SOCIAL RELATIONSHIPS AND PROGRESSION OF FRAILTY: EXPLORING THE RECIPROCAL ASSOCIATION OF SOCIAL TIES AND PHYSICAL VULNERABILITY IN LATER LIFE

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ABSTRACT

SOCIAL RELATIONSHIPS AND PROGRESSION OF FRAILTY:
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Frailty is described as a state of heightened vulnerability and functional impairment due to the cumulative declines across multiple physiological systems. When faced with stressor events, older adults with frailty are in higher risk of adverse health outcomes. While the prevalence of frailty generally increases with age, there are considerable heterogeneity in onset and progression of frailty among older population. Growing attention is given to identifying the psychosocial factors related to the development of frailty. Social relationships often serve as a vital context of health, where older adults experience multidimensional and dynamic exchange with close others as they age. In this dissertation, two studies are conducted to investigate complex and reciprocal nature of social relationships and frailty progression in older adulthood. The data are from the six waves (2006-2016) of the Health and Retirement Study (HRS), a nationally representative study of older adults in U.S. aged 50 and older.

The first study utilized the convoy model of social relations to provide comprehensive investigation of how different aspects of social relations are associated with frailty progression over a decade. There were three distinctive subpopulations following a different frailty progression trajectory. When social network and relational quality with spouse, children, family, and friends were examined, higher frequency of contact with friends were associated with lesser frailty. Negative relationship quality with social ties were detrimental to frailty progression, such that strain with spouse and kin (children and extended family) had an additive effect on belonging to high frailty or steep increase frailty trajectory groups. The perceived loneliness

partially explained the negative effect of spousal strain, but the negative effect of large family size and strain with kin were independent from loneliness.

The second study explored the health contexts of older couple's marital quality, specifically focusing on the presence of frailty and depression within- and across-person in the marital relationship. Using three waves of dyadic data from HRS, I found that one's own and partner's higher frailty and higher depression all had independent associations with one's higher marital strain. For one's marital support, one's own higher frailty, higher depression, and partner's higher depression had negative effects. There was an across-person interaction effect of frailty, such that one's marital quality was affected by their partner's higher level of frailty only when their own health was good. Having a husband with higher frailty was associated with higher marital strain for wives. Most effects were stable over time.

Overall, the findings illustrate the significance of social relationship context as a predictor for different trajectory of frailty progression. The size, frequency of contact, positive, and negative quality were linked to frailty differentially by relationship type, underscoring the benefits of comprehensive examination of social experiences. Further, the level of frailty and depression were linked to perceived marital quality of both members of the couple, especially in damaging manner when healthier spouse is faced with partner's health problems. Taken together, my dissertation demonstrated the importance of studying linked lives in context of health conditions prevalent in older adulthood. The findings can be useful to practitioners and policy makers in understanding the intricate link between social relations and frailty as well as in identifying modifiable factors for frailty prevention.

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CHAPTER 1. INTRODUCTION

Introduction to the dissertation

The term frailty describes a physical state of heightened vulnerability due to the decline in many physiological systems and reserve (Carlson et al., 1998; Fried et al., 2001; Walston et al., 2006). It is associated with a considerably increased risk of falls, disability, hospitalization and mortality (Song, Mitnitski, & Rockwood, 2010; Kojima, 2016, 2017; Crow et al., 2018). Increasingly, geriatricians are focusing on finding the biopsychosocial factors related to the onset and progression of frailty. Social relationships often serve as a vital context of health for older adults, with considerable heterogeneity between individuals (Berkman, Glass, Brissette, & Seeman, 2000; House, Landis, & Umberson, 1988; Thoits, 2011; Uchino, 2006). Identifying the multidimensional and potentially modifiable risk factors in social relationship will be an important agenda in understanding and preventing frailty in older populations. The current research will address the gaps in the literature by exploring the longitudinal associations between social relationship and frailty progression in later adulthood. The study will utilize six waves (2006-2016) from Health and Retirement Study (HRS), a nationally representative study of older adults in U.S. In this dissertation, two studies will jointly address the complex and reciprocal nature of close relationship quality and frailty progression.

The first study utilized the convoy model of social relations (Antonucci, Ajrouch, & Birditt, 2013; Antonucci & Akiyama, 1987) to provide comprehensive investigation of how different aspects of social relations are associated with frailty progression. This study will address the following: 1) identify distinctive trajectories of frailty among older adults; 2) examine the differences in the trajectory memberships in relation to the social network (i.e.

relationship types and frequency of contact), relationship quality (i.e. both support and strain) with various social partners, and individual's perception of loneliness.

The second study will focus on married older adults and the effects of frailty and often accompanied depression on their marital quality. Dyadic data from HRS will be examined utilizing actor-partner interdependence models to describe the actor, partner, and multiplicative effects of the level of frailty and depression within- and across-person on the quality of marital interaction (i.e. marital support and strain) over an eight-year period.

Before presenting the two studies, the following introductory section provides overview of the literature on frailty. I describe the conceptualization, measurement models, prevalence, and outcomes of frailty.

Overview of Frailty

Definition and conceptualization of frailty

The word 'frail' has long been used to describe declining health status associated with aging. Clinicians may have been intuitively categorizing weak and slow walking older patients as frail (Sternberg, Wershof-Schwartz, Karunananthan, Bergman, & Clarfield, 2011). The concept of frailty was first described in 1968 (O'Brien, Roberts, Brackenridge, & Lloyd, 1968). However, there was no scholarly consensus in the definition of the term nor empirically tested models to identify frailty in geriatric patients and older populations. It was only in the mid 1990's that scholars have started to recognize the need for conceptualizing and defining frailty as a clinical syndrome (Gealey, 1997; Kaufman, 1994; Ory et al., 1993; Rockwood, Fox, Stolee, Robertson, & Beattie, 1994; Schulz & Williamson, 1993). The operationalization of physical frailty by Fried and colleges (2001) was a seminar work that embarked the next generation of research in systematic assessment and conceptualization of frailty. Over the last two decades,

scholars have gained more understanding of the significance in the physiological factors and processes underlying the expressions of frailty (Yang & Lee, 2010).

Frailty has been defined as an age-related biological syndrome of decreased physiological reserves and increased vulnerability to stressors or perturbations, resulting from cumulative declines across multiple physiologic systems (Carlson et al., 1998; Fried et al., 2001; Fried, Ferrucci, Darer, Williamson, & Anderson, 2004; Markle-Reid and Browne, 2003). A gradual depletion of the physiological reserve may occur with aging itself. However, in conceptualizing frailty, the deregulation across multiple physiological systems is accumulated over the lifetime. The gradual depletion of physiological capacities can cause poor recovery from a stressor event. For example, a non-frail person will soon find a resolution of homeostasis after a small event such as change of medication or minor viral infections, but a frail individual will experience larger deterioration of the function and may not fully recover to the prior state (Carlson et al., 1998; Clegg, Young, Iliffe, Rikkert, & Rockwood, 2013). The condition can be understood as a vicious cycle responsible for the onset of negative health-related outcomes. A person with higher frailty is more likely to experience sudden disproportionation in health state, such as becoming functionally dependent, delirious, or prone to falls due to loss of postural stability (Ensrud et al., 2009). Also, the frailty has been referred to as "unstable disability" as a frail elderly person often shows oscillations of dependency level. Although there are similarities, frailty has been conceptualized to be distinctive from disability, with a different biological basis, potentially carrying a higher risk for disability or a pre-disability state (Fried et al., 2004).

In effort to unify the definition of physical frailty, a consensus group consisting of delegates from six major international, European, and US societies agreed to operationalize physical frailty as "a medical syndrome with multiple causes and contributors that is

characterized by diminished strength, endurance, and reduced physiologic function that increases an individual's vulnerability for developing increased dependency and/or death." (Morley et al., 2013, p. 393). Although there is ambiguity and controversy over diagnosis and operationalization of frailty for research and clinical studies, it is established that frailty is a condition frequently observed in older adults, serving as a warning sign for high risk of adverse health outcomes later. Among geriatric patients, the level of frailty is predictive of post-operative complications, the length of hospital stays, and the likelihood to be discharged to a skilled or assisted-living facility rather than returning home after the surgery (Robinson et al., 2011). Thus, in clinical settings, frailty screening is increasingly being used as a diagnostic tool to assess risk and stratify older patients (Makary et al., 2010).

In population health literature, more researchers are focusing on frailty as comprehensive syndrome representing a transitional phase between successful aging and disability (Cesari, Gambassi, van Kan, & Vellas, 2014; Clegg et al., 2013). Global network of researchers have recognized frailty as a key concept to implement in the latest version of WHO World Report on Aging and Health (Cesari et al. 2016). In preventing and managing age-related conditions, the researchers suggest that frailty may represent a novel and person-tailored way to conceptualize "biological age", replacing the concept of chronological age (Cesari et al. 2016).

Models of frailty

There are two main conceptual models of frailty that have been widely accepted by the researchers: (a) the biological or phenotypic approach, which focuses on the physical aspects of frailty and (b) the multi-domain approach, which includes a combination of physical measures, syndromes, diseases, and psychosocial factors.

Phenotypic model of frailty (PF). Phenotypic frailty model proposed by Fried and colleges views that frailty originates from cumulative declines across multiple physiological systems, so that there is decreased reserve to recover from stressors (Fried et al., 2001, 2004). Mainly focused on the physical domain, thus referred as a model of physical frailty, the phenotypic frailty measure uses criteria related to skeletal muscle, nutrition, and energy metabolism. Specifically, the original phenotype of frailty model includes five criteria: (a) unintentional weight loss, (b) weak grip strength, (c) slow gait speed, (d) presence of exhaustion, and (e) low physical activity. Each criteria was operationalized based on measurements available in Cardiovascular Health Study (CHS; Fried et al., 1991) in which the model was first developed. The scores were dichotomous on the presence (1) or the absence (0) of the five symptoms. An ordinal frailty score, ranging 0-5, was created by summing the points for each criteria. As a diagnostic tool, the model classifies individuals into three categories such as robust, pre-frail, and frail. A person who scores zero from the five frailty components are referred as non-frail or robust (0), those who score one to two is pre-frail (1-2), and individuals who score three or higher are categorized as frail (3-5). Fried and colleagues (2001) initially validated the measure by demonstrating strong relationships between frailty and a range of adverse health outcomes (i.e. incident falls, decreased mobility, activities of daily living, incident hospitalization, and death). There are criticisms for this model, such that the initial variables were selected from a prospective cohort study not designed to study frailty, the model excluded samples with Parkinson's disease, previous stroke, cognitive impairment, or depression, and that cognitive impairment was not included as the phenotype when the measure was validated (Clegg et al., 2013; Rothman, Leo-Summers, & Gill, 2008). Despite these limitations, the phenotypic frailty model is one of the most widely used measures of frailty (Dent, Kowal, & Hoogendijk, 2016).

Frailty Task Force of the American Geriatrics Society adopted this model as its conventional operational definition (Lang, Michel, & Zekry, 2009).

Accumulation of deficit model of frailty (Frailty Index). The accumulation of deficits model of frailty, often termed the frailty index (FI), views that frailty is a nonspecific age-associated vulnerability that is reflected in an accumulation of deficits in symptoms, signs, disabilities, and diseases (Mitnitski, Mogilner, & Rockwood, 2001; Rockwood et al., 2004). The model posits that the frailty can be measured by counting an individual's health problems or deficits across several different domains of health. These domains include current illnesses, ability to manage activities of daily living (ADL), and physical signs (Rockwood et al., 2005). The model involves that accumulation of deficits in multiple physiological systems which make the person vulnerable to stressors, leading to higher risk of health problems. Compared to phenotypic model of frailty, FI emphasizes trajectories of gradual deterioration and accumulation, which is also reflected in its measurement (Clegg et al., 2013). As a measure, an index score is calculated as the ratio of deficits present in an individual from the total number of deficits measured. The presence of the deficits for a given person is counted as binary (0 = absent, 1 = present) and a given person's deficit is divided by total deficits considered. FI score is presented as continuous variable ranging between 0-1 (e.g. 10/40 = 0.25).

Unlike the phenotypic frailty model, the deficits that can be included in the frailty index are not pre-determined. The flexibility of FI allows for application and comparison across multiple surveys (Rockwood & Mitnitski, 2007). The original study used a total of 70 deficits from the Canadian Study of Health and Aging (CSHA) data, covering the presence and severity of current diseases, ability in ADLs and physical signs from clinical and neurologic exams (Rockwood et al., 2005). Searle and colleagues (2008) have shown that versions with 30-40

deficits yielded comparable predictive validity to the original index when deficits are selected based on the following criteria. They recommended that when frailty index is constructed, (a) the selected deficit must generally accumulate with age, (b) a deficit must be related to health status in a biologically plausible way (e.g. graying hair is an age-related symptom, but not a health status), (c) a deficit must not become saturated at an early age (e.g. presbyopia are age-related but nearly universal by age 55, thus saturates too early to be considered as deficit), and (d) the deficits together must represent a wide range of bodily systems (e.g. not one domain should dominate the items; Searle et al., 2008). Song and colleagues (2010) illustrate that although each deficit items have the same weight; the severity and burden can be represented in the number of items in the deficit count. For example, if a person who has a history of *hypertension*, *heart disease*, *diabetes mellitus*, and *glaucoma* severe enough to cause *vision problems* so that help was needed with *heavy housework* (each deficits are italicized). This person would have six deficits, and their FI score will be 6/26 (0.17). However, if this person was able to get treatment for glaucoma and is not experiencing visions problems anymore, and no longer needing help with housework, the person would have four deficits resulting in 4/26 (0.11; Song et al., 2010).

Studies have found that FI is highly predictive of risk of institutionalization and mortality when at least 30 variables are included (Rockwood & Mitnitski, 2007). Also, FI are known to show consistent maximum level such that in most cases, the most deficit anyone may have in the sample will be about 2/3 of the total deficit (Rockwood & Mitnitski, 2007). FI score over 0.25 are considered as equivalent to belonging in the frail category in the phenotypic frailty model (Cigolle et al., 2009; Rockwood, Andrew, & Mitnitski, 2007).

