154	0.95	0.91 – 1.00	0.038

1: 4455 observations from 1298 participants for mobility disability and 4501 observations from 1302 participants for Nagi disability
2: P-value based on change in -2 x log-likelihood

In summary, the binary logistic regression analyses suggested that better relatives networks are significantly associated with a decrease in the risk of transition to mobility disability and Nagi disability.

6.3.2 Proportional odds and multinomial logistic regression

At each of Waves 2 through 6, some participants had died. Excluding death as a possible category of response may give a misleading impression concerning the effects of social networks on disability. Thus death was included as an (absorbing) state of

disability in the classification of disability status. The proportional odds regression model

$$\text{logit Pr}(Y_{ij} \le b \mid \boldsymbol{Y}^{*}_{ij\text{-}1} = \boldsymbol{y}^{*}_{ij\text{-}1}) = \theta_b + \sum_{k=0}^{1} \alpha_{kb} y^{*}_{ij\text{-}1;k} + \boldsymbol{x}_{ij}'\beta + \boldsymbol{x}_{ij}' \sum_{k=0}^{1} \gamma_k y^{*}_{ij\text{-}1;k}$$

described in Equation (4.4) was fit initially to the data. In the present application, the $\boldsymbol{x}_{ij}$ term consisted of age group, gender, propensity score stratum, Wave, and the social network variable. The five two-way interactions, described in Section 6.3.1, were also included in the fitted proportional odds regression model. The $y_{ij\text{-}1}$ term corresponded to either mobility disability status at Wave j-1 or Nagi disability status at Wave j-1, depending on which of the disability response variables was under consideration.

To assess the assumption of proportional odds, the changes in -2 x log-likelihood between the generalized ordinal logit and the proportional odds model were calculated for each social network variable (as described in Section 4.3), with each model including the aforementioned terms. The resulting likelihood ratio tests are summarized in Table 6.6. For each of the social network variables and for both mobility disability and Nagi disability, there was a significant difference between the generalized ordinal logit and the proportional odds model ($P<0.001$ in each case), indicating violation of the proportionality assumption. Therefore, proportional odds models were not considered any further in the analyses of disability.

Table 6.6: Summary of likelihood ratio tests between generalized ordinal logit model and proportional odds model for mobility disability and Nagi disability

		Categorical		Continuous	
Disability variable	**Network**	**LR**	**df**	**LR**	**df**
Mobility[1]	Children	370.9	49	363.2	43
	Relatives	375.1	49	370.8	43
	Friends	351.7	49	348.1	43
	Confidants	367.3	73	360.2	67
	Total	364.3	49	355.3	43
Nagi[2]	Children	321.4	49	313.6	43
	Relatives	327.0	49	317.1	43
	Friends	319.9	49	309.6	43
	Confidants	335.8	73	329.8	67
	Total	330.3	49	327.8	43

1: 5079 observations from 1391 participants for mobility disability
2: 5122 observations from 1393 participants for Nagi disability

Multinomial logistic regression models of the form

$$\text{logit Pr}(Y_{ij} = b \mid Y_{ij\text{-}1} = y_{ij\text{-}1}) = \boldsymbol{x}_{ij}'\beta_b + y_{ij\text{-}1}\boldsymbol{x}_{ij}'\alpha_b$$

as described in Equation (4.5) were therefore fit to the data. As for the simpler binary case, the initial models fit for each disability variable and each social network variable included age group, gender, propensity score stratum, Wave, the social network variable under consideration, $y_{ij\text{-}1}$, and five two-way interaction terms. To recap, these interaction terms were $y_{ij\text{-}1}$ x Wave, $y_{ij\text{-}1}$ x network, $y_{ij\text{-}1}$ x propensity score, Wave x network, and Wave x propensity score.

Backwards elimination was again undertaken for the multinomial logistic regression models to obtain parsimonious fitted models. Age group, gender, propensity score, Wave, network variable, $y_{ij\text{-}1}$, $y_{ij\text{-}1}$ x Wave and $y_{ij\text{-}1}$ x propensity score were significant

and retained in all of the final fitted models. In the model fitting process for the total social network for mobility disability and Nagi disability, the Wave x propensity score interaction was also significant and retained in the final fitted model. This interaction term was also necessary in the final fitted models for the continuous relatives social network variables for both mobility disability and Nagi disability, for the categorized confidants social network variable for mobility disability, and for the categorized relatives social network variable for Nagi disability. LR tests were then undertaken to test the effect of each social network variable on mobility disability and Nagi disability.

The effects of the categorized social network variables on mobility disability are presented in Table 6.7. As the results show, there was a marginally significant protective effect of relatives social networks against transition to mobiliy disability and death overall, with the odds ratio for disability for those in the upper compared to the lower category equal to 0.83 (95%CI 0.67 – 1.02). Networks with children, friends, confidants and total social networks did not have a significant effect on transitions to mobility disability. Also of note from Table 6.7 is the apparent protective effect of networks with friends and networks with confidants against mortality, as well as the protective effect of total networks against mortality. More detailed analyses concerning the effects of social networks on survival are presented in Chapter 8 of this thesis.

Table 6.7: Effects of categorized social network variables in multinomial logistic regressions for mobility disability[1]

Network	Category	OR	95%CI	P-value[4]
Children[2]	*Disability*			0.685
	33⅓ – 66⅔rd	1.06	0.87 – 1.28	
	66⅔ – 100th	1.07	0.89 – 1.30	
	Dead			
	33⅓ – 66⅔rd	0.95	0.73 – 1.23	
	66⅔ – 100th	0.89	0.69 – 1.16	
Relatives[2]	*Disability*			0.060
	33⅓ – 66⅔rd	0.98	0.81 – 1.19	
	66⅔ – 100th	0.83	0.67 – 1.02	
	Dead			
	33⅓ – 66⅔rd	0.92	0.71 – 1.18	
	66⅔ – 100th	1.06	0.80 – 1.40	
Friends[2]	*Disability*			0.008
	33⅓ – 66⅔rd	1.06	0.87 – 1.28	
	66⅔ – 100th	0.90	0.74 – 1.10	
	Dead			
	33⅓ – 66⅔rd	0.90	0.70 – 1.15	
	66⅔ – 100th	0.63	0.48 – 0.83	
Confidants[3]	*Disability*			0.037
	33⅓ – 66⅔rd	0.89	0.74 – 1.08	
	66⅔ – 100th	0.92	0.75 – 1.13	
	Dead			
	33⅓ – 66⅔rd	0.77	0.59 – 1.00	
	66⅔ – 100th	0.66	0.51 – 0.87	
Total[3]	*Disability*			0.136
	33⅓ – 66⅔rd	0.97	0.80 – 1.18	
	66⅔ – 100th	0.89	0.73 – 1.09	
	Dead			
	33⅓ – 66⅔rd	0.75	0.58 – 0.98	
	66⅔ – 100th	0.79	0.60 – 1.03	

1: 5079 observations from 1391 participants for mobility disability

2: Final model included age group, gender, $y_{ij\text{-}1}$, Wave, propensity score, network variable, $y_{ij\text{-}1}$ x Wave, and $y_{ij\text{-}1}$ x propensity score

3: Final model included age group, gender, $y_{ij\text{-}1}$, Wave, propensity score, network variable, $y_{ij\text{-}1}$ x Wave, $y_{ij\text{-}1}$ x propensity score, Wave x propensity score

4: P-value based on change in -2 *x* log-likelihood

Figure 6.1 presents an alternative view of the analyses summarized in Table 6.7, demonstrating the effects for friends, confidants and total social networks on survival as well as the lack of an effect for the children social networks on disability.

Figure 6.1: Summary of multinomial logistic regressions of effects of social networks on mobility disability

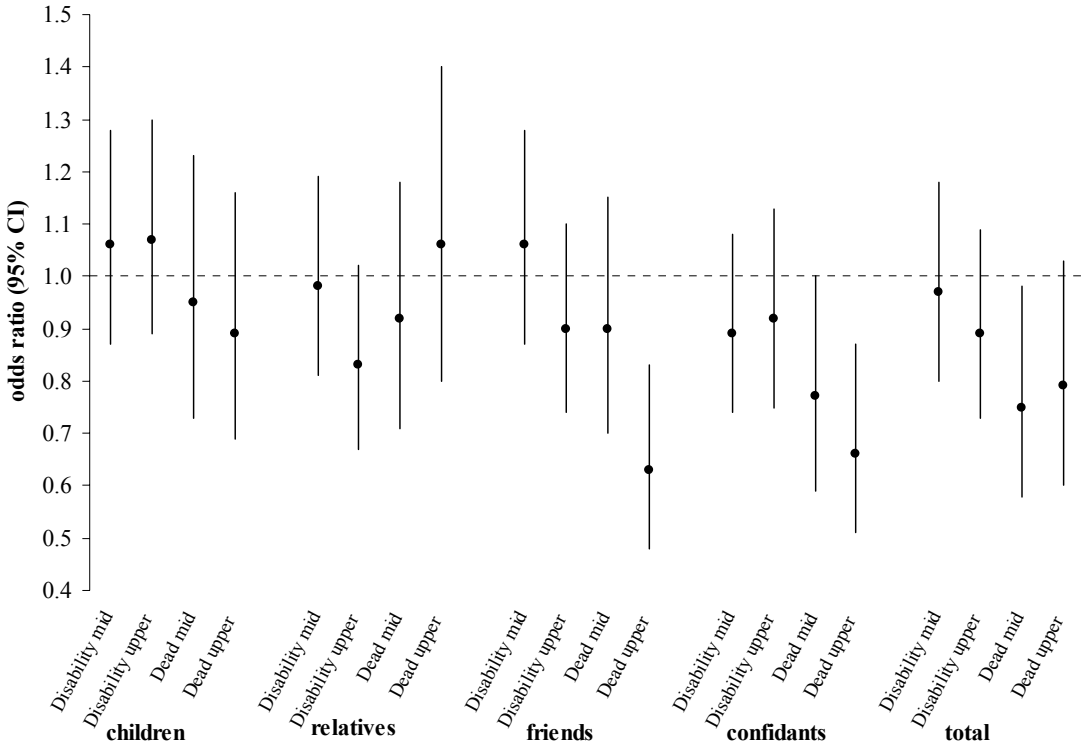


Table 6.8 summarizes the effects when the social network variables were included as continuous variables in the models. As was demonstrated for the categorized network variables, there were significant effects of the friends social networks and confidants social networks on mobility disability, apparently related to their effects on risk of death. Relatives social networks (OR 0.93; 95%CI 0.84 – 1.03) and total social networks (OR 0.98; 95%CI 0.93 – 1.03) again showed a marginally significant protective effect against developing mobility disability. The effect of children social networks on mobility disability was not statistically significant.

Table 6.8: Effects of social networks as continuous variables in multinomial logistic regression for mobility disability

Social network	Outcome state[1]	OR	95%CI	P-value[4]
Children[2]	No disability	1.00		0.548
	Disability	1.03	0.93 – 1.15	
	Dead	0.97	0.84 – 1.11	
Relatives[3]	No disability	1.00		0.086
	Disability	0.93	0.84 – 1.03	
	Dead	1.05	0.92 – 1.20	
Friends[2]	No disability	1.00		0.001
	Disability	0.93	0.84 – 1.03	
	Dead	0.76	0.66 – 0.88	
Confidants[2]	No disability	1.00		0.021
	Disability	0.99	0.88 – 1.10	
	Dead	0.82	0.71 – 0.95	
Total[3]	No disability	1.00		0.088
	Disability	0.98	0.93 – 1.03	
	Dead	0.93	0.87 – 0.99	

1: 5079 observations from 1391 participants
2: Final model included age group, gender, $y_{ij\text{-}1}$, Wave, propensity score, network variable, $y_{ij\text{-}1}$ x Wave, and $y_{ij\text{-}1}$ x propensity score
3: Final model included age group, gender, $y_{ij\text{-}1}$, Wave, propensity score, network variable, $y_{ij\text{-}1}$ x Wave, $y_{ij\text{-}1}$ x propensity score, Wave x propensity score
4: P-value based on change in -2 x log-likelihood

Table 6.9 summarizes the effects of the categorized social network variables in multinomial logistic regression models for Nagi disability. The results were similar, although stronger, to those observed for mobility disability. The upper category of networks with relatives had a significant protective effect against developing Nagi disability in comparison with the lower relatives category (OR 0.79; 95%CI 0.65 – 0.96). As was demonstrated for mobility disability, participants in the upper category of friends networks had a lower risk of death than participants in the lower category of friends networks. A similar, albeit marginally significant, effect was noted for confidants and for the mid category of total social networks. The effect of social networks with children was not statistically significant.

Table 6.9: Effects of categorized social network variables in multinomial logistic regressions for Nagi disability[1]

Network	Category	OR	95%CI	P-value[4]
Children[2]	*Disability*			0.917
	33⅓ – 66⅔rd	0.93	0.77 – 1.12	
	66⅔ – 100th	0.97	0.81 – 1.17	
	Dead			
	33⅓ – 66⅔rd	0.90	0.69 – 1.17	
	66⅔ – 100th	0.93	0.71 – 1.22	
Relatives[3]	*Disability*			0.021
	33⅓ – 66⅔rd	0.98	0.82 – 1.18	
	66⅔ – 100th	0.79	0.65 – 0.96	
	Dead			
	33⅓ – 66⅔rd	0.99	0.76 – 1.28	
	66⅔ – 100th	1.02	0.77 – 1.37	
Friends[2]	*Disability*			0.009
	33⅓ – 66⅔rd	1.03	0.85 – 1.25	
	66⅔ – 100th	0.89	0.73 – 1.07	
	Dead			
	33⅓ – 66⅔rd	0.90	0.69 – 1.17	
	66⅔ – 100th	0.63	0.48 – 0.83	
Confidants[2]	*Disability*			0.063
	33⅓ – 66⅔rd	0.98	0.81 – 1.18	
	66⅔ – 100th	0.94	0.78 – 1.13	
	Dead			
	33⅓ – 66⅔rd	0.79	0.61 – 1.02	
	66⅔ – 100th	0.68	0.52 – 0.90	
Total[3]	*Disability*			0.075
	33⅓ – 66⅔rd	0.96	0.79 – 1.16	
	66⅔ – 100th	0.85	0.70 – 1.06	
	Dead			
	33⅓ – 66⅔rd	0.74	0.56 – 0.97	
	66⅔ – 100th	0.76	0.57 – 1.01	

1: 5122 observations from 1393 participants for Nagi disability
2: Final model included age group, gender, $y_{ij\text{-}1}$, Wave, propensity score, network variable, $y_{ij\text{-}1}$ x Wave, and $y_{ij\text{-}1}$ x propensity score
3: Final model included age group, gender, $y_{ij\text{-}1}$, Wave, propensity score, network variable, $y_{ij\text{-}1}$ x Wave, $y_{ij\text{-}1}$ x propensity score, Wave x propensity score
4: P-value based on change in -2 x log-likelihood

Figure 6.2 presents an alternative way of viewing the effects of the categorized social network variables on Nagi disability. The figure demonstrates the significant effect of relatives social networks on Nagi disability. Figure 6.2 also illustrates the significant effects of friends, confidants and total social networks on survival as well as the lack of an effect for the children social networks on Nagi disability.

Figure 6.2: Summary of multinomial logistic regressions of effects of social networks on Nagi disability

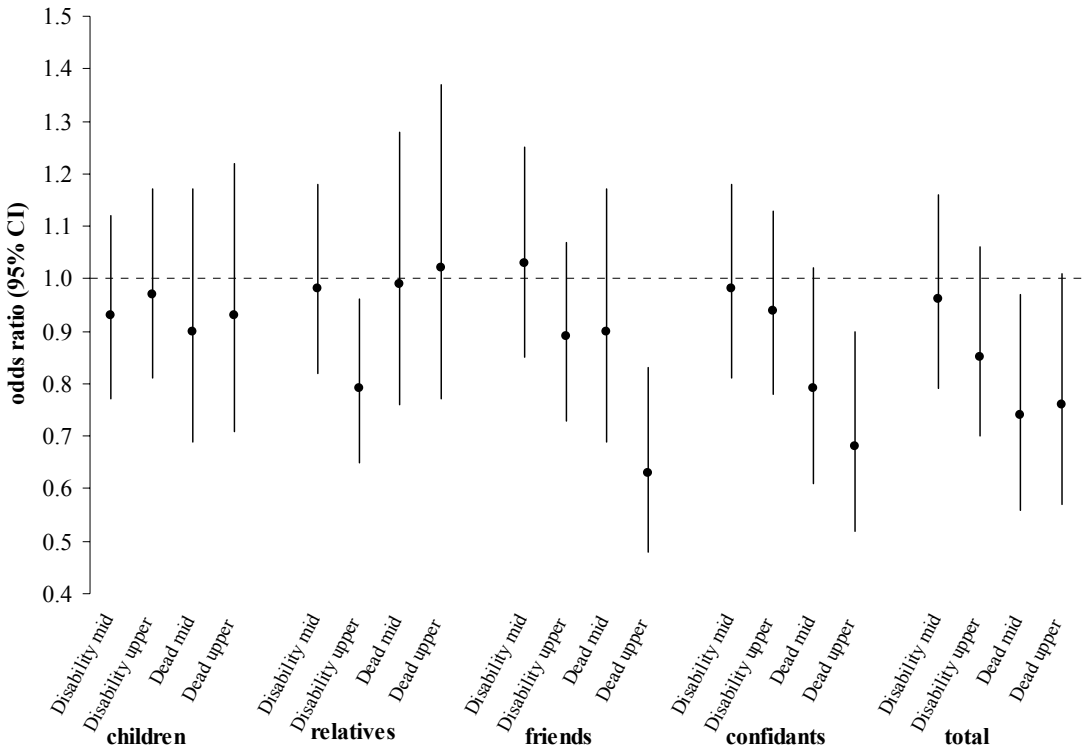


Table 6.10 summarizes the effects of the social network variables when included as continuous variables in the analyses of transitions in Nagi disability. The results showed that relatives networks appeared to protect against transitions to Nagi disability (OR 0.91; 95%CI 0.83 – 1.00), as did total social networks (OR 0.96; 95%CI 0.91 – 1.00). Significant effects of friends social networks and confidant social networks were

also observed, due to the apparent protective effects of these specific social networks against mortality.

Table 6.10: Effects of social networks as continuous variables in multinomial logistic regression for Nagi disability

Social network	Outcome state[1]	OR	95%CI	P-value[4]
Children[2]	No disability	1.00		0.834
	Disability	1.00	0.90 – 1.10	
	Dead	0.96	0.83 – 1.11	
Relatives[3]	No disability	1.00		0.021
	Disability	0.91	0.83 – 1.00	
	Dead	1.02	0.89 – 1.17	
Friends[2]	No disability	1.00		0.001
	Disability	0.92	0.83 – 1.02	
	Dead	0.76	0.66 – 0.87	
Confidants[2]	No disability	1.00		0.049
	Disability	0.96	0.86 – 1.06	
	Dead	0.83	0.72 – 0.96	
Total[3]	No disability	1.00		0.044
	Disability	0.96	0.91 – 1.00	
	Dead	0.92	0.86 – 0.99	

1: 5122 observations from 1393 participants
2: Final model included age group, gender, $y_{ij\text{-}1}$, Wave, propensity score, network variable, $y_{ij\text{-}1}$ x Wave, and $y_{ij\text{-}1}$ x propensity score
3: Final model included age group, gender, $y_{ij\text{-}1}$, Wave, propensity score, network variable, $y_{ij\text{-}1}$ x Wave, $y_{ij\text{-}1}$ x propensity score, Wave x propensity score
4: P-value based on change in -2 x log-likelihood

Overall, the results from the multinomial logistic regressions suggest there is a significant association between better relatives networks and a reduced risk of transition to disability. The results also suggest a significant association between friends networks

and mortality, and confidants networks and mortality. This point is examined further in Chapter 8.

6.3.3 Threshold effects

Table 6.11 summarizes the analyses that tested for the threshold effects at the tertiles in the final binary and multinomial logistic regression models for both mobility and Nagi disability, using the bent-line regression approach outlined in Section 4.6. Only those social network variables that were significant in the primary analyses were considered in the analyses for threshold effects. The results suggested little evidence of threshold effects with only a marginally significant result at the upper category for total social networks for Nagi disability.

Table 6.11: Summary of threshold effects on disability in binary and multinomial logistic regression models

	Cutpoint	**33⅓rd**		**66⅔rd**	
	Social network	**LR test**	**P-value**	**LR test**	**P-value**
			Binary[1]		
Mobility disability	Relatives	0.00	0.947	1.38	0.239
Nagi disability	Relatives	0.20	0.654	0.38	0.539
	Total	0.47	0.493	0.69	0.406
			Multinomial[2]		
Mobility disability	Friends	0.82	0.662	1.42	0.491
	Confidants	2.98	0.226	1.40	0.498
Nagi disability	Relatives	0.95	0.622	0.51	0.774
	Friends	0.65	0.723	0.42	0.810
	Confidants	2.50	0.286	0.95	0.623
	Total	3.91	0.141	5.72	0.057

1: 4455 observations from 1298 participants for mobility disability and 4501 observations from 1302 participants for Nagi disability; LR test on 1 df

2: 5079 observations from 1391 participants for mobility disability and 5122 observations from 1393 participants for Nagi disability; LR test on 2 df

6.4 DISCUSSION

The effects of structural components of social relationships on disability in mobility and Nagi tasks over nine years of follow-up were analysed. After controlling for a wide range of personal, environmental and health-related factors through propensity score adjustment, there were apparently protective effects of networks with relatives against tra nsitions in both mobility disability and Nagi disability. Total social networks were significantly associated with a reduced risk of developing Nagi disability. There were no significant effects of children social networks found in the analyses of mobility disability nor Nagi disability.

In considering the effects of social networks upon physical disability, most authors have analysed changes in ADL disability. Two of the recent studies by Mendes de Leon and colleagues (Mendes de Leon et al., 1999, 2001) considered the effects of social relationships upon mobility disability. In contrast to the results for the ALSA cohort, Mendes de Leon et al. (2001) found more frequent contact with friends decreased the risk of developing mobility disability. These authors reported no significant effect of networks with relatives upon the development of mobility disability. Mendes de Leon et al. (1999) found no statistically significant effects of either specific or total social networks on mobility disability. The analysis of the ALSA data suggest that specific social networks are associated with preventing mobility disability, and suggest that better networks with relatives may have particular protective effects against the development of disability in mobility.

In both the binary and multinomial analyses, the effect of relatives networks was statistically significant for Nagi disability. Unger et al. (1999) found that a greater number of social ties was associated with less Nagi disability at seven year follow-up,

but these authors did not present a breakdown of the effects of specific types of social networks on Nagi disability. Few other authors have considered the effects of social networks on Nagi disability. Because Nagi disability will occur earlier for an individual than ADL disability (Verbrugge and Jette, 1994), the identification of variables that have an effect on Nagi disability are important, from both individual and policy perspectives.

Taken together, the findings suggest that social networks with relatives may be particularly important in preventing the development of disability or promoting recovery from mobility and Nagi disability. This adds to the work of Seeman et al. (1996), who showed that women with more close ties with relatives were less likely to experience the onset of new or recurrent ADL disability. The present findings suggest that social networks with relatives have statistically significant effects in the disablement process that precede the onset of ADL disability and that disability in mobility and Nagi tasks are affected by social networks with relatives in particular.

The finding that networks with children had no effect on developing or recovering from disability was somewhat unexpected, but the result is consistent with other recent work concerning mobility (Mendes de Leon et al., 1999, 2001). Networks with children may have competing effects, because children can provide supportive resources as well as be a source of stress to their older parents. This negation of any benefits of social networks with children could be one reason for the lack of effect of children network on mobility disability and Nagi disability. Another explanation may be that older persons turn to their children first when anticipating a decline in health, and this decline in health offsets any benefits that might come from networks with children (Mendes de Leon et al., 1999). This effect is also consistent with Carstensen's socioemotional selectivity

theory (Carstensen, 1991, 1992, 1995), in which those closest are turned to first in times of need, such as the development of disability in everyday tasks reflected in mobility and Nagi items.

The results also suggested that there were no significant effects of friends networks on disability status. Earlier work in this area has been equivocal with respect to the effect of friends networks on mobility disability**,** and little previous work has considered the effect of social networks upon Nagi disability. Mendes de Leon et al. (1999) found friends networks were not associated with mobility disability. In contrast, the findings of Mendes de Leon et al. (2001) suggested contacts with friends were protective against developing mobility disability. Given these results, coupled with the results from ALSA presented in this chapter, the balance of evidence suggests the effect of friends networks upon the development of mobility or Nagi disability is fairly minimal.

No significant effects of confidant networks upon either mobility disability or Nagi disability were found in the present study. This supports the similar findings of Mendes de Leon et al. (1999) who found no significant effects of confidant networks on ADL disability and mobility disability. It may be that, like children, confidants are turned to when a decline in health is anticipated or experienced, and this decline in health and associated greater needs could negate any benefits with respect to disability that might come from networks with confidants.

There were no significant threshold effects for any of the specific social network variables for either mobility disability and Nagi disability. Of the 18 models that were compared in the analyses of threshold effects, one likelihood ratio test were marginally significant (for the total social network variable). Overall, there is little to suggest that

there are threshold effects of specific social networks on mobility disability or Nagi disability.

The analyses presented here suggest consistent effects of networks with relatives on preventing mobility disability and Nagi disability. Few other reports have systematically compared the effects of different social networks using a range of analyses. The convergence of findings from the different analyses undertaken in this chapter lends support to the consistency of effects of relatives networks.

The results raise important questions about how social networks with relatives impact upon disability in later life. The model proposed by Berkman et al. (2000) suggests that the effects of relatives networks on disability arise because of micro-level and subsequent pathway effects of social networks.

The micro-level of the effects of relatives social networks may operate through the provision of social support, social influence, social engagement, or access to goods and resources. Social influence appears the most plausible of these in terms of the micro-level effects of relatives networks on disability. For example, it may be that relatives in particular offer advice about better health habits and access to health services, and act as role models. Similarly, diet and exercise and help-seeking behaviour may be affected by the micro-level of the influence of relatives networks. ALSA participants may have been more influenced by advice offered by relatives than by advice offered by a child, spouse, or friend. Siblings and grandchildren may be particularly important in this regard.

The micro-level of social network function may influence health through a number of pathways, including health behaviours, psychological and physiological routes. Cognitive and emotional states such as self-esteem, coping, depression and sense of well-being may be affected by the psychosocial mechanisms. From a physiological perspective, psychosocial mechanisms may be important due to their influence on immune system function or neuroendocrine activation (Seeman and McEwen, 1996, Seeman, 2000; Uchino et al., 1996, Uchino, 2006).