Prevalence of frailty

Frailty is common in later life. Based on data from National Health and Aging Trends Study, a nationally representative data of persons aged 65 or older in U.S., the prevalence of physical frailty was estimated at 15%. Among the rest, 46% were categorized as pre-frail and 39% were robust (based on phenotypic frailty model, Bandeen-Roche et al., 2015). Similarly, a review of research that examined 21 published studies on the frailty of community-dwelling older adults reported that the prevalence of frailty and pre-frailty was approximately 11% and 42%, respectively (Collard, Boter, Schoevers, & Oude Voshaar, 2012). However, there were variations in the frailty prevalence among reviewed studies, mostly due to the differing operationalization of frailty and inclusion criteria. Studies that used phenotypic frailty approach reported a generally lower prevalence rate ranging from 4% to 17%, but studies that used the multi-domain definition of frailty reported prevalence rate ranging from 22% to 59% (Collard et al., 2012; Shamliyan, Talley, Ramakrishnan, & Kane, 2013). Compared to the community-dwelling older adults, the patients in nursing homes had higher prevalence of frailty. A study that pooled the estimates of frailty of nine studies has reported that the prevalence of frailty reaches about 52% for nursing home patients (Kojima, 2015). However, the study also noted that about 40% of the patients were pre-frail, suggesting that this patient group can be targeted for interventions for frailty prevention.

Prevalence of frailty steeply increased with age. The prevalence was about 9% for adults aged 65-69, but it increased to about 38% for age over 90 (Bandeen-Roche et al., 2015). Even when limiting the estimation to community-dwelling older adults, thus excluding the older adults in nursing homes, the prevalence almost doubles in advanced age (16%; age 80-84, 26%; age 85+, Collard et al., 2012).

Prevalence of frailty differs by sociodemographic determinants. Frailty was more prevalent among women, racial/ethnic minorities, lower SES, and for those living in residential care settings (Bandeen-Roche et al., 2015; Shamliyan et al., 2013). For example, women had higher frailty prevalence than men (17% vs. 13%), 65%–85% higher for blacks and Hispanics than whites (23-25% vs. 14%). Lowest income quartile had fourfold higher prevalence than the highest income quartile (26% vs. 6%) and those living in the residential care settings (excluding nursing home patients) were twice-likely to be frail than community-dwelling adults (30% vs. 15%). Interestingly, frailty prevalence were twice as high for those living in the inland south of the U.S. than those living in mountain/desert west, independent of the demographic characteristics (22% vs. 10%; Bandeen-Roche et al., 2015). In Europe, being female and lower education level were predictors of increased risk of worsening in frailty state two years later (Etman, Burdorf, Cammen, Mackenbach, & Lenthe, 2012). Ntanasi and collegues (2018) found that advanced age and low education level were associated with increased risk of frailty across the five different frailty measures (i.e. Fried phenotypic frailty, Frailty Index, FRAIL Scale, Tilburg Frailty Indicator, and the Groningen Frailty Index).

Progression of frailty

For any health symptoms, the prognosis or the change in severity over time is important to document and understand. In order to investigate the trajectory of frailty development, the likelihood of transitioning into more severe state over time was examined in a few studies that used the phenotype frailty model (Fried et al., 2001). The study that first validated the frailty phenotype measure reported that 7% of the initially robust participants developed frailty over four years (Fried et al., 2001). Similarly, a study based on data with Mexican Americans showed that 8% of the initially robust participants developed frailty over the course of seven years (Ostir,

Ottenbacher, & Markides, 2004). Study with community-dwelling American Men reported that over 10% of the men who were pre-frail become frail in three years (Cawthon et al., 2007). Regarding the various symptoms that comprise the frailty syndrome in Fried's phenotypic model, recent study based on two decade-long data from Netherlands and Italy found that the feelings of exhaustion tend to emerge early in the process of frailty and weight loss near the onset of frailty (Stenholm et al., 2019). One study investigated the dynamic nature of the transition between physical frailty states and whether recovery into a less severe state is possible (Gill, Gahbauer, Allore, & Han, 2006). Using the data from 754 community-living older adults, the study found that about half (58%) of the participants transitioned into different frailty category over 4.5 years. During 18-month intervals, transitioning into a more severe state was very common; robust to a pre-frail rate of transition was up to 43%, pre-frail to frail rates up to 58%. On a positive note, some participants transitioned into a less severe category; pre-frail to a robust rate of up to 16.5%, and frail to pre-frail transition rate up to 23%. However, there was no case where a person categorized as frail to transitioned into a robust state (Gill et al., 2006).

Overall, these findings show that frailty progresses in a gradual manner, with fluctuations between categories when measured on a categorical instrument, but a full recovery from frailty is rare. However, descriptions using categorical transitions provides limited information about the rate of change. A notable finding was reported based on the 8-year follow up study from the Rush Memory and Aging Project (Buchman, Wilson, Bienias, & Bennett, 2009). Using a continuous measure, they found that the rate of change in frailty varied among individuals, and that baseline frailty and the longitudinal change in frailty were independently associated with all-cause mortality. For example, controlling for baseline level of frailty, each 1-unit increase in annual change in frailty was associated with an almost 5 times the risk of death. Recognizing the

need for longitudinal investigations of various progression path of frailty, a small number of recent studies used panel data and more sophisticated statistic methods to identify heterogeneous trajectories of frailty progression (Chamberlain et al., 2016a, 2016b; Hsu & Chang 2015; Peek, Howrey, Ternent, Ray, & Ottenbacher, 2012).

Chamberlain and colleagues (2016a, 2016b) applied a cluster modeling for longitudinal population data of Olmsted County, Minnesota (N = 16,443) from 2005 to 2012. They analyzed frailty trajectory for each decade of age groups. They found that people in their 60's had three distinct trajectories with low, middle, and high level of frailty. For those in their 70's and 80's, there were two trajectories of low and high level of frailty with about half of the sample in each trajectory. The higher frailty trajectories in all age group were associated with greater rates of emergency room visits, hospitalization, and deaths. They also found that among social and health behavior factors, the alcohol consumption (i.e. Concerns from relatives/friends about alcohol consumption) and having less than high school level of education were associated with increased likelihood to being in a high frailty trajectory. The associations were stronger for older adults in their 60's and 70's, but not significant for those in their 80's when adjusted for age, gender, and baseline frailty level. Hsu and Chang (2015) examined the frailty trajectories in the population level with data from Taiwanese Longitudinal Survey on Aging (TLSA) spanning 14-years. Using group-based model of trajectory analysis for samples aged 64 or older, they identified three frailty trajectory groups: maintaining non-frailty (44%), developing frailty (39%), and high risk of frailty (18%) groups. Similar to the cross-sectional reports, being female, older, and having a lower education level was associated with increased likelihood of being the developing frailty group or high risk of frailty group. Chronic diseases, physical disability, having a depressive mood, and lower cognitive function were also associated with a higher probability of frailty.

Similar trajectories were reported from 12-year period panel data of older Mexican Americans (Peek et al., 2012). Using group-based model of trajectory analysis as well, the researchers identified steady low frailty, progressive moderate frailty, and progressive high frailty groups. Across the three groups, the number of chronic conditions, financial strain, and health event history were associated with an increase in frailty.

Health outcomes and mortality of frailty

Frailty status has been shown to predict a host of adverse health outcomes across assessment instrument, target populations, and settings (Cesari et al., 2016). Based on the 3-year prospective study, individuals with frailty were at an almost two-fold increase in the risk of recurrent falls and hip fracture (Ensrud et al., 2007). Being frail independently predicted an increased risk of ADL disability and hospitalizations (Woods et al., 2005; Kojima, 2016, 2017). Astonishingly, odds of having four or more comorbid conditions were more than 40 times higher among frail person than the non-frail (Bandeen-Roche et al., 2015).

Additionally, frailty was associated with increased decline in cognitive health. A prospective cohort study that followed initially healthy older adults over 12 years, researchers found that physical frailty was associated with greater risk of developing a mild cognitive impairment (hazard ratio= 1.63, 95% CI = 1.27–2.08) and higher level of frailty was associated with a faster decline in global cognition (Boyle, Buchman, Wilson, Leurgans, & Bennett, 2010). A similar adverse association was also found for incident Alzheimer's disease, such that baseline frailty level and the rate of change in frailty was both associated with increased risk of Alzheimer's disease (i.e. one tenth of a unit increase on the frailty scale at baseline was associated with >9% increased risk of AD; each one tenth of a unit increase in annual rate of change in frailty was associated with a 12% increased risk of AD; Buchman, Boyle, Wilson,

Tang, & Bennett, 2007). The increasing awareness of the comorbidity of physical decline and cognitive decline has led to scholarly efforts into including a concept of cognitive frailty into the assessment of multi-domain frailty (Vella Azzopardi et al., 2018).

In terms of health service use, the risk of admission to long-term care was more than two-fold for those with mild and severe frailty, compared to non-frail individuals (Rockwood et al., 2004). Furthermore, community-dwelling older men with frailty were more likely to use health and community services, such as consulting a doctor, visits by a nurse, meal and housework service, and spending at least one night in a hospital or nursing home (Rochat et al., 2010). Among surgical geriatric patients, frailty predicted worsened prognosis. The pre-operation physical frailty status was associated with increased risk of post-operation complications (OR 2.54; 95% CI = 1.12–5.77), longer hospital stays (incidence rate ratio 1.69; 95% CI = 1.28–2.23), and discharge into assistive care facility for those previously living at home (OR 20.48; 95% CI = 5.54–75.68; Makary et al., 2010). Finally, frailty is related to increased risk of death. A meta-analysis study reports that individuals with frailty has 50% greater mortality rate than robust individuals (Shamliyan et al., 2013). Recent study added that frailty was associated with 84% increased chance in mortality and even pre-frailty was associated with considerably higher risk of mortality (64%) than a robust individual (Crow et al., 2018). It is estimated that 3–5% of deaths among older adults could be delayed if frailty was prevented (Shamliyan et al., 2013).

The high prevalence of frailty in advanced age, increased risk for multimorbidity, and cost burden of health care system all points to the need to investigate the mechanism of frailty progression and to find the modifiable socio-environmental targets for intervention.

CHAPTER 2. SOCIAL RELATIONS AND FRAILTY TRAJECTORY IN LATER ADULTHOOD

Introduction

The investigation of social context of frailty progression needs to be grounded in theoretical perspectives that explains the relationships and processes of various social experiences. In recent decades, social science scholars have established that social support is positively related to physical and mental health (Berkman et al., 2000; Holt-Lunstad, Smith, & Layton, 2010; House et al., 1988; Uchino, 2004, 2006, 2012). In efforts to explain the links between social relationships and health, largely two models have been proposed. Main-effect model posits that a higher level of social integration is beneficial for health as itself (Berkman et al., 2000). In this framework, the social relationship contributes to health, via psychological and physiological mechanisms. Psychologically, greater social support and integration were known to provide the individual a sense of purpose, identity, security, as well as social control to reinforce health-promoting behaviors, all promote health (Thoits, 2011). Physiological mechanisms, such as processes through cardiovascular, neuroendocrine, and immune-mediated inflammatory processes have been studied to link social support to health (Uchino, 2006). In another school of thought, scholars have proposed the stress-buffering model to explain the health promoting effects of social ties (Cohen & Wills, 1985). This model suggest that social support buffers the harmful health effects of stress exposure, either by providing direct mitigation to ease the stressor or indirectly by reducing the degree of stress appraisals (Uchino, 2004). However, it has been documented that the buffering effects are less consistent or strong as the main effects of social ties on health (Uchino, 2004). Furthermore, there was a need for an encompassing framework to describe and model the social context in human lives.

The convoy model of social relations recognized that individuals are embedded within a context of close others throughout their life course (Antonucci et al., 2013; Antonucci & Akiyama, 1987). The convoy is described as the layers of family and friends who protect and promote the well-being of the person as they experience changing needs. Several concepts incorporated in the convoy model are useful in capturing the complex of the nature of human's social relations. First, the convoy model emphasizes the multidimensional aspects of social relationships. A person's relationship within their social convoy vary in their structure such as the size, composition, contact frequency, geographic proximity. The quality of the social relationships can include positive features such as emotional support and perceived availability, and the negative aspects of relationships such as conflict and stress (Umberson, & Montez, 2010). The social ties may serve different functions, such as affection, instrumental, and functional aid and exchanges. Considerable evidence supports that both structural aspects (i.e. large social network and cohesion), as well as functional aspects (i.e. emotional support) of social support are associated with better health, and that some quantity of relations are necessary to have high quality relationships (Antonucci et al., 2013; Xing, Zhang, and Cheng, 2017). Second, the multidimensional aspects of convoy model also reflect the notion that one's experiences and evaluations of social relationship can by both objective and subjective. For example, the same number of social network size (i.e. objective) can be perceived as lacking or sufficient depending on the person's perspective (i.e. subjective), with differential implication for their well-being. Third, the convoy model is dynamic and focuses on the entire lifespan, theorizing that personal (i.e. age, gender, race) and situational factors (i.e. roles, norms, values) influences the dynamic changes in the structure, quality, and function of social relationships over the life course (Antonucci et al., 2013). Previous research on the predictors of frailty onset and

progression have been mainly focused on co-developing health conditions, socio-economic status. Moreover, the most studies have not been designed based on theorical models focused on the complex nature of social experiences to understand the effect on frailty.

Thus, the current study is based on the convoy model of social relations (Antonucci et al., 2013) to investigate the multidimensional nature of social relationships in relations to frailty progression. The main purpose of the study is to first examine the heterogeneous trajectories of frailty progression and to explore the multifaceted social relations predictors of frailty trajectory in later adulthood by using longitudinal data from Health and Retirement Study (HRS). In the following literature review, I assessed the empirical findings on the associations of structural, quality, and types of social relationships and frailty.

Literature Review

Structure of social relations and frailty

Within the literature that investigated antecedents, correlates, and outcomes of frailty in older population, relatively few studies included measures related to social relationships. Most of these studies tested the role of social support as a potential factor of frailty progression, or as a modifier in the link between frailty and adverse health outcomes. However, there are inconsistent findings regarding the effects of social relationship on frailty (Duppen, Van der Elst, Dury, Lambotte, & De Donder, 2017).

First, among the structural characteristics of one's social relationship, indicators of marital status and living arrangement had the most robust association with frailty. Generally, being married status was consistently associated with lower level of frailty, across five types of measurement models of frailty in a cross-sectional data (Ntanasi et al., 2018). In a longitudinal study, the effect of marital status on the progression of frailty differed by age (Chamberlain et

al., 2016b), For example, for those in their 60's and 70's, non-married status was associated with belonging in a high frailty trajectory group. For those in their 70's, in addition to being un-married, living alone or with other family members was related to belonging in high frailty group. However, marital status or living arrangement was not a predictive factor of frailty progression for those in their 80's, when the baseline frailty was controlled (Chamberlain et al., 2016b). Moreover, gender differences are suggested in the link between marital status and frailty progression. In a study that investigated non-frail adults in Italian sample, there was a higher risk of onset of frailty for never married and widowed men, compared to married men after 4.4 years. However, for women, those who were widowed had significantly lower risk of becoming frail than married women (Trevisan et al., 2016). Overall, marital status seems to influence the onset and progression of frailty, with some gender and age differences.