Burg and Seeman (1994) reviewed some of the negative effects of family members on health, and suggested spouses in particular may promote poor health habits. It is possible that relatives, in contrast to children and spouses, offer health advice less frequently which makes its reception more welcome. In this way, it could be that participants with larger relatives networks have a lower risk of developing disability because of the preventive health behaviours that the networks with relatives foster.

An alternative explanation is that relatives, in particular siblings, also possibly share early-life environments and health behaviours. Thus the relatives network may be a proxy measure of genetic factors, including the predisposition to survivorship and good or poor health. Another possible pathway through which relatives networks may affect disability status is a direct link between physiological mechanisms and social networks, with subsequent beneficial effects upon disability. Any physiological advantage that stems from networks with relatives probably reflects a mixture of genetic factors and health behaviours that are shaped by early-life environments. A further refinement of social networks with specific relatives aside from those with children and spouses is warranted from these results. Disentangling the effects of networks with different kinds of relatives, such as siblings, grandchildren and nieces or nephews, may provide further

insight into the way in which relatives networks act to prevent or reverse disability in mobility and Nagi tasks.

In summary, the results in this chapter have suggested the protective role that specific social relationships may have in the disablement process prior to the development of ADL disability. Having a strong social network, particularly with relatives, is significant in protecting against disability in mobility and disability in Nagi tasks.

7 THE EFFECTS OF SOCIAL NETWORKS ON USE OF RESIDENTIAL CARE

7.1 INTRODUCTION

This chapter describes the effects of specific and total social networks on use of residential care at successive waves of the ALSA study. Section 7.2 presents the observed proportions for transitions in place of residence at each wave of ALSA, classified according to total social network categories as well as overall. In Section 7.3.1, the effects of social networks on any use of low-level residential care (hostels) or high-level residential care (nursing homes) over the nine years of follow-up are presented. The longitudinal analyses concerning the effects of the specific and total social networks on place of residence at each of the six study waves are then presented in Section 7.3.2. Analyses that investigated the existence of threshold effects of social networks on place of residence were also conducted, and these are presented in Section 7.3.3. The chapter concludes with a discussion of the findings in Section 7.4.

7.2 USE OF RESIDENTIAL CARE

The observed proportions for transitions in place of residence between waves are shown in Table 7.1. The transitions are classified according to the categories of the total social network variable and overall.

Table 7.1 shows that overall the proportion of participants who continued to live in the community between waves decreased over the course of the study, from 0.92 between Waves 1 and 2 to 0.71 between Waves 5 and 6. A lower proportion of those in the lower category of total social networks continued to live in the community at the

subsequent wave than those in either the mid or upper categories of total social networks.

Transitions from low-level care at one wave to community living at the next wave were observed, albeit rarely, and appeared more likely among the participants in the mid and upper categories than in the lower category of total social networks. However, as the absolute number of surviving participants who lived in either low-level or high-level care at any wave was less than 80, small differences in the absolute numbers of participants in residential care who made changes in their place of residence between waves will have a large effect on the observed proportions.

Transitions from high-level care to either low-level care or community living were rare in all subgroups of total social networks. A large proportion of participants who were in high-level care at any given wave were dead by the next wave. There was little obvious difference between the three categories of total social networks in terms of transitions from living in high-level residential care.

The patterns of transition in place of residence between Waves 2 and 3 appear to differ from the other waves. For example, the proportion of participants who made the transition from low-level care at Wave 2 to community living at Wave 3 was 0.16 overall, yet for other waves the comparable figure did not exceed 0.07. A similar finding was noted for participants who were in high-level care at Wave 2. This apparent anomaly was thoroughly investigated but no specific cause was found, and therefore analyses proceeded with the observed data.

Table 7.1: Transitions in place of residence over six waves of ALSA for lower, mid and upper total social networks and overall

Residential status at Wave *j*-1	Wave *j*	Residential status at Wave *j*				Total n
		Comm-unity	Low-level care	High-level care	Dead	
Community						
Lower	2	0.88	0.01	0.03	0.08	370
	3	0.86	0.03	0.03	0.08	314
	4	0.87	0.01	0.04	0.08	270
	5	0.70	0.05	0.04	0.22	216
	6	0.63	0.02	0.06	0.29	125
Mid	2	0.95	0.01	0.00	0.04	406
	3	0.90	0.02	0.01	0.07	365
	4	0.91	0.01	0.01	0.07	333
	5	0.79	0.04	0.02	0.15	285
	6	0.72	0.03	0.04	0.20	189
Upper	2	0.94	0.01	0.01	0.05	443
	3	0.91	0.02	0.01	0.06	400
	4	0.93	0.01	0.01	0.06	363
	5	0.84	0.03	0.03	0.10	318
	6	0.75	0.03	0.05	0.17	222
Overall	2	0.92	0.01	0.01	0.05	1219
	3	0.89	0.02	0.02	0.07	1079
	4	0.91	0.01	0.02	0.07	966
	5	0.78	0.04	0.03	0.15	819
	6	0.71	0.03	0.05	0.21	536
Low-level care						
Lower	2	0.00	0.92	0.03	0.05	37
	3	0.11	0.62	0.14	0.14	37
	4	0.00	0.73	0.16	0.11	37
	5	0.04	0.36	0.25	0.36	28
	6	0.10	0.14	0.29	0.48	21
Mid	2	0.00	0.86	0.04	0.11	28
	3	0.21	0.39	0.21	0.18	28
	4	0.05	0.77	0.05	0.14	22
	5	0.05	0.45	0.14	0.36	22
	6	0.05	0.15	0.30	0.50	20
Upper	2	0.00	0.92	0.00	0.08	12
	3	0.20	0.73	0.07	0.00	15
	4	0.00	0.94	0.00	0.06	17
	5	0.06	0.33	0.00	0.61	18
	6	0.06	0.19	0.31	0.44	16
Overall	2	0.00	0.90	0.03	0.08	77
	3	0.16	0.56	0.15	0.13	80
	4	0.01	0.79	0.09	0.11	76
	5	0.04	0.38	0.15	0.43	68
	6	0.07	0.16	0.30	0.47	57

Table 7.1 (continued): Transitions in place of residence over six waves of ALSA for lower, mid and upper total social networks and overall

Residential status at Wave *j*-1	Wave *j*	Residential status at Wave *j*				Total n
		Comm-unity	Low-level care	High-level care	Dead	
High-level care						
Lower	2	0.00	0.00	0.80	0.20	35
	3	0.05	0.15	0.54	0.26	39
	4	0.00	0.03	0.68	0.30	37
	5	0.00	0.08	0.31	0.61	36
	6	0.00	0.04	0.16	0.80	25
Mid	2	0.00	0.11	0.67	0.22	9
	3	0.00	0.13	0.50	0.38	8
	4	0.00	0.07	0.57	0.36	14
	5	0.00	0.10	0.40	0.50	10
	6	0.08	0.00	0.31	0.62	13
Upper	2	-	-	-	-	0
	3	0.00	0.00	1.00	0.00	3
	4	0.00	0.11	0.56	0.33	9
	5	0.00	0.00	0.33	0.67	9
	6	0.00	0.00	0.08	0.92	12
Overall	2	0.00	0.02	0.77	0.20	44
	3	0.04	0.14	0.56	0.26	50
	4	0.00	0.05	0.63	0.32	60
	5	0.00	0.07	0.33	0.60	55
	6	0.02	0.02	0.18	0.78	50

7.3 THE EFFECTS OF SOCIAL NETWORKS ON RESIDENTIAL CARE USE

7.3.1 Binary logistic regression model

Participants' use of residential care at any point over the course of the study was first considered, to enable comparison with previous Australian research in this area by Wang et al. (2001) and McCallum et al. (2005). Accumulated across all study waves, low-level residential care was known to have been used over the study period by 189 participants (13%) – that is, at one or more waves, 189 of the ALSA participants were interviewed in low-level care. A total of 914 participants (62%) were not in low-level care at any of the study waves or died without using low-level care at any previous wave. Of these, 136 participants (9% of total) were either in high-level care at Wave 1 or moved directly to

high-level residential care from the community. Information on use of low-level care could not be ascertained for 374 participants (25%), because these participants missed an interview, but were known to be alive, for at least one of Waves 2 to 6. An example which could have led to this indeterminate outcome is for a participant to have been living in the community at Wave 1, missing at Wave 2 and dead at Wave 3. The participant may have used high or low-level care between Wave 1 and death, but this cannot be determined from the available data.

Over the course of the study, a total of 195 participants (13%) were in high-level residential care at some time. A total of 883 participants (60%) were not in high-level care at any wave or died without using high-level residential aged care at any previous wave. Use of high-level care could not be ascertained for the remaining 399 participants (27%).

Binary logistic regression models were fit to ascertain the effects of social networks on any use of low-level residential care over the six study waves, and similar models were fit separately for any use of high-level residential aged care. The initial model fit for each social network variable was

$$\text{logit Pr}(Y_i = 1) = \boldsymbol{x}_i'\beta$$

where the $\boldsymbol{x}_i$ terms consisted of the propensity score strata, age group, gender and the social network variable, and β corresponded to the vector of regression coefficients. LR tests were used to determine the significance of the social network in question.

Table 7.2 summarizes the effects of the categorical social networks variables on any use of low-level care and any use of high-level care. Consider first the analyses of any low-level residential aged care use. This table shows that there were no statistically

significant effects of relatives or friends networks on use of low-level care. However, there were significant effects of children (mid category OR 1.63, 95%CI 1.07 – 2.47), confidants (mid category OR 1.67, 95%CI 1.13 – 2.47) and total social networks (mid category OR 1.70; 95%CI 1.13 – 2.56) on use of low-level care. It appeared that the participants in the mid category for each of these types of social networks were at increased risk of any use of low-level care in comparison to those in the lower or upper categories. It is not clear as to why an increase in risk of low-level care is associated with the mid social network categories for these specific networks.

The analyses of any use of high-level care suggested that participants with better social networks with children (upper category OR 0.59; 95%CI 0.38 – 0.90), confidants (upper category OR 0.52; 95%CI 0.32 – 0.86) and total social networks (mid category OR 0.57; 95%CI 0.38 – 0.85; upper category OR 0.58; 95%CI 0.37 – 0.90) were protected against any use of high-level care, after adjusting for the propensity score quintiles, age group and gender.

Table 7.2: Summary of effect of social networks upon any low-level or high-level care use. Lower category is referent category in all analyses.

	Low-level care[1]			High-level care[2]		
	OR	**95%CI**	**P-value[3]**	**OR**	**95%CI**	**P-value[3]**
Children						
0 – 33⅓rd	1.00		0.036	1.00		0.008
33⅓ – 66⅔rd	1.63	1.07 – 2.47		1.08	0.72 – 1.60	
66⅔ – 100th	1.04	0.68 – 1.60		0.59	0.38 – 0.90	
Relatives						
0 – 33⅓rd	1.00		0.874	1.00		0.575
33⅓ – 66⅔rd	1.07	0.71 – 1.62		0.83	0.56 – 1.23	
66⅔ – 100th	1.13	0.72 – 1.77		0.82	0.52 – 1.27	
Friends						
0 – 33⅓rd	1.00		0.152	1.00		0.326
33⅓ – 66⅔rd	1.11	0.73 – 1.67		0.83	0.56 – 1.23	
66⅔ – 100th	1.40	0.91 – 2.16		0.73	0.47 – 1.12	
Confidants						
0 – 33⅓rd	1.00		0.037	1.00		0.019
33⅓ – 66⅔rd	1.67	1.13 – 2.47		0.70	0.48 – 1.03	
66⅔ – 100th	1.28	0.77 – 2.13		0.52	0.32 – 0.86	
Total						
0 – 33⅓rd	1.00		0.034	1.00		0.008
33⅓ – 66⅔rd	1.70	1.13 – 2.56		0.57	0.38 – 0.85	
66⅔ – 100th	1.43	0.90 – 2.27		0.58	0.37 – 0.90	

1: Complete data available for 1103 cases
2: Complete data available for 1078 cases
3: P-value based on -2 x change in log-likelihood

The findings from the analyses that included the social network variables as continuous variables (Table 7.3) suggested that there were no statistically significant effects of specific nor total social networks on use of low-level care (which would be consisted with a non-monotonic effect of children and confidants networks on use of low-level care). In keeping with the analyses of the categorized social network variables, for high-level care there were statistically significant protective effects of children networks (OR 0.80; 95%CI 0.65 – 0.99) and total social networks (OR 0.87; 95%CI 0.79 – 0.97). In addition, marginally significant effects were observed for confidant social networks (OR 0.81; 95%CI 0.64 – 1.03) and friends social networks (OR 0.80; 95%CI 0.65 – 1.00) on any use of high-level residential care.

Table 7.3: Effects of continuous social network variables on use of low-level and high-level residential care over nine years

	Low-level care[1]			High-level care[2]		
Network	**OR**	**95%CI**	**P-value[3]**	**OR**	**95%CI**	**P-value[3]**
Children	0.94	0.76 – 1.17	0.587	0.80	0.65 – 0.99	0.044
Relatives	1.03	0.83 – 1.28	0.814	0.92	0.74 – 1.14	0.426
Friends	1.16	1.92 – 1.46	0.198	0.80	0.65 – 1.00	0.051
Confidants	1.09	0.84 – 1.41	0.518	0.81	0.64 – 1.03	0.088
Total	1.06	0.96 – 1.18	0.244	0.87	0.79 – 0.97	0.008

1: Complete data available for 1103 cases
2: Complete data available for 1078 cases
3: P-value based on -2 x change in log-likelihood

Given that overall use of low-level or high-level care could not be ascertained for around one quarter of the ALSA participants, some crude sensitivity analyses were conducted to compare the effects of different assumptions regarding missing data. All missing values were first imputed as being never used for the relevant type of residential care. A second set of analyses was run in which all missing values were imputed as if the participants with missing data had used residential care. The results of the sensitivity analyses for

low-level care are summarised in Figure 7.1. In this figure, the estimates presented in Table 7.2 are contrasted with those obtained from analyses with the missing values imputed as described above. Figure 7.2 presents a summary of the sensitivity analyses for high-level care based on the categorized social network variables.

Not unexpectedly, the results demonstrate that the assumptions regarding missing values affected the estimates of the effects of social networks for low-level care. This was most obvious for the children social networks and total social networks, particularly the point estimates of the mid categories. As demonstrated in Table 7.4, the substantive conclusions remained the same for the children and confidant social networks for any use of low-level care. However, the effect of total social networks when all missing values were imputed as used low-level residential care were not statistically significant, and this was different to the results for the observed data or when all missing values were imputed as never used residential care. For high-level care, the imputation most obviously affected the estimates of the confidant social network effects, again in the instance when the participants with missing data were imputed to have used high-level care. This also affected the significance of the effect, as demonstrated in Table 7.4.

As already noted, the sensitivity analyses described here present the extremes in terms of assumptions regarding missing values, and the 'true' results lie somewhere between these extremes. Across the sensitivity analyses and observed results, the only consistently significant findings for both the categorized and continuous variables were for the effect of total social networks on any use of high-level residential care.

Given that overall use of low-level or high-level care could not be ascertained for around one quarter of the ALSA participants, the differences between participants with

missing values and with complete data were explored. This showed that the participants with missing status for use of low-level care were significantly younger at Wave 1 (mean 78.1 years; SD 6.1) than the participants with complete data concerning use of low-level care (mean 80.2 years; SD 6.5). Similarly, participants with missing data concerning use of high-level care were significantly younger (78.5 years; SD 6.2) than those with complete data (mean 80.1; SD 6.5). However, there was no significant difference in the gender profile of those with and without missing data concerning any use of either low-level or high-level care. Similarly, there was no significant difference between those with and without missing data for any use of low-level or high-level care in any of the social network variables. The results from the sensitivity analyses suggest that the impact from the likely pattern of missing values on the results is probably small.

Overall, it appears that the upper category for children social networks and total social networks were associated with a reduced risk of any use of high-level residential care.

Figure 7.1: Summary of odds ratios and 95% confidence intervals from sensitivity analyses for any use of low-level care

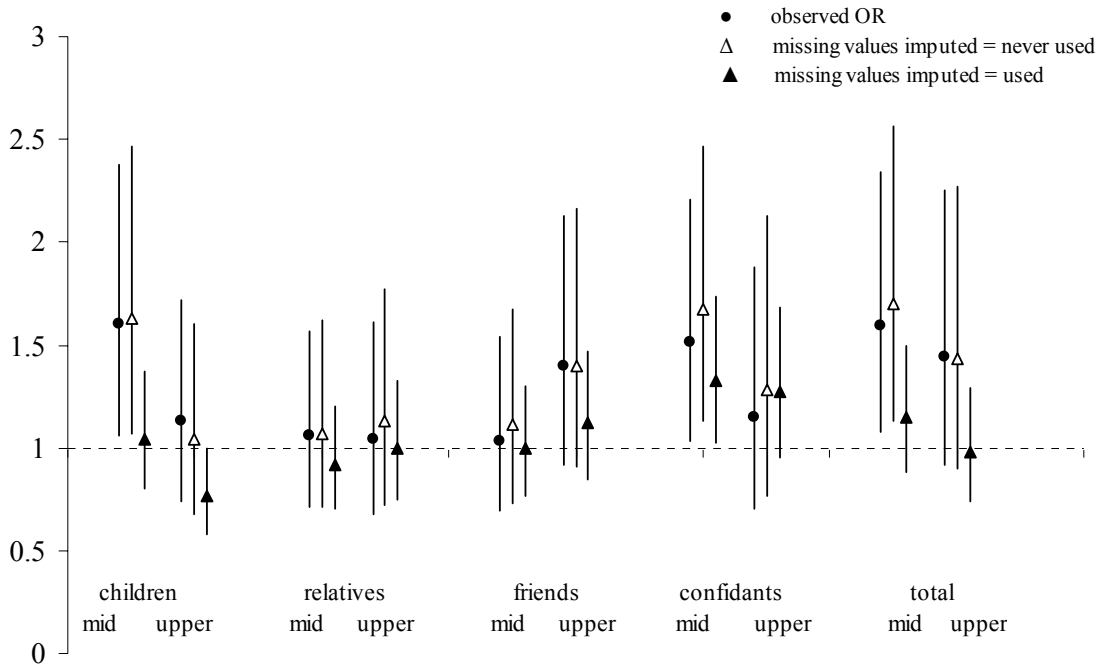


Figure 7.2: Summary of odds ratios and 95% confidence intervals from sensitivity analyses for any use of high-level care

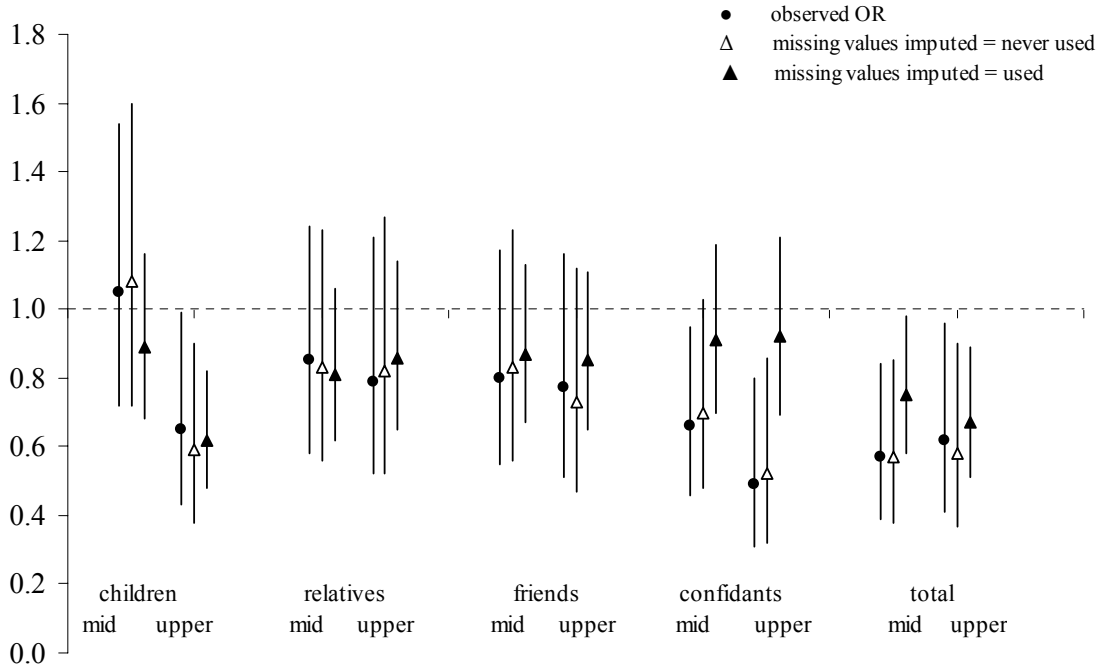


Table 7.4: Summary of likelihood ratio tests for sensitivity analyses

		Low-level care		High-level care	
Social network	**Data**	**LR**[1]	**P-value**	**LR**	**P-value**
Categorized					
Children	Observed	6.63	0.036	9.61	0.008
	Imputed never used	5.59	0.061	6.31	0.043
	Imputed used	6.34	0.042	13.15	0.001
Relatives	Observed	0.27	0.874	1.11	0.575
	Imputed never used	0.07	0.963	1.27	0.529
	Imputed used	0.61	0.737	2.37	0.305
Friends	Observed	3.76	0.152	2.24	0.326
	Imputed never used	2.89	0.235	2.02	0.364
	Imputed used	0.88	0.645	1.62	0.444
Confidants	Observed	6.59	0.037	7.90	0.019
	Imputed never used	4.68	0.096	10.40	0.006
	Imputed used	5.23	0.073	0.59	0.743
Total	Observed	6.76	0.034	9.76	0.008
	Imputed never used	5.85	0.054	9.49	0.009
	Imputed used	1.81	0.404	8.56	0.014
Continuous					
Children	Observed	0.30	0.587	4.07	0.044
	Imputed never used	0.01	0.908	2.02	0.155
	Imputed used	7.06	0.008	13.47	<0.001
Relatives	Observed	0.06	0.814	0.63	0.426
	Imputed never used	0.00	0.976	0.82	0.364
	Imputed used	0.11	0.738	0.99	0.321
Friends	Observed	1.65	0.198	3.79	0.051
	Imputed never used	1.85	0.173	3.22	0.073
	Imputed used	0.28	0.594	2.52	0.112
Confidants	Observed	0.42	0.518	2.92	0.088
	Imputed never used	0.21	0.650	3.33	0.068
	Imputed used	0.35	0.551	0.95	0.329
Total	Observed	1.36	0.244	6.54	0.011
	Imputed never used	1.53	0.216	5.37	0.021
	Imputed used	0.94	0.333	11.43	0.001

1: Likelihood ratio test calculated as -2 x change in log-likelihood

7.3.2 Proportional odds and multinomial logistic regression

As illustrated in Table 7.1, participants could be living in the community, low-level residential care, or high-level residential care at each wave, or have died. The effects of social networks on transitions in use of residential care were modelled through initially fitting the proportional odds regression model, defined in Equation (4.4) as

$$\text{logit}(Y_{ij} \le b \mid \boldsymbol{Y}^{*}_{ij\text{-}1} = \boldsymbol{y}^{*}_{ij\text{-}1}) = \theta_b + \sum_{k=0}^{1} \alpha_{kb} y^{*}_{ij\text{-}1;k} + \boldsymbol{x}_{ij}'\beta + \boldsymbol{x}_{ij}' \sum_{k=0}^{1} \gamma_k y^{*}_{ij\text{-}1;k} \ .$$

In the present application, the $y_{ij\text{-}1}$ term corresponded to residential status at Wave j-1 for participant i. The $\boldsymbol{x}_{ij}$ terms consisted of age group, gender, the propensity score strata, Wave, and one of the social network variables. The five two-way interactions, described in Section 6.3.1, were also included in the initial fitted proportional odds regression model.

To assess the assumption of proportional odds, the procedure outlined in Section 4.3 was followed – that is, the likelihood ratio test for the difference between the generalized ordinal logit and the proportional odds model was calculated for each social network variable. In every case, the generalized ordinal logit model did not converge when interaction terms were included in the model. This appeared to be due to cell counts that were zero or close to zero when interactions were considered. Therefore the comparison of the proportional odds model and generalized ordinal logit model was made for fitted models that excluded the two-way interaction terms.

The resulting likelihood ratio tests are summarized in Table 7.5. For each of the social network variables (whether categorized or continuous), there was a significant difference between the generalized ordinal logit model and the proportional odds model

(P<0.001 in each case). This indicated the assumption of proportional odds was not met in each instance. Consequently, proportional odds models were not considered any further in the analyses of use of residential care.

Table 7.5: Summary of likelihood ratio tests of generalized ordinal logit model versus proportional odds model for residential care use[1]

	Categorized		Continuous	
Network	**-2 x change in log-likelihood**	**df**	**-2 x change in log-likelihood**	**df**
Children	1147.81	32	1134.53	30
Relatives	1152.12	32	1149.86	30
Friends	1132.07	32	1125.44	30
Confidants	1163.62	40	1161.90	38
Total	1127.05	32	1113.78	30

1: 5236 observations from 1396 participants

Multinomial logistic regression models, as outlined in Equation (4.5), were therefore fit to the data. The initial model fit was of the form

$$\text{logit Pr}(Y_{ij} = b \mid Y_{ij\text{-}1} = y_{ij\text{-}1}) = \boldsymbol{x}_{ij}'\beta_b + y_{ij\text{-}1}\boldsymbol{x}_{ij}'\alpha_b$$

where the $\boldsymbol{x}_{ij}$ terms consisted of propensity score stratum, age group, gender, Wave, and the social network variable and $y_{ij\text{-}1}$ corresponded to residential care status at the previous wave. The five two-way interaction terms corresponding to $y_{ij\text{-}1}$ x Wave, $y_{ij\text{-}1}$ x network, $y_{ij\text{-}1}$ x propensity score, Wave x network, and Wave x propensity score strata were also included in this model.

For every one of the specific and total social network variables, this model that included the five two-way interactions failed to converge. Excluding the interactions that involved $y_{ij\text{-}1}$ also resulted in a model fit that failed to converge for the relatives and

confidants social network variables, irrespective of whether the categorical or the continuous form of these variables was considered. Therefore, a decision was made to exclude the interactions terms from the final fitted model for every social network variable. Thus the fitted model in each case was

$$\text{logit Pr}(Y_{ij} = b \mid Y_{ij\text{-}1} = y_{ij\text{-}1}) = \boldsymbol{x}_{ij}'\beta_b + y_{ij\text{-}1}\alpha_b$$

where the $\boldsymbol{x}_{ij}$ terms were propensity score stratum, age group, gender, Wave, and the social network variable, and $y_{ij\text{-}1}$ was residential care status at the previous wave.