Second, having few social ties or having infrequent contact with the social ties are indicators of social isolation. Some studies have found significant associations between social isolations and frailty. In a longitudinal study that studied a cohort of adults from 1965 and followed up with them every ten years, being socially isolated had elevated risk for frailty (Strawbridge, Shema, Balfour, Higby, & Kaplan, 1998). Especially, the risk was present for those who reported being socially isolated for more than one wave, suggesting that a long-term social isolation contributes to frailty. Also, there were gender differences. The high level of social isolation (i.e. living arrangement, the frequency of contact, and participation in social organizations) was a risk for physical frailty in men, but not in women (Gale, Westbury, & Cooper, 2017). The frequency of contact with relatives were linked to lower frailty rates in women, but not in men (Woo, Goggins, Sham, & Ho, 2005).

Third, there were no significant findings for the role of social network factors and frailty. Two studies by Hoogendijk and colleagues (2014a, 2014b) examined size of network, emotional connection and frequency of contact with seven role types (e.g., friends, family and neighbors) using multiple waves of the Longitudinal Aging Study Amsterdam (LASA). They report that social network size of the baseline did not moderate the link between lower education level and frailty (Hoogendijk et al., 2014a), nor moderated the link between frailty and functional decline and mortality (Hoogendijk et al., 2014b). Contact with the network also failed to contribute in buffering the effect of low education. Although informative, the two studies only tested the mediating effect of social participation, leaving the main effect still untested.

Quality of social relations and frailty

Studies that investigated the influence of social support reported both significant and non-significant findings. Lurie and colleagues (2015) investigated whether social support was predictive of frailty development in survivors of myocardial infraction a decade later. They included a measure for functional social support (e.g. instrumental, financial, and emotional support) and further separated the reception of social support (e.g. social interactions) and the perceived social support (e.g. perception of availability of support). The study reports that only the lower level of perceived social support was associated with a greater risk of frailty. Similarly, in 12-year period frailty trajectory study for older Mexican Americans (Peek et al., 2012), higher social support (i.e. two items: can count on family and friends, can talk about deepest problems with at least some of your family and friends) was associated with a less-steep increase in frailty over time, but only for those who were moderately frail. The beneficial effect of social support was not found for consistently high frailty group. However, there were similar amounts of non-significant findings for the effect of social support. In a cross-sectional study of Canadian

sample, the perception of available support or receiving emotional support were not associated with frailty (St John, Montgomery, & Tyas, 2013). Similarly, in a cross-sectional study with Mexican sample, a sum measure of social support and social contact was not associated with frailty (Kawano-Soto, García-Lara, & Avila-Funes, 2012). They did find that frail persons were more likely to leave important decisions to other family members than pre-frail or non-frail persons.

Although informative, there are very little work that incorporated the complex nature of the quality of social relationships. In any social relationships, there are potentially positive and negative aspects, often the experience with a social tie can be ambivalent. The positive aspects may include perceived emotional, instrumental, and practical support from the social ties and perception of having a relationship one can confide in when problems arise. In contrast, negative social relationship may include criticism, conflict, and the lack of reciprocity (Rook, 1984). In a longitudinal study with both positive and negative interaction with social ties included, the emotional support or having a person to confide in were not linked with frailty (Gale, Syddall, Bergman, Brunner, Cooper, & Sayer, 2012). But the negative interaction with the social ties was associated with increased in frailty, only in women. This finding corresponds with the reports that in both positive and negative exchange in intimate relationships, the negative exchange was more salient to health (Ingersoll-Dayton, Morgan, & Antonucci, 1997; Newsom, Nishishiba, Morgan, & Rook, 2003; Newsom, Rook, Nishishiba, Sorkin, & Mahan, 2005). Further study is needed to examine to the extent to which the perceived quality of social relationship is linked to frailty progression.

Additionally, there is a lack of study that looked at both the quantity and quality aspects of social relationships. Scholars emphasize that both constructs are connected to health outcomes

and suggest that some quantity of relations are needed in order to have high quality relations (Antonucci et al., 2013). For example, a recent study on Chinese older adults' social network found that quality of emotionally close social partners influenced mental health the most, the number of peripheral relationships were associated with positive mental health, and that those with larger size of emotionally close social partners had a stronger association between relationship quality and mental health (Xing et al., 2017). Thus, the study of frailty progression would require an examination of both quantity (i.e. social network size and contact frequency) and quality of social relationships.

Types of social relations and frailty

The differentiation between relationship types would provide more detailed picture of how social relationship may affect frailty progression. Many of the existing social support measures ask general questions regarding the sum of one's social ties. While the perceived quality or size of the whole social network would have indications for individuals' health and well-being, differential social support may be derived from many different relationships including the spouse, other close family, friends, and others (Fiori, Antonucci, & Cortina, 2006). One study measured social support from different sources in predicting frailty in later life (Woo et al., 2005). In this cross-sectional study, the effect of relationship type on the levels of frailty was examined for family members and neighbors. Notably, the support from the neighbors were related to lower frailty for both genders, social support from family members was associated with lower levels of frailty only for men.

Especially, the investigation into complex experiences in marital relationship is much warranted in frailty literature. So far, there is no study that looked into qualities of the marital relationship, except for simple marital status. There are many reasons that spousal relations are

important for health in later life. Spouses and children make up a greater portion in social network of older adults than younger adults (Aartsen, van Tilburg, Smits, & Knipscheer, 2004), and the spouse is the most frequently nominated close person in older age (Antonucci, Akiyama, & Takahashi, 2004; Cornwell, Laumann, & Schumm, 2008). Spouses share considerable time together, share the same living space and financial and tangible assets (Carr & Springer, 2010). Spouses take a central role in social control for individuals in terms of regulating and monitoring health behaviors such as diet, exercise, and medication adherence. With the close proximity and the shared health in couples (Meyler, Stimpson, & Peek, 2007), the negative interaction with the spouse might be a significant source of stress. Recent research consistently finds that the health benefits of marriage may depend considerably on the quality of the marriage (Kiecolt-Glaser & Newton, 2001; Liu & Waite, 2014; Robles, Slatcher, Trombello, & McGinn, 2014). Mixed positive and negative feelings are particularly high for the spousal relationship (Fingerman, Hay, & Birditt, 2004).

Loneliness and frailty

In later adulthood, loneliness is a key risk factor for morbidity and mortality, known to be linked to increase in depression, impaired cognitive performance, functional limitations, and cardiovascular diseases (for a review: Cacioppo & Cacioppo, 2014, Holt-Lunstad, Smith, Baker, Harris, & Stephenson, 2015). Conceptually, loneliness can be understood as the perception of social isolation, where there is a dissatisfaction with the mismatch between desired and actual social relationship experienced by the person (Peplau & Perlman, 1982). Loneliness captures the subjective appraisal that there is a lack of closeness and affection with a significant other (i.e. emotional loneliness) or to close family and friends (i.e., relational loneliness; Ong, Uchino, &

Wethington, 2016). Thus, loneliness is distinct construct from social isolation, living alone, or solitude which captures more objective state (Luo, Hawkley, Waite, & Cacioppo, 2012).

There is a possibility that loneliness is associated with frailty. Based on the evidence of biological pathways linking loneliness to poor health, some scholars have theorized that "loneliness contributes to, and accelerates, age-related decreases in physiological resilience through its influences on health behaviors, stress exposure, psychological and physiological stress responses, and restorative processes that replenish physiological reserves and fortify against future stress" (Hawkley & Cacioppo, 2007, 187p.). However, there are very few studies that investigated the effect of loneliness in frailty progression. In a cross-sectional study, lonely Mexican adults were more likely to be frail, compared to robust or pre-frail (Herrera-Badilla, Navarrete-Reyes, Amieva, & Avila-Funes, 2015). Recent longitudinal study with data from English Longitudinal Study of Aging (ELSA) found that the high level of loneliness, but not social isolation (i.e. living alone, frequency of contact, and social participation), increased the risk of becoming frail four years later (Gale et al., 2017). This finding suggests that loneliness, the subjective social isolation, predicted worsening of frailty, but not social isolation itself. However, this study tested social isolation and loneliness in separate models, leaving the comparative effects between social isolation and loneliness on frailty un-tested.

Overall, prior studies have investigated social network, social participation, isolation, and support, not always finding statistically significant associations of social relationship with frailty outcomes. However, previous findings are limited by their cross-sectional study design, non-representative samples, and varying measures of frailty. Additionally, investigations of social relationship characteristics were bounded by the available variables in the data, limiting the inquiry into more nuanced effect that a social experience may have on frailty in later life. A

group of studies focused on creating index of social vulnerability or social frailty in context of physical frailty progression (Bessa, Ribeiro, & Coelho, 2018; de Jesus, Orlando, & Zazzetta, 2018; de Labra et al., 2018; Makizako et al., 2018). For example, social frailty was conceptualized with various indicators (e.g. who went out less frequently, rarely visited friends, felt less like helping friends or family, lived alone and did not talk to another person every day). They found that those who were socially frail were 2.5 to 3.9 times more likely to develop physical frailty four years later (Makizako, et al, 2018). Although informative, these measures may not be helpful in the context of social relations. One of the benefits of convoy model is that in the process of examining multiple domains simultaneously, it is possible to identify the "active ingredients" that has most implications for the health outcome (Zahodne, Ajrouch, Sharifian, & Antonucci, 2019).

Research questions and Hypotheses

Based on the convoy model of social relations (Antonucci et al., 2013), the current study aims to investigate the multidimensional nature of social relationships in relations to frailty progression. The main purpose of the study is to examine the heterogeneous trajectories of frailty progression and explore the multifaceted social relations predictors of frailty trajectory in later adulthood. Further, the role of loneliness in the link between social relations and frailty trajectory is examined. Below are the three research questions.

Research question 1: How does the frailty progress over time in older age and are there different groups of trajectories? Here, I examine the trajectory of frailty progression for adults over the age of 65. To address the heterogeneous expression of frailty among older populations,

(a) I explore whether there are distinctive subpopulations of older adults who share similar frailty trajectory. I hypothesize that groups are distinguished by the severity at baseline and differing rate of progression of frailty.

(b) I expect that there will be group differences in demographic (age, gender, race, education), baseline health conditions (depressive symptoms, cognitive functioning), social network and quality (network size, contact frequency, support, strain, loneliness) among identified frailty trajectory groups.

Research question 2: What are the baseline social network predictors of the frailty progression trajectory? I examine the influences of network size and contact frequency with different social ties on frailty progression group membership, when controlling for perceived loneliness.

(a) Controlling for demographic and health conditions, I expect that larger network size will be associated with healthier frailty trajectory group.

(b) Controlling for demographic and health conditions, I hypothesize that more frequent contact with social ties will be associated with belonging to a healthier frailty trajectory group.

(c) Regarding different social ties, I expect that friend relationship has stronger association with frailty than children and family members.

(d) Additionally, I hypothesize that when loneliness is accounted for in the model, the effect of network size and contact frequency will be diminished.

Research question 3: What are the baseline social relationship quality predictors of the frailty progression trajectory? I examine the extent to which the relationship support and strain

with spouse, children, family, and friend predict frailty progression group membership, when controlling for perceived loneliness.

(a) I hypothesize that reporting higher support and lower strain with spouse, children, family, and friend will be associated with belonging to a healthier frailty trajectory group.

(b) I also hypothesize that the overall effect on frailty progression will be stronger for spousal relationship quality, compared to relationships with children, family, and friends.

(c) Additionally, I hypothesize that when loneliness is accounted for in the model, the effect of social support and strain will be diminished.

Methods

Data

Data for the current study were drawn from Health and Retirement Study (HRS). HRS is a nationally representative longitudinal study of Americans over the age of 50. The study launched in 1992 and surveys were conducted every two years. HRS collects data on physical/health, family, employment, retirement, and wealth. The recent introduction of data on biomarkers, genetics, and psychosocial information makes HRS an ideal data for a multidisciplinary study of aging (Sonnega et al., 2014).

The HRS participants were sampled based on multi-stage area probability sample design at the household level (Heeringa & Connor, 1995). From the household, the age-eligible (birth year 1931-1941 for original HRS sample) respondent and their spouse or partner in any age was recruited. Additionally, HRS included oversampling of African American and Hispanic households (Ofstedal &, Weir, 2011). The sample of HRS has expanded over time to recruit new

cohorts of older adults. Initial cohorts were surveyed at 1992-93 and merged at 1998 to create one biannual survey ("HRS cohort" born 1931-1941 and "AHEAD cohort" born 1890–1923). Sub-sample cohorts were recruited subsequently, such as Children of the Depression cohort (CODA, born 1924–30) and the War Babies (born 1942–47). HRS now adds new 6-year birth cohort to the sample every six years to replenish younger cohorts. Early Baby Boomers (born 1948–53) were added in 2004, Mid Baby Boomers (born 1954–59) were added in 2010, and Late Baby Boomers (born 1960–65) were added in 2016 wave (Sonnega et al., 2014).

All participants of HRS are read a confidentiality statement at recruitment and given written informed consent document for each interview. For current dissertation, MSU IRB approval was exempted because publicly available HRS datasets do not include individual identifiers. The current study utilized HRS public release data accessible from the online HRS data repository. RAND HRS File is a cleaned and imputed for easy-to-use dataset based on the HRS core data This file was developed at RAND with funding from the National Institute on Aging and the Social Security Administration. Specifically, I used RAND HRS Longitudinal File 2014 (V2) released on February 2018 (RAND, 2018), RAND HRS Fat Files for waves from 2006-2014 (RAND, 2018). Additionally, this analysis uses Early Release data from the Health and Retirement Study, (HRS 2016 Core (Early V2.0), July 2018), sponsored by the National Institute on Aging (grant number NIA U01AG009740) and conducted by the University of Michigan. These data have not been cleaned and may contain errors that will be corrected in the Final Public Release version of the dataset.

Analytic sample

This study used six waves of data of older adults from the Health and Retirement Study (HRS). In HRS, self-report questions on participant's psychosocial characteristics and social

relationships were collected via leave-behind questionnaire starting from 2006 (Smith, Ryan, Sonnega, & Weir, 2017). In 2006, a random half of the sample received a face-to-face interview enhanced with physical, biological measures, and leave-behind questionnaire. The other half-sample completed the same enhanced interview on 2008. Thus, the baseline social network, relationship quality, and loneliness measures are collected in 2006 for half of the sample and in 2008 for the other half of the study sample. Health indicators that are used to create frailty measure were available in each biannual wave from 2006 to 2016.