LR tests were used to test for the effects of the social network variables, and the categorical and continuous social network variables were investigated in separate models.

Table 7.6 summarizes the longitudinal analyses of the effects of the categorical social networks on residential care use. As these results suggest, the specific and total social networks did not have a significant effect overall upon low-level care residential use, although there was some evidence of a protective effect of the upper children category in comparison to the lower children category (OR 0.67; 95%CI 0.47 – 0.96). Participants in the mid and upper category for confidant networks versus the lower category appeared to be protected against use of high-level care (mid OR 0.71; 95%CI 0.52 – 0.99; upper OR 0.53; 95%CI 0.35 – 0.81). Similarly, significant effects of total social networks were found for high-level care use for participants in the mid (OR 0.60; 95%CI 0.42 – 0.85) and upper (OR 0.68; 95%CI 0.46 – 0.99) categories. There was a marginally significant effect of friends social networks, although this was confined to participants in the upper category who appeared to have a reduced risk of death compared to participants in the lower category for friends network (OR 0.73; 95%CI

0.56 – 0.94). This point is returned to in Chapter 8, where survival is considered in detail.

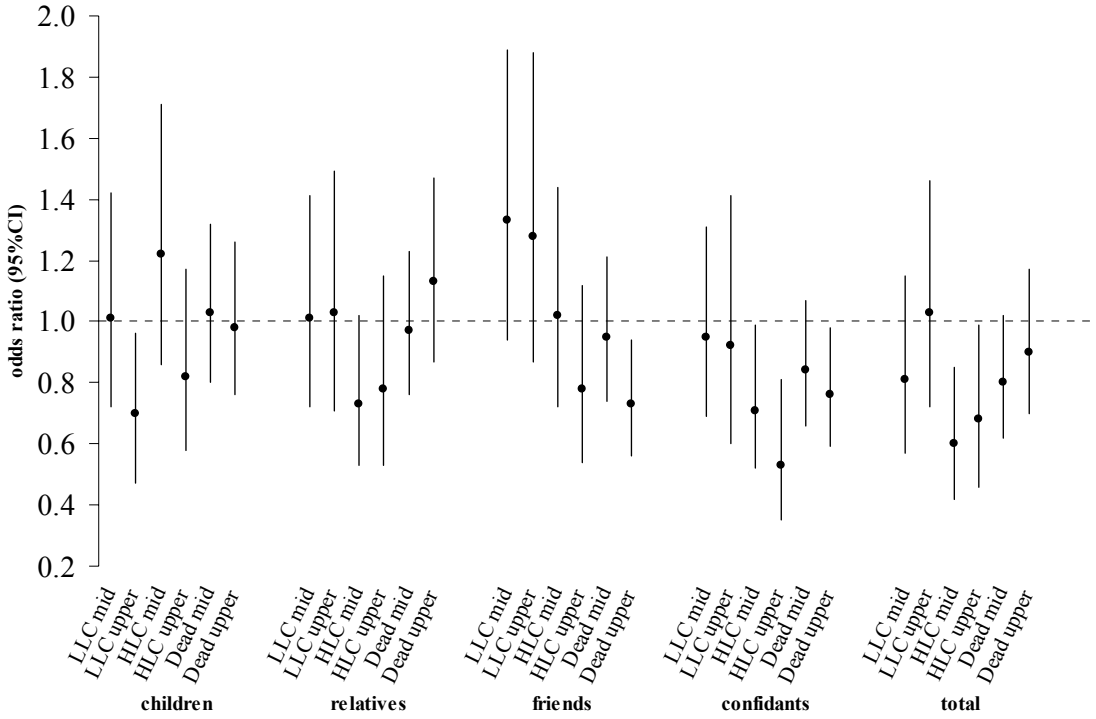
Table 7.6: Summary of effects of categorized social networks on transition in place of residence across study period[1,2]

	Low-level care		High-level care		Dead		
	OR	**95%CI**	**OR**	**95%CI**	**OR**	**95%CI**	**P-value**[3]
Children							
0 – 33⅓rd	1.00		1.00		1.00		0.241
33⅓ – 66⅔rd	1.01	0.72 – 1.42	1.22	0.86 – 1.71	1.03	0.80 – 1.32	
66⅔ – 100th	0.67	0.47 – 0.96	0.82	0.58 – 1.17	0.98	0.76 – 1.26	
Relatives							
0 – 33⅓rd	1.00		1.00		1.00		0.473
33⅓ – 66⅔rd	1.01	0.72 – 1.41	0.73	0.53 – 1.02	0.97	0.76 – 1.23	
66⅔ – 100th	1.03	0.71 – 1.49	0.78	0.53 – 1.15	1.13	0.87 – 1.47	
Friends							
0 – 33⅓rd	1.00		1.00		1.00		0.051
33⅓ – 66⅔rd	1.33	0.94 – 1.89	1.02	0.72 – 1.44	0.95	0.74 – 1.21	
66⅔ – 100th	1.28	0.87 – 1.88	0.78	0.54 – 1.12	0.73	0.56 – 0.94	
Confidants							
0 – 33⅓rd	1.00		1.00		1.00		0.087
33⅓ – 66⅔rd	0.95	0.69 – 1.31	0.71	0.52 – 0.99	0.84	0.66 – 1.07	
66⅔ – 100th	0.92	0.60 – 1.41	0.53	0.35 – 0.81	0.76	0.59 – 0.98	
Total							
0 – 33⅓rd	1.00		1.00		1.00		0.120
33⅓ – 66⅔rd	0.81	0.57 – 1.15	0.60	0.42 – 0.85	0.80	0.62 – 1.02	
66⅔ – 100th	1.03	0.72 – 1.46	0.68	0.46 – 0.99	0.90	0.70 – 1.17	

1: Lower category is referent category in all analyses; community-dwelling is referent response category
2: 5236 observations from 1396 participants
3: Overall P-value based on likelihood ratio test

Figure 7.3 is an alternative representation of the relationship between the categorized social network variables and transitions in residential care use. This illustrates the protective effect of confidants and total social networks against high-level care use across the six study waves. It also shows the apparent effect of children networks on low-level care use, and the lack of an effect for relatives social networks on use of residential care over the six study waves.

Figure 7.3: Summary of multinomial logistic regressions of effects of social networks on transitions in residential care



LLC=low-level care; HLC=high-level care

The effects of the continuous social network variables on use of residential care are presented in Table 7.7. The results were broadly consistent with those from Table 7.6. Overall, only the friends network variable was statistically significant. Participants with better friends networks had reduced odds of a transition to high-level care (OR=0.80; 95%CI 0.66-0.96). While the effect of confidants networks was only marginally

significant overall, participants with higher scores for confidants networks had reduced odds of transitions to high-level care (OR=0.79; 95%CI 0.66 – 0.94).

Table 7.7: Effects of social networks as continuous variables in multinomial logistic regression for residential care use

	OR	95%CI	P-value[2]
Children			
Community	1.00		0.644
Low-level	0.89	0.75 – 1.06	
High-level	0.92	0.77 – 1.10	
Dead	0.99	0.87 – 1.13	
Relatives			
Community	1.00		0.393
Low-level	1.03	0.87 – 1.23	
High-level	0.94	0.77 – 1.14	
Dead	1.09	0.96 – 1.23	
Friends			
Community	1.00		0.003
Low-level	1.04	0.85 – 1.27	
High-level	0.80	0.66 – 0.96	
Dead	0.81	0.71 – 0.92	
Confidants			
Community	1.00		0.096
Low-level	0.91	0.73 – 1.12	
High-level	0.79	0.66 – 0.94	
Dead	0.88	0.76 – 1.01	
Total			
Community	1.00		0.178
Low-level	0.98	0.90 – 1.07	
High-level	0.90	0.82 – 0.99	
Dead	0.96	0.90 – 1.03	

1: 5236 observations from 1396 participants
2: P-value based on likelihood ratio test

Thus overall, better confidants social networks and total social networks appear to confer most benefit in preventing transitions from the community to high-level residential care. There was weak evidence of an effect of better children social networks in preventing transitions to low-level residential care.

7.3.3 Threshold effects

The existence of threshold effects for the specific and total social networks was tested using the method described in Section 4.6. As shown in Table 7.8, there was no evidence of a significant threshold effect for the children, friends nor confidant social networks.

Table 7.8: Summary of threshold effects on residential care use in multinomial logistic regression models

Percentile	**33⅓rd**		**66⅔rd**	
Social network	**LR**	**P-value**	**LR**	**P-value**
Children	2.12	0.548	6.79	0.079
Friends	1.05	0.790	0.79	0.851
Confidants	2.37	0.499	2.18	0.535

7.4 DISCUSSION

The effects of specific and total social networks upon residential care use were examined over a nine year period, using propensity score methods to adjust for a broad range of covariates. The results suggested specific and total social networks had little effect on use of low-level residential care over the period of the study. The longitudinal analyses showed that better confidant networks and better total social networks were associated with reduced odds of high-level care use over the course of the study. The longitudinal analyses suggested there was no significant effect of children networks on use of high-level care. There was no significant effect of social networks with other

relatives upon use of low-level or high-level residential aged care. There was some evidence of an effect of friends networks, although this appeared to be largely due to an effect of friends social networks on survival.

Confidant social networks had a statistically significant effect on use of high-level care. The analyses of the continuous variables also suggested that friends networks had significant protective effects against use of high-level care, but this finding did not concur with the results from the analyses of the categorical form of the friends social network variable. Together, these results suggest that a close, emotionally supportive relationship with another person is beneficial in preventing or delaying nursing home use. The importance of a confidant to mental and physical health is well known (Bowling, 1997; Buckwalter, 2001; Lowenthal and Haven, 1968) but the translation of that effect to a reduction in risk of nursing home use has not been shown previously. Further research is clearly warranted to examine the repeatability of this finding in other settings and countries.

Those with fewer non-kin social supports may have smaller networks of human resources to draw upon for maintenance of community living status (Wolinsky and Johnson, 1991; Wolinsky et al., 1992). Other research has shown significant protection against nursing home use arises through having living daughters and siblings (Freedman, 1996). The results in this chapter suggests that the core network of confidants is more significant than other specific networks in delaying or preventing use of high-level care in Australia. The striking impact of absence of confidants may reflect the consequences of reduced emotional support that permitted continued residence in the community.

The analyses found that overall there was little evidence for a threshold effect of any of the specific or total social networks on use of residential care, as none of the tests of threshold effects at the tertiles were statistically significant. This suggests that the apparently protective effects of stronger specific networks continue to accrue. There does not appear to be critical level for networks beyond which no additional advantage in terms of a reduction in risk of residential care use is gained.

Previous research in this area has been dominated by studies conducted in the United States and findings concerning the effects of social networks on use of residential care have been equivocal. For example, six previous studies reported no significant effects of social networks on use of residential care (Branch and Jette, 1982 ; Kraus et al., 1976; Marottoli et al., 1994; Hanley et al, 1990; Speare et al., 1991; Steinbach, 1992). In contrast, a number of authors (Coward et al., 1996; Freedman, 1996; Freedman et al., 1994; McCoy and Edwards, 1981; Palmore, 1976; Pearlman and Crown, 1992; Salive et al., 1993; Shapiro and Tate, 1985; Vicente et al., 1979; Wan and Weissert, 1981; Wilmoth, 2000; Wolinsky et al., 1992) have shown that better social relationships are significantly protective against residential care use. What is not clear from this literature in general is which aspects of social networks are of most benefit.

Better networks with children were not found to be associated with high-level residential care use in the longitudinal analyses in the present study, in contrast to results reported by Freedman (1996) and Wilmoth (2000). However, Wolinsky et al. (1992) also demonstrated no significant effect of kin networks on admission to nursing homes, but found a protective effect of better non-kin relationships. In ALSA, confidants were most commonly spouses (39%) but 18 per cent of participants reported their confidant was a male or female friend. The absence of an effect of children and

relatives networks but a protective effect of confidants networks suggests it is not the bloodline of the relationship – that is, kin or not kin – that is important in preventing use of high-level residential care. Rather, it is the closeness and consequential support afforded by a confiding relationship that acts to protect against nursing home use.

It is important to bear in mind that the effects of instrumental and emotional support were not directly investigated in the present study. Questions pertaining to social support in ALSA were only asked of participants who were living in the community at Wave 1. The receipt of more instrumental support at baseline has been generally shown to be a risk factor for use of residential care (Coward et al., 1996; Greene and Ondrich, 1990; Hanley et al., 1990; Newman et al., 1990; Wilmoth, 2000), although other authors have reported instrumental support to be protective against residential care use (Greenberg and Ginn, 1979; Liu et al., 1991). These findings come from studies with two waves of data with the exception of the cross-sectional study by Greenberg and Ginn (1979). Findings concerning emotional support are scant. Salive et al. (1993) pooled emotional support with instrumental support to create an overall support measure. More research in the Australian residential care context that carefully distinguishes between different types of social support as well as the specific types of social networks appears warranted based on the results in this chapter.

Thus with respect to the Berkman et al. (2000) model, the effects of confidants and total social networks are important in protecting against use of high-level residential aged care, and they most plausibly operate at the micro-level. It remains unclear as to exactly why confidants in particular, as opposed to children, other relatives or friends, confer the most protective effects against residential care use. The effects of confidants networks probably act to buffer against adverse health events as well as have direct

health benefits through the provision of the type of support that is needed and thereby delay or prevent admission to residential care.

ALSA took place against a background of reforms in Australian aged care (Department of Health and Ageing, 1997) that may have impacted on the use of residential care services independent of the risk factors considered in the propensity scores and social network variables. One of the most significant reforms saw the assessment for entry to low-level and high-level residential aged care merged into one system in 1997. An individual's eligibility for residential aged care is assessed by Aged Care Assessment Teams against standardized criteria that include functional status, health and living arrangements. The persistent effects of social networks on use of high-level care over a long period of follow-up and over and above the effects of a range of other variables suggest that an individual's social milieu needs to be reflected more strongly in eligibility criteria, particularly for admission to high-level residential care. The results in this chapter also highlight the importance of recognising that social networks go beyond a simple ascertainment of marital status or number of children. Policymakers may need to reconsider whether social relationships have been given adequate weight in the current assessment and entry process.

The effects of social networks upon residential care use have not been previously examined in an Australian context. The results in this chapter have shown that social networks with confidants can protect against the use of high-level residential care over nine years in a large cohort of older Australians. Policy needs to reflect the importance of these particular relationships, and incorporate these along with the expectations of future cohorts of older people about where they want to live in later life.

8 THE EFFECTS OF SOCIAL NETWORKS ON MORTALITY

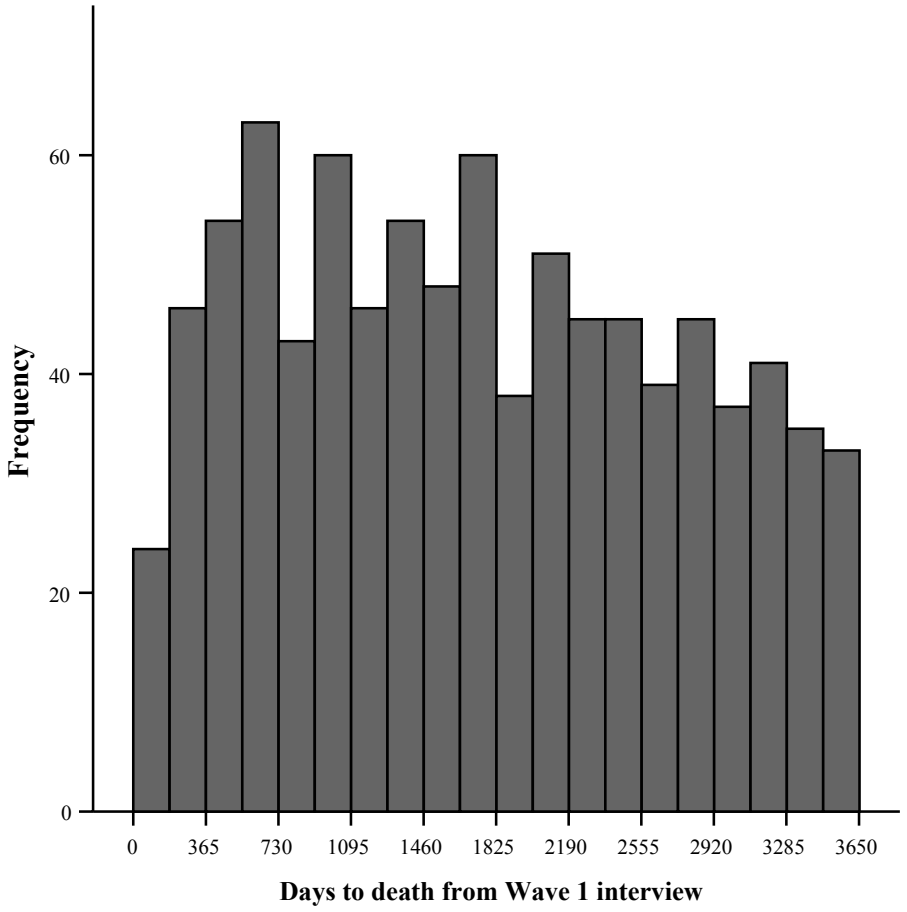
8.1 INTRODUCTION

In this chapter, the effects of specific and total social networks on mortality over a 10 year follow-up period are investigated. First, descriptive statistics pertaining to the survivors and decedents are presented. Following this, in Section 8.3.1 the effects of social networks on mortality are described. Results for both the categorical and continuous forms of the social network variables are presented. The existence of threshold effects of social networks on mortality was also investigated, and these analyses are summarized in Section 8.3.2. The findings are discussed in Section 8.4.

8.2 CHARACTERISTICS OF SURVIVORS AND DECEDENTS

At the tenth anniversary of their Wave 1 interview, 570 ALSA participants were alive and the remaining 907 participants had died. Among the participants who died, the average time from Wave 1 interview to death was just under five years (1747.0 days, SD 996.8). A histogram of days to death from Wave 1 interview is shown in Figure 8.1, which suggests a relatively uniform distribution of the time to death from six months post-interview onwards among the participants who died. The smaller number of deaths in the first six months possibly reflects an initial 'healthy participant' effect, in that frail people within a few months of death may have refused participation in the study. This would have resulted in an interviewed sample who were less likely to die in the period shortly after their Wave 1 interview.

Figure 8.1: Distribution of survival times for ALSA decedents (n=907)



Descriptive statistics for the social network variables along with a range of demographic, health, psychological and lifestyle variables are presented for survivors and decedents in Table 8.1. As this table shows, the mean specific and total network scores were higher for the participants who survived 10 years after the Wave 1 interview than for the participants who died in the intervening decade. For every specific social network and total social network, there was a higher proportion of survivors who were in the upper category of each social network than there were decedents. The survivors were generally younger, more likely to be married at baseline, had completed more years of education, were less likely to have disability, had better self-rated health and were less likely to be sedentary than those participants who died during the decade following the Wave 1 interview.

Table 8.1: Summary statistics for 570 survivors and 907 decedents among 1477 ALSA participants

Characteristic (at Wave 1)	n[1]	Survivors (n=570)	Decedents (n=907)
Children networks mean (SD)	1477	0.08 (0.77)	-0.04 (0.81)
Children networks upper n (%)	1477	220 (38.6)	285 (31.4)
Relatives networks mean (SD)	1477	0.11 (0.80)	-0.06 (0.79)
Relatives networks upper n (%)	1477	227 (39.8)	266 (29.3)
Friends networks mean (SD)	1477	0.19 (0.73)	-0.11 (0.78)
Friends networks upper n (%)	1477	244 (42.8)	251 (27.7)
Confidants networks mean (SD)	1477	0.10 (0.71)	-0.04 (0.74)
Confidants networks upper n (%)	1477	213 (37.4)	253 (27.9)
Total networks mean (SD)	1477	0.48 (1.60)	-0.26 (1.76)
Total networks upper n (%)	1477	247 (43.3)	246 (27.1)
Age mean (95%CI)	1477	76.2 (5.1)	82.0 (6.3)
Gender male n (%)	1477	326 (57.2)	602 (66.4)
Community dwelling n (%)	1477	554 (97.2)	786 (86.7)
Married n (%)	1477	330 (57.9)	441 (48.6)
Left school ≤14 yrs n (%)	1463	306 (53.8)	524 (58.6)
Household income ≤$12,000 n (%)	1369	212 (37.2)	378 (41.7)
# Morbid conditions mean (SD)	1477	1.5 (1.1)	1.7 (1.2)
Mobility disability	1455	113 (19.9)	393 (44.3)
Cognitive function poor n (%)	1440	36 (6.4)	183 (20.9)
Self-rated health fair/poor n (%)	1472	116 (20.4)	353 (38.9)
Depressive symptoms n (%)	1400	59 (10.4)	160 (18.6)
Hearing difficulty n (%)	1472	253 (44.4)	493 (54.7)
Vision difficulty n (%)	1410	92 (16.7)	283 (32.9)
Alcohol problem n (%)	1466	28 (4.9)	37 (4.1)
Current smoker n (%)	1461	31 (5.4)	92 (10.3)
Ex smoker n (%)	1461	253 (44.5)	424 (47.5)
Pack-yrs smoking mean (SD)[2]	800	34.9 (33.5)	41.3 (33.3)
Sedentary n (%)	1457	214 (37.6)	449 (50.6)

1: number of non-missing observations out of possible total of 1477
2: calculated for current or ex-smokers only

8.3 THE EFFECTS OF SOCIAL NETWORKS ON MORTALITY

8.3.1 Cox proportional hazards model

As described in Section 4.4, survival analysis techniques were used in the analysis of the mortality data. For each of the four specific social network variables and the total social network variable, separate Cox proportional hazards models were fit. The assumption of proportional hazards was explored graphically, using the Nelson-Aalen cumulative hazard estimator. The assumption was also tested formally, using the method outlined by Grambsch and Therneau (1994).

The general form of the Cox proportional hazards model that was initially fit was $h_i(t;\boldsymbol{x}_i) = h_0(t)e^{x_i'\beta}$ as described in Equation (4.6). The $\boldsymbol{x}_i$ consisted of the propensity score stratum, age group, gender, and the social network variable. No interactions were included in the initial model. Each of the categorical and continuous versions of the social network variables were considered in separate analyses.

Turning first to the analyses of effects of the categorical social network variables, the observed Nelson-Aalen cumulative hazard estimates in days from the Wave 1 interview for the specific and total social networks are shown in Figure 8.2. The lower panes in the figure suggest that there was greater divergence in the cumulative hazard estimates between the categories for friends, confidants and total networks than was evident for either the children or relatives networks. Furthermore, there appears to be a crossing of the cumulative hazard estimates for the mid and upper categories for the children networks and confidants networks, suggesting some non-proportionality of hazards.

Figure 8.2: Summary of Nelson-Aalen cumulative hazard estimates by type of social network

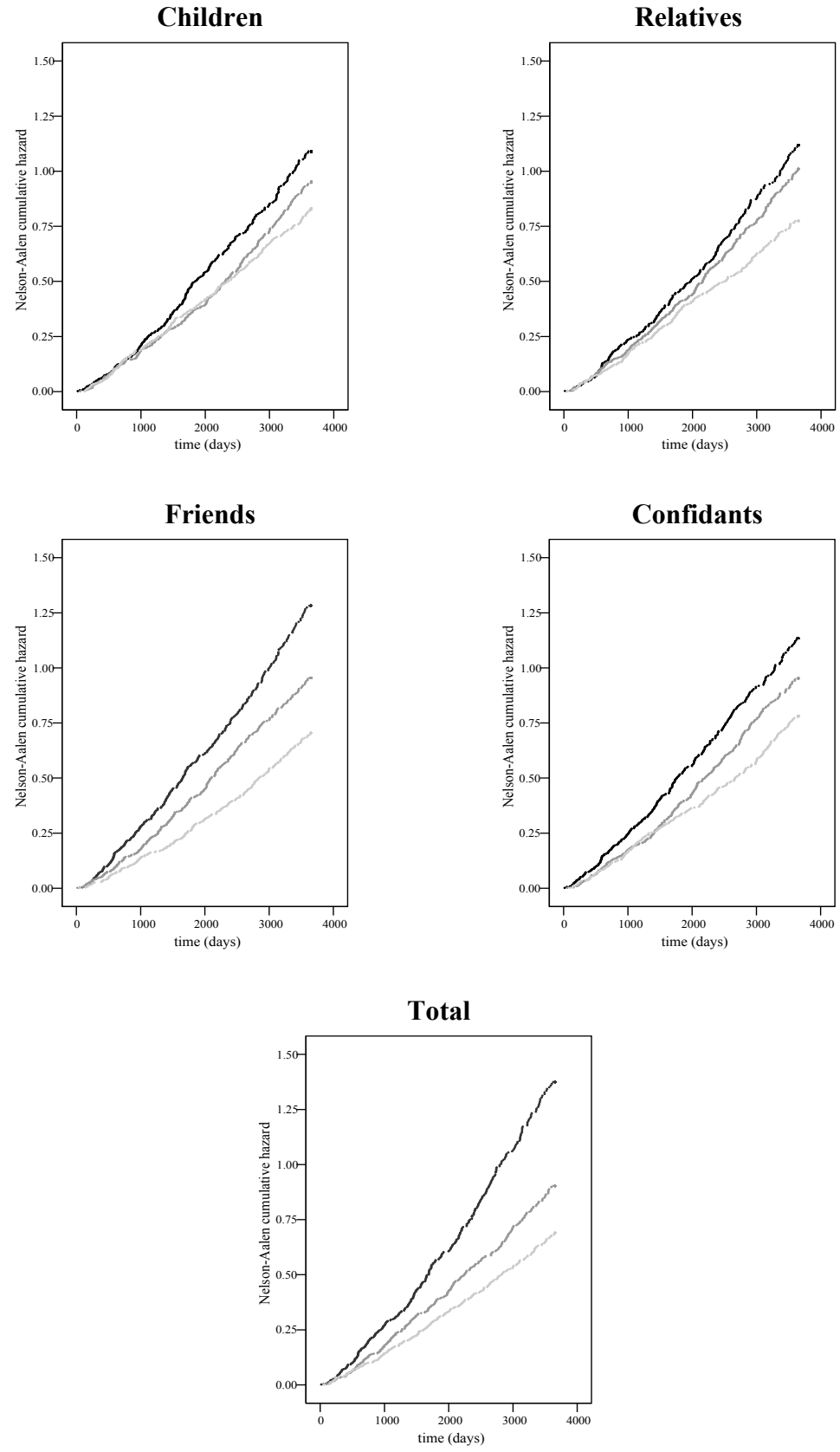


Table 8.2 presents the results of the analyses that formally assessed the assumption of proportional hazards (Grambsch and Therneau, 1994). As this table shows, the assumption of proportional hazards was not significantly violated for the relatives, friends, confidants and total social networks. Therefore the analyses proceeded as planned for these variables. However for the children social network variable, the test of non-zero slope was significant for several of the propensity score quintiles, and the global test was significant (P=0.049). Hence for the analysis of the effect of children social networks on survival, the fitted model was stratified by propensity score. For this stratified model, the global test of the proportional hazards assumption was non-significant (X^2=8.01 on 6df; P=0.237), as were the tests corresponding to the children social network variable, age group, and gender.