Inclusion criteria for the current study were: 1) those who have completed leave-behind questionnaire in baseline (2006/08) and 2) aged over 65 at baseline. Although the HRS includes participants over the age 50, the current study limited the sample to those aged over 65 so the sample characteristics are comparable to the existing frailty literature's population. The final analytic sample was 8,892 individuals. As shown in Table 2.1., compared to the excluded sample, the analytic sample were more likely to be male ($\chi^2(1) = 38.56$, $p < .001$), less likely to be married ($\chi^2(1) = 356.38$, $p < .001$), more likely to be White, non-Hispanic ($\chi^2(1) = 85.12$, $p < .001$). The analytic sample were older (t (14,256.22) = −169.39, $p < .001$), had less years of education (t (14,560) = 16.83, $p < .001$), had fewer depressive symptoms (t (10,636.31) = 4.81, $p < .001$), and lower cognitive functioning score (t (12,252.41) = 31.83, $p < .001$) than the excluded sample.

Among the selected sample, about a half (49%, N = 4,397) of the participants had frailty scores for all six waves, 14% (N = 1,238) has frailty scores available for five waves, 12% (N = 1,034) for four waves, 10% (N = 866) for three waves, 11% (N = 986) for two waves, and 4% (N = 371) for single wave. On average, the participants had 4.68 (SD = 1.60) time points of frailty data over six waves.

Measures

Frailty. Accumulation of deficit model of frailty index (FI) was adopted to measure frailty in each wave (Mitnitski, et al, 2001; Rockwood et al., 2004, 2007). As a multi-domain approach to frailty, FI views frailty as an accumulated burden of diseases, functional disabilities, and other health-related deficits and symptoms (Rockwood et al., 2004). FI is designed to provide flexible approach to the components of the measurement, enabling the application to multiple surveys and comparisons (Searle et al., 2008). The total number of deficit selection in the current study will be informed by previous studies that used HRS data (Lohman, Mezuk, & Dumenci, 2017; Lohman, Dumenci, & Mezuk, 2014, 2016; Lohman & Mezuk, 2013; Mezuk, Lohman, Rock, & Payne, 2016). The selected items follow the inclusion criteria suggested by Searle and colleagues (2008). As a result, a total of 32 items were selected. The total list of items and coding schemes are presented in Table A1. A continuous variable was created as the sum of present deficits divided by the valid number of items each individual had. For example, if a person had deficits in 8 items and had valid responses in all 32 items, their frailty index is 0.25. Note that depressive symptoms item from CES-D is not included in FI.

Social network size. In HRS leave-behind questionnaire, the questions regarding particular relationship types has been prompted with asking if the respondents have those relationships (Smith et al, 2017). The questions asked if they had spouse ("Do you have a husband, wife, or partner with whom you live?"), children ("Do you have any living children?"), family members ("Do you have any other immediate family, for example, any brothers or sisters, parents, cousins or grandchildren?"), and friends ("Do you have any friends?"). The answer options were 'yes' and 'no'. Further questions about that relationship type were skipped if the respondent answered no to having those relationships. Next, respondents were asked about the

number of close people for each relationship type in following question: “How many of your children/family members/friends would you say you have a close relationship with?” The answers were documented in raw numbers in HRS. For the analysis, the answers for network size were capped at 20 for each relationship type because very few participants (1% for children, 2% for family, 2.6% for friends) have reported having more than 20 close relationships of each type. Additionally, those who have reported to have no close children, family, and friends in the prompting question were given zero for network size of that relationship type.

Frequency of contact. Frequency of contact with the children, family members, and friends were based on total contacts using three modes of communication. The question was preceded by the following statement: “On average, how often do you do each of the following with any of your children/any of these family members/ friends, not counting any who live with you?”. The modes of communication included (a) meet up (include both arranged and chance meetings), (b) speak on the phone, and (c) write or email. Each items were measured in 6-point scale, (1) three or more times a week, (2) Once or twice a week, (3) once or twice a month, (4) every few months, (5) once or twice a year, (6) less than once or twice a year or never. Scores were reversed coded so that higher values indicated more frequent contact. Scores of the three types of communication modes were averaged for each type of relationship (Smith et al, 2017). Because the spousal relationship was defined as spouses who lives together with the respondent, the question about frequency of contact is not applicable to spouses.

Social relationship quality. Respondents were asked to rate the perceived support and strain with four relationship types: (a) spouse, (b) children, (c) family members, and (d) friends. The items for social support and strain was used by previous studies on social exchanges in older

populations and were found to be reliable (e.g. Ingersoll-Dayton et al., 1997; Ryan, Wan, & Smith, 2014; Schuster, Kessler, & Aseltine, 1990; Walen & Lachman, 2000).

First, relationship support was assessed using the following three items: (a) "How much do they (spouse, children, family, and friends) really understand the way you feel about things?" (b) "How much can you rely on them if you have a serious problem?" and (c) "How much can you open up to them if you need to talk about your worries?". The original response options ranged from 1 (a lot) to 4 (not at all). The responses were reverse coded so that higher values indicated higher levels of support (1 = not at all, 2 = some, 3 = a little, 4 = a lot). Relationship support was calculated as the average across three items per relationship types. In the current sample, internal consistency ranged from .79 to .86 (α_{spouse} = .79/ .81, $\alpha_{children}$ =.80/ .81, α_{family} = .85/ .86 , $\alpha_{friends}$ = .83/ .82, 2006/08 wave, respectively).

Second, relationship strain asked as the perceived negative support they receive from the relationships. Strain included following four items: (a) "How much do they (spouse, children, family, friends) criticize you?", (b) "How much do they let you down when you are counting on them?", (c) "How often do they make too many demands on you?", and (d) "How much do they get on your nerves?". All responses were reverse coded so that higher values indicated higher levels of strain (1 = not at all, 2 = some, 3 = a little, 4 = a lot). Four items were averaged to construct a relationship strain score. Internal consistency ranged from .72 to .79 in the current sample (α_{spouse} = .77/ .79, $\alpha_{children}$ = .77/ .77 , α_{family} = .77/ .77, $\alpha_{friends}$ = .74/ .72, 2006/08 wave, respectively). Response with missing values for two or more items on the support scale and three or more items on the strain scale were treated as missing (Smith et al., 2017).

Loneliness. Loneliness were measured using a shortened version of the UCLA Loneliness Scale, which has been tested for its validity with HRS sample (Hughes, Waite,

Hawkley, & Cacioppo, 2004). The three items were assessed with the following questions: "How often do you feel that you lack companionship?", "How often do you feel left out?", "How often do you feel isolated from others?" The response options were 1 (often), 2 (some of the time), and 3 (hardly ever or never). Scores were reversed coded so that higher values indicated higher loneliness. The average of the three items were calculated and score were set to missing if there was more than one missing item (Smith et al., 2017). The internal consistency of this measure was good (α = .82/.80, 2006/08 wave, respectively).

Demographic and health status covariates. Baseline demographic and health conditions covariates are included in the analyses. Participant's *gender* as coded as binary variable (male = 0, female = 1). *Race* was assessed by two questions: "Do you consider yourself primarily White or Caucasian, Black or African-American, American-Indian, or Asian?" and "Do you consider yourself Hispanic or Latino?''. The responses were recoded to represent four categorical variables of race/ethnicity (0 = White, non-Hispanic, 1 = Black, non-Hispanic, 2 = Hispanic, 3 = Other, non-Hispanic). As a covariate in growth mixture modeling of frailty, a binary race variable was used (0= all other, 1 = White, non-Hispanic). *Education level* was a continuous variable based on self-reported years of education. *Age* was a continuous variable of respondent's age calculated based on birthdate.

Depressive symptoms were measured with a summary score of a shortened 8-item version of the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). The 8-item version has been validated for high reliability and validity in older adult populations (Karim, Weisz, Bibi, & Rehman, 2015). The participants were asked to respond 'yes' or 'no' to six negatively worded items ("feeling depressed", "feeling that everything is an effort", "restless sleep", "feeling lonely", "feeling sad", and "could not get going") and two positively worded

items ("feeling happy" and "enjoying life") based on their experiences for the preceding week. Two positively worded items were reverse coded and the responses for the eight items were added resulting in scores ranging from 0 to 8. High scores indicate more depressive symptoms.

Cognitive functioning was assessed in HRS using modified version of the Telephone Interview for Cognitive Status (TICS; Brandt, Spencer, & Folstein, 1998; Crimmins, Kim, Langa, & Weir, 2011). Items for the modified TICS include: delayed and immediate word recall of ten words (range 0-20 points), serial 7s (subtracting 7 from 100 five times; range 0-5 points), and backwards counting (counting from 20 to 10; range 0-2 points) for a possible total summed score of 27 (Crimmins et al, 2011). HRS RAND data included cognitive functioning values that were imputed to replace missing values, refusals, and any non-applicable responses (Fisher, Hassan, Faul, Rodgers, & Weir, 2017).

Analysis Plan

The sample data construction and descriptive analyses were conducted using SPSS 24 (IBM Corp., 2016). Main analyses were conducted in structural equation modeling framework using Mplus 8 (Muthén & Muthén, 1998–2017).

First, preliminary analysis was conducted to explore the characteristics of the sample. Univariate sample means, standard deviations, and analytic sample size, and bivariate correlation among study variables were examined.

Second, growth mixture modeling (GMM) was applied to identify the unobserved sub-populations (i.e. latent classes) that follows distinctive trajectory patterns of frailty progression. GMM assumes that the heterogeneity within data may indicate that there are qualitatively different groups that follows different pattern of change and that the group memberships for individuals are not known *a priori* or based on observable variables (Muthén & Muthén, 2000). The goal of GMM is to identify multiple latent classes, to infer the class membership for each

individual based on the posterior class membership probability, to describe longitudinal change within latent classes, and to examine the differences in change among latent classes (Ram & Grimm, 2009; Berlin, Parra, & Williams, 2014). Four step procedures (i.e. problem definition, model specification, model estimation, and model selection and interpretation) were used to implement GMM analysis (Ram & Grimm, 2009).

In step 1, the goal is to determine the growth model for the overall sample. To determine the model of change, I fitted the unconditional latent growth curve model (LGCM) of frailty in no-growth, linear, quadratic, and latent basis model, sequentially. Latent basis model is an adequate choice if the data doesn't fit in typical linear or quadratic patterns. In latent basis model, the first loading was fixed to zero and the last was fixed to one. The free estimation of the remaining factor loading reflected the relative percent of growth achieved by the last time period (Grimm & Ram, 2009). Next, a set of model fit indices was be used to determine the goodness-of-fit of the models. This include model chi-square statistics (χ^2/df), comparative fit index (CFI; Bentler, 1990), and root mean square error of approximation (RMSEA; Steiger & Lind, 1980). Using combination of absolute and relative fit indices is recommended to strengthen the reliability of the model selection. Cutoff values to determine good fit of the model are CFI (good models >= .95), RMSEA (good models <= .06; Hu and Bentler, 1999; Ferrer & McArdle, 2010). Based on the fit indices I determined the forms of the growth model. This growth model served as a starting point from which further latent classes were explored in later steps.

In step 2, I generated hypothesis about the number of latent classes and how the classes will differ in terms of mean change, interindividual differences in change, and pattern of change. In this step, the number of classes and group differences are specified reflecting the theory and previous empirical findings. I fitted models estimating one to six classes sequentially. It was

recommended to fit models with at least one more class (c+1) than the hypothesized number of classes and review the changes in model fit. In this way, if the fit of the c+1 model is worse than the model with c classes, there is statistical evidence to reject the c+1 model and to select c class model. Regarding group differences, Ram and Grimm (2009) suggest exploring models that differ in terms of means, means+covs, and means+covs+pattern for each number of classes. The first means group difference model allows for the classes to differ on the intercept and slope mean. The second means+covs group difference model additionally allows for variances and covariances of the intercept and slope factors to vary across groups. The third means+covs+pattern group difference model additionally allows for differences in the shapes and patterns (e.g. one group is linear, and another group is quadratic).

In step 3, I selected estimation method for GMM. The expectation-maximization (EM) estimation procedure was used to obtain maximum likelihood estimates of all model parameters, and individual posterior probabilities (Ram & Grimm, 2009). Specifically, full information maximum likelihood estimation (FIML) was be used with Mplus (Muthén & Muthén, 1998–2017). FIML can accommodate the missing data on the outcome variable when the missingness condition is at least missing at random (MAR; Enders, 2001). Under the MAR condition, the probability of missingness in one variable may depend on other observed variables (Rubin, 1976). FIML utilizes all available information from the covariance matrix of other observed variables to estimate the mean and variance of the incomplete variables' missing portions (Enders, 2001). In addition to MAR, FIML requires multivariate normality for the joint distribution of all the variables. When the two assumptions are met, FIML is shown to yield unbiased parameter estimates and valid model fit information (Enders, 2001, 2011; Enders & Bandalos, 2001). Furthermore, the models were estimated using the maximum-likelihood

method with robust standard errors (MLR) to handle the possibility of non-normality and non-independence of observations in the data.

In step 4, the best model was selected based on examining the model fit indices and the interpretability of the classes and estimates. A set of model fit indices that are applicable for mixture modeling was used (Ram & Grimm, 2009). First, models were compared with relative fit information criteria such as Bayesian information criteria (BIC), and adjusted BIC. Better fitting model would have lower values for these indices (Nylund, Asparouhov, & Muthén, 2007). Second, entropy is a summary indicator useful in determining the confidence of class membership of each individuals (Jedidi, Ramaswamy, & Desarbo, 1993; Muthén, 2004). The higher value of entropy (ranging from 0.00 to 1.00) indicates that there is adequate separation between latent classes. Third, likelihood ratio tests such as the adjusted Lo–Mendell–Rubin likelihood ratio test (Adjusted LRT) was used to compare the number of classes (Lo, Mendell, & Rubin, 2001; Nylund et al., 2007). The significance ($p < .05$) of the tests represents that a model with c number of classes is better fitting than the model with one less (c-1) classes. Additionally, the proportion of each classes was considered so that no class has less than 1% of the sample (Jung & Wickrama, 2008). Additionally, GMM fit indices were examined for both unconditional GMM and conditional GMM. It is advised that antecedent information in the form of covariates should be included in the model to improve the results of the GMM (Muthen, 2004). Thus, in the conditional GMM, gender, race, age, and education years were included in the model as covariates predicting the intercept and slope factors and class factor (Muthen, 2004). The decisions regarding the model fit indices from one to six class solutions were made based on both unconditional and conditional model. After selecting the best fitting model, the group trajectories were plotted and inspected regarding the hypotheses.