Table 8.2: Summary of tests of proportional hazards assumption

Variable	Category	X^2	df	P-value
Children				
Network	Mid category	1.46	1	0.226
	Upper category	0.01	1	0.928
Age group	75-79 years	3.13	1	0.077
	80-84 years	0.43	1	0.514
	≥ 85 years	0.01	1	0.943
Gender	Female	1.63	1	0.165
Propensity score	Stratum 2	5.42	1	0.020
	Stratum 3	1.00	1	0.318
	Stratum 4	0.34	1	0.559
	Stratum 5	7.29	1	0.007
Global		18.37	10	0.049
Relatives				
Network	Mid category	0.06	1	0.801
	Upper category	2.67	1	0.102
Age group	75-79 years	3.61	1	0.058
	80-84 years	1.82	1	0.178
	≥ 85 years	0.60	1	0.440
Gender	Female	1.52	1	0.218
Propensity score	Stratum 2	3.77	1	0.052
	Stratum 3	0.19	1	0.666
	Stratum 4	0.04	1	0.849
	Stratum 5	1.25	1	0.263
Global		14.37	10	0.157

(continued)

Table 8.2 (continued): Summary of tests of proportional hazards assumption

Variable	Category	X^2	df	P-value
Friends				
Network	Mid category	0.00	1	0.970
	Upper category	1.17	1	0.278
Age group	75-79 years	2.58	1	0.109
	80-84 years	1.30	1	0.254
	≥ 85 years	0.61	1	0.435
Gender	Female	2.16	1	0.142
Propensity score	Stratum 2	0.01	1	0.909
	Stratum 3	2.02	1	0.155
	Stratum 4	0.46	1	0.499
	Stratum 5	1.71	1	0.191
Global		13.64	10	0.190
Confidants				
Network	Mid category	2.21	1	0.138
	Upper category	0.28	1	0.595
Age group	75-79 years	2.64	1	0.104
	80-84 years	1.04	1	0.307
	≥ 85 years	0.30	1	0.586
Gender	Female	0.26	1	0.609
Balancing score	Stratum 2	0.00	1	0.998
	Stratum 3	0.34	1	0.561
	Stratum 4	0.08	1	0.778
	Stratum 5	2.03	1	0.155
	Stratum 6	1.21	1	0.271
	Stratum 7	0.74	1	0.390
	Stratum 8	0.22	1	0.638
	Stratum 9	1.40	1	0.236
Global		13.99	14	0.451
Total networks				
Network	Mid category	0.04	1	0.840
	Upper category	0.22	1	0.639
Age group	75-79 years	3.39	1	0.066
	80-84 years	1.48	1	0.224
	≥ 85 years	0.41	1	0.521
Gender	Female	1.36	1	0.244
Propensity score	Stratum 2	1.17	1	0.280
	Stratum 3	0.92	1	0.338
	Stratum 4	1.02	1	0.313
	Stratum 5	0.11	1	0.742
Global		11.45	10	0.324

Plots of the Schoenfeld residuals versus time for each of the terms in the initial fitted models are presented in Appendix 3. These plots demonstrate that overall there was

little indication of non-proportional hazards. Nonetheless, stratification by propensity score was undertaken for the analysis of children social networks, as already noted.

LR tests were used to test for the significance of each social network variable in the fitted proportional hazards models.

The hazard ratios that demonstrate the effects of the categorized social network variables on mortality, adjusted for age group, gender and the propensity scores, are shown in Table 8.3. As this table shows, there were significant effects of friends, confidants and total social networks on mortality overall. The effect of the friends network on survival was largest for those with the greatest networks of friends (that is, upper category of friends social network HR 0.75; 95%CI 0.63 – 0.89). The effect of the confidant social network appeared significant for those in both the middle (HR 0.81; 95%CI 0.69 – 0.95) and upper categories (HR 0.77; 95%CI 0.64 – 0.91) of confidant networks. There was also a significant effect of better total social networks on mortality (mid category HR 0.83; 95%CI 0.71 – 0.97; upper category HR 0.83; 95%CI 0.70 – 0.99). The effects of networks with children and networks with relatives on survival were not statistically significant.

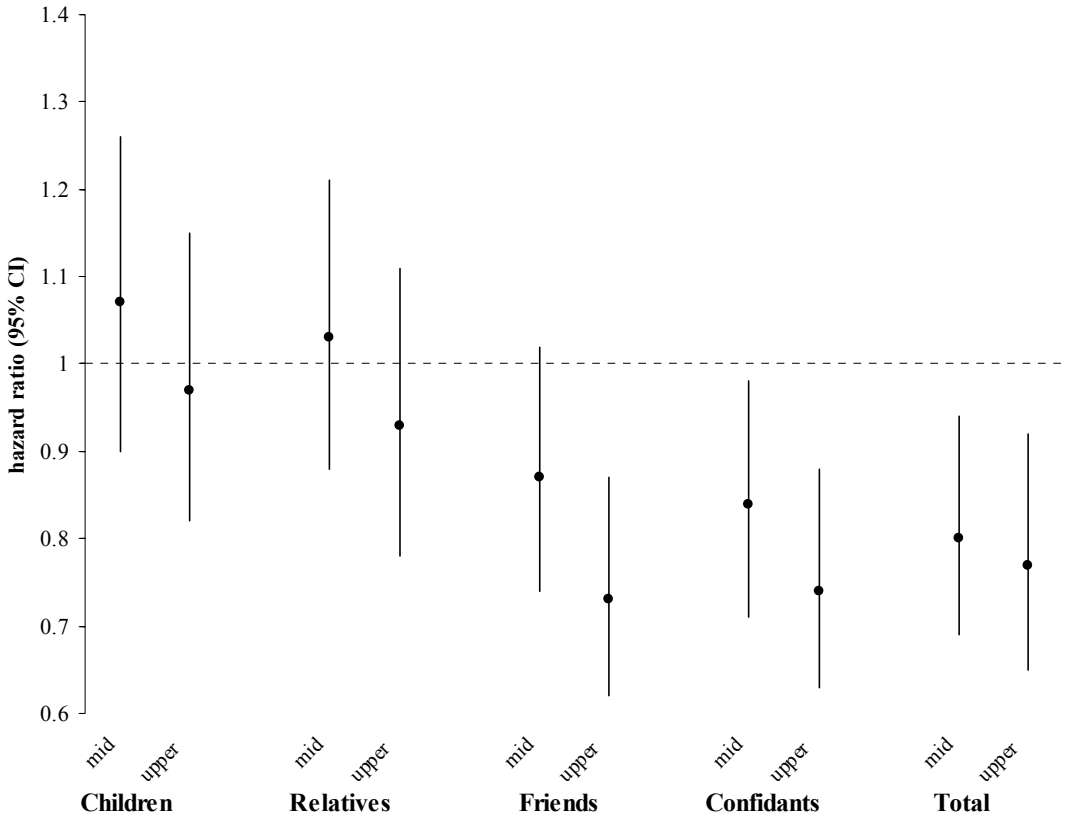
Table 8.3: Summary of adjusted hazard ratios for categorized social network variables

Network	Category	HR	95% CI	P-value[1]
Children	0 – 33⅓rd	1.00		0.993
	33⅓ – 66⅔rd	1.01	0.86 – 1.20	
	66⅔ – 100th	0.98	0.83 – 1.16	
Relatives	0 – 33⅓rd	1.00		0.560
	33⅓ – 66⅔rd	1.01	0.86 – 1.19	
	66⅔ – 100th	0.93	0.78 – 1.11	
Friends	0 – 33⅓rd	1.00		0.004
	33⅓ – 66⅔rd	0.90	0.77 – 1.05	
	66⅔ – 100th	0.75	0.63 – 0.89	
Confidants	0 – 33⅓rd	1.00		0.004
	33⅓ – 66⅔rd	0.81	0.69 – 0.95	
	66⅔ – 100th	0.77	0.64 – 0.91	
Total	0 – 33⅓rd	1.00		0.034
	33⅓ – 66⅔rd	0.83	0.71 – 0.97	
	66⅔ – 100th	0.83	0.70 – 0.99	

1: P-value from likelihood ratio test

Figure 8.3 gives an alternative presentation of the results for the categorized social network variables. The figure emphasises the protective effect of better social networks with friends and confidants, as well as total social networks. Furthermore, the lack of a significant effect for networks with children and relatives is clear from Figure 8.3.

Figure 8.3: Summary of Cox proportional hazards models of effects of categorized social networks variables on mortality



Cox proportional hazards models that investigated the effects of the continuous social network variables were then fit. As before, the initial model fit was $h_i(t;\boldsymbol{x}_i) = h_0(t)e^{x_i'\beta}$ as described in Equation (4.6). The $\boldsymbol{x}_i$ consisted of the propensity score stratum, age group, gender, and the social network variable. No interactions were included in the initial model. LR tests were again used to examine the significance of each social network variable in question.

The results for the continuous social network variables are summarized in Table 8.4. There were significant protective effects of better friends social networks, confidants social networks and total social networks against mortality. As was found for the

categorized social network variables, the effects of social networks with children and relatives were not statistically significant.

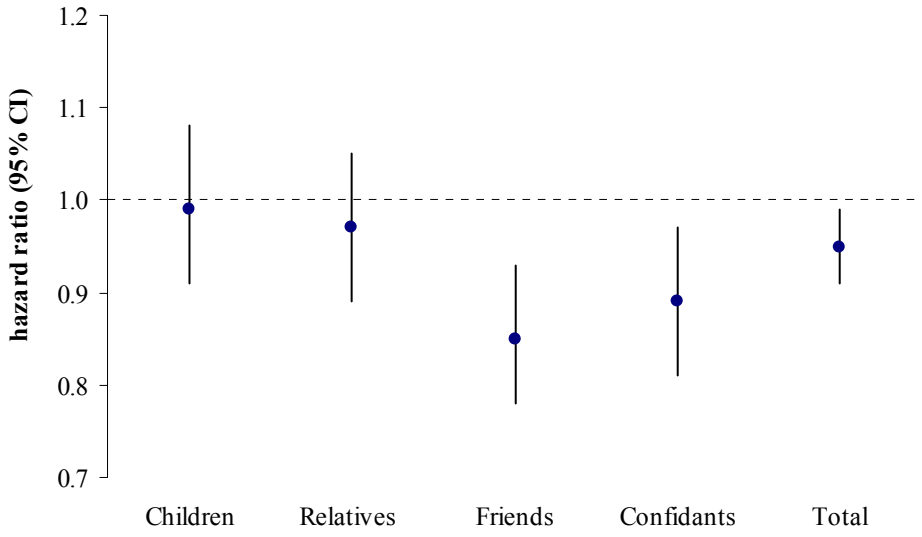
Table 8.4: Summary of adjusted hazard ratios for continuous social network variables

Network	HR	95% CI	P-value[1]
Children	0.99	0.91 – 1.08	0.851
Relatives	0.97	0.89 – 1.05	0.451
Friends	0.85	0.78 – 0.93	<0.001
Confidants	0.89	0.81 – 0.97	0.010
Total	0.95	0.91 – 0.99	0.008

1: P-value from likelihood ratio test

Figure 8.4 provides an alternative presentation of the results for the continuous social network variables.

Figure 8.4: Summary of Cox proportional hazards models of effects of continuous social networks variables on mortality



Thus overall, the results suggest better friends and confidants networks are associated with a reduced hazard of mortality over a 10 year period.

8.3.2 Threshold effects

The existence of threshold effects at the first and second tertiles for the friends, confidants and total social network variables was investigated, using the approach described in Section 4.6. As shown in Table 8.5, there was no evidence of a threshold effect at either tertile for either of the specific social network variables. There was, however, a statistically significant potential threshold effect at the upper tertile for the total social network variable.

Table 8.5: Summary of lower and upper threshold effects on mortality

	33⅓rd percentile		66⅔rd percentile	
Network	**LR**	**P-value**	**LR**	**P-value**
Friends	0.00	1.000	0.01	0.928
Confidants	0.48	0.486	0.40	0.528
Total	2.69	0.101	5.11	0.024

8.4 DISCUSSION

This study builds on previous work concerning social relationships and mortality. Most other studies have used *ad hoc* measures of social networks. Furthermore, there is a paucity of research that has examined the effects of specific social networks upon mortality. The present results suggest that greater social networks with friends and confidants had significant protective effects against mortality over a 10 year follow-up period. Networks with children and relatives were not significant predictors of mortality over the same follow-up period. This highlights the importance of

disaggregating kin and non-kin networks, rather than relying on measures of total social networks.

The finding that total social networks are protective against mortality suggests overall social integration is important, and reinforces findings from other studies of older people (Blazer, 1982; Penninx et al., 1997; Seeman et al., 1987; Sugisawa et al., 1994; Welin et al., 1985). Previous Australian studies (Jorm et al., 1991; Korten et al., 1999; McCallum et al., 1994; Simons et al., 1996) have not demonstrated a consistent effect of social networks on mortality in older adults. However, these studies were generally smaller or did not consider the specific types of social networks that were investigated in the present study. Differences in the definitions of social relationships, different covariates, different follow-up times, and different analytic approaches may have contributed to the disparities in previous reports.

Earlier research has shown social relationships with close friends and/or relatives were protective against mortality in older adults (Seeman et al., 1987), and subsequent research (Seeman and Berkman, 1988) also pointed to the importance of a confidant in the perceived adequacy of social support. Recent studies have found no significant effect of children networks on mortality (Rasulo et al., 2005; Guilley et al., 2005), while earlier studies suggested a significant protective benefit of children networks (Zuckerman et al., 1984; Yasuda et al, 1997). By differentiating between friends, children, confidants and other relatives, the results in this chapter suggest that it is networks with friends and confidants, rather than children or relatives, which confer most benefit to survival in later life. This suggests that 'discretionary relationships', with friends and confidants, as opposed to relationships where there is less choice

concerning interaction, such as those with children and relatives, have important positive effects on survival.

There has been limited previous research that has examined the existence of a threshold of effect of social networks on mortality. Schoenbach et al. (1986) and Blazer (1982) concluded that there were threshold effects of total social networks on mortality in older people, whereas Welin et al. (1985) and Orth-Gomér and Johnson (1987) found the effects of social networks continued to increase with 'dose'. The results in this chapter suggest that there was a gradation in response for specific social networks with friends and confidants. The significant bent-line effect that was found for total social networks suggests that there was little advantage for those in the upper category of total social networks compared to the mid category. Thus those older people in the lower category of total social networks were at greatest risk of mortality over a decade, with little difference between those in the mid and upper categories of total social networks. For friends and confidants social networks, those in the upper category had a survival advantage compared with those in the mid category, and in turn those in the mid category were less likely to die over a decade of follow-up than those in the lower category of friends or confidants social networks.

The results from the present study raise important questions about how social networks with friends in particular impact upon mortality. The exact mechanisms through which social networks affect health (and vice-versa) are not well understood. The Berkman model (Berkman et al., 2000) proposed that culture, socioeconomic factors, politics and social change condition the extent, shape and nature of social networks. In turn, social networks provide opportunities for 'psychosocial mechanisms' which include social support, social influence, social engagement, interpersonal contact and access to

financial and health care resources. Psychosocial mechanisms may impact upon health through behavioural, psychological, and physiological pathways (Berkman et al, 2000). If social networks are considered within this framework, networks with friends may exert an influence upon health behaviours such as smoking, alcohol consumption and exercise, variables that were controlled for in the analyses reported here through propensity scores. Friends possibly also encourage health seeking behaviour, which in turn can impact survival. Friends can have effects on depression (Anstey and Luszcz, 2002), self-efficacy, self-esteem (Lee and Shehan, 1989), coping and morale (Litwin, 2001) or a sense of personal control (Anstey et al., 2002), possibly through social engagement by reinforcing social roles (Berkman and Glass, 2000) or because interactions with friends stem from choice (Lee and Ishii-Kuntz, 1987) or selectivity (Carstensen et al., 2003).

Social network variables exerted an effect on mortality 10 years after they were measured at baseline in this study, and after a broad range of demographic, health, psychological and lifestyle variables were taken into account in the analyses. These baseline effects persisted even though many other changes may have occurred for participants in the decade following the Wave 1 interview, including widowhood, deaths of friends, siblings, children, or geographical relocation of some members of their overall social network. What these analyses do not reflect is changes in social networks among ALSA participants, and the impact of any changes upon mortality.

In summary, better social networks with friends and confidants predict survival over the following decade among ALSA participants. Strong social networks of discretionary relationships may be important for longer survival.

9 SUMMARY AND CONCLUSIONS

9.1 INTRODUCTION

The notion that one's social circumstances can have a profound impact on health is undeniably attractive, and there are numerous studies that investigate this broad hypothesis in the gerontological and epidemiological literature. Yet there is considerable variation between studies in this area in terms of design, definitions, and results. In much of the published literature, only a limited set of covariates has been controlled for in the analyses. Moreover, inadequate attention has been paid in many published studies to the issue of confounding of covariates with the effects of social relationships.

To date, there has not been a synthesized analysis of the effects of social relationships using a single, consistent operational definition, across a range of different health outcomes. Furthermore, the extant literature has not satisfactorily addressed the important question of whether different kinds of social relationships have different effects on health.

In this thesis, the effects of structural measures of social relationships on disability, residential care use, and mortality were examined. Measures that reflected total social networks and specific social networks with children, relatives, friends, and confidants were constructed, and the effects of these variables on the three health outcomes were presented.

Contemporary statistical techniques were used in the analyses and the effects of a broad range of covariates were adjusted for in the analyses using propensity scores. In the

applied epidemiological literature, propensity scores are generally calculated for binary treatment variables. There are few examples in which propensity scores have been derived for categorical variables with three or more levels. The analyses presented in this thesis represent one of the first efforts in the gerontological literature to use propensity score adjustment in a longitudinal study with a categorical, non-dichotomous, 'treatment' variable.

In this chapter, the results are considered as a whole in Section 9.2. In Section 9.3, limitations in the research conducted in this thesis are discussed. Avenues for future research are presented in Section 9.4, before the overall conclusions are presented in the final section.

9.2 SUMMARY OF MAJOR FINDINGS

9.2.1 Social networks

Based on a measurement model developed by Glass et al. (1997), CFA applied to the ALSA data demonstrated that the four specific social network types of children, relatives, friends and confidants were tenable for the ALSA study. Three alternative methods for calculating each specific social network variable were compared, and these showed that there was little difference between averaging, differential weighting or $\boldsymbol{E}(\xi \mid \mathbf{X})$. Therefore, the average of the standardized observed variables was an appropriate basis for the specific social network variables, and a total social network variable was then derived by summing the four specific social network variable scores.

9.2.2 Disability

The effects of structural components of social relationships upon disability in mobility and Nagi tasks over nine years of follow-up were analysed. After controlling for a wide

range of personal, environmental and health-related factors through propensity score adjustment, there were persistent significant effects of networks with relatives, that appeared to protect against transitions to both mobility disability and Nagi disability. Total social networks also protected against Nagi disability. There was evidence of a gradient of effect of relatives and total social networks in protecting against both Nagi disability and mobility disability. Threshold effects of these social networks were not statistically significant for the disability variables.

9.2.3 Residential care use

The effects of specific and total social networks upon residential care use were examined over Waves 1 through 6. Similar to the analyses carried out for disability, propensity score methods were used to adjust for a broad range of covariates.

The results presented in this thesis represent the first time that the effects of social networks upon residential care use in Australia have been examined. Only two previous Australian studies (McCallum et al. 2005; Wang et al. 2001) have presented a longitudinal analysis of the risk factors for nursing home admission, but neither of these studies considered the effects of social relationships. There have been no previous studies concerning the effects of social networks on low-level care use in Australia, and remarkably few studies in the international literature were found, although differences in the definition of residential care and implementation of residential care policies in different countries hamper comparisons.

The results showed specific and total social networks had little effect on use of low-level residential care over the period of the study, although there was some indication of an association between better children networks and reduced risk of transition to low-

level care. Better friend networks, better confidant networks and better total social networks were associated with reduced odds of transitions to high-level care over the course of the study. The effects of children networks on high-level care use were not significant. There was no evidence of threshold effects for any of the social network measures.

9.2.4 Mortality

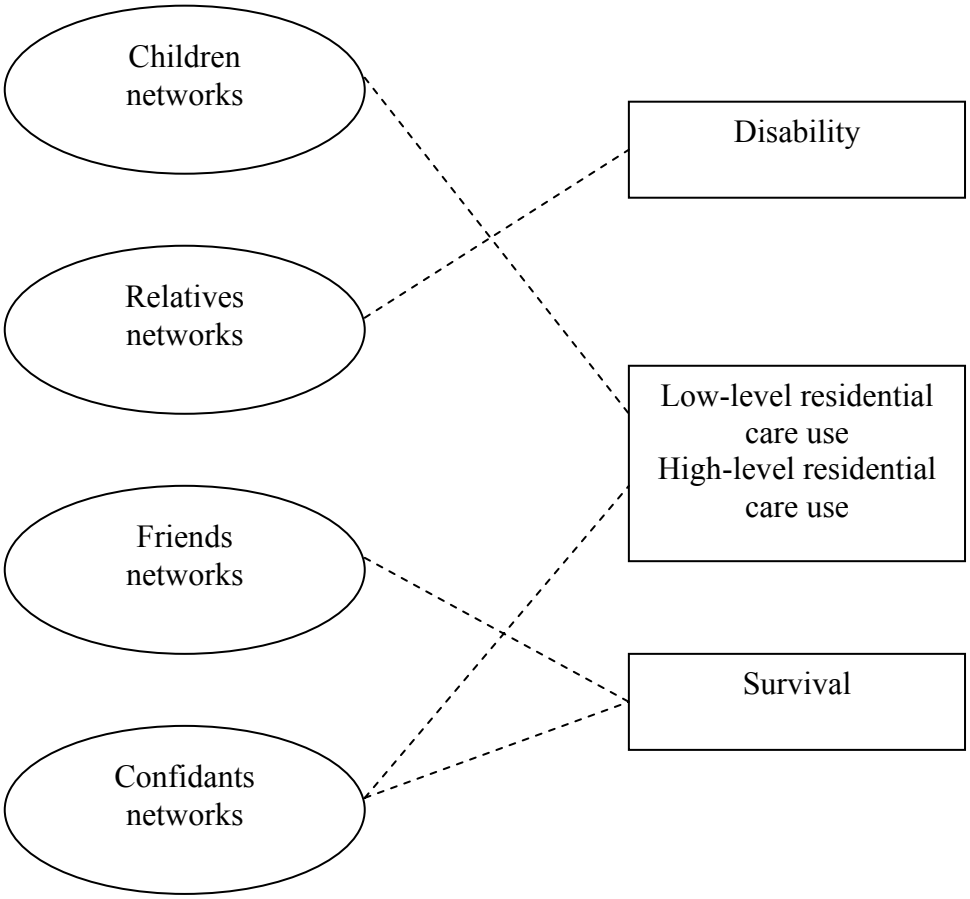
Although there is an extensive literature concerning the effects of social relationships on mortality, there is a paucity of research that has examined the effects of specific social networks upon mortality. There are four published Australian studies (Jorm et al., 1991; McCallum et al., 1994; Simons et al., 1996; Korten et al., 1999) that have examined the effects of social relationships on mortality, but none of these has compared the effects of different types of social networks on mortality.

In this thesis, it was demonstrated that greater social networks with friends and confidants had significant protective effects against mortality over a 10 year follow-up period. Networks with children and relatives did not have significant effects on mortality over the same follow-up period. This highlights the importance of disaggregating kin and non-kin networks, rather than relying on measures of total social networks. Social networks that comprise ties with whom there may be less choice about contact, such as children and other relatives, were not significant predictors of mortality in the ALSA cohort. Discretionary social networks, with friends and confidants, had a significant protective effect against death among the ALSA participants. There was a significant threshold effect for total social networks at the upper tertile.

9.2.5 Summary

There are significant and differing effects of specific social networks on the three health outcomes of disability, residential care and mortality that were considered in this thesis. Better total social networks had statistically significant effects on all three of the health outcomes considered here. Networks with relatives appear protective in terms of disability prevention and recovery. Better networks with friends and better networks with confidants were suggested to be protective against high-level residential care use and death over a decade of follow-up. Figure 9.1 summarises the major results for effects of the specific social networks on health.

Figure 9.1: Summary schema for effects of specific social networks on disability, residential care use and survival



Equally interesting is what this thesis failed to show. Children networks did not have significant effects on disability or survival, and there was only limited evidence of an effect on low-level residential care. It is possible that the measure of children networks used in this thesis did not adequately capture participants' social networks with their children. The social networks variables were deliberately constructed to measure the structural components of social relationships – the mezzo level in the Berkman model – and were operationally defined through the number and proximity of children and frequency of telephone and personal contact. Thus the nature of the social exchange with children was not captured in the social network variable.

An alternative explanation is that there is a true absence of an effect of children networks on the health outcomes considered here. As posited by Mendes de Leon et al. (1999), who similarly found no effect of children networks on disability, it may be that older people draw close to their children in anticipation of a decline in health, with an implicit expectation that children will provide some or all of the instrumental social support that is necessary. This closing of ranks in later life, more broadly explored in terms of socio-emotional selectivity theory (Carstensen et al., 2003), may mean that positive effects of social networks with children for some participants in ALSA were cancelled out by negative effects of children networks for other participants.

With respect to threshold effects, previous studies that have reported on the existence of such effects (e.g. Berkman and Syme, 1979; Blazer, 1982; Welin et al., 1985) have not been based within a framework that formally tested for threshold effects. No previous studies were identified that have investigated threshold effects of specific social networks. The results presented in this thesis are the first to formally test for the existence of such effects, and the results consistently showed that threshold effects for children, relatives, friends and confidant social networks did not exist. There was, however, evidence of a potential threshold effect at the upper tertile for mortality for total social networks, suggesting the hazard of mortality does not continue to decline for those in the upper category of total social networks.

Taken together, the findings presented here reinforce that disentangling the effects of different kinds of social networks on health is critical to understanding the impacts of social networks on the health of older people. Such research may enable the development of better interventions that target the appropriate types of social relationships for specific health outcomes.

9.3 LIMITATIONS

There are overall limitations in the methods and results presented in this thesis, as well as specific limitations within each of the three studies pertaining to disability, residential care use and mortality. The overall limitations are discussed first, before the limitations specific to each health outcome are addressed.