Third, after frailty groups were identified with GMM, I examined the characteristics of each frailty classes on their demographics, health status, and social relationship variables. Chi-Square and Analysis of Variance (ANOVA) was conducted to examine the differences between classes. Post hoc comparison methods were used according to the Levene statistics result for testing the assumption of homogeneity of variances: Tamhane's T2 (when the assumption of equal variances was not met) and Tukey HSD (when the assumption of homogeneity of variances were met).

Finally, a series of multinomial logistic regression were conducted to determine the predictors of frailty progression classes. Multinomial logistic regression estimates the link between a given predictor characteristics and the relative probability of belonging in a particular frailty progression class compared to the reference class. The reference group was rotated so that all possible comparisons were made between frailty groups. I used sequential modeling strategy to evaluate demographic, health, and social relationship factors on frailty progression. In the model for testing the research question regarding social network characteristics, first I entered the baseline demographic, health status, and social network (size and contact frequency) variables to the model. In the second model, I added loneliness variable. Next, in the model testing the research question regarding the relationship quality, first I entered baseline demographic, health status, social network characteristics, and social relationship quality with different social ties in the model. In the second model, I added loneliness variable.

Results

Descriptive analyses

Descriptive statistics for the study variables are displayed in Table 2.2. The mean score for frailty index in first wave (2006) was 0.24 and the mean score increased over the six waves.

The sample was relatively balanced in gender (female 58%), had mean age of 74, had high school or more education, and mostly White (79%). About 59% of the sample were living with their spouse or partner. Bivariate correlations between the study variables are shown in Table 2.3. Among the control variables, depressive symptoms had moderate level of correlation with the frailty scores (r = .37-.49). Among the social predictor variables, moderate to high level of correlations were found between the social support between relationship types and social strain between relationship types (e.g. children strain and family strain, r = .56).

Changes in frailty in older adulthood

The study first explored the changes in frailty over the six waves using latent growth curve modeling. The model fit indices are presented in Table 2.4. The growth curve was modeled without covariates (unconditional). Among the growth models, the latent basis model (χ^2 (12) = 749.36, p < .001, CFI = .964, RMSEA = .083) and quadratic growth model (χ^2 (12) = 724.69, p < .001, CFI = .965, RMSEA = .082) showed good model fit. Although the quadratic model had similarly desirable model fit, the latent basis model was chosen for subsequent analysis. First reason is the detailed growth rate that latent basis model provides, and second, the relatively simpler model interpretation compared to the quadratic model (e.g. it is difficult to empirically interpret intercept, linear, and quadratic terms at the GMM stage when some of the terms are non-significant). Results for the latent basis growth model indicated that frailty increased slightly (intercept mean= 0.24, linear slope mean = 0.1), with steady growth pattern over the waves (factor loading = 0, 0.046, 0.415, 0.511, 0.841, 1, for the six waves).

Identifying group trajectories of frailty

To answer the first research question, the study explored whether there are heterogeneous groups of frailty progression using growth mixture modeling. The final estimation specification

allowed for the classes to differ on the means for latent variables (e.g., intercept and slope), but same across the classes for other parameters (estimated the variances of the latent variables, variances and residual variances for observed variables, and covariance among the latent variables). The model fit indices of the one- to six-class models are displayed in Table 2.5. The model used the latent basis growth curve model which was established for overall growth model of frailty. Both unconditional and conditional GMM model with demographic covariates (gender, race, age, and education) were run to aid the class selection. The changes in BIC, ABIC, and entropy, and LMR test suggested that 3-class solution or 4-class solution had best fit. Although the 4-class solution are tested to be better fit according to the LMR test but deemed problematic for two reasons: for the fourth class, only 2% of the sample was assigned and the parameter estimates were different between unconditional model and conditional model. Specifically, the fourth class' slope factor mean was considerably different when covariates are included in the model. This is possible because small sample of the fourth class may have low power to detect the significance of slopes within trajectory classes (L. Muthén & Muthén, 2002; Frankfurt et al., 2016). Further, 3-class solution had high average probability of class membership for the three classes (0.816 - 0.929) and displayed similar pattern with the previous empirical findings. Thus, the 3-class solution was selected. Parameter estimates for the three-class model is shown in Table 2.6. The illustration of the estimated trajectory of the three classes are depicted in Figure 2.1.

Among three groups, the majority (81%) of the sample belonged to the group with the low initial frailty level with small increase over time (intercept factor mean = 0.2, slope factor mean = 0.09). Thus, I labeled this group as "average frailty group". Second group included 12% of the sample and had high initial frailty level and small increase pattern (intercept factor mean =

0.45, slope factor mean = 0.07). This group had much higher frailty score compared to the average group but kept a relatively steady level of frailty over time. Thus, I labeled this group as "high frailty group". Lastly, 8% of the sample belonged to the group with low initial frailty level with steep increasing pattern over time (intercept factor mean = 0.23, slope factor mean = 0.49). This group was labeled "steep increase frailty group".

Description of frailty trajectory Groups

The demographic, health status, and social variables were described and compared in Table 2.7. There were more women in high frailty group (68%) than the average (56%) or steep increase group (59%, χ^2 (2) = 50.66, $p < .001$). As expected, high frailty and steep increase frailty group were older (F(2, 8887) = 309.24, $p < .001$), had less education (F(2, 8887) = 123.04, $p < .001$), higher depressive symptoms (F(2, 8569) = 708.25, $p < .001$), and low cognitive functioning than the average frailty group (F(2, 8572) = 237.12, $p < .001$). There were more persons who live with their spouse/partner in average frailty group (64%) than high (49%) and steep increase (49%) frailty group (χ^2 (2) = 124.889, $p < .001$).

In terms of the social network characteristics, there were no group differences in overall network size, the number of children, and family members. Average frailty group had more close friends than the high frailty group (F(2, 8035) = 11.85, $p < .001$). In general, average frailty group had higher frequency of contact in social relationships than the other two groups. In terms of the social support, high frailty group reported perceiving low support than the other two groups from kin (Spouse, F(2, 5531) = 53.56, $p < .001$; Children F(2, 8127) = 32.45, $p < .001$; Family F(2, 8169) = 4.51, $p = .011$). There were no group differences in friends' support. Similarly, the high frailty group reported more strain with all social relationships (Spouse, F(2, 5503) = 23.13, $p < .001$; Children F(2, 8103) = 78.26, $p < .001$; Family F(2, 8137) = 60.97, p

= .011; Friends $F(2, 8106) = 13.28$, $p < .001$). and higher loneliness than average frailty or steep increase frailty group ($F(2, 8562) = 235.27$, $p < .001$).

Predicting frailty trajectory groups by social network characteristics

To answer the second set of research questions and related hypotheses, I examined the associations between baseline social network characteristics and frailty progression groups. The multivariate logistic regression results are shown in Table 2.8.

In model 1, baseline demographic factors, health conditions, social network size, and contact frequency is entered to predict frailty group membership. For the demographic and health variables, women were more likely to belong in the high frailty group than the average frailty group ($b = 0.38$, $p < .001$) or the steep increase group ($b = -0.34$, $p = .005$). Older age was predictive of belonging to the steep increase frailty group rather than the average frailty ($b = 0.11$, $p < .001$) or the high frailty group ($b = 0.11$, $p < .001$). Hispanic older adults were more likely to belong in the average frailty group ($b = -0.45$, $p = .005$) or the steep increase frailty group ($b = 0.46$, $p = .033$) than the high frailty group, compared to Whites. Higher depressive symptoms were associated with belonging to the high frailty group than to the average frailty ($b = 0.44$, $p < .001$) or the steep increase group ($b = -0.40$, $p < .001$). Those with better cognitive functioning were most likely to belong in the average frailty group than the other two less healthy group (compared to High frailty group $b = -0.07$, $p < .001$; Steep increase frailty group $b = -0.09$, $p < .001$).

In terms of the social network variables, living together with spouse or partner was not associated with frailty trajectory groups. Having higher number of close family members were associated with belonging to the high frailty group than the average frailty group ($b = 0.02$, $p = .043$). Frequency of contact with friends were associated with belonging to the average frailty

group than other two less healthy group (compared to High frailty group b = -0.18, p < .001; Steep increase frailty group b = -0.14, p = .005).

In model 2, the loneliness variable was added to the model to examine whether feeling lonely would explain the associations between social network features on the probability of frailty group membership. Adults who felt lonely were more likely to belong in the high frailty (b = 0.40, p < .001) and the steep increase frailty group (b = 0.30, p = .001) than the average frailty group. Interestingly, the notable effects mentioned above (e.g. being Hispanic, depressive symptoms, cognitive functioning, number of family members, and contact frequency with friends) were stable even when loneliness was controlled, suggesting their unique effects on frailty group membership.

Predicting frailty trajectory groups by relationship quality characteristics

Lastly, to answer the third set of research questions, the associations between baseline social relationship qualities with social ties (i.e. spouse, children, family, and friends) and frailty progression groups were examined. The multivariate logistic regression results are shown in Table 2.9. In model 1, the information about the social network size and contact frequencies are kept in the model to account for their influences on frailty group membership. Additionally, loneliness was added in model 2.

In model 1, in general, perceived support from social ties was not associated with frailty group membership. Unexpectedly, those who were perceiving higher support from friends were more likely to belong in the steep increase frailty group than the average frailty group (b = 0.15, p = .047). Those who perceive higher strain with their spouse were more likely belong in the steep increase frailty group rather than in the average frailty (b = 0.22, p = .034) or the high frailty group (b = 0.36, p = .006). Strain with children was associated with belonging to the high

frailty group than average frailty group (b = 0.29, p = .001). Strain with family members were associated with belonging to the high frailty group than the average (b = 0.22, p = .007) or the steep increase frailty group (b = -0.28, p = .026).

In model 2, loneliness was added to the model to test for possible changes in the effect of social network and relationship quality predictors. Loneliness were associated with belonging to the high frailty group than the average frailty group (b = 0.33, p < .001). When loneliness was accounted, spousal strain effect on the likelihood of belonging to the steep increase frailty group than average group disappeared, but the effect was present for the higher chance to belong in steep increase frailty group than high frailty group (b = 0.37, p = .010). Largely, the existing effects of children and family strain on high frailty group membership remained significant after loneliness were controlled in the model.

Discussion

Based on the convoy model of social relations (Antonucci et al., 2013), the current study investigated the multidimensional nature of social relationships in relations to frailty progression. First, I found that there were three distinctive subpopulations of older adults who share similar frailty trajectory, namely the average frailty group, high frailty group, and steep increase frailty group. Second, when social network and relationship quality predictors were examined, living with spouse, network size, and frequency of contact with kin were not influential in the probabilities for belonging to different frailty groups. Only higher frequency of contact with friends were associated with lesser frailty. When contact frequency was controlled, having a larger family was associated with belonging to higher frailty group. Third, when the positive and negative relationship quality with different relationship types was examined, only the negative quality (i.e. strain) was influential to the frailty progression. Perceived strain with the spouse,

children, and family members all had additive influence on the membership to the higher or steep increase frailty group, compared to the average frailty group. Fourth, greater loneliness was associated with belonging to the high frailty group. Loneliness influenced the link between spousal strain and the steep increase frailty group membership. Overall, the negative effect of large network size, children and family strain remained influential to having higher frailty, even with controlling for loneliness.

Changing patterns of frailty

Although the three frailty progression groups from this study reflects the general findings from the existing literature, but also have differences in the grouping patterns and measurement model of frailty. I found that "average frailty group" was the largest with low initial frailty level with small increase over time. About 12% belonged to the "high frailty group" with much higher initial frailty level, accompanied by small increase in frailty over time. The "steep increase frailty group" was the smallest group (8%) with low initial frailty level but exhibited steep increase trend in frailty, reaching the most problematic level of frailty over the 11-year study period.

Among the prior studies that examined longitudinal change patterns of frailty with large-scale data, Chamberlain and colleagues (2016a, 2016b) used a continuous measure of frailty based on frailty index (32 items, yearly from 2005-2012) similar to the current study. They stratified the older population by age: 60–69, 70–79 and 80–89 years, and found three group solutions for 60–69 age group and two group for older groups. The authors note that there is greater variability in frailty in younger ages (Chamberlain et al., 2016a). Two other studies have also reported three distinctive groups. With Taiwanese data spanning 14-years, Hsu and Chang (2015) found a largest group that maintained non-frailty (44%), developing frailty group with sharp increase (39%), and high-risk frailty group with high level but slight decrease in later time

points (18%). With Mexican older adults, Peek and colleagues (2012) found one group with lowest level of frailty throughout the 12 years, but with smallest proportion. Their largest group had higher level of frailty with sharp increase in later years, and other high frailty group with temporary slight decline during the middle years. However, the two studies have used Fried's model of phenotypic frailty with ordinal variable and group-based mixture modeling in SAS PROC TRAJ (Jones & Nagin, 2007), which may suggest the exact comparison is difficult.

In terms of the demographic and health characteristics of frailty groups, the current investigation found that being female, older age, less education, higher depression, and low cognitive functioning were associated with belonging to high and/or steep increase frailty group. These demographic and health differences generally match with the frailty literature (Clegg et al., 2013; Collard et al., 2012; Hsu & Chang, 2015; Mezuk et al., 2012).

Social engagement and frailty: getting together with friends

The current study extends the previous findings by examining the differential effects of structure and quality of social relationship types. Living with spouse, the number of children, the number of friends, and the frequency of meeting with kin (i.e. children and family members) were not influential in frailty progression. Rather, the frequency of meeting friends was the defining factor in predicting frailty trajectory group, such that the more you meet with friends, the less likely you were to be in high frailty or steep increase frailty group. This positive effect of friendship contact was not diminished when loneliness was included in the model.

Network size itself was not important for frailty. Previous research also indicated neither network size predicted onset of chronic disease (Hill, Weston, & Jackson, 2014) nor mediated the link between education attainment and frailty (Hoogendijk et al., 2014a). These findings resonate with the socioemotional selectivity theory in social relationships in later adulthood

(SST; Carstensen, 1992). The SST supports the idea that older adults may experience a change in time perspective, shifting their focus into prioritizing emotionally meaningful relationships and a sense of meaning. Starting at midlife, network size of tends to shrink as adults choose to "prune out" less rewarding and peripheral relationships (Carstensen, Isaacowitz, & Charles, 1999; Lang, 2000). However, selected relationships are kept close, older adults report experiencing more positive and fewer negative emotions with their network members, compared to younger adults (English & Cartensen, 2014). Reflecting this notion, it is reported that adults keep a similar frequency of visits with families over the adult years, but the non-family (i.e. neighbors, friends, and acquaintances) visits declined starting from middle adulthood, with strong decline after mid 60's (Sander, Schupp, & Richter, 2017). Thus, the network size itself, or a smaller network size, is not an indicator of undesirable social condition for frailty progression.