9.3.1 General limitations

Several limitations in the design of the ALSA study and the analyses presented in this thesis must be acknowledged, and this means the findings in this thesis must be interpreted with several caveats.

First, a wide range of covariates were included in the derivation of the propensity scores, but complete data were unavailable for some potentially important factors, such as diet or nutrition. However, given that diet and nutrition contribute to overall health, the propensity scores indirectly capture these potential effects through the inclusion of a range of health measures. It is possible that other important covariates were not measured in ALSA, and thus were not adjusted for in the analyses presented in this thesis. Nonetheless, the analyses presented in this thesis include one of the most comprehensive sets of covariates that have been considered in this area to date.

A second limitation is that ALSA was not explicitly designed to examine the effects of social networks on health. The analyses are based on self-reported data, and the propensity scores were derived from baseline data collected at Wave 1. However, these same limitations are true of the majority of studies that have considered social relationships and health in older adults. The cost of developing and running a

longitudinal study in a large cohort of older people necessitates that many disciplinary areas are included in an interview schedule to justify the financial investment, as well as the investment of time by the participants. The obvious trade-off is that depth must be sacrificed to ensure breadth of questions. Thus a new study that captures a very detailed description of social relationships *and* many covariates *and* a range of health outcome measures on close to 1500 older people is unlikely to take place in Australia in the foreseeable future.

A third limitation to consider is the possibility of non-response bias. ALSA non-respondents at Wave 1 may have been more socially isolated than those who chose to be participants. The likely effect of this is difficult to quantify. However, non-response bias has been demonstrated as minimal in other analyses of ALSA data (Anstey and Luszcz, 2002, Luszcz et al., 1997, Andrews et al., 2002). It is, however, an important point to recognize in terms of generalizability of the results.

Another point to be made is that social networks may have changed over time, but only baseline data were available in the present set of analyses. The questions used to calculate social networks in this thesis were not asked at every wave of ALSA, and thus the change in social networks from wave to wave could not be calculated. Disentangling the effects of changing social networks on transitions in health status becomes further complicated, and would further reduce the available sample size for analysis. Total network size in older people has been demonstrated as relatively stable in Dutch (Van Tilburg, 1998) and North American cohorts (Stoller and Pugliesi, 1988; Glass et al., 1995 cited in Mendes de Leon et al., 1999). The effects of changes in specific and total social networks on health remains an important issue for future research in this area.

Another limitation is the derived measures of social networks themselves. The measures developed by Glass et al. (1997) were replicated as closely as possible in this thesis and then used in subsequent analyses. There were, however, limitations in the available data and this may have affected the derivation of the social network variables. For example, participants with no living children were in the lowest category for each of the four observed variables that were used in the derivation of the children social network variable. It was also assumed that the 74 participants who had never married were never parents and these participants were not asked the questions concerning children in the ALSA Wave 1 interview.

The information available for the derivation of the confidants network variable in this study was limited to binary indicators, and did not capture the frequency of contact with confidants. It is not known how richer data concerning confidants may have changed the findings reported here.

Another possible limitation in this thesis is that adjustments for multiple comparisons were not made, adopting Rothman's position that such adjustments may be unnecessary (Rothman, 1990). The effects of social networks on disability, residential care use and survival were *a priori* expected to be relatively small (in that they were unlikely to be of the same order of magnitude as the effect of say smoking on risk of cardiovascular disease), and investigation of the different effects of social networks on health was the overall aim of this thesis. For this reason, a decision was made not to adjust for multiple comparisons. Nonetheless, like the majority of studies in this field, the possibility of inflated Type I errors due to the number of statistical tests performed cannot be ruled out.

These limitations are to some extent balanced by ALSA's strengths. The baseline data collection in the ALSA study was very rich, and captured extensive details about the older participants' lives. The inclusion of residents in aged care facilities at baseline is also a strength of the study. Many other longitudinal studies of ageing exclude older people (e.g. EPESE, LSOA, Dubbo) who live in residential care from their design. This limits the generalizability of findings to only those older people who live in the community. The heterogeneity of the ALSA sample can be viewed as a feature that distinguishes it from many other longitudinal studies of ageing. A further strength of ALSA is the breadth of the data collected from participants. Few Australian studies have captured similar amounts of information from an older cohort.

9.3.2 Limitations in analyses of disability

A limitation in the analyses of the disability data is the timing of study waves. The six waves of ALSA data considered in this thesis were collected at annual interview for the first four waves, and then roughly every three years for the final two waves. Fluctuations in the disability status of ALSA participants over a shorter time period less than one year cannot be ascertained from the available data. It is important to note that these same limitations are true in other longitudinal studies of older people, where short-term fluctuations in health status cannot be reflected in more widely-spaced interviews. Moreover, the primary interest from a policy perspective is in long-term disability, which the spacing of ALSA interviews was appropriate to detect.

9.3.3 Limitations in analyses of residential care use

One limitation in the analyses of residential care is the lack of information concerning date of entry to residential care. Thus residential care use between study waves is not

reflected in the data reported in this thesis. Participants who were alive and living in the community at one wave and dead by the next wave may have been admitted to low-level or high-level care (or both) in the intervening period, but the data available from ALSA do not capture the period spent in residential care before death.

There is some potential to capture this information for the participants who have died, since an address at the time of death is recorded on the death certificate and is potentially available. However, there are inconsistencies in the completion of this address by the certifying physicians. If a resident of an aged care facility dies shortly after admission to hospital, their address may be recorded as the hospital in some instances and the residential aged care facility in other instances.

A second limitation is that respite and permanent admissions to residential care were not differentiated in the analyses in this thesis, and this may have impacted on the results. However, less than 2% of the available places in Australian residential aged care facilities were used for respite in 2004-05 (Australian Institute of Health and Welfare, 2006) and therefore the impact of the failure to separate respite and permanent admissions in the results reported in this thesis was likely to be small.

Theoretically, separate funding and ethical approval could be sought to ascertain if an individual has at least one record in the Australian Government Department of Health and Ageing's Aged Care Payments Database, denoting use of low-level or high-level residential care and date of admission(s). However, Australian privacy laws have evolved and become much stricter since ALSA began in 1992. The original informed consent that people gave is insufficient to allow the relevant data to be extracted, even if the Department of Health and Ageing were agreeable to their release. Thus such a study

is not likely to prove feasible in this particular instance, although the possibilities that exist through data linkage are discussed further in Section 9.4.

9.3.4 Limitations in analyses of mortality

As with all studies of mortality among older people, the survival status of participants in this study was left-censored. Survival to the point at which people entered ALSA (a minimum age of 70 years) was necessary to be included in this study. It is possible that people most adversely affected by poor social networks do not survive to older age, and are thus not reflected in the data presented here.

Another limitation in the results presented here is that only all-cause mortality was considered. There was substantial variability in the way in which cause of death was completed by the certifying doctor, and this precluded a reliable coding of cause of death. For example, smoking was listed as the main cause of death on several death certificates, and it was impossible to determine if the participant died from cancer or a cardiovascular event or some other cause. In any case, there may not be sufficient statistical power to examine the effects of specific social networks on individual causes of death such as cardiovascular disease, cancer and stroke if the number of events in each category was small.

9.4 FUTURE RESEARCH

The results presented in this thesis suggest several avenues of inquiry that may be fruitful in future research endeavours. These include extensions to the analyses carried out here and the consideration of additional outcome measures. Identifying individuals with poor social networks and then targeting interventions to address the effect of improving this on subsequent health outcomes could also be explored. The potential to

link the ALSA survey data with administrative databases such as aged care and hospital morbidity records could also be examined. Some work that linked the ALSA survey data to the Medicare Benefits Schedule and Pharmaceutical Benefits Schedule in the subset of survivors in 2001 has already been conducted (Luszcz et al., 2007) and this approach could be extended to later points in time in the ALSA study.

Multilevel statistical techniques might be useful in analyses that have the Berkman model as a conceptual foundation. The incorporation of macro level variables, such as socio-economic status based on postcode of residence and the availability of regional health services, could be included in a model with social networks and micro level measures of social support. There have been few such reports concerning the effects of social relationships on health in older people identified to date. This approach also has the potential to shed new light on the effects that specific social networks have on health.

Jorm (2005) contended that there is sufficient evidence to support an intervention trial of the effects of befriending schemes in older people on mortality. When considered altogether, the results in this thesis suggest several distinct intervention trials for different health outcomes. While friends and confidants networks were shown to be significant for survival, other relatives networks were indicated as having significant protective effects against disability. Confidants and friends networks were also shown to be important in maintaining the ability to live in the community and not enter residential aged care. Trials need to move beyond interventions targeted towards spouses and children and look at the opportunities that may exist for promoting discretionary social networks and those with other relatives.

Another important avenue for research concerning the effects of friends networks on health could be in the ascertainment of the effects of friendships that formed relatively recently compared to those friendships that are long-standing. While data on the length of friendships are not available from the ALSA study, other longitudinal studies of ageing may be able to address this question.

The opportunity to examine the effects of social networks on other health outcomes among ALSA participants aside from the three outcomes considered in this thesis remains. For example, the effects of social networks on self-rated health has been reported recently in young (Melchior et al., 2003) and older (Litwin, 2006) people. These studies were limited however in that Litwin's (2006) research used cross-sectional data and the study by Melchior et al. was based on only one year of follow-up. Additional insights into this relationship could be gleaned from analysis of the effects of social networks on the self-rated health of ALSA participants. The effects of social networks on changes in cognitive status would also appear worthy of further investigation, as called for by Fratiglioni et al. (2000) in their seminal work on this topic. Research that investigates the effects of social networks on physiological outcomes may also be helpful in better understanding the pathways through which social networks exert their influence on health.

Finally, as noted by Kelman, Bass and Holman (Kelman et al. 2002), the increasing number and availability of health data resources in Australia provides a tremendous opportunity for researchers to value-add to studies of older people such as ALSA. Data linkage is particularly attractive in studies that involve older groups of people, because cognitive and other health issues can lead to significant difficulties in interviewing a representative sample of people. The $20 million investment in the national 'Population

health and clinical data linkage' funded from the National Collaborative Research Infrastructure Strategy will help to realise the full potential of health data linkage to add to existing studies of human health, and build on the extensive expertise in data linkage that exists in New South Wales and Western Australia in particular. Plans for a cross-agency data linkage unit are underway for each Australian state or territory. However, in the case of ALSA the potential of data linkage to value-add is somewhat limited, because many of the administrative health records from 16 years ago would simply no longer be available in electronic form (e.g. hospital records, aged care utilization). The greatest benefits of data linkage will come in other studies that are yet to commence, where consent processes for data linkage that ensure an individual's privacy can be incorporated from the outset of the study.

9.5 CONCLUSIONS

In this thesis, the effects of different social network types on three different health outcomes have been established. The results showed that networks with relatives were protective in terms of transitions to disability. Better networks with confidants were important in protecting against high-level residential care use and mortality. Networks with friends were shown to be protective against death over a decade of follow-up. Better total social networks were shown to be significant for all three health outcomes. There was no evidence of a threshold of effects of any type of specific social networks, suggesting there is no ceiling effect inherent in the action of specific social networks on health. There was evidence of a significant threshold effect at the second tertile of total social networks for mortality. This suggests that the positive effect of total social networks on mortality does not continue past the mid category, such that participants in mid and upper categories of total social networks had a similar hazard of mortality.

The results in this thesis also demonstrate the importance of recognizing that social networks go beyond a simple ascertainment of marital status or number of children. Whether specific kinds of social relationships, beyond spouses and children, have been given adequate weight in current policy frameworks that address the health of older people is not immediately clear. Clinicians may also usefully profit from these findings, and consider non-filial social ties when attempting to discern patient's sources of social support.

Finally, the results presented in this thesis highlight the insights into the health of older people that can be gained through the analysis of a large and complex study like ALSA. All surviving participants in the study are now aged at least 85 years, and the opportunity to collect additional information concerning the health and well-being of these older people in the very near future should not be missed.

APPENDIX 1: PEER-REVIEWED PUBLICATIONS ARISING FROM THIS THESIS

Giles, L.C., Metcalf, P.A., Anderson, C.S and Andrews, G.R. (2002) Social networks among older Australians: a validation of Glass's model.
Journal of Cancer Epidemiology and Prevention, v. 7 (4), pp. 195-204, 2002

The Effects of Social Networks on Disability in Older Australians

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Objective: To investigate the effects of total social networks and specific social networks with children, relatives, friends, and confidants on disability in mobility and Nagi functional tasks. **Methods:** Six waves of data from the Australian Longitudinal Study of Ageing were used. Data came from 1,477 participants aged 70 years or older. The effects of total social networks and those with children, relatives, friends, and confidants on transitions in disability status were analyzed using binary and multinomial logistic regression. **Results:** After controlling for a range of health, environmental, and personal factors, social networks with relatives were protective against developing mobility disability (OR = 0.89; 95% CI = 0.79 to 1.00) and Nagi disability (OR = 0.85; 95% CI = 0.74 to 0.96). Other social subnetworks did not have a consistent effect on the development of disability. **Discussion:** The effects of social relationships extend beyond disability in activities of daily living. Networks with relatives protect against disability in mobility and Nagi tasks.

Keywords: *transitions; Australian Longitudinal Study of Ageing; activity limitations; disability; social networks*

Understanding the factors that are associated with disability in older persons has been deemed a critically important public health issue

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(Guralnik, Fried, & Salive, 1996). Several theoretical models have been put forward to explain differing levels of physical disability (e.g., Pope & Tarlov, 1991; Verbrugge & Jette, 1994; World Health Organization, 1980, 2002). All of these models share the concept that disability results from a complex relationship between an individual's health, environment, personal attributes, and psychosocial factors. In this study, we incorporate these elements in specifically analyzing the effects of social networks on disability in older people. We used the International Classification of Functioning, Disability and Health (ICF; World Health Organization, 2002) as the conceptual framework in this study, and have adopted the ICF terminology in this article.

A wide range of instruments have been used for assessing activity limitations in older persons, including activities of daily living (ADL), mobility (Rosow & Breslau, 1966), and Nagi physical function tasks (Nagi, 1976). The ADL scale covers basic personal self-maintenance tasks, such as eating and toileting. Questions concerning mobility usually include whether the respondent can walk half a mile and climb stairs without help (Rosow & Breslau, 1966). In contrast, the Nagi tasks address difficulty in moving large objects, stooping, carrying heavy weights, lifting arms above shoulder level, and fine joint movement (Nagi, 1976). The factors that affect these three forms of disability may differ. Disability in mobility and Nagi tasks usually precede ADL disability in the disablement process (Verbrugge & Jette, 1994). Thus, identifying factors that protect against developing mobility and Nagi disability and promote recovery from such forms of disability may provide additional insight into the process of disability.

One aspect of environmental and personal factors that has often been overlooked in studies of transitions in disability is the effect of social relationships on the development of, and recovery from, disability. Most authors who have considered the effect of social relationships on disability have used only two waves of data (e.g.,

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Strawbridge, Cohen, Shema, & Kaplan, 1996; Unger, McAvay, Bruce, Berkman, & Seeman, 1999) and fairly crude measures of social relationships (as discussed by Glass, Mendes de Leon, Seeman, & Berkman, 1997).

Two recent studies have considered the effects of social relationships on disability using multiple waves of data (Mendes de Leon et al., 1999; Mendes de Leon, Gold, Glass, Kaplan, & George, 2001). Both of these studies showed that structural components of social relationships reduced the odds of developing ADL disability and increased the odds of recovery from ADL disability. Mendes de Leon et al. (1999) also found no effect of social networks on developing or recovering from disability in mobility. Using a different population of older persons, Mendes de Leon et al. (2001) reported that size of network and frequency of contact with friends were protective against both ADL and mobility disability, whereas no effect of network with relatives was evident. Given the differences between these findings for mobility, examination of another sample of older persons is warranted to further assess how social relationships affect mobility disability. Furthermore, the effects of social relationships on disability in Nagi tasks using multiple waves of data have not been rigorously studied in the extant literature.

The primary aim of the present study was to estimate the effects of structural aspects of social relationships on mobility and Nagi disability. A secondary aim was to compare the results obtained from binary logistic regression, comparing states of no disability to disability, to the results obtained from multinomial logistic regression, when "missing" and "deceased" were also included as response categories. The present study also adds to the existing literature in this area, as it reports findings from a longitudinal study of aging conducted in Australia.

Methods

SAMPLE

Data for this research came from the Australian Longitudinal Study of Ageing (ALSA), a large epidemiological study of aging carried out

in Adelaide, South Australia. The study has been described in detail elsewhere (Andrews, Clark, & Luszcz, 2002; Finucane et al., 1997). In brief, the ALSA sample was randomly selected from the South Australian Electoral Roll in 1992 and was stratified by geographic area of residence, gender, and 5-year age groups from 70 to 74 years through to 85 years and older. Older males were deliberately oversampled to provide sufficient numbers for longitudinal follow-up, and both community-dwelling and persons living in residential care participated. Of the eligible sample of 2,703 persons, 1,477 (55%) agreed to participate. Sample weights, adjusted for nonresponse, were calculated to reflect the population totals on December 31, 1992, in each age group, gender, and geographic area. An examination of the use of health services and record of doctors' visits suggested little difference between the responders and nonresponders in terms of health status (Andrews et al., 2002).

Ethical approval for the study was obtained from the Clinical Investigation Committee of Flinders Medical Centre in South Australia. Written informed consent was given by each participant in the study.

DATA COLLECTION AND MEASURES

To date, six waves of data have been collected from these participants. Waves 1 to 4 were annual interviews beginning in 1992, Wave 5 occurred in 1998, and Wave 6 was conducted in 2000 to 2001. Waves 1, 3, and 6 involved both clinical assessments and detailed personal interviews that captured biomedical, behavioral, economic, social, and environmental aspects of participants' lives. Waves 2, 4, and 5 consisted of a short telephone interview that captured information on major domains of health and lifestyle, including physical function.

DISABILITY

Two complementary measures of self-reported disability were considered in the present study. The first of these, mobility, is derived from Rosow & Breslau (1966). Participants were defined as having no mobility disability if they reported they were able to walk up and down a flight of stairs and walk half a mile without help. If either or both of these activities could not be completed, they were classified as

having a mobility disability. Other studies have used a similar dichotomous measure of mobility (Beckett et al., 1996; Guralnik et al., 1993; Mendes de Leon et al., 1999).

The second disability measure was derived from questions developed by Nagi (1976). Participants reported their level of difficulty in performing five tasks (pushing or pulling large objects, stooping or crouching or kneeling, lifting or carrying 10 pounds, reaching or extending arms, and writing or handling small objects). There were five response categories for each task, namely, "no difficulty," "a little difficulty," "some difficulty," "a lot of difficulty," or "just unable to do it." Participants were defined as having no Nagi disability if they reported no more than a little difficulty for all five Nagi questions. Participants who reported at least some difficulty for at least one of the five questions were classified as having a Nagi disability (Beckett et al., 1996).

For Waves 1 through 6, participants could have missing values on one or more of the component disability questions. Participants with missing values for one or both of the mobility questions at any wave were coded as missing for that wave, unless one of the nonmissing items indicated mobility disability. In this case, participants were coded as having a mobility disability (Mendes de Leon et al., 1999). Similarly, the response for participants with missing values for at least one of the five Nagi tasks within a wave was coded as missing for that wave, unless a nonmissing response to one of the tasks indicated disability. In this case, participants were classified as having a Nagi disability. Participants' mortality status was tracked throughout the study via searches of official death certificates conducted by the Epidemiology Branch of the Department of Human Services in South Australia.

SOCIAL NETWORKS

Structural measures of social networks were hypothesized as predictors of transitions to and from disability. Following Glass et al. (1997), confirmatory factor analyses of the Wave 1 data were used to develop measures of social networks, and the analyses showed that children, relatives, friends, and confidants were important social subnetworks for the ALSA participants (Giles, Metcalf, Anderson, & Andrews, 2002). The children subnetwork combined information on

the number of children, proximity of children, and frequency of personal and phone contact with children. The relatives subnetwork was composed of the number of relatives, apart from spouse and children, to whom the participant felt close, and the frequency of personal and phone contact with such relatives. Similarly, the friends subnetwork captured the number of close friends, personal contact and phone contact. The confidant subnetwork reflected the existence of confidants and whether the confidant was a spouse. A total social network score was calculated as the sum of the children, relatives, friends, and confidant subnetwork scores. All of the component variables, such as number of children and frequency of contact with children, were self-reported and standardized prior to the derivation of the social network variables.

COVARIATES

The effects of a number of personal, environmental, and health-related factors on transitions to and from disability were considered in the analyses to control for confounding. These covariates were derived from self-reported data from the Wave 1 interview and were operationalized as follows.

Age group was classified as 70 to 74 years, 75 to 79 years, 80 to 84 years, and 85 years or older on December 31, 1992. Gender was also included as a covariate, as was geographic area of residence. Place of residence was classified as community or residential care. Current marital status was classified as married/partnered or not married. Household income was coded as less than or equal to $AUD12,000 per annum, more than $AUD12,000 per annum, or missing. This cut-off point for income was chosen because it was similar to the single persons' aged pension rate in 1992. The age at which the participant left full-time education was categorized as less than or equal to 14 years of age or more than 14 years of age.

Self-rated health was classified as excellent/very good, good, and fair/poor. The number of chronic conditions was derived from self-reported information on whether each participant had ever suffered from arthritis, cancer (excluding non-melanocytic skin cancer), chronic bronchitis or emphysema, diabetes, fractured hip, heart attack, heart condition, hypertension, myocardial infarction, or

osteoporosis. Hearing difficulty and difficulty with corrected vision were based on self-report.

Depressive symptomatology was assessed using the 20-item CES-D Scale (Radloff, 1977), with scores of 17 or more out of a possible 60 suggesting symptoms of depression. Cognitive function was assessed using a subset of items from the Mini–Mental State Examination. Items included those assessing orientation, registration, attention and calculation, and recall (see Teng, Chui, Schneider, & Metzger, 1987) for a maximum score of 21, with scores of 16 or below (out of a possible 21) indicative of cognitive impairment (Luszcz, 1998; Luszcz, Bryan, & Kent, 1997).

Participants were classed as current, former, or never smokers based on their responses to questions concerning smoking. Participants were classified as having a hazardous drinking problem if their score on the 10-item AUDIT scale was 8 or more (Barbor, de la Fuente, Saunders, & Grant, 1992). Questions about the exercise undertaken in the previous fortnight enabled participants to be classified as exercisers or sedentary (Finucane et al., 1997).

ANALYSIS

To adjust for the complex sampling design, all descriptive analyses were based on weighted data. The covariates were included in all of the analyses. As the covariates were defined using Wave 1 data, social network variables and other covariates were treated as time invariant in the analyses.

The disability variables were treated as either dichotomous variables, with categories of no disability and disability, or polytomous variables, with response categories of no disability, disability, missing, and deceased. When disability was analyzed as a dichotomy, generalized estimating equations, assuming an unstructured form for the working correlation matrix, were used to fit binary logistic regressions that accounted for the correlation between observations from the same participant (Liang & Zeger, 1986). When the polytomous disability variables were analyzed, unordered multinomial logistic regression models were fitted. In these models, the Huber-White robust variance estimator was used to account for the multiple observations from each participant (Huber, 1967; White, 1982).

Two approaches were used to ascertain the effects of social relationships on mobility and Nagi disability. First, the participants who were not disabled at the previous wave were selected. The effects of social relationships on the transition to disability (binary logistic regression) or disability/missing/deceased (multinomial logistic regression) at the subsequent wave were estimated. These models were of the form logit $\Pr(Y_{ij} = 1 | Y_{ij-1} = 0) = x_{ij}'\beta_0$. Similarly, participants who were disabled at the beginning of each wave were selected, and the effects of social relationships on remaining disabled (binary logistic regression) or disabled/missing/deceased (multinomial logistic regression) were estimated. These models were of the form logit $\Pr(Y_{ij} = 1 | Y_{ij-1} = 1) = x_{ij}'\beta_1$. Second, models that used all data were fitted to estimate the effects of social networks on disability (Diggle, Heagerty, Liang, & Zeger, 2002). Disability status in the previous wave was used as a covariate in this second set of models. These models are written as logit $\Pr(Y_{ij} = 1 | Y_{ij-1} = y_{ij-1}) = x_{ij}'\beta + y_{ij-1}x_{ij}'\alpha$. Simpler nested models that excluded the interaction between disability in the previous wave and covariate terms were also assessed and fitted where appropriate. We assumed that the missing data mechanisms were ignorable and missing at random (Beckett et al., 1996; Diggle et al., 2002). Stata was used in all analyses (StataCorp, 2001).

Results

The responses to the covariates for the 1,477 participants are summarized in Table 1. The average age at selection was 77.2 ± 5.9 years, and females represented 61% of the weighted sample. More than half of the participants had left school before the age of 15 years, and half of the sample was married/partnered. Although the majority of participants lived in the community, 137 of the participants were in residential care. Participants most commonly had one morbid condition, and 14% of the participants showed signs of cognitive impairment. Almost half of the participants did not exercise. The social network scores had ranges of –1.67 to 1.17 (children), –1.05 to 2.02 (other relatives), –1.54 to 1.19 (close friends), –1.68 to 0.77 (confidants), and –5.69 to 4.97 (total).

Tables 2 and 3 present the wave-to-wave transitions in mobility and Nagi disability during the course of the study. Table 2 shows that for

Table 1
Frequencies (%) for Covariates Used in Modeling Disability Measures

Covariate	n	*Unweighted %*	*Weighted %*
Age at selection			
70 to 74	379	25.7	39.4
75 to 79	352	23.8	29.1
80 to 84	341	23.1	18.5
85+	405	27.4	13.0
Gender			
Male	928	62.8	39.4
Female	549	37.2	60.6
Place of residence			
Community	1,340	90.7	93.5
Institution	137	9.3	6.5
Marital status			
Married	771	52.2	51.5
Not married	705	47.8	48.5
Missing	1		
Age left school			
≥ 15 years	633	43.3	44.4
≤ 14 years	830	56.7	55.6
Missing	14		
Household income			
> $12,000	779	52.7	51.9
< $12,000	590	39.9	41.2
Missing	108	7.3	6.8
Number of morbid conditions			
0	264	17.9	16.4
1	494	33.4	31.5
2	421	28.5	30.0
3	190	12.9	14.4
≥4	108	7.2	7.7
Cognitive impairment			
No impairment	1,246	84.8	86.2
Impairment	199	15.2	13.8
Missing	32		
Self-rated health			
Excellent/very good	563	38.2	39.2
Good	440	29.9	31.3
Fair/poor	469	31.9	29.5
Missing	5		
Depressive symptomatology			
No depressive symptoms	1,205	81.6	83.9
Depressive symptoms	195	13.2	12.0
Missing	77	5.2	4.1

(continued)

Table 1 (continued)

Covariate	n	*Unweighted %*	*Weighted %*
Hearing difficulty			
No	726	49.3	56.2
Yes	746	50.7	43.8
Missing	5		
Vision difficulty			
No	1,035	73.4	77.4
Yes	375	26.6	22.6
Missing	67		
Alcohol problem			
No	1,401	95.6	96.0
Yes	65	4.4	4.0
Missing	11		
Smoking status			
Never smoker	661	45.2	52.8
Ex-smoker	677	46.3	38.3
Current smoker	123	8.4	8.8
Missing	16		
Sedentary			
No	794	54.5	55.2
Yes	663	45.5	44.8
Missing	20		

mobility, between 15% and 19% of participants made the transition from no disability to being disabled at the subsequent wave. Conversely, between 7% and 24% of participants made the transition from a mobility disability to no disability at the next wave. For Nagi tasks (Table 3), between 21% and 38% of participants made the transition from no disability to disability at the following wave. Between 5% and 21% of the participants recovered from disability to no disability at the subsequent wave, and the proportion recovering also generally decreased in time. For both disability measures, the proportion of participants developing disability increased in time, and the proportion recovering from disability decreased with subsequent waves.