Furthermore, the friends that are kept close were particularly important for older adults' frailty. Studies consistently found that having balanced proportion of friends in one's social network was found to promote mental health (Fiori et al., 2006; Litwin & Shiovitz-Ezra, 2011), cognitive health (Li & Dong, 2017), and reduce mortality (Litwin & Shiovitz-Ezra, 2006). How do friendships contribute to better health? Unlike family ties that are more predetermined, friendships are formed in more voluntary and informal manner. This also means that it takes more active efforts to maintain friendships (i.e. communication, shared activities; Roberts & Dunbar, 2011). When engaging with social partners, it is particularly beneficial if the older adults have the sense of agency in the process (Herzog, Franks, Markus, & Holmberg, 1998). Friendships can also provide the sense of belonging, especially when relationships based on employment or organizations may diminish in older age (Stevens, Martina, & Westerhof, 2006; Ten Bruggencate, et al. 2018). Thus, even though it may require more effort, older adult can

exercise their agency and social identity through relationship with friends. Further, the relationship with friends are motivated by pleasure and companionships (Pinquart & Sorenson, 2000). Friends usually share similar values, roles, and interests, which makes the companionship enjoyable and rewarding (Blieszner & Roberto, 2004). Indeed, it was shown that spending time with friends had greater effect on life satisfaction than family members for older adults (Huxhold, Miche, & Schüz, 2013). It can be explained that friends serve as interaction partners for informal social activities without the sense of roles and obligations, which may be present in time with family members (Blieszner & Roberto, 2004). Moreover, friendships can provide tailored support to the adult's needs. Friends tend to be age peers and those who share similar experiences, which may be the reason that people are more likely to confide in or seek solace in friends (Blieszner & Roberto, 2004). For example, among spouses of dementia patients, resilient spouses were more likely to receive informational support from groups and existing friends with shared experiences (Donnellan, Bennett, & Soulsby, 2017).

Regarding these psychological and emotional aspects of friendships, the study hypothesized that reduced loneliness as a potential mechanism to link friend contact to lower frailty. Previous studies showed that support from friends improves well-being via alleviating loneliness (Chen & Feeley, 2014; Nicolaisen & Thorsen, 2017). A cross-sectional study found that older adults with frailty were lonelier, if they had less friends in their network (Hoogendijk, Suanet, Dent, Deeg, & Aartsen, 2016). However, the current study found that the positive effect of contact with friends was strong even when loneliness was accounted for. Thus, it is possible that the mechanisms are more cognitive and physical pathways. Frequent contact with friends may promote mental and physical activity which may linked to lesser frailty. Recent work found that frequent contact with friends had protective effects on memory decline, net of other

structural and quality aspects of social relations (Zahodne et al., 2019). They suggest that social interactions with friends may be mentally stimulating, such that it requires multiple domains of cognitive functioning such as attention, memory, socio-emotional regulation as well as processing of new information (Zahodne et al., 2019). Further, older adults preferred doing physical activities with people with similar age and less-structured format (Burton, Khan, & Brown, 2012), which might imply that friends can function as a facilitator of physical activity for older adults.

Social Stress and frailty: strain with family as a chronic stressor?

Based on previous research on the complex quality of social relationships, both support and strain were modeled simultaneously to examine their relative influence (Ingersoll-Dayton et al, 1997; Newsom et al., 2003, 2005; Rook, 1990). Corresponding to the literature, higher social strains were influential to frailty, but not social support (Gale et al., 2012). Scholars explain that negative exchanges with the social partner generally occur infrequently, but there are stronger physical and psychological impact when they do occur, sometimes outweighing the positive exchanges (Brooks & Dunkel Schetter, 2011; Rook, 2015). For example, it was reported that frequent negative interactions with social relationships were associated with increased risk for incident hypertension four years later (Sneed & Cohen, 2014) and risk of mortality by stroke (negative interaction with family members; Tanne, Goldbourt, & Medalie, 2004).

Differentially by relationship types, the results indicated that spousal strain was associated with belonging to the steep increase frailty group. The strain with children and family members were independently associated with belonging to the high frailty group. In comparison, spousal strain was associated with sharp worsening pattern of frailty, which is different than high frailty pattern associated with children and family member. This difference may imply that stress

from marital relationship are uniquely harmful to frailty. Spouses are most proximal person and involve in daily interaction regarding shared environment. The chronic and proximal nature of conflict with the spouse may provoke the development of frailty with amplified stress.

Overall, the negative interaction (i.e. conflict, irritation, or criticism) with kin members can be a salient source of chronic stress that undermines health. Although the reasons for how and why strain with kin members becomes a stress were not addressed in this study, existing theories and research may provide some insight. The convoy model reminds us that interaction quality with social relationships are built from previous experiences and cumulative over time (Antonucci et al., 2013). The children, and family members who the respondents report their perceived support and strain are those who are in long-term kin relationship together. Unlike non-kin relationship such as friends which can be chosen and pruned out when deemed stressful, kin relationships such as children and extended family are expected to be continued even when the quality is low. Also, appraisal process mediates the stressful events or interactions to affect the health outcomes (Newsom et al, 2005). It is possible that changing needs and situational factors related to later life (e.g. retirement, informal care needs) might brought disruptions to the existing role expectation, creating new conflict. While current study did not examine whether the social strain is long-term or short-lived, a previous study reports that prolonged higher levels of negative social exchanges significantly predicted lower self-rated health, greater functional limitations, and more health conditions over two years (Newsom, Mahan, Rook, & Krause, 2008).

Physiological mechanisms could provide a possible pathway for linking social stress to the development of frailty. While the pathophysiological processes underlying the development of frailty are not clearly established, evidence suggests that abnormal inflammatory responses

(i.e. hyper-responsivity to stimuli and persisting response after the removal of initial stimuli) play a major role in developing frailty symptoms (Clegg et al., 2013; Hubbard, O'Mahony, Savva, Calver, & Woodhouse, 2009; Yao, Li, & Leng, 2011). A systematic review of research suggested that higher levels of inflammatory biomarkers, such as C-reactive protein (CRP) and Interleukin-6 (IL-6), are linked to frailty and pre-frailty (Soysal et al., 2016). There is mounting evidence that social negativity has a particularly strong association with the dysregulation of endocrine, immune, and inflammation functions (review by Kiecolt-Glaser, Gouin, & Hantsoo, 2010; Wright & Loving, 2011). For example, a longitudinal study of midlife older adults found that strain with family substantially increased risks of heightened inflammation biomarkers, and that the negative influence of social strain was stronger than the positive effect of social support (Yang, Schorpp, & Harris, 2014). Similarly, negative interactions with spouse and family members was associated with elevated allostatic load (i.e. an index of physiological risks in multiple systems) in older adults (Brooks et al., 2014; Priest et al., 2015). Thus, it is possible that social strain is associated to frailty development via interrupting the regulation of the inflammatory responses.

Limitations

Findings of the current study should be understood in context of the study limitations. First, the study used longitudinal data available from HRS for social relationship measures and frailty index. Due to the implementation of leave-behind questionnaire in 2006-2008 waves, the prospective trajectory of frailty was modeled with data collected afterwards. While longitudinal data spanning 11 years of time is valuable, it may not be sufficient to track developmental trajectory of frailty. Especially that frailty was modeled based on the accumulated deficit model (frailty index, Mitnitski et al., 2001; Rockwood et al., 2004), the incremental progression frailty

could be modeled better with long-term data. Second, the sample selection limits the generalizability of the study to those who are community-dwelling older adults. Noting that frailty is more prevalent in residents of care institutions and highly related to hospitalization (Kogima, 2016; Rockwood et al., 2006), the study was only modeling frailty trajectory and social predictors from a relatively healthy and functionally independent population. Third, the chosen methodology for longitudinal analysis has weakness. Growth mixture modeling was useful to identify heterogeneous groups of older adults that develop frailty and comparing distinctive groups in their predictors. However, if the future studies were to focus on mechanisms that include multiple antecedents and health outcomes of frailty, other longitudinal models such as latent growth curve modeling would be appropriate to test mediation and moderation processes. Fourth, while my study comprehensively included and compared different aspects of social relations, there are many remaining questions to be answered on how social relationships shape frailty. For example, I find that contact with friends were beneficial for remaining low on frailty. However, the measure only asked how often they contact with friends, not revealing the contexts of friendship activity. The current measure averaged meeting in person, phone call, and writing to contact friends, so differential effect of the mode of communication is not analyzed in the study. Further, the nature or goal of friend contact is also missing. Because the study did not separately measured social participation activity (i.e. volunteering, religious groups etc.), it is possible that the contact with friends also include these types of activity. Fifth, the social support types are not included in current study. Current study's measures of social support and strain are focused on the psychological aspects of the quality (i.e. understand the way you feel about things?). Future study should investigate whether different type of support, such as instrumental, knowledge, affirmation, or the exchange of social support (i.e. provision of support and receiving

support) are associated with frailty progression. Prior study found that inability to provide support to family and friends were related to increased frailty (King, Fillenbaum, & Cohen, 2017), which hints that active social role, rather than being a recipient of support could be protective of frailty. Finally, study design with network member specificity would extend the findings from this study. Because the question regarding social relationship types are asked about the overall relationship (i.e. overall family, overall friends), it is difficult to distinguish whether there is specific person who has the most influence or the relationship types are the factor of the perceived social quality. Future studies can benefit from social network roaster method to investigate the interaction quality and overall social relationship composition to further explore my findings.

Conclusion

Using a large, national sample of older adults, this study identified heterogeneous trajectory of frailty progression over 11 years. Employing a convoy model of social relations, the study is the first to comprehensively examine the social relationship predictors of frailty development. It demonstrated that the frequent contact with friends were protective of frailty development, whereas larger family size, relationship strain with spouse, children, and family members were independently associated with higher frailty or steeply increasing frailty trajectory. These relationship effects and loneliness independently predicted frailty progression. The pathology and contextual antecedents of frailty is still much unknown, but frailty is recognized as a warning sign for high risk of adverse health outcomes. This study contributes to the literature by identifying potentially protective and hazardous factors of frailty progression in social relationship domain. Given that social relationship experiences are dynamic and can be improved by intervention, the findings can be useful to intervention design in frailty prevention,

such as promoting activities involving friends. Future population studies on frailty can benefit from incorporating social mechanisms to model the onset and prognosis of frailty and related health outcomes.

CHAPTER 3. EXPLORING THE EFFECTS OF FRAILTY AND DEPRESSION ON MARITAL QUALITY: A DYADIC LONGITUDINAL INVESTIGATION

Introduction

Frailty is prevalent in advanced age and the progression pattern differs considerably in older population (Cesari et al., 2016; Chamberlain et al., 2016; Collard et al., 2012). Chapter 2 reports evidence that when multidimensional characteristics of social relationship were examined, the perceived quality of the relationship had independent associations with the trajectory of frailty progression. To further explore potentially reciprocal link between social relationship and frailty, it is also important to investigate the influence of frailty on social experiences. Marital relationship is understood as a critical social context in one's health and well-being across the lifespan. Spouses are often the most proximal figure in one's social convoy, with close daily interactions, understanding of each other's strengths and weaknesses, and shared socioeconomic resources (Robles et al., 2014). Because the well-being of spouses is interconnected, health problems such as an onset of illness can have ramifications for the lives of both partners (Berg & Upchurch, 2007; Hoppmann & Gerstorf, 2016). Research from the dyadic perspective conceptualize that health problems of one or both spouses are likely to impact the couple's homeostasis, and that the couple is challenged to activate their coping resources and renegotiate their role to regain balance (Burman & Margolin, 1992). The couple may perceive positive or negative changes in their marital quality as they strive to cope with the health problem of their own or their partners (Korporaal, Broese van Groenou, & Tilburg, 2013). However, there is limited knowledge of how the level of frailty may influence the marital quality in couples. Moreover, frailty is often accompanied by depressive symptoms (Lohman, Mezuk, & Dumenci, 2017), but the comorbidity of both frailty and depression has yet been studied in terms

of their consequences in social domain. Given that depression is known as a particularly taxing health context for both partners in a marital dyad (Benazon & Coyne, 2000; Pruchno, Wilson-Genderson, & Cartwright, 2009), it is important to consider the co-existence of frailty and depression within a person or across partners as a complex health context that challenges marital relationship quality.

The current study examines how couple's health context, the level of frailty and depression, is associated with perceived marital quality in older couples. Using the dyadic longitudinal data from married couples in Health and Retirement Study (HRS), I apply actor–partner interdependence model (APIM; Kenny et al., 2006) to examine the dyadic the effects of own and partner's level of frailty and depression on the level and change of perceived marital support and strain over an eight-year period. Further, the assessment of multiplicative effects of the presence of both frailty and depression within- and across- person in the dyad illustrates nuanced nature of the couple's health context in relation to their marital quality. Lastly, the gender differences among the observed associations are examined. The findings highlight the importance of exploring spousal interactions of chronic health conditions in later adulthood.

Literature review

Frailty and depression: a double threat?

Frailty and depression are prevalent and pervasive health conditions in older age. The prevalence of frailty is estimated to be about 11% (ranging from 4-59%; Collard et al, 2012) and the prevalence of depression ranged from 10-20% in community-dwelling older adults (Rodda, Walker, & Carter, 2011). Recent systematic review studies report the high co-existence of frailty and depression in older population, such that approximately 40% of individuals with depression have frailty and a similar proportion of those with frailty have depression (Buigues et al., 2015;

Soysal et al., 2017). Many studies found evidence for bidirectional risk between frailty and depressive symptoms (Mezuk et al., 2012). Early studies found that, for example, persons with repeatedly elevated depressive symptoms were 3.2 times more likely to be frail in follow up (Strawbridge et al., 1998), and conversely, the onset and persistence of frailty were stronger predictors of depressive symptoms in over 6 years (Yang & George, 2005). Meta-analysis of the strength of reciprocal associations between frailty and depression revealed that, older adults with frailty were 2.6-4.4 times more likely to have depression than non-frail people. Similarly, those with depression were 3.7-4 times more likely to have frailty than non-depressed adults (Soysal et al., 2017). There is evidence that comorbidity of frailty and depression leads to elevated risk for detrimental health outcomes, such as cognitive impairment (Potter, McQuoid, Whitson, & Steffens, 2016). Faster increase in frailty and depressive symptoms were associated with higher likelihood of both nursing home admission and serious falls over time (Lohman et al., 2017).