The relationship between the mobility and Nagi disability variables was weak (Spearman's *r* ranged from .38 to .52 across the six waves), showing that although there is some overlap, the mobility and Nagi variables are capturing different aspects of disability.

Table 2
Transitions From Each Mobility State During Six Waves of ALSA

	Disability Status				
Disability Status at Previous Wave	*No Disability (%)*	*Disability (%)*	*Missing (%)*	*Deceased (%)*	*Total* n
No disability					
1-2	73.2	15.6	8.8	2.4	987
2-3	77.0	15.0	5.5	2.5	841
3-4	75.0	14.6	7.4	3.0	761
4-5	65.9	17.5	8.6	8.0	663
5-6	54.0	19.4	19.3	7.3	510
Disability					
1-2	24.3	57.7	10.4	7.6	473
2-3	18.3	65.7	5.2	10.8	429
3-4	15.1	66.7	7.2	11.0	441
4-5	13.4	54.1	11.5	21.0	418
5-6	7.0	44.0	17.6	31.4	357
Missing					
1-2	23.1	52.9	8.8	5.2	17
2-3	24.4	23.3	44.3	8.0	145
3-4	19.8	11.4	65.7	3.1	134
4-5	9.5	10.6	67.8	12.1	179
5-6	8.6	10.1	49.4	31.9	230
Deceased					
2-3					62
3-4					141
4-5					217
5-6					380

Note. Results were weighted according to the sampling scheme. ALSA = Australian Longitudinal Study of Ageing.

DISABILITY AS A DICHOTOMOUS VARIABLE

Results are presented for participants (a) who were not disabled at the previous wave, (b) who were disabled at the previous wave, and (c) using all available data.

The binary logistic regression analyses, summarized in Table 4, showed there was a protective effect of relatives network on developing mobility disability in the overall analysis and a marginal effect of

Table 3
Transitions From Each Nagi State During Six Waves of ALSA

	Disability Status				
Disability Status at Previous Wave	*No Disability (%)*	*Disability (%)*	*Missing (%)*	*Deceased (%)*	*Total* n
No disability					
1-2	66.2	22.2	8.2	3.4	577
2-3	61.2	31.9	4.9	2.0	569
3-4	69.9	22.0	5.0	3.1	444
4-5	64.8	20.7	6.8	7.7	442
5-6	35.9	37.6	19.4	7.1	376
Disability					
1-2	20.9	65.1	9.4	4.6	879
2-3	11.8	74.5	5.7	8.0	714
3-4	15.4	70.8	6.4	7.4	758
4-5	12.5	60.6	10.4	16.5	655
5-6	5.7	51.8	18.3	24.2	495
Missing					
1-2	23.1	52.9	8.8	15.2	21
2-3	24.4	23.3	44.3	8.0	132
3-4	19.8	11.4	65.7	3.1	134
4-5	9.5	10.6	67.8	12.1	163
5-6	8.6	10.1	49.4	31.9	226
Deceased					
2-3					62
3-4					141
4-5					217
5-6					380

Note. ALSA = Australian Longitudinal Study of Ageing.

relatives network protecting against disability among the group who were disabled at the previous wave. The effect of networks with children, friends, and confidants was far from statistical significance in all binary analyses of mobility disability.

Networks with relatives and the total social network had significant protective effects against developing Nagi disability for those participants who were not initially disabled (Table 4). However, for the initially disabled participants, there were no effects of social networks on Nagi disability. The analysis of the overall data showed a significant protective effect of networks with relatives and total networks. As was

Table 4
Dichotomous Disability Analyses: Effects of Social Network Variables for Transitions to Mobility and Nagi Disability for Participants Initially Not Disabled, Initially Disabled, and Overall

	Mobility			*Nagi*		
	OR	*95% CI*	p	*OR*	*95% CI*	p
Initially not disabled						
Children	0.97	0.83-1.14	.720	0.93	0.79-1.10	.380
Relatives	0.91	0.77-1.07	.236	0.84	0.73-0.98	.026*
Friends	1.01	0.86-1.20	.862	0.97	0.82-1.16	.766
Confidant	1.13	0.94-1.35	.190	0.90	0.75-1.08	.258
Total network	0.99	0.92-1.07	.811	0.91	0.84-0.99	.022*
Initially disabled						
Children	1.14	0.92-1.41	.229	0.94	0.79-1.11	.472
Relatives	0.81	0.66-1.01	.063	0.91	0.76-1.09	.313
Friends	0.91	0.72-1.16	.450	0.91	0.76-1.09	.297
Confidant	0.97	0.75-1.27	.847	1.03	0.85-1.24	.793
Total network	0.97	0.87-1.08	.579	0.94	0.87-1.02	.140
All data						
Children	1.05	0.93-1.19	.394	0.98	0.85-1.12	.761
Relatives	0.87	0.76-0.98	.027*	0.84	0.74-0.96	.012*
Friends	1.02	0.90-1.16	.756	0.91	0.79-1.05	.210
Confidant	1.09	0.95-1.25	.223	0.96	0.82-1.12	.625
Total network	1.00	0.94-1.06	.880	0.92	0.86-0.98	.014*

Note. Analyses adjusted for all covariates.
*$p < .05$.

noted for mobility, there was no effect of networks with children, friends, or confidants on Nagi disability in any of the binary analyses.

DISABILITY AS A POLYTOMOUS VARIABLE

Table 5 shows the effects of social networks on transitions to disability using all data in multinomial logistic regressions.

For those participants who were not initially disabled in mobility, networks with children had a significant protective effect against being missing (Table 5). The effect of networks with relatives was a significant predictor of death. Total social networks were protective against having a missing response.

Table 5
Polytomous Disability Analyses: Effects of Social Network Variables for Transitions to Mobility Disability and Nagi Disability for Participants Initially Not Disabled, Initially Disabled, and Overall

	Mobility			*Nagi*		
	OR	*95% CI*	p	*OR*	*95% CI*	p
Initially not disabled						
Children						
Disabled	0.98	0.84-1.15	.823	0.95	0.80-1.12	.522
Missing	0.78	0.66-0.93	.005*	0.81	0.64-1.02	.074
Deceased	0.97	0.78-1.20	.750	0.94	0.71-1.25	.692
Relatives						
Disabled	0.89	0.76-1.04	.149	0.85	0.73-0.98	.027*
Missing	0.92	0.77-1.10	.337	0.94	0.75-1.18	.592
Deceased	1.36	1.10-1.68	.005*	1.13	0.87-1.46	.364
Friends						
Disabled	1.03	0.88-1.21	.735	0.97	0.82-1.15	.755
Missing	0.89	0.74-1.08	.231	1.17	0.91-1.52	.225
Deceased	0.93	0.73-1.17	.511	0.93	0.71-1.21	.565
Confidants						
Disabled	1.12	0.94-1.34	.195	0.90	0.75-1.07	.220
Missing	0.99	0.82-1.20	.950	0.90	0.68-1.19	.442
Deceased	1.00	0.77-1.29	.978	1.00	0.71-1.41	.984
Total Network						
Disabled	0.99	0.92-1.07	.842	0.92	0.85-0.99	.027*
Missing	0.90	0.83-0.98	.014*	0.95	0.84-1.07	.361
Deceased	1.04	0.92-1.17	.552	1.01	0.87-1.16	.905
Initially disabled						
Children						
Disabled	1.13	0.92-1.39	.244	0.97	0.82-1.15	.742
Missing	0.92	0.70-1.21	.532	0.74	0.60-0.92	.007*
Deceased	1.10	0.86-1.39	.446	0.96	0.79-1.18	.713
Relatives						
Disabled	0.82	0.67-1.00	.054	0.91	0.76-1.08	.288
Missing	0.90	0.68-1.20	.481	0.94	0.74-1.20	.638
Deceased	0.88	0.69-1.12	.283	1.05	0.84-1.32	.645
Friends						
Disabled	0.92	0.73-1.16	.470	0.93	0.78-1.11	.430
Missing	0.82	0.61-1.11	.205	0.75	0.60-0.95	.017*
Deceased	0.74	0.56-0.97	.032*	0.78	0.62-0.98	.035*
Confidants						
Disabled	0.96	0.74-1.26	.784	1.02	0.84-1.23	.842
Missing	0.89	0.63-1.24	.482	1.05	0.82-1.34	.724
Deceased	0.83	0.60-1.13	.233	0.93	0.73-1.18	.560

Table 5 (continued)

	Mobility			*Nagi*		
	OR	*95% CI*	p	*OR*	*95% CI*	p
Total Network						
Disabled	0.96	0.87-1.07	.491	0.95	0.88-1.03	.239
Missing	0.92	0.80-1.06	.240	0.88	0.79-0.97	.012*
Deceased	0.92	0.81-1.04	.162	0.93	0.84-1.04	.201
All data						
Children						
Disabled	1.04	0.93-1.16	.530	0.99	0.86-1.14	.895
Missing	0.82	0.71-0.94	.005*	0.79	0.67-0.93	.005*
Deceased	1.00	0.86-1.15	.967	0.97	0.82-1.15	.758
Relatives						
Disabled	0.89	0.79-1.00	.042*	0.85	0.74-0.96	.011*
Missing	0.94	0.81-1.08	.379	0.88	0.75-1.04	.137
Deceased	1.07	0.93-1.24	.354	1.00	0.84-1.18	.959
Friends						
Disabled	1.03	0.92-1.15	.646	0.96	0.84-1.10	.579
Missing	0.91	0.78-1.06	.231	0.91	0.77-1.08	.301
Deceased	0.86	0.73-1.00	.051	0.83	0.70-0.99	.034*
Confidants						
Disabled	1.08	0.95-1.23	.225	0.96	0.83-1.11	.589
Missing	1.00	0.85-1.17	.964	0.94	0.78-1.13	.529
Deceased	0.93	0.79-1.10	.414	0.90	0.74-1.10	.298
Total network						
Disabled	1.00	0.94-1.05	.900	0.94	0.88-1.00	.036*
Missing	0.92	0.86-0.99	.022*	0.89	0.82-0.96	.003*
Deceased	0.97	0.90-1.05	.440	0.93	0.85-1.01	.085

Note. Analyses adjusted for all covariates.
*$p < .05$.

Among participants who were initially disabled in mobility, there was a marginally significant protective effect of relatives networks against being disabled at the subsequent wave. There was also a protective effect of networks with friends against dying by the subsequent wave.

When all mobility data were considered, the results showed that children and total social networks both had protective effects against a missing response. Networks with relatives had a significant protective effect against being disabled at the subsequent wave. Networks with

friends had a marginally significant protective effect against being deceased by the subsequent wave.

Networks with confidants had no effects on mobility disability in the multinomial logistic regression analyses.

Similar analyses were undertaken for disability in Nagi tasks (Table 5). For the participants who were not initially disabled, there was a protective effect of relatives and total social networks against becoming disabled.

Restricting the analyses to those participants who were initially disabled in Nagi tasks showed a significant protective effect of the children, friends, and total social networks against a missing response. The friends network was also a significant protective factor against death. Neither relatives nor confidant networks had an effect on any of the outcomes among the initially Nagi-disabled participants.

When all available data were analyzed, a significant protective effect of larger children network on missing status was observed. Networks with relatives had a significant protective effect against Nagi disability. Networks with friends protected against death by the subsequent wave. Total social networks had a significant protective effect against being disabled or missing. The effect of networks with confidants was not significant for any of the three outcome states of Nagi disability.

Discussion

The effects of structural components of social relationships on disability in mobility and Nagi tasks were analyzed in this study. After controlling for a wide range of personal, environmental, and health-related factors, there were persistent protective effects of social networks on disability. However, the protective effects varied according to the type of social network and were strongest for relatives.

For mobility disability, the results differed depending on whether the analyses were based on the two-category (binary logistic regression) or the four-category (multinomial logistic regression) definition of disability. The strongest effect of social subnetworks was that for relatives in the binary logistic regression. However, the multinomial logistic regression suggested an effect of relatives in preventing

mobility disability among those not already disabled as well as overall. No other subnetworks had a significant effect on the development of or recovery from mobility disability. In contrast, Mendes de Leon et al. (2001) found that more frequent contact with friends decreased the risk of developing mobility disability, and contact with relatives had no effect on the development of mobility disability. Earlier work by these authors suggested the effects of social subnetworks, and total social networks on mobility were not statistically significant (Mendes de Leon et al., 1999). Our analysis shows that specific social networks are of moderate importance in preventing mobility disability and suggest that networks with relatives may have particular effects on the development of mobility disability.

Both the binary and multinomial analyses showed that relatives networks were important in protecting against Nagi disability. Unger et al. (1999) reported that more social ties was associated with less Nagi disability 7 years later but did not present a breakdown of the effects of specific types of social networks on Nagi disability. Given the paucity of literature concerning the effects of social networks on Nagi disability, we believe our results represent an important first step in the consideration of specific types of social networks on Nagi disability, and that additional research in this area is necessary.

Taken together, our findings show that social networks with relatives may be particularly important in preventing the development of disability or promoting recovery from mobility and Nagi disability. This adds to the work of Seeman, Bruce, and McAvay (1996), who showed that women with more close ties with relatives were less likely to experience the onset of new or recurrent ADL disability. Our findings suggest that social networks with relatives have effects in the disablement process that precede the onset of ADL disability and that disability in mobility and Nagi tasks are affected by social networks with relatives.

The analyses showed that networks with children and total networks were protective against having a missing response for both outcomes at subsequent waves. Children were important contacts in the ALSA study when tracing participants at Waves 2 through 6, and this may explain the protective effect of children network against missing status. It is also possible that networks with children provide more

instrumental support than do networks with friends or relatives, and this support led to the participants' being less likely to refuse and easier to trace if they had changed addresses between study waves. However, the total social network was also protective against a missing response, suggesting that not only children but general social integration is an important enabling factor for participation in a longitudinal study.

The finding that networks with children had no effect on developing or recovering from disability was somewhat unexpected, but the result is consistent with other recent work concerning mobility (Mendes de Leon et al., 1999; Mendes de Leon et al., 2001). Networks with children may have competing effects because children can provide supportive resources as well as be a source of stress to their older parents. This negation of any benefits of social networks with children could be one reason for the lack of effect of children network on mobility disability and Nagi disability. Another explanation may be that older persons turn to their children first when anticipating a decline in health, and this decline in health offsets any benefits that might come from networks with children (Mendes de Leon et al., 1999).

The findings also showed that there were significant protective effects of friends on becoming deceased but no significant effects of friends networks on disability status. Earlier work in this area has been equivocal with respect to the effect of friends networks on mobility disability, and little previous work has considered the effect of social networks on Nagi disability. Mendes de Leon et al. (1999) showed that friends networks were not associated with mobility disability. In contrast, Mendes de Leon et al. (2001) showed that contacts with friends were protective against developing mobility disability. Given these findings, the balance of evidence suggests that the effect of friends networks on the development of mobility or Nagi disability is fairly minimal.

No effects of confidant networks on either mobility or Nagi disability were found in the present study. This supports the similar findings of Mendes de Leon et al. (1999) who showed no effect of confidant networks on ADL disability and mobility disability. It may be that, like children, confidants are turned to when a decline in health is anticipated or experienced, and this decline in health and associated greater

needs could negate any benefits that might come from networks with confidants.

Our statistical approach has extended previous work concerning disability transitions (Anderson et al., 1998; Beckett et al., 1996; Mendes de Leon et al., 1997; Mendes de Leon et al., 1999; Mendes de Leon et al., 2001; Rudberg, Parzen, Lamond, & Cassel, 1996). The findings from both sets of analyses presented here suggest consistent effects of networks with relatives on preventing Nagi disability and slight evidence of an effect of relatives networks on preventing mobility disability. To our knowledge, this is the first report to systematically compare these analytic approaches in examining the effect of social networks on transitions in disability.

The results from the present study raise important questions about how social networks with relatives affect disability in later life. Several pathways have been suggested to explain the effects that social relationships have on health (Berkman, 1985). Under one hypothesized pathway, social ties influence the adoption of health behaviors, both positive and detrimental, and also influence the success of behavior change. One interpretation of our findings is that relatives, in particular, offer advice about better health habits and access to health services, and act as role models. As well, participants may be more receptive to advice offered by relatives than to advice offered by a child, spouse, or friend. Burg and Seeman (1994) reviewed some of the negative effects of family members on health and showed that spouses in particular may promote poor health habits. It is possible that relatives, in contrast to children and spouses, offer health advice less frequently, which makes its reception more welcome. In this way, it is possible that participants with larger relatives networks have a lower risk of developing disability because of the preventive health behaviors that the networks with relatives foster. Relatives also possibly share early-life environments and health behaviors, so that relatives networks may be a proxy measure of genetic factors, including survivorship and health.

Another possible pathway is a direct link between physiological mechanisms and social relationships, with subsequent beneficial effects on disability. More positive, supportive relationships with relatives may be associated with beneficial physiological effects, which in turn could promote recovery from disability and prevent transitions to

disability. Why social networks with relatives would have differential physiological effects to other network types remains unclear, and this is an area that could potentially benefit from future research.

The findings reported here should be interpreted recognizing that there are several potential strengths as well as limitations of the present study. The strengths of the study include its longitudinal design, the richness of the baseline data, an Australian setting, and the population base for the study cohort. Limitations of our study include the basing of definitions of social networks and covariates on Wave 1 data only, and that the research reported here is a secondary analysis of data not expressly collected to examine social networks and disability. As well, the waves of the study were unequally spaced, but the differing time between waves was not incorporated in our analyses.

In summary, the present research has confirmed the protective role of specific social relationships in the disablement process prior to the development of ADL disability. Having a strong social network, particularly with relatives, is important in protecting against the development of disability, particularly in Nagi tasks.

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Effect of social networks on 10 year survival in very old Australians: the Australian longitudinal study of aging

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Do social networks affect the use of residential aged care among older Australians?

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Abstract

Background: Older people's social networks with family and friends can affect residential aged care use. It remains unclear if there are differences in the effects of specific (with children, other relatives, friends and confidants) and total social networks upon use of low-level residential care and nursing homes.

Methods: Data were drawn from the Australian Longitudinal Study of Ageing. Six waves of data from 1477 people aged ≥ 70 collected over nine years of follow-up were used. Multinomial logistic regressions of the effects of specific and total social networks on residential care use were carried out. Propensity scores were used in the analyses to adjust for differences in participant's health, demographic and lifestyle characteristics with respect to social networks.

Results: Higher scores for confidant networks were protective against nursing home use (odds ratio [OR] upper versus lower tertile of confidant networks = 0.50; 95%CI 0.33–0.75). Similarly, a significant effect of upper versus lower total network tertile on nursing home use was observed (OR = 0.62; 95%CI 0.43–0.90). Evidence of an effect of children networks on nursing home use was equivocal. Nursing home use was not predicted by other relatives or friends social networks. Use of lower-level residential care was unrelated to social networks of any type. Social networks of any type did not have a significant effect upon low-level residential care use.

Discussion: Better confidant and total social networks predict nursing home use in a large cohort of older Australians. Policy needs to reflect the importance of these particular relationships in considering where older people want to live in the later years of life.

Background

At any point in time in Australia, around one in ten older people have left their home to receive either respite or permanent care in a residential care facility [1]. The Australian aged care system is a tiered system that comprises both community and residential aged care places. Residential aged care may be provided as either 'high-level' or 'low-level' care, depending on clients' needs. In the Australian aged care system, high-level care is equivalent to nursing home care in other countries, and reflects high levels of medical and personal care needs. Low-level residential care (also referred to as 'hostel care' in the Australian sys-

tem) provides help and housing to older people who do not need continual, high level access to nursing care but have physical, medical, psychological or social care needs that cannot be met through living in the community [2].

Both high-level and low-level residential aged care services are predominantly funded and regulated by the Australian Government [1]. Currently a total of 88 residential aged care places per 1,000 people aged 70 years or more is provided in the Australian aged care system [1]. Religious and charitable organizations deliver the majority of residential aged care services in Australia, although publicly listed companies and small community-based organizations also deliver residential aged care services to a significant number of older people.

Unlike some other countries with specific taxation levies or social insurance programs, the Australian Government funded services are financed from general taxation revenue and user contributions [1]. From both individual and societal perspectives, there are high personal and financial costs associated with admission to residential care [3]. For the Australian Government, the costs of supplying aged care services are forecast to increase from $7.8 billion in 2002–2003 to $106.8 billion in 2042–2043 [4].

A substantial body of U.S. dominated research has identified factors including increasing age, female gender, lack of a marital partner, greater income, better education, lack of home ownership, more comorbid conditions, poorer self-rated health, prior nursing home use, more physical disability, and poorer cognitive status as significant predictors of residential care use, as reviewed recently [5,6]. The findings have been drawn from both cross-sectional and longitudinal studies with follow-up time that varied between one and twenty years, with the median length of follow-up equal to three years.

Social networks with family and friends may be particularly important in providing care to older people, and may thereby delay or prevent admissions to residential care [7-12]. However, few studies have distinguished between social networks with family and those with friends and separately examined their effects on use of residential care. Among those studies that have made this distinction, findings are conflicting. For example, Wolinksy et al. [13] reported non-kin networks were protective but kin networks were not, whereas Freedman [9] demonstrated networks with daughters and siblings, but not sons, were protective against nursing home use. The meta-analysis by Gaugler et al. [5] demonstrated that a greater number of children was protective against nursing home admission, although it is worth noting that only three studies were available for pooling in their analysis of the effects of children, reflecting the paucity of evidence in this area.

It is also likely that social networks are themselves related to some of the factors that have been demonstrated as predictors of residential care use. Therefore it is difficult to make a clear interpretation of the effects of specific and total social networks on residential care based on the existing literature.

Surprisingly little is known about the factors that predict residential care use in Australia. Two recent Australian studies have examined some health, function and lifestyle risk factors for entry to nursing homes. McCallum et al. [13] showed increasing age, incontinence, impaired respiratory flow, more disability, depression, male gender and lower alcohol intake were associated with nursing home use over a 14 year period. Wang et al. [14] found older age, poorer self-rated health, walking disability, current smoking, and lower alcohol consumption were risk factors for admission to nursing homes. However, both studies were set in narrowly defined geographic regions in the same Australian state and were focussed on cardiovascular and ophthalmologic factors respectively. Thus the generalisability of the findings to the wider Australian population remains unclear. Furthermore, social networks and low-level residential care were not considered in these studies. These important gaps in knowledge are addressed in the research reported here.

The primary aim of the present study was to consider the effects of specific types of networks (i.e. those with children, other relatives, friends, confidants, and total social networks) upon use of both low-level residential care and nursing homes in a large sample of older Australians, adjusting for the effects of a wide range of health, function and lifestyle variables. A secondary aim was to examine the effects of putative risk factors on use of nursing homes to add to the knowledge gained from the two previous Australian studies.

Methods

Sample

This study uses data from the Australian Longitudinal Study of Ageing (ALSA), a large epidemiological study which aims to increase our understanding of how social, biomedical, behavioural, economic and environmental factors are associated with age-related changes in the health and social well-being of older persons. The study has been described in detail elsewhere [15,16]. In brief, ALSA began in 1992 and is continuing with survivors of the original cohort. The primary sample for ALSA was randomly selected from the South Australian Electoral Roll, and was stratified by Local Government Area (LGA), gender, and five year age groups from 70–74 years through to 85 years and over. Older males aged 85 years or more were deliberately over-sampled to provide sufficient numbers of males for longitudinal follow-up. Persons identified

through the Electoral Roll were defined as eligible for the study if they were resident in the Adelaide Statistical Division and were aged 70 years or more on 31 December 1992. Both community-dwelling and people living in residential care were eligible to take part in ALSA, although the majority of participants (91%) were living in the community at baseline interview. A total of 1477 eligible people took part in wave 1 (56% response rate).

Ethical approval for the study was granted by the relevant institutional ethics committee, and each study participant provided written informed consent.

Data collection and measures

Eight waves of data have been collected from participants between 1992 and 2005, with fieldwork for a ninth wave due to commence in late 2007. In the present article, data from the first six waves were analysed. Waves 1 to 4 were annual interviews that began in 1992, and consenting participants were re-interviewed in 1993, 1994 and 1995. Wave 5 occurred in 1998, and wave 6 was conducted in 2000–2001. Waves 1, 3 and 6 involved detailed personal interviews that covered demographic, medical, psychological, social and economic areas of participants' lives. As well, clinical assessments of participants were carried out in these waves. The clinical examination included anthropometric, neuropsychological, physical performance, balance, and gait measures. Both the interview and clinical assessment were carried out in the participant's usual place of residence. Waves 2, 4 and 5 each consisted of a brief telephone interview that concentrated mainly on health and lifestyle.

Residential care use

At each interview, participants were classified as living in the *community*, *low-level residential care*, or *nursing home*. For waves 2 through 6, participants were classified as *missing* if they refused an interview or were untraceable. Ongoing searches of the database of official death certificates identified the participants who had *died*, and this approach has been validated previously by the authors [17].

Variables that summarized any use of low-level care or nursing homes over the nine-year study period were also created. For low-level care use, participants were classified as *never using*, *using low-level care*, *already in nursing home at wave 1*, or *missing*. Participants who died without known use of residential care were classified as *never using*. An analogous variable was created to reflect nursing home use. Participants' status was classified as missing if use of the relevant residential care could not be ascertained from the available data.

Social networks

Adapting the approach of Glass and colleagues [18], confirmatory factor analyses of the wave 1 data were used to develop measures of social networks with children, other relatives, friends, confidants and total social networks. The derivation of the social network variables has been reported previously [19]. Briefly, the children network combined information on the number and proximity of children, and frequency of personal and phone contact with children. The relatives network was composed of the number of relatives, apart from spouse and children, the participant felt close to, and the frequency of personal and phone contact with such relatives. Similarly, the friends network captured the number of close friends, and frequency of personal and phone contact with friends. The confidant network reflected the existence of confidants and whether the confidant was a spouse. A total social network score was calculated as the sum of the children, relatives, friends, and confidant scores. Social network variables were then categorised according to their tertiles, and the tertile classification for each social network was used in further analyses.

Propensity scores

A range of personal, health, and lifestyle variables were considered important covariates (see Table 1). Geographic area (with 24 levels that designate locality) was also included as a covariate, but is excluded from Table 1 for space considerations. There were many covariates and their distributions were unbalanced among the social network categories, in that participants in different social network categories (i.e. low, medium or high) tended to have different demographic, health and lifestyle characteristics.

In randomized controlled trials, group assignment is, by definition, randomly allocated and so the differences in observed covariates between treatment groups should be minimized. However, in observational studies such as ALSA, there is no manipulation of 'treatment' assignment, and so there is the potential for large differences between observed covariates in the different treatment groups. Ignoring these differences could potentially lead to biased estimates of treatment effects. Traditional methods of adjusting for observed covariates in analyses, such as matching or stratification, may be difficult to use if there are a large number of covariates. Regression adjustment can also be problematic. Missing values in one or more covariates for an individual will result in all data for that individual being dropped from a regression analysis unless estimation of the missing covariate values is carried out. Another potential problem in regression adjustment is that finding and fitting an appropriate functional form for each covariate may be difficult.

Table 1: Summary of baseline covariates and association with any nursing home use over study period

Variable	Classification	n (%)	Odds ratio (95%CI) (n = 909)[a]
Age group	70–74	379 (25.7%)	Referent
	75–79	352 (23.8%)	4.2 (1.7 – 10.3)
	80–84	341 (23.1%)	3.7 (1.5 – 9.0)
	85+	405 (27.4%)	4.1 (1.7 – 9.8)
Gender	Male	928 (62.8%)	Referent
	Female	549 (32.2%)	0.7 (0.4 – 1.2)
Education	Left school >14 yrs	633 (42.9%)	Referent
	Left school ≤14 yrs	830 (56.2%)	1.0 (0.6 – 1.5)
	Missing	14 (0.9%)	
Marital status	Married/de facto	771 (52.2%)	Referent
	Widowed	586 (40.0%)	1.2 (0.5 – 2.8)
	Single	120 (8.1%)	1.4 (0.8 – 2.4)
Household income	>$AUD12,000	779 (52.7%)	Referent
	≤$AUD12,000	590 (39.9%)	2.0 (1.2 – 3.2)
	Missing	108 (7.3%)	
Home ownership	Owns home	1038 (71.0%)	Referent
	Renting	242 (16.4%)	0.8 (0.5 – 1.5)
	Other	50 (3.4%)	0.4 (0.1 – 1.8)
	In residential care	137 (9.3%)	4.8 (2.5 – 9.0)
Number of chronic conditions[b]	0	264 (17.9%)	Referent
	1	494 (33.4%)	1.3 (0.7 – 2.4)
	2	421 (28.5%)	0.9 (0.5 – 1.8)
	3+	298 (20.2%)	0.6 (0.3 – 1.2)
Self-rated health	Excellent/very good	563 (38.1%)	Referent
	Good	440 (29.8%)	1.2 (0.7 – 2.0)
	Fair/poor	469 (31.8%)	1.4 (0.8 – 2.5)
	Missing	5 (0.3%)	
Hearing difficulty	No	726 (49.2%)	Referent
	Yes	746 (50.5%)	1.5 (1.0 – 2.3)
	Missing	5 (0.3%)	
Difficulty with (corrected) vision [43]	No	1035 (70.1%)	Referent
	Yes	375 (25.4%)	1.3 (0.8 – 2.1)
	Missing	67 (4.5%)	
Mobility disability [44,45]	No disability	949 (64.3%)	Referent
	Disability	506 (34.3%)	1.5 (0.9 – 2.4)
	Missing	22 (1.4%)	
Depressive symptoms CES-D [46]	<17/60	1181 (80.0%)	Referent
	≥17/60	219 (14.8%)	1.2 (0.7 – 2.1)
	Missing	77 (5.2%)	
Cognitive function [40, 47]	>16/21	1221 (82.7%)	Referent
	≤16/21	219 (14.8%)	1.6 (0.9 – 2.7)
	Missing	37 (2.5%)	
Alcohol consumption (AUDIT) [48]	<8/10	1401 (94.9%)	Referent
	≥8/10	65 (4.4%)	1.0 (0.3 – 3.0)
	Missing	11 (0.7%)	
Exercise status [16, 49]	Exerciser	794 (53.8%)	Referent
	Sedentary	663 (44.9%)	1.0 (0.7 – 1.6)
	Missing	20 (1.4%)	
Smoking status	Never	661 (44.8%)	Referent
	Former	667 (45.2%)	0.6 (0.4 – 1.0)
	Current	123 (8.3%)	0.5 (0.2 – 1.2)
	Missing	16 (1.1%)	

a: analysis based on data from 909 participants with complete information on both nursing home use (n = 1078) and risk factors (n = 1243)
b: self-reported ever suffering from arthritis, cancer, chronic bronchitis or emphysema, diabetes, fractured hip, heart attack, heart condition, hypertension, osteoporosis, stroke

Propensity scores have been proposed as an alternative method to adjust for a set of covariates [20,21]. Most applications of propensity scores to date have involved simple cross-sectional studies with binary treatments. More recent work [21] that extends the derivation of propensity scores to treatments with multiple categories is applicable in the present study. In our study the 'treatment', social network tertile, has three categories corresponding to the low, mid, or high tertile of the relevant social network score for each of the specific and total social networks.

We turn now to a more formal definition of propensity scores, and first consider their derivation for a binary treatment. Let Z_i be an indicator variable of assignment to a treatment for individual i, such that $Z_i = \begin{cases} 1 & \text{if treated} \\ 0 & \text{if control} \end{cases}$.

The propensity score $p(\boldsymbol{x}_i)$ is defined as the conditional probability of assignment to treatment versus control given a vector of observed covariates $\boldsymbol{x}_i$. More formally $p(\boldsymbol{x}_i) = \Pr(Z_i = 1 | \boldsymbol{X}_i = \boldsymbol{x}_i)$ under the assumption that, given the $\boldsymbol{X}_i$, the Z_i are independent – that is, $\Pr(Z_1 = z_1, ..., Z_n = z_n \mid \boldsymbol{X}_1 = \boldsymbol{x}_1, ..., \boldsymbol{X}_n = \boldsymbol{x}_n) = \prod_{i=1}^{n} p(\boldsymbol{x}_i)^{z_i} \{1 - p(\boldsymbol{x}_i)\}^{1-z_i}$. In other words, $p(\boldsymbol{x}_i)$ is a measure of the probability that an individual would have been treated based on only the individual's covariate information [22]. Propensity scores 'balance' the observed covariates, in that the conditional distribution of $\boldsymbol{X}_i$ given $p(\boldsymbol{x}_i)$ is the same for individuals, irrespective of whether they receive treatment or control. In other words, Z_i and $\boldsymbol{X}_i$ are conditionally independent given $p(\boldsymbol{x}_i)$. The success of the propensity scores in balancing the covariates can be checked through simple comparisons of the treatment and control groups that adjust for the propensity scores in the analyses [22].

Applications of propensity score adjustment with more than two treatment categories have not been widely reported. For three or more levels of treatment, Joffe and Rosenbaum [21] showed that if the distribution of treatment doses given $\boldsymbol{X}_i$ is accurately described by McCullagh's proportional odds model, then stratifying on $b(\boldsymbol{x}_i) = \boldsymbol{x}_i'\beta$ where β is a $p \times 1$ vector of parameters, will balance $\boldsymbol{X}_i$ across several dose groups. More generally, it is possible that the distribution of doses Z given a large number of covariates may depend on the covariates through only a small number of linear functions of $\boldsymbol{X}$, say $\boldsymbol{X}\mathbf{G}$ for some matrix $\mathbf{G}$. Then controlling for the several variables in $\boldsymbol{X}\mathbf{G}$ will 'tend to balance the ... variables in $\boldsymbol{X}$' [21]. For example, if a multinomial logistic regression model was adequate to describe $\Pr(Z_i = z | \boldsymbol{X}_i = \boldsymbol{x}_i)$ for some $z = 0, 1, ..., c$, then $\boldsymbol{X}\mathbf{G}$ would be an $n \times c$ matrix in which each of the c columns defined a propensity score for level c of the treatment dose. Because of the linear dependence of the c^{th} propensity score on the first c-1 propensity scores, analyses would adjust for the first c-1 propensity scores.

To obtain propensity scores in the present analysis, an ordinal logistic regression of each of the social network tertiles on the covariates was initially fit for participants with complete covariate data (n = 1243). A pragmatic approach was adopted so that if, for a given participant, an observation was missing for at least one of the covariates, a propensity score was estimated using the subset of covariates with complete data for that participant. In this way, a propensity score was estimated for every participant, not only those participants with complete data for all covariates, and a propensity score was estimated for each pattern of missing covariate observations. Thus for every participant, the propensity scores were estimated using the maximum covariate information available for that participant.

Ordinal logistic models were appropriate for the distribution of the children, relatives, friends and total social network variables given the observed covariates, but not for confidants. For the confidant network variable, a multinomial logistic regression model was used. The resulting conditional probabilities of being in each of the three confidant network categories defined the three propensity scores for the confidant social network [23,24]. Because of the linear dependence of the third propensity score on the other two, only the first two propensity scores were included in subsequent analyses of the effect of the confidant social network upon use of residential care.

Rosenbaum and Rubin [20], based on work by Cochran [25], stated that five strata based on the propensity score would remove over 90% of the bias in each of the covariates. Thus when an ordinal logistic model was used, participants were sub-classified into quintiles based on the propensity scores. When the multinomial logistic model was used, participants were sub-classified into nine strata based on the joint distribution of their first two propensity scores. The propensity score strata for each participant was included in all analyses.

The balance of the covariates in each of the propensity score strata in the present study was examined by chi-square tests of association of each covariate with each of the categorised social network variables [23]. A total of 25 out of 484 comparisons for balance status of the covariates (5%) were statistically significant at $P < 0.05$. This

indicated that the propensity score method produced balance in the observed covariates similar to that which would be expected by randomization of these covariates across the social network tertiles. On this basis, it was determined that the propensity scores provided an adequate adjustment.

Statistical analysis

Several analyses of place of residence were conducted. First, the effects of the putative risk factors on any nursing home use over the study period were examined to enable comparison with previous Australian studies [13,14]. An unordered, multinomial, multiple logistic regression model that included the factors shown in Table 1 and geographic area was fit.

Second, separate logistic regression models of i) any low-level care use and ii) nursing home use across the study period on each social network variable were fit, adjusting for propensity score strata. Sensitivity analyses, in which missing values were imputed as never used (most conservative) or all used (most extreme), were conducted to compare the effects of different assumptions regarding missing values with the analyses that used only available data.

Finally, the place of residence across the six study waves was longitudinally analysed, with response categories of community, low-level care, nursing home, or dead possible at each wave. A separate multinomial logistic regression model of place of residence at waves 2–6 on each social network was fit, adjusting for propensity score strata, study wave, and place of residence at the previous wave. The Huber-White robust variance estimator was used to account for the repeated observations ($n \leq 5$ observations corresponding to waves 2–6) from each participant [26,27].

Results

Table 1 summarises the baseline characteristics of the 1477 participants. The average age at selection was 79.8 years (SD = 6.9), and close to two-thirds of the sample were male. More than half of the participants had left school before the age of 15 years, and approximately half of the sample was married/partnered. Participants most commonly had one morbid condition, and 15% of participants showed some signs of cognitive deficits. More than half of the participants were former or current smokers, and almost half of the participants were sedentary. Also shown in Table 1 are odds ratios that describe the association of any nursing home use with each of the risk factors. These results are described later in this section.

Across the entire study period, a total of 778 participants (53%) did not use low-level care, or died without use, and a further 136 participants (9%) were either in a nursing home at wave 1 or moved directly to a nursing home from the community. Low-level care was known to have been used over the study period by 189 participants (13%). Information on use of low-level care could not be ascertained for 374 participants (25%) because they were alive but not interviewed at one or more waves, and thus their use of residential care at the missing wave(s) could not be determined.

Over the nine years of the study, 883 participants (60%) never used a nursing home or died without use, while 195 participants (13%) used a nursing home. Nursing home use could not be ascertained for the remaining 399 participants (27%), for the same reason as those with missing low-level care information.

The place of residence at each wave is shown in Table 2. The percentage of the surviving cohort living in the community decreased over the nine years from 91% at wave 1 to 82% at wave 6. Between 6% and 8% of participants lived in low-level care at each of the waves. The proportion of participants who were resident in nursing homes increased over time, from 3% at wave 1 to 12% at wave 6.

A total of 909 participants had complete data concerning any nursing home use across the nine-year study period and the putative risk factors. As summarized in Table 1, age group, lower household income, lack of home ownership and hearing difficulty were significant risk factors for nursing home use over the study period.

The effects of social networks on use of low-level care and nursing home use were then explicitly considered. As shown in Table 3, better social networks with children, confidants and total social networks appeared protective

Table 2: Place of residence at each wave

Year	Wave	Community	Low-level care	Nursing Home	Missing	Dead
1992	**1 (n)**	1,340	92	45	0	0
	(% all)	91	6	3	0	0
1993	**2 (n)**	1,126	83	51	137	80
	(% all)	76	6	4	9	5
	(% alive)	89	7	4		
1994	**3 (n)**	1,030	80	61	113	193
	(% all)	70	5	4	8	13
	(% alive)	88	7	5		
1995	**4 (n)**	900	74	62	150	291
	(% all)	61	5	4	10	20
	(% alive)	87	7	6		
1998	**5 (n)**	646	64	51	210	506
	(% all)	44	4	4	14	34
	(% alive)	85	8	7		
2000	**6 (n)**	412	28	60	215	762
	(% all)	28	2	4	14	52
	(% alive)	82	6	12		

Shown are number and per cent of all participants (% all) and surviving participants (% alive).

Table 3: Summary of effect of social networks upon any nursing home use and any low-level care use

	Low-level care[a]		Nursing Home[b]	
	OR	**95% CI**	**OR**	**95% CI**
Any use over study period				
Children				
Mid tertile	1.44	0.97 – 2.15	1.02	0.69 – 1.50
Upper tertile	0.97	0.64 – 1.46	0.60	0.40 – 0.90
Overall χ^2_2 [c]	4.81		8.60*	
Relatives				
Mid tertile	0.98	0.66 – 1.44	0.81	0.56 – 1.18
Upper tertile	1.00	0.65 – 1.53	0.76	0.50 – 1.17
Overall χ^2_2	0.02		1.81	
Friends				
Mid tertile	1.05	0.71 – 1.55	0.78	0.54 – 1.14
Upper tertile	1.29	0.85 – 1.95	0.70	0.46 – 1.06
Overall χ^2_2	1.56		3.13	
Confidants				
Mid tertile	1.53	1.04 – 2.24	0.67	0.46 – 0.97
Upper tertile	1.10	0.67 – 1.79	0.49	0.31 – 0.77
Overall χ^2_2	5.16		10.75*	
Total				
Mid tertile	1.49	1.01 – 2.21	0.55	0.37 – 0.81
Upper tertile	1.25	0.80 – 1.95	0.54	0.35 – 0.83
Overall χ^2_2	4.05		12.21*	

Lower tertile is referent category in all analyses
a: Complete data available for 1103 cases.
b: Complete data available for 1078 cases.
c, *: χ^2on 2 df;values > 5.99 significant at P < 0.05

against any nursing home use across the study period, after adjusting for propensity score strata. However, only the upper tertile of children networks in comparison to the lower tertile had a significant effect on any nursing home use. Moreover, there was no evidence of a gradient of the effect of children networks on any nursing home use.

There was no significant effect of the specific or total social network variables upon low-level care use. The findings were robust to assumptions regarding the use of residential care by participants with missing data, as the sensitivity analyses did not differ substantively from the main results.

Table 4 summarises the longitudinal analysis of the effect of social networks on place of residence, adjusted for propensity score strata, study wave, and residence at previous wave. As these results show, specific and total social networks did not have a significant effect upon low-level care use. The significant effect observed for the friends network was due to the protective effect of better friend networks upon survival. Higher scores for confidant networks appeared protective against nursing home use (odds ratio [OR] upper versus lower tertile of confidant networks = 0.50; 95%CI 0.33–0.75). Similarly, a significant effect of upper versus lower tertile for the total social network was observed (OR = 0.62; 95%CI 0.43–0.90).

Discussion

The effects of specific and total social networks on residential care use were examined over a nine year period, using propensity score methods to adjust for a broad range of covariates. Longitudinal analyses showed better confidant networks and better total social networks were associated with reduced odds of nursing home admission over the course of the study. There was weaker evidence of a significant effect of better children networks on reduced odds of nursing home use, and there was no evidence of an effect of children networks in the longitudinal analyses. There was no significant effect of social networks with other relatives or friends on nursing home use. Furthermore, the results suggested specific and total social networks had little effect on use of low-level residential care over the period of the study.

Increasing age, lower income, and hearing difficulty were shown to be significant risk factors for nursing home use across the course of the study, adding to previous Australian research in this area. The finding regarding hearing difficulty adds more evidence to the need for adequate assessment of sensory impairments at the time of assessment for nursing home placement [14] and ongoing monitoring of auditory acuity. In contrast to visual acuity, hearing difficulties may go unnoticed, as the behavioural consequences may not be immediately obvious in the context of competing demands on staff time and attention. The effect of income on risk of nursing home admission is equivocal in the international literature, with some authors reporting reduced income to increase risk [28,29], while others have shown higher income is a risk factor for nursing home admission [30-33]. The results for income reported here possibly reflect that older Australians with a lower income may not be able to purchase support services to assist them to continue to live in the community, and so are more likely to move to residential care. Furthermore, higher income may be a disincentive to nursing home use in Australia. Substantial entry costs or ongoing costs in addition to the Australian Age Pension can be levied by individual facilities according to means-tested criteria. Issues of equity and access to residential care must remain high on the agenda for Australian aged care policy makers.

Table 4: Summary of effects of social networks upon place of residence across study period

	Low-level care OR	95% CI	Nursing home OR	95% CI	Dead OR	95% CI
Children						
Mid tertile	1.03	0.74 – 1.44	1.24	0.89 – 1.72	1.11	0.87 – 1.42
Upper tertile	0.68	0.48 – 0.96	0.85	0.60 – 1.21	1.02	0.79 – 1.31
Overall χ^2_6 = 8.6[a]						
Relatives						
Mid tertile	0.93	0.67 – 1.29	0.72	0.53 – 1.00	0.95	0.75 – 1.20
Upper tertile	0.92	0.64 – 1.33	0.74	0.51 – 1.07	1.07	0.82 – 1.38
Overall χ^2_6 = 13.5						
Friends						
Mid tertile	1.28	0.91 – 1.81	0.99	0.70 – 1.38	0.92	0.73 – 1.17
Upper tertile	1.22	0.83 – 1.78	0.74	0.52 – 1.07	0.72	0.56 – 0.93
Overall χ^2_6 = 26.4						
Confidants						
Mid tertile	0.95	0.69 – 1.32	0.70	0.51 – 0.97	0.84	0.67 – 1.06
Upper tertile	0.86	0.57 – 1.31	0.50	0.33 – 0.75	0.73	0.56 – 0.94
Overall χ^2_6 = 31.0						
Total						
Mid tertile	0.77	0.55 – 1.08	0.57	0.40 – 0.81	0.76	0.60 – 0.97
Upper tertile	0.94	0.67 – 1.34	0.62	0.43 – 0.90	0.84	0.65 – 1.08
Overall χ^2_6 = 30.7						

Lower tertile is referent category in all analyses. Community dwelling is referent response category.

a: χ^2_6 test of effect of social network variable; values > 12.59 significant at P < 0.05.

Confidant networks were significantly protective against nursing home use in this study, suggesting a close, emotionally supportive relationship with another person is beneficial in preventing or delaying nursing home use. The importance of a confidant to mental and physical health is well known [34-36] but the translation of that effect to a reduction in risk of nursing home use has not been shown previously. Further research is clearly warranted to examine the repeatability of this finding in other settings and countries.

Social networks with relatives and friends had no significant effect on use of residential care. Children networks appeared to have some protective effect against any nursing home use over the study period, but this finding did not extend to the results from the longitudinal analyses. Those with fewer non-kin social supports may have smaller networks of human resources to draw upon for maintenance of community living status [12,37]. Other research has shown significant protection against nursing home use arising from having daughters and siblings [8]. Our research suggests that the core network of confidants, and to a lesser extent children, is more important than other specific networks in delaying or preventing use of nursing homes in Australia. The striking impact of absence of confidants may reflect the consequences of reduced emotional support that permitted continued residence in the community, which would be consistent with Carstensen's socioemotional selectivity theory [38]. Social networks of any of the types considered here had minimal effect upon use of low-level care.

Several limitations to the study must be acknowledged. ALSA non-respondents may have been more socially isolated than participants, although non-response bias has been demonstrated as minimal in other analyses of ALSA data [15,39,40]. The analyses were based on self-reported data and adjusted for covariates that were measured at wave 1. Social networks may have changed over time, but the social networks considered in the present study were based on only wave 1 data. However, total network size has been demonstrated as relatively stable over a long follow-up period in a study of older Dutch people [41]. Furthermore, disentangling the effects of time-varying social networks may be difficult as changes in social networks may be a consequence of changes in place of residence. A final limitation is that date of entry to residential care was not available, and thus residential care use between study waves was not reflected in the data.

Arguably these limitations are balanced by ALSA's strengths, which include the rich baseline data that enabled propensity score adjustment, the broad sample, and the Australian setting, which expands the generalisability

of the role of social networks in the use of residential care. The follow-up time in the present study is also notably longer than that of many other international studies in this area. Our results add not only to the general body of knowledge concerning risk factors for residential care use, but also extend the literature to encompass the specific role played by social networks in this important transition. In future research, we will track place of death for study decedents which will reduce the proportion of missing data concerning the use of residential care over time.

ALSA took place against a background of reforms in Australian aged care [42] that may have had an impact on the use of residential care services independent of the risk factors considered here. One of the most significant reforms saw the assessment for entry to low-level and high-level residential aged care merged into one system in 1997. It is important to note that the policy changes did not affect an individual's eligibility for residential aged care, but streamlined the administrative processes concerning assessment criteria. An individual's eligibility for residential aged care is ascertained by Aged Care Assessment Teams (ACAT) against standardized criteria that include functional status, health and living arrangements. The persistent effects of social networks on use of nursing homes over a long period of follow-up and over and above the effects of a range of other variables suggest that an individual's social milieu needs to be reflected more strongly in eligibility criteria, particularly for high-level residential care. The results of the present study also highlight the importance of recognizing that social networks go beyond a simple ascertainment of marital status or number of children. It may be possible to incorporate the findings from the present study in better screening assessments by ACATs for residential care eligibility. Policymakers may need to reconsider whether social relationships have been given adequate weight in the current assessment and entry process.

The effects of social networks on residential care use have not been examined previously in an Australian context. We have shown that social networks with children and total social networks, especially those with confidants, predict nursing home use over nine years in a large cohort of older Australians. Policy needs to reflect the importance of these particular relationships, and incorporate these along with the expectations of future cohorts of older people about where they want to live in later life.

Competing interests

The author(s) declare that they have no competing interests.

Authors' contributions

LG conceived of the present study, participated in the design and conduct of the statistical analysis plan and had primary responsibility in drafting the manuscript. GG participated in the design of the study and the statistical analysis plan and participated in drafting of the manuscript. ML participated in the design of the study and in drafting of the manuscript. GA conceived of and directed the Australian Longitudinal Study of Ageing, and participated in the design of the present study.

LG, GG and ML read and approved the final manuscript. GA commented on early drafts of the manuscript but was unable to approve the final version due to his death in May 2006.

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APPENDIX 2: ETHICAL APPROVAL

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Bedford Park South Australia 5042

Telephone (08) 204 5511
International 618 204 5511

Committee on Clinical Investigation
Extension 4507
AV:CMH

Facsimile (08) 204 4006 International 618 204 4006

21 January 1992

Professor G.R. Andrews
Centre for Ageing Studies
254 Greenhill Road
GLENSIDE S.A. 5065

Dear Professor Andrews

re: Research Application 9/88 - "Australian Longitudinal Study of Ageing

The amendments to your research application which was originally approved 22 February 1988 (Minute 1462) and annually reviewed, have been received. The application, as amended, has been approved.

This approval is for a period of one year. Application for re-approval must be made annually. Please note that if this trial involves normal volunteers it will be necessary for you to keep a record of their names and you will be required to supply this list with your annual report.

You are reminded that the Committee on Clinical Investigation must approve the content and placement of advertisements for the recruitment of volunteers.

The Committee must be notified and approve of any changes (e.g. additional procedures, modification of drug dosage, changes to inclusion or withdrawal criteria, changes in mode and content of advertising) in the investigational plan particularly if these changes involve human subjects.

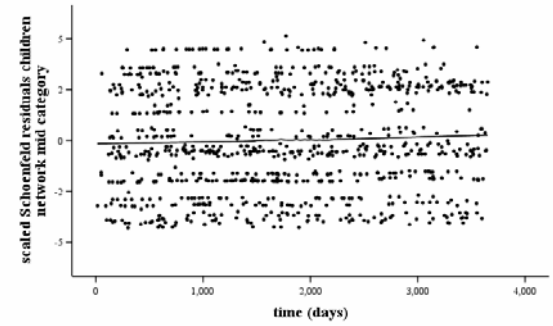
The safe and ethical conduct of a trial is entirely the responsibility of the investigators. While the Committee on Clinical Investigation takes care to review and give advice on the conduct of trials, approval by the Committee on Clinical Investigation is not an absolute confirmation of safety, nor does approval alter in any way the duties, obligations and responsibilities of investigators. It is the duty of the chief investigator to notify the Committee on Clinical Investigation should there be a change in the base for a decision made by the Committee.