Some reasons for higher comorbidity between frailty and depression may be from overlapping diagnostic criteria that taps in to similar symptoms, differing conceptual views and measurement of frailty, and the shared etiology (i.e. pathophysiological alterations and inflammation biomarkers; Buigues et al., 2015). Recent studies report that measures of frailty and depression identify large overlapping populations of older adults, with symptom of exhaustion important factor for the high correlation (Lohman et al., 2014). However, empirical evidence points to frailty and depression as interrelated constructs, but not as a single syndrome. A confirmatory latent class analysis showed that the frailty and depression represent distinct syndromes, rather than a single construct (Mezuk et al., 2013). Further, the strong correlation of frailty and depression exists across various measures of frailty and the shared symptomology did not fully explained the high correlation (Lohman et al., 2015). Researchers suggest that when

studying the determinants, progression, or consequences of frailty, it should not be examined in isolation from depression (Lohman et al., 2014). Thus, exploring the separate and interactive influence of frailty and depression may add to the literature on how the two health problems co-exist in context of social relations.

Adverse effects of frailty on marital quality in later life

The existing literature on the adverse effects of frailty has mostly focused on the health outcomes, such as increased risk of falls, functional decline, hospitalization, and mortality (Ensrud et al., 2009; Kogima, 2016; Rockwood et al., 2004). However, when examining social context of frailty, there is limited knowledge about the effects of frailty on social outcomes (Monin et al, 2016). There was only one study that investigated the effect of physical frailty on social functioning, reporting that the high level of frailty was associated with an increase in loneliness over 3 years. They found no further decline in the network size, emotional, and instrumental support (Hoogendijk et al., 2016). The finding illuminates the importance of investigating the social relational dynamics precipitated by frailty.

Marital relationship is a central social relationship to examine in the context of health problems. In later life, spouse is a key member in person's social network (Antonucci et al., 2004), sharing considerable time, living space, financial, and tangible assets (Carr & Springer, 2010). Spouses often serve as the most immediate person to provide emotional, instrumental, and informational support for older adults to manage their health events (Bookwala, 2012). Considering that frailty often leads to disease onset, disability, and institutionalization, the lack of, or negative marital relationship may become the risk factor for further decline in health. Indeed, marital conflict has been found to accelerate the decline in physical and mental health

and linked to risk of mortality (Birditt & Antonucci, 2008; Waite, Luo, & Lewin, 2009; Wickrama, Lorenz, Conger, & Elder, 1997).

Further, the concept of patient-caregiver dyad may not be suitable for studying frailty or depression. So far, most of the studies regarding health effects on marital relationship comes from spousal caregiving literature. Often these studies assume distinction among the couple as a patient and a caregiver and has specific illness for the study population. However, in older couples, both persons may be dealing with health conditions of their own, making the distinction of patient-caregiver inaccurate description of the couples' experience (Korporaal et al., 2013). Moreover, both persons may be diagnosed and managing multiple conditions simultaneously (Polenick, Renn, & Birditt, 2018). Research on multimorbidity shows that the presence of a mental health disorder increased as the number of physical conditions increased, exacerbating the complexity of conditions (Barnett et al., 2012).

Theoretically, previous studies conceptualized that the onset of illness or health decline as a stressor to the couple (Davila, Bradbury, Cohan, & Tochluk, 1997). In this systems perspective, the illness is the stress factor that challenges the homeostasis of the relationship, requiring role changes and adaptation (Burman & Margolin, 1992). Poor physical illness causes stress, stress increases the risk of destructive spousal interaction, resulting in decline in marital quality (Davila et al., 1997). In detail, Booth and Johnson (1994) reported potential mechanisms linking decline in health to decline in marital quality, such as declines in marital activity, financial adjustments, shifts in division of household labor, and problematic interpersonal behavior of the person with illness. Recent work confirmed these mechanisms in couple-level data, finding that decline in sexual activity and worsened mental health mediated the effect of own and partner's poor physical health on declined marital quality (Galinsky & Waite, 2013).

The marital couples' health context: Actor, partner, and actor-partner interactions

When examining the health effects on marital quality, the effects of ones' own health to their own marital quality (actor effect) and the effect of partner's health to one's marital quality (partner effect) can be differentiated (Kenny et al., 2006). Previous work with couple data illustrated the benefit of examining actor, partner effects, as well as the interaction of actor and partner's health conditions.

Regarding actor effects, previous findings are mixed on whether health problems influence one's own marital quality. For example, some studies find lowered marital quality when the person has vision impairment (Strawbridge, Wallhagen, & Schema, 2007), but other studies found no marital quality change from cancer (Langer et al, 2010) or end-life renal disease (Pruchno, Wilson-Genderson, & Cartwright, 2009). Some even reported an improved marital quality on the onset of disability (Yorgason, Booth, & Johnson, 2008).

Regarding partner effects, existing studies provide strong evidence that partner's health problems are associated with one's own decreased marital quality. For example, the decline in self-rated health of the partner was strongly damaging to the person's marital quality, especially for wives (Yorgason et al, 2008; Booth & Johnson, 1994). Similarly, negative partner effects were reported on latent factor of overall health issues (Korporaal et al, 2013), vision impairment (Strawbridge et al, 2007), cancer (Langer et al, 2010), and end-life renal disease (Pruchno et al., 2009).

Lastly, it is possible for both partners to have health issues. Marital quality may be related to one's own and partner's health as in actor and partner effect, but those effects may not necessarily capture the complex interactions between the two (Carr, Freedman, Cornman, & Schwarz, 2014). Research shows that couples often experience concordance of health to each

other (interdependence; Hoppmann & Gerstorf, 2009; Hoppmann, Gerstorf, & Hibbert, 2011), and often tackle health problems of both as a unit (i.e. dyadic goal pursuit; Berli, Bolger, Shrout, Stadler, & Scholz, 2017). Recent work found an evidence for spousal interdependence of frailty and depression in older couples (Monin et al., 2016). They found that individual's higher frailty predicted partner's higher frailty, and a similar partner effect for depression. The finding that frailty and depression are interrelated in couples supports the need to test if the spousal interaction of these health problems results in particularly undesirable social outcomes. For example, a study investigated the actor-partner interaction effect of health problems on marital satisfaction, observing that spousal health problems were detrimental to one's marital satisfaction, but only when their own health was good (Korporaal et al., 2013). Adding that frailty and depression also often co-occur, it is possible that frailty and depression may interact within the person or across the partners.

Research questions and hypotheses

The current study aims to further test the dyadic model of couple's health effects on marital outcomes in later life, and to investigate the frailty and depression as a specific couple's health context. Below are the three research questions for this study.

Research question 1: Does one's own level of frailty and depression associated with their marital quality over time? I examined the "actor effects" of baseline frailty and depression on the level and change of marital support and strain. I hypothesized that there are independent actor effects for frailty and depression and moderation of time on actor effects, such that;

(a) one's higher level of frailty is linked to their own lower level and greater decline in martial support;

(b) one's higher level of depression is linked to their own lower level and greater decline in martial support;

(c) one's higher level of frailty is linked to their own higher level and greater increase in martial strain;

(d) one's higher level of depression is linked to their own higher level and greater increase in martial strain.

Research question 2: Does one's partners' level of frailty and depression associated with one's marital quality over time? I examined the "partner effects" of baseline frailty and depression on the level and change of marital support and strain. I hypothesized that over and above the actor effects, there will be partner effects for frailty and depression and moderation of time on partner effects, such that;

(e) partner's higher level of frailty is linked to one's own lower level and greater decline in martial support;

(f) partner's higher level of depression is linked to one's own lower level and greater decline martial support;

(g) partner's higher level of frailty is linked to one's own higher level and greater increase in martial strain;

(h) Partner's higher level of depression is linked to one's own higher level and greater increase in martial strain.

Research question 3: What are the multiplicative effects of frailty and depression within- and across-individuals on marital quality over time? To investigate the effect of co-existence of frailty and depression within the couple, I examined "interaction effects" of actor and partner's frailty and depression within- and across-individuals on the level and change of

marital support and strain. I hypothesized that interaction effects of high frailty and high depression will result in negative level and decline in marital quality (i.e. low martial support and high marital strain), such that;

(i) combination of one's own higher frailty and higher depression is associated with actor's own negative level and decline in marital quality (Actor F × Actor D);

(j) combination of actor's own and partner's higher frailty is associated with actor's own negative level and decline in marital quality (Actor F × Partner F);

(k) combination of own higher frailty and partner's higher depression is associated with actor's negative level and decline in marital quality (Actor F × Partner D);

(l) combination of own higher depression and partner's higher frailty is associated with actor's own negative level and decline in marital quality (Actor D × Partner F);

(m) combination of own and partner's depression is associated with actor's own negative level and decline in marital quality (Actor D × Partner D);

(n) combination of partners' high frailty and high depression is associated with actor's own negative level and decline in marital quality (Partner F × Partner D);

Research question 4: Do these effects differ by gender? I examined whether the actor, partner, and interactive effects of frailty and depression on marital quality differ by the gender of the individual. Given the findings from the literature that shows marital experiences differ fundamentally for men and women (Kiecolt-Glaser & Newton, 2001) and stronger partner effects found from the previous dyadic studies (Korporaal et al, 2013; Yorgason et al, 2008), I expect that wives' marital quality will be more influenced by their own and partner's frailty and depression than for husbands' marital quality.

Methods

Data and Analytic sample

Data for the current study were drawn from Health and Retirement Study (HRS). HRS is a nationally representative longitudinal study of Americans over the age of 50. The study launched in 1992 and surveys were conducted every two years. HRS sampling method is based on multi-stage area probability sample design at the household level, where age-eligible respondent and their spouse or partner were both recruited (Heeringa & Connor, 1995). The HRS (Health and Retirement Study) is sponsored by the National Institute on Aging (grant number NIA U01AG009740) and is conducted by the University of Michigan.

This study used six waves of longitudinal data from heterosexual married and partnered couples from the Health and Retirement Study (HRS). In 2006, a random 50% of HRS respondents were selected for an enhanced face-to-face interview followed by a self-administered questionnaire (SAQ) with questions including the assessment of marital quality. In 2008, the remaining 50% of HRS respondents were visited for an enhanced face-to-face interview and completed the self-administered questionnaire. In coupled households, both members are asked to complete the SAQ in the same wave. Husbands and wives completed the questionnaire privately. In following waves, respondents received a SAQ every other wave (every four years) that they completed and mailed to the University of Michigan. Measures related to frailty and depression was collected every wave (ever two years). As a result of this survey structure, there are eight-year longitudinal data for those who completed SAQ in 2006 (Cohort 1: Assessed in 2006, 2010, 2014) and in 2008 (Cohort 2: Assessed in 2008, 2012, 2016). In order to increase the sample size and strengthen the statistical power, I stacked both cohort

into one sample. Thus, in the combined data, Wave 1 reflects assessments in 2006/08, Wave 2 is assessments in 2010/12, and Wave 3 is assessments in 2014/16.

From this dataset, the analytic sample is selected by following criteria: (a) HRS core participant at Wave 1(2006/08); (b) randomly selected and completed SAQ at Wave 1; (c) participant aged 50 and older at Wave 1; (d) alive and remained in the HRS study through Wave 3 (2014/16); (e) married or partnered at Wave 1; (f) married to the same spouse through Wave 3; and (g) both members of the couple meet the criteria above and have valid data on SAQ. Sample sizes for each step are described in Table 3.1. The final sample size was 4,418 individuals, including 2,209 husbands (age M = 66.5, SD = 7.8) and their wives (age M = 63.6, SD = 7.6) who were continuously married to each other with average of 36.4 years (SD = 38.8, Range = 0.3-65.2) of marriage.

Measures

Martial support and strain. The outcome variables of this study were the perceived marital support and marital strain. Prior literature emphasized the multifaceted nature of marital experience, suggesting that positive and negative dimensions of marriage are distinctive constructs and often not exclusive of each other (Liu & Waite, 2014; Umberson, Williams, Powers, Liu, & Needham, 2006). Thus, the marital quality was measured with two separate dimensions: marital support and strain. The items for support and strain was used by previous studies on social exchanges in older populations and were found to be reliable (e.g. Ingersoll-Dayton et al., 1997; Ryan et al., 2014; Walen & Lachman, 2000).

Marital support was assessed using the following three items: (a) “How much does your spouse really understand the way you feel about things?” (b) “How much can you rely on them if you have a serious problem?” and (c) “How much can you open up to them if you need to talk

about your worries?". The original response options ranged from 1 (a lot) to 4 (not at all). The responses were reverse-coded so that higher values indicated higher levels of support (1 = not at all, 2 = some, 3 = a little, 4 = a lot).

Marital strain asked as the perceived negative support they receive from their spouse with four items: (a) "How much does your spouse criticize you?", (b) "How much do they let you down when you are counting on them?", (c) "How often do they make too many demands on you?", and (d) "How much do they get on your nerves?". All responses were reverse coded so that higher values indicated higher levels of strain (1 = not at all, 2 = some, 3 = a little, 4 = a lot). Four items were averaged to construct a strain score. Response with missing values for two or more items on the support scale and three or more items on the strain scale were treated as missing (Smith et al., 2017).

Frailty. Accumulation of deficit model of frailty index (FI) was used to measure frailty at Wave 1 (Mitnitski, et al, 2001; Rockwood et al., 2004, 2007). As a multi-domain approach to frailty, FI views frailty as an accumulated burden of diseases, functional disabilities, and other health-related deficits and symptoms (Rockwood et al., 2004). FI is designed to provide flexible approach to the components of the measurement, enabling application to multiple surveys and comparisons (Searle et al., 2008). The total number of deficit selection in current study was be informed by previous studies that used HRS data (Lohman et al., 2013, 2014, 2016, 2017; Mezuk et al., 2016 2016). The selected items follows the inclusion criteria suggested by Searle and colleagues (2008). As a result, a total of 32 items were selected. The total list of items and coding schemes are presented in Table A1. A continuous variable was created as the sum of present deficits divided by the valid number of items each individual had. For example, if a

person had deficits in 8 items and had valid responses in all 32 items, their frailty index is 0.25. Note that depressive symptoms item from CES-D is not included in FI.

Depressive symptoms. Depressive symptoms were measured at Wave 1 with a summary score of a shortened 8-item version of the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). The 8-item version has been validated for high reliability and validity in older adult populations (Karim et al., 2015). The participants were asked to respond 'yes' or 'no' to six negatively worded items ("feeling depressed", "feeling that everything is an effort", "restless sleep", "feeling lonely", "feeling sad", and "could not get going") and two positively worded items ("feeling happy" and "enjoying life") based on their experiences for the preceding week. Two positively worded items were reverse coded and the responses for the eight items were added resulting in scores ranging from 0 to 8. High scores indicate more depression.

Demographic and health status covariates. Demographic and cognitive health status measured at Wave 1 were included as covariates in the analyses. *Age* was a continuous variable of respondent's age calculated based on birthdate. *Education* was based on self-reported years of education. M*arital length* was based on the self-reported years of marital duration with the current spouse. Age, education, and marital length were centered at the sample mean for the analyses. *Race/ethnicity* was assessed by asking the participants two questions, 'Do you consider yourself primarily White or Caucasian, Black or African-American, American-Indian, or Asian?' and 'Do you consider yourself Hispanic or Latino?'. The responses were recoded into a binary variable (1 = Minority, -1 = White, non-Hispanic).

Additionally, *cognitive functioning* was assessed in HRS using modified version of the Telephone Interview for Cognitive Status (TICS; Brandt et al., 1998; Crimmins et al., 2011). Items for the modified TICS include: delayed and immediate word recall of ten words (range 0-20 points), serial 7s (subtracting 7 from 100 five times; range 0-5 points), and backwards counting (counting

from 20 to 10; range 0-2 points) for a possible total summed score of 27 (Crimmins et al, 2011). Data for the cognitive functioning was drawn from the HRS dataset in which values were imputed to replace missing values, refusals, and any non-applicable responses (Fisher, Hassan, Faul, Rodgers, & Weir, 2017).

Analytic plan

To answer the research questions, the data analyses were conducted in the following order using SPSS 24 (IBM Corp., 2016). First, descriptive statistics of the key study variables were calculated for husbands and wives. Paired t- test was performed to provide comparison between the husband and wife on the continuous study variables. Additionally, Pearson's r, the Pearson product-moment correlation coefficient was estimated to describe the linear correlation between husbands' and wives' scores on the study variables. Pearson's r was used as the indicator of the level of spousal concordance for distinguishable dyads (Kenny et al., 2006).

Second, actor–partner interdependence model (APIM; Kenny et al., 2006) in multilevel modeling framework (MLM) was used to estimate own and partner's health influence on marital quality over time. The analyses were conducted using MIXED procedure in SPSS Version 24 (IBM Corp., 2016). Dataset was reconstructed into long format, suitable for longitudinal analysis in multilevel modeling in SPSS. There are two levels in multilevel modeling framework for longitudinal dyadic data. The lower level represents variability from repeated measures within individuals, and the upper level represents between-couple variability (Kenny et al., 2006). APIM accounts for the nonindependence of the observations among couples by allowing the errors to be correlated between husbands and wives in each wave. To do so, the heterogeneous compound symmetry (CSH) error structure was applied in MIXED procedure. The study variables were coded as below. In heterosexual couples, each member is distinguishable based on one's gender (1 = woman, -1 = man). Continuous variables were grand-mean centered and

binary variables were effect-coded (e.g. race; 1=Minority, -1 = White, Non-Hispanic). This way, parameter estimates specify the deviation of each level of the variable from the grand mean of the outcome (Kenny et al., 2006). Frailty and depression at baseline were treated as time-invariant predictors for time-varying marital quality outcomes. Time was modeled as linear (W1 = -1, W2 = 0, W3 = 1).

To answer the research questions on two dimensions of marital quality, separate models were conducted for marital support and marital strain. First, the Model 1 included the actor effects, partner effects, and multiplicative effects on marital quality concurrently and over time. The actor effect represented the intra-individual effects of husbands' and wives' frailty and depression on their own marital quality. The partner effect referred to the inter-individual effect of one's partner's frailty and depression on one's own marital quality. Because the actor and partner effect of both frailty and depression were included in the same model, the significance of each effect can be examined controlling for variance explained by other effects. The six interaction terms of actor and partner's frailty and depression within- and across-individuals showed the multiplicative effects of given conditions (i.e. Actor frailty × Actor depression; Actor frailty × Partner frailty; Actor frailty × Partner depression; Actor depression × Partner frailty; Actor depression × Partner depression; Partner frailty × Partner depression). Interaction term with time tested whether the strength of a frailty or depression on marital quality changes in strength over time. Demographic and cognitive functioning covariates were included in the analytic model. Second, in Model 2, gender interaction terms were introduced to actor, partner, and multiplicative effects of frailty and depression on marital quality. This model tested whether any of the concurrent effects differed for husbands and wives. Due to the possibility of spurious effects of including too many interaction terms, Model 2 tested gender moderation in cross-

sectional level (i.e. interaction term with time was excluded). Lastly, significant interaction terms from model 1 and 2 were further examined by plotting and testing the simple slopes using the predictor variables from one standard deviation below and above the mean (Aiken & West, 1991; (Preacher, Curran, & Bauer, 2006). Depending on the findings, if there are significant gender differences in actor and partner effects based on gender moderation terms, results can be presented for husbands and wives using two-intercept model by gender (Kenny et al., 2006). However, if there is no significant gender difference, it is advised to present as actor and partner effects. Effect sizes (*r*) are presented in the table and used in interpretation along with unstandardized coefficients (*b*).

Results

Descriptive analyses

Descriptive statistics and intraclass correlations of key variables for husband and wives are displayed in Table 3.2. Paired t-test results showed that husbands were older (t (2,205) = 29.96, $p < .001$) and had more years of education than wives (t (2,202) = 2.46, $p = .014$). In terms of baseline health, wives had better cognitive function (t (2,134) = -11.31, $p < .001$), but also reported higher frailty (t (2,208) = -4.24, $p < .001$) and depressive symptoms (t (2,133) = -4.85, $p < .001$) than husbands. Marital support did not differ by gender in early waves, but husbands had higher level of perceived marital support than wives at W3 (t (1,786) = 2.81, $p = .005$). Wives reported consistently higher level of marital strain than husbands in all three waves. Pearson's r provided that there were small to moderate correlations between husbands and wives in frailty ($r = .25$) and depression ($r = .13$). Marital support and strain were also in moderate correlation for husbands and wives ($r = .28 – .36$), suggesting that there is moderate degree of spousal concordance in health and marital quality indices.

Additionally, intercorrelations of the study variables are separately presented for husbands and wives in Table 3.3. At the bivariate level, frailty and depression were positively and moderately correlated for husbands (r = .40) and wives (r = .44). Frailty and depression both had small correlations with marital support and strain variables. Marital support and strain were moderately correlated (among husbands, r = -.36 – -.36; among wives, r = -.43 – -.45), which may reflect that both variables tap into distinct aspects of perceived marital quality.

Associations of individuals' frailty and depression with marital quality over time

Model 1 examining the associations of individual and partner frailty and depression with marital support (left column) and marital strain (right column) are presented in Table 3.4. The model included actor, partner, and multiplicative effects and time moderation of these effects. Regarding the outcome variables, perceived marital support (b = -0.02, p < .001, r = 0.10) and marital strain (b = -0.02, p < .001, r = 0.08) both decreased over time. Gender difference were not found for marital support. But there was a significant mean-level difference in marital strain by gender, such that wives reported higher marital strain than husbands (b = 0.03, p < .001, r = 0.10).

Addressing the first research question on actor effects, there was evidence of actor effects of frailty and depression on marital support and strain. Individuals' own higher level of frailty were associated with perceiving low levels of marital support (b = -0.30, p < .001, r = 0.06). Additionally, one's own higher level of depression was linked to experiencing low marital support (b = -0.04, p < .001, r = 0.10). Similar patterns emerged for marital strain, such that one's own higher levels of frailty (b = 0.45, p < .001, r = 0.08) and more depressive symptoms (b = 0.06, p < .001, r = 0.15) were associated with higher levels of marital strain. All time interaction with actor frailty and actor depression terms were not statistically significant,

indicating that the actor effects of frailty and depression on marital quality did not change over time.

Associations of partners' frailty and depression with marital quality over time

Addressing the second set of research question on partner effects, a depression partner effect was found for marital support. The partner effect indicated that one's partner's higher depression was associated with lower levels of marital support for the person (b = -0.04, p < .001, r = 0.09). There was no significant partner effect of frailty on marital support. Interestingly, partner effect was present for both frailty and depression for marital strain. Having a partner with higher levels of frailty was associated with higher marital strain for the person (b = 0.34, p < .001, r = 0.06) and partner's depression was also uniquely associated with one's higher marital strain (b = 0.04, p < .001, r = 0.15). Additionally, there was a moderation effect of partner depression by time. Contrary to the expectation, having a partner with depression at baseline were associated with decline in marital strain over time (b = -0.01, p = .003, r = 0.05).

Multiplicative effects of individuals' and partners' frailty and depression with marital quality over time

To answer the third set of research questions, multiplicative effects of frailty and depression within- and across-individuals were examined. When six interaction terms were included in the same model, a significant actor × partner frailty effect has emerged for both marital support (b = 2.25, p = .005, r = 0.06) and marital strain outcomes (b = -2.49, p = .006, r = 0.06). This means that the effect of partners' frailty on one's marital support (strain) was conditional on their own frailty. The plots for actor × partner frailty moderation effects are illustrated in Figure 3.1. Simple slopes analysis shows that, when the person's own frailty is high, their partner's frailty (b = 0.11, p = .316, r = 0.02) is not significantly influential to one's marital

support. However, when the person's own frailty is low, their partner's level of frailty (b = -0.37, p = .003, r = 0.05) was associated with lower marital support. Similarly, for marital strain, when the person's own frailty is high, their partner's frailty (b = 0.07, p = .569, r = 0.01) is not influential, but when the person's own frailty is low, their partner's level of frailty (b = 0.6, p < .001, r = 0.08) were linked to higher marital strain.

Additionally, some of the multiplicative effects were moderated by time. First, there were time moderation in within-person interaction of actor's frailty and depression on their marital support (b = 0.06, p = .017, r = 0.04). Plotting the three-way interaction term revealed that, except for when actor had low frailty and depression, other three interaction conditions where actor had either or both frailty and depression, they experienced slight decrease in marital support (Figure 3.2.). Second, similarly, the within-person interaction of partner's frailty and depression on the person's marital support changed over time (b = 0.07, p = .002, r = 0.05). All four interaction conditions' effect on marital support decreased over time, but the decrease in marital support was most drastic for the person when their partner had high frailty with low depression (Figure 3.3.). Third, there was a time moderation of multiplicative effect on marital strain. In the between-person interaction of partner's frailty and actor's depression (b = -0.08, p = .025, r = 0.04), surprisingly, the interaction effects on marital strain decreased over time for three conditions. Only when actor's depression is low, and their partner's frailty was high, there was an increase in the person's marital strain (Figure 3.4.). The multiplicative effect of actor depression and partner depression also changed over time, but given the low statistical significance, the effect is not investigated further (b = -0.004, p = .048, r = 0.04).

Gender differences in actor, partner, and multiplicative effects of frailty and depression with marital quality

Finally, I examined whether the actor, partner, and interactive effects of frailty and depression on marital quality differed for husbands and wives. In Model 2, gender interaction term was introduced to the set of actor, partner, and multiplicative effect terms of frailty and depression. As shown in Table 3.5., the results showed that most effects did not differ by gender. Only the partner effect of frailty on marital strain were moderated by gender (b = 0.20, p = .031, r = .004). Decomposing the partner frailty × gender interaction revealed that, partner frailty was associated with increased marital strain for wives (b = 0.56, p < .001, r = 0.09), but not influential for husbands' marital strain (b = 0.16, p = .203, r = 0.03).

Discussion

The present study explores the complex health contexts of older couple's marital quality, specifically focusing on the presence of frailty and depression within- and across-person in the marital relationship. There is relatively little research on the health effects on marital quality that focused on both persons in the marital relationship, especially looking at frailty and often accompanied depression. This study addresses this gap by using large-scale dyadic longitudinal data spanning over eight years. The results demonstrated that one's own and partner's higher frailty and higher depression all had independent associations with one's higher marital strain. For one's marital support, one's own higher frailty, higher depression, and partner's higher depression had negative effects. There was an across-person interaction effect of frailty, such that one's marital quality was affected by their partner's higher level of frailty only when their own health was good (low frailty). Most actor and partner effects were stable over time. The within-person interaction of frailty and depression were associated with decrease in one's own

and partner's marital support. Finally, while most effects did not differ by gender, but having a husband with higher frailty was associated with higher marital strain for wives.

Association of frailty and depression on marital quality at individual levels

In terms of the actor effects, I found that one's own frailty and depression were independently associated with their marital support and strain. Further, the interaction of frailty and depression within person showed that having either frailty or depression added to perceiving lower marital support. The study tested all possible terms of frailty and depression interaction within- and between-person, but the interaction of two symptoms (i.e. frailty and depression) seem to exist in within-person manner, as evidenced by these terms (Act Frailty × Actor Depression × Time and Partner Frailty × Partner Depression × Time).

Prior studies have reported mixed evidence for actor effects, some found decline in marital quality but other found no change or even improvement in marital quality (Langer et al, 2010; Pruchno et al., 2009; Strawbridge et al., 2007; Yogarson et al., 2008). The reason for significant negative actor effect may be related to the characteristics of frailty and depression. Yorgarson and colleagues (2008) found that for self-rated decline in health had declining marital quality, but onset of disability did not result in lowered marital quality. The onset of disability or the diagnosis of cancer may have a treatment plan, which provides clarity about the health condition for themselves and their partner. A qualitative study found that among patients with chronic pain, those who discuss their symptoms more with the spouse had higher marital satisfaction (Newton-John & Williams, 2006). However, frailty and depression symptoms, especially as my study adopted continuous measure rather than the diagnostic measure used in clinical setting, can be gradual and ambiguous in their symptoms. It is possible that the person

experiencing frailty and depressive symptoms has increased need for spousal support, but often the need is unmet without a diagnosis or communication regarding the symptoms.

Association of frailty and depression on marital quality at couple levels

I found that partner's higher level of frailty was associated with high marital stain for the person. Partner's higher depression were linked to both low marital support and high marital strain. The negative partner effect of depression has been found robust in the literature and my finding corresponds to prior studies (Booth & Johnson, 1994; Benazon & Coyne, 2000; Pruchno et al, 2009; Rehman, Gollan, & Mortimer, 2008). The effect sizes of the partner effects were slightly smaller than the actor effects but having both significant actor and partner effects in one model indicates that one's own and partner's frailty and depression independently adds to influence how one perceives their marital