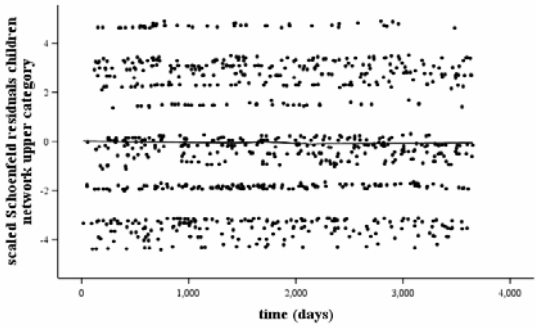
Yours sincerely,

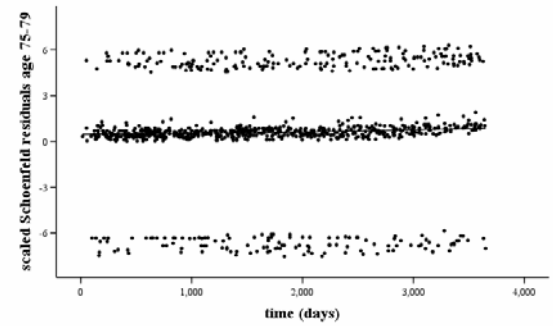
A. Vedig
Chairman
Committee on Clinical Investigation

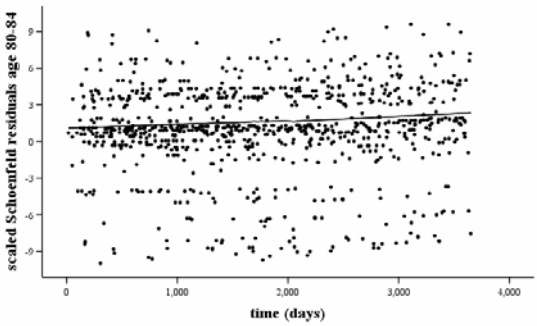
APPENDIX 3: PLOTS OF SCHOENFELD RESIDUALS FOR FITTED PROPORTIONAL HAZARDS MODELS

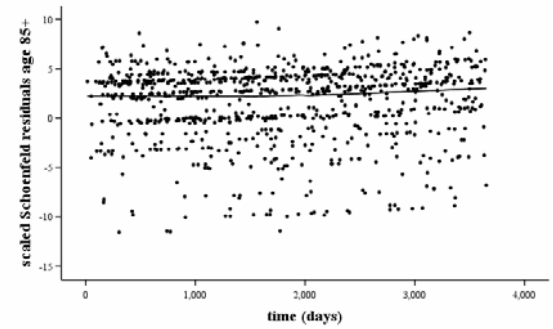
Children social networks

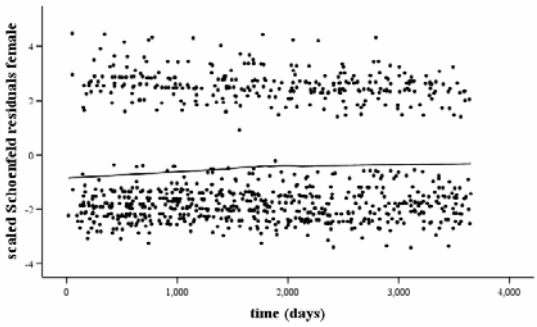


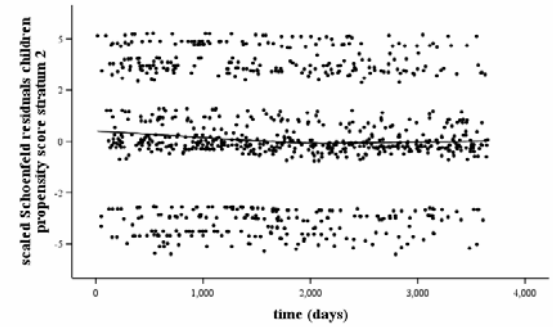


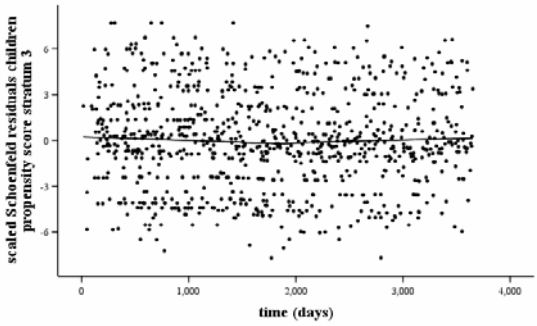


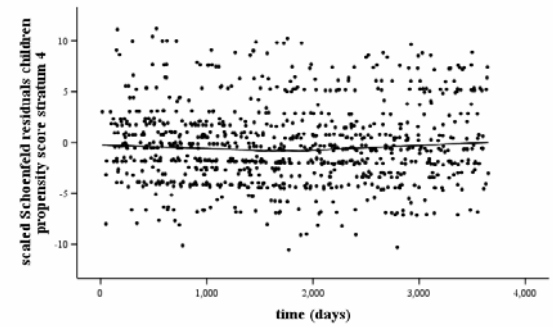


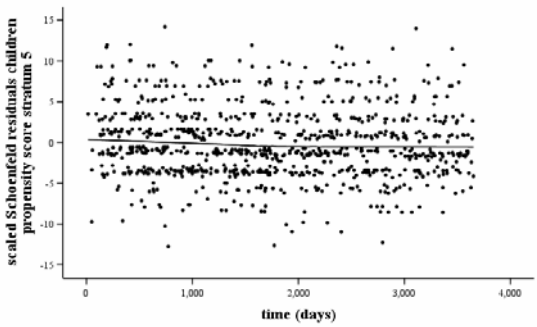




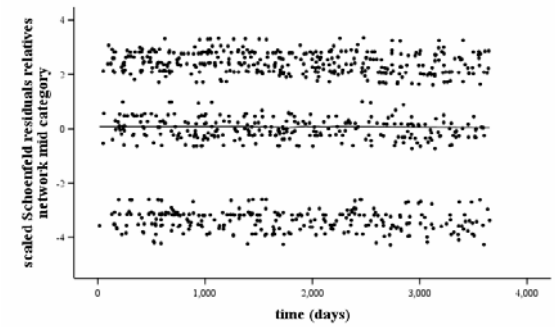


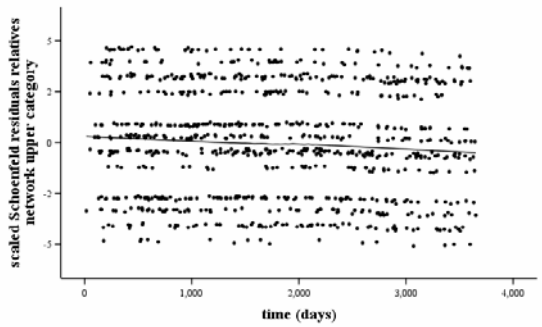


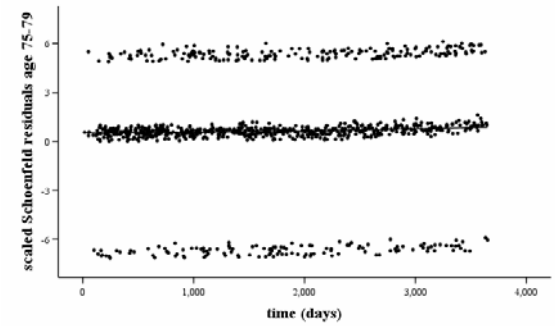


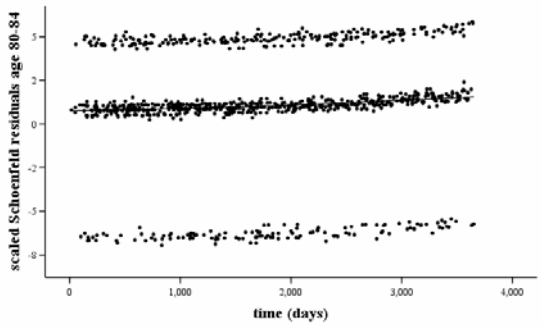


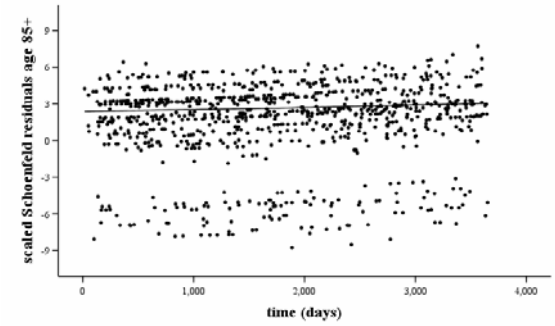
Relatives social networks

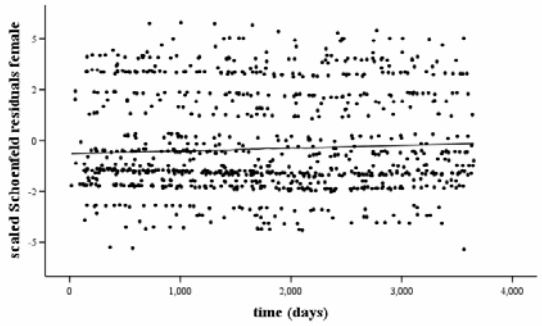


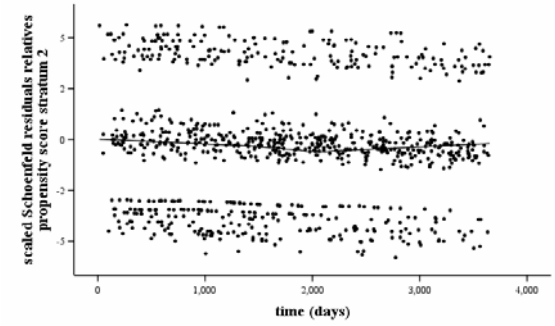


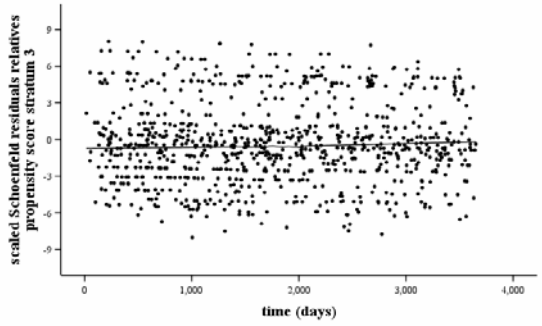


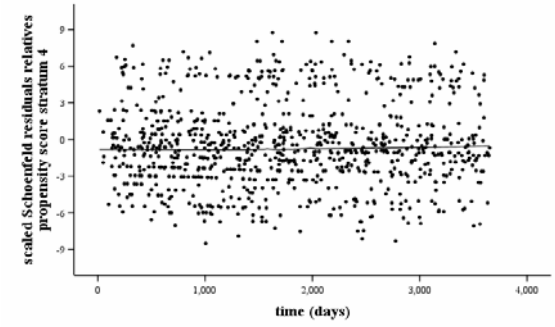


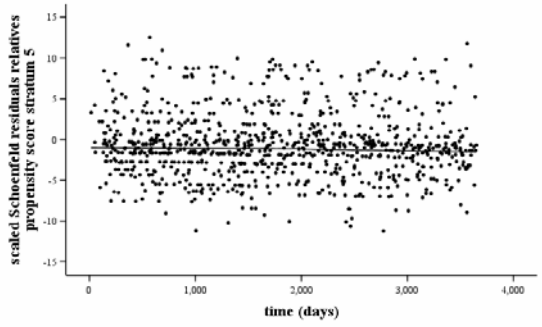












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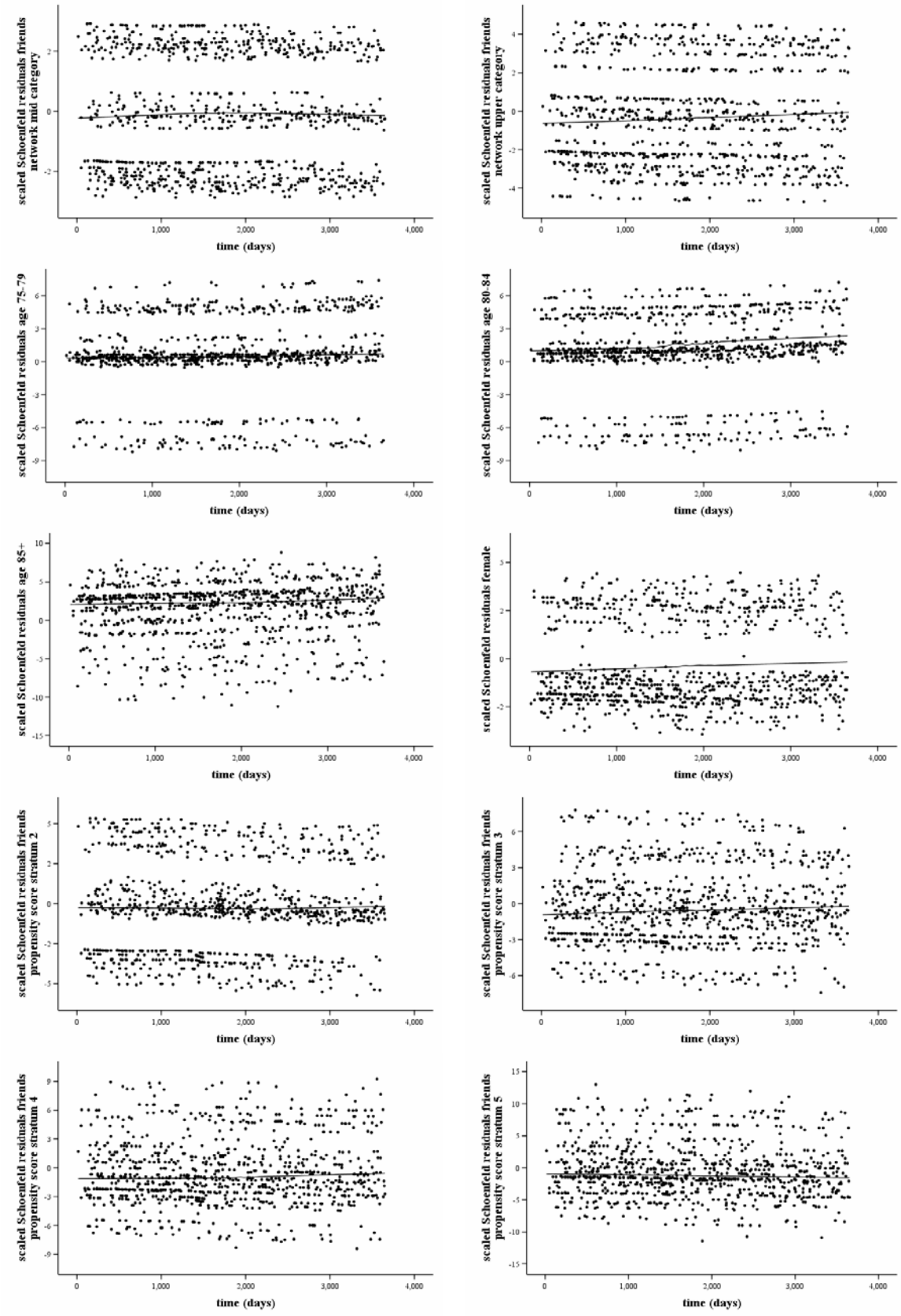

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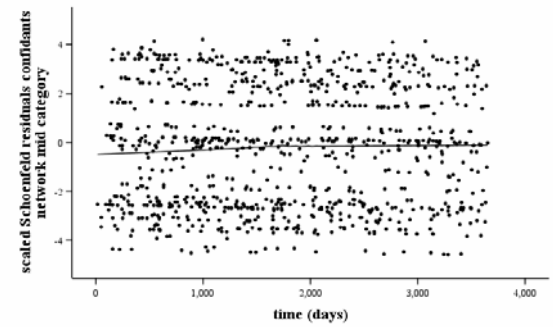

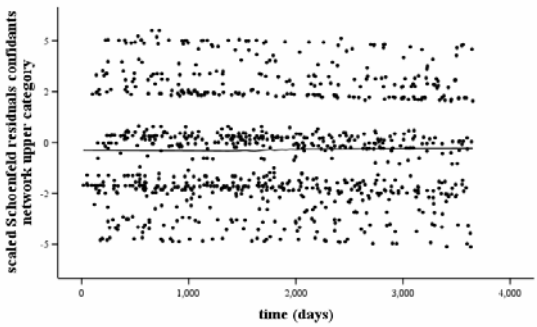

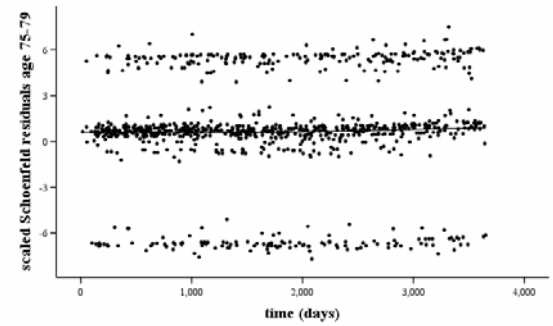

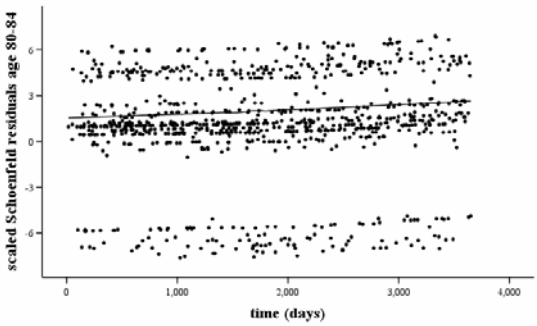

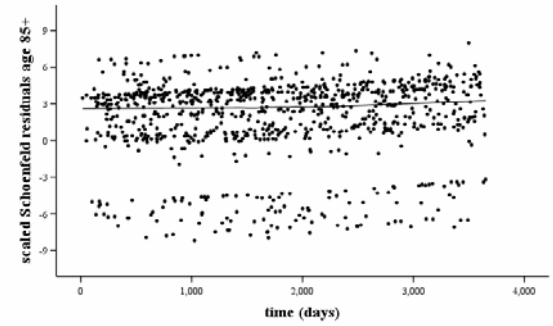

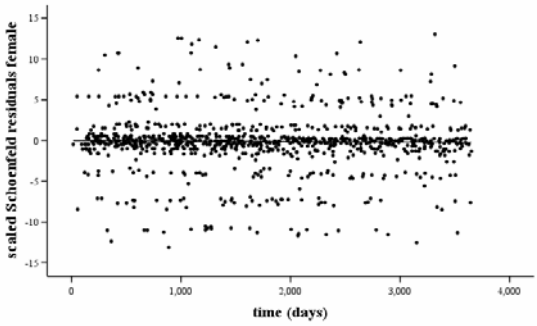

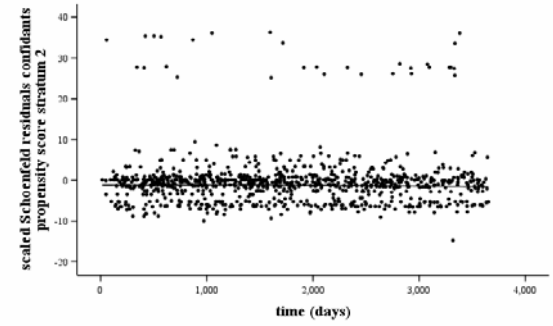

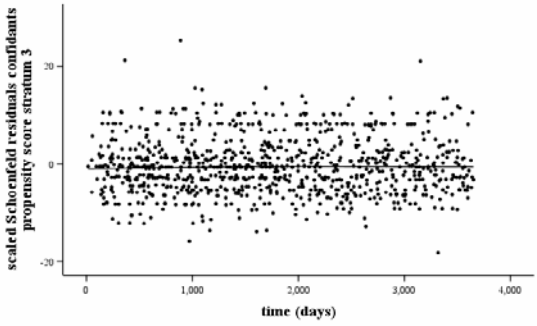

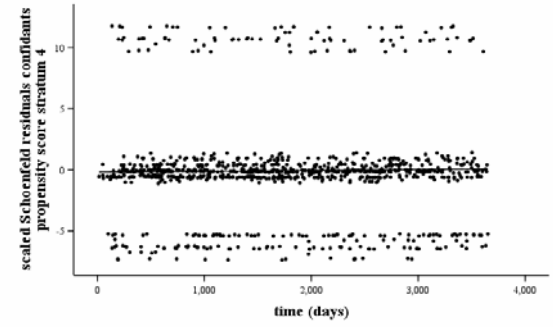

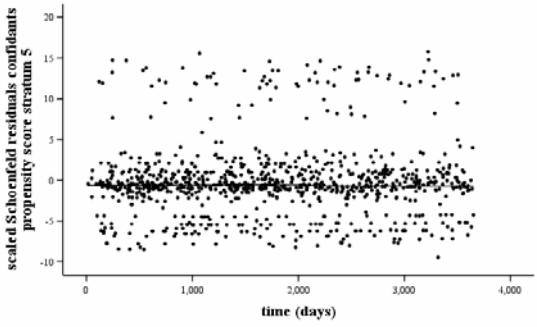

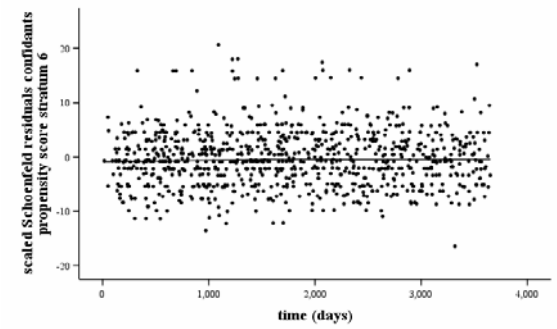
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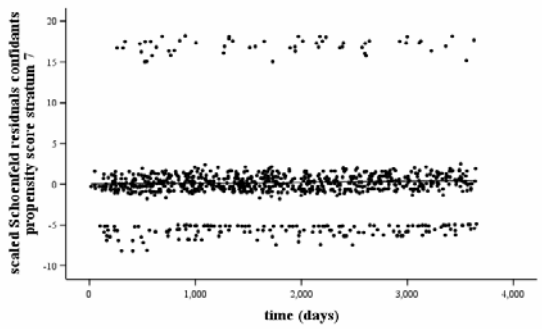
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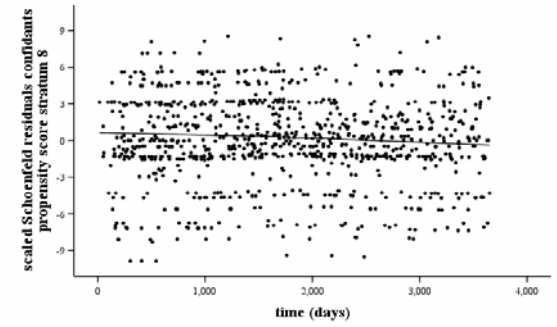
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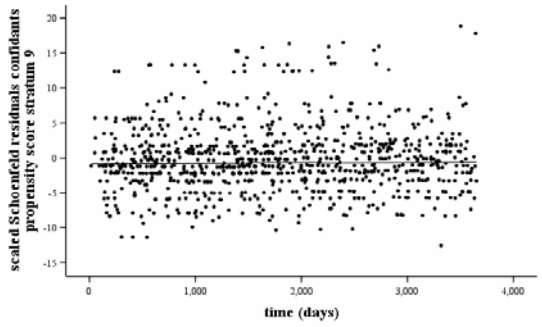
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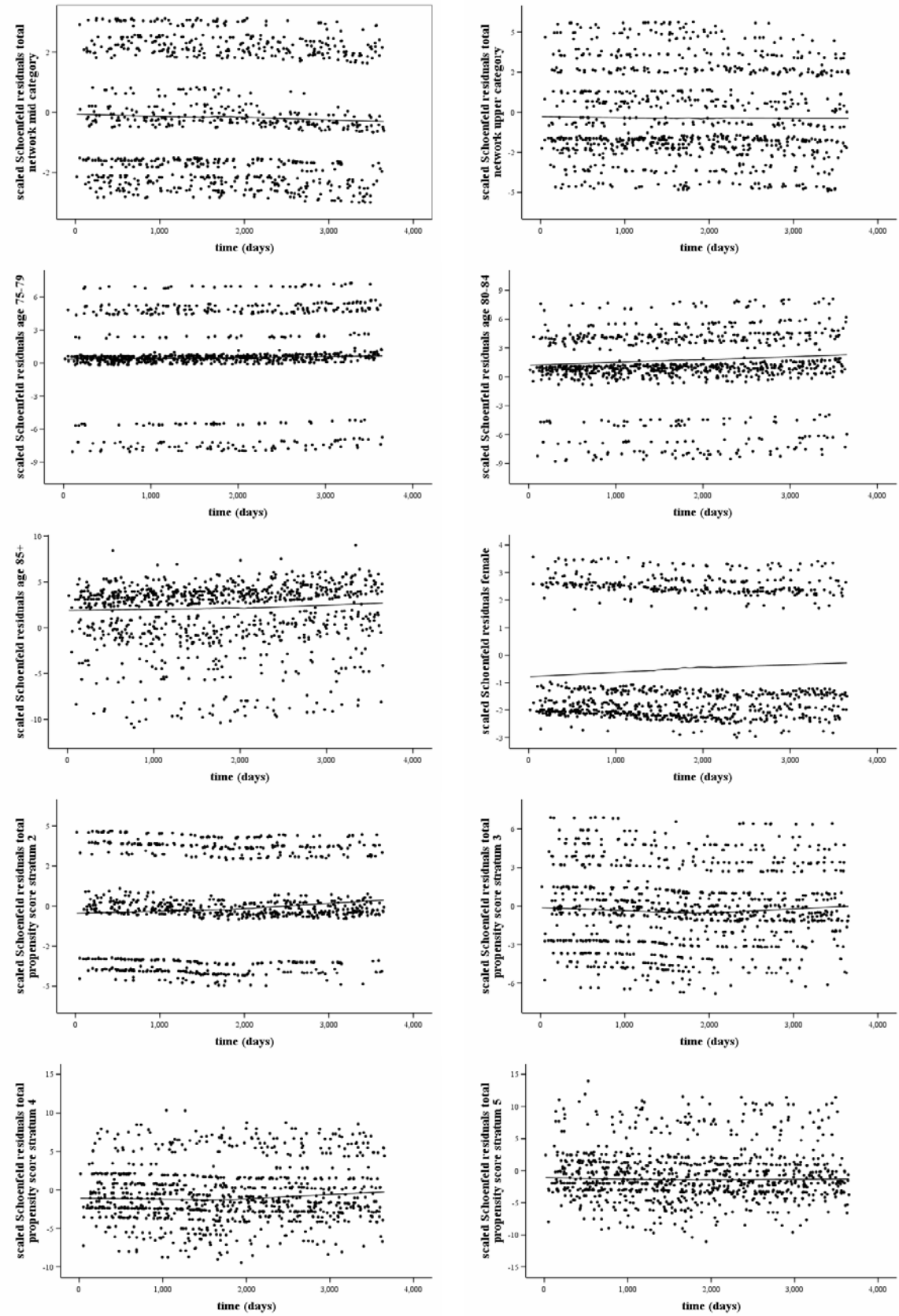
time (days)

Total social networks



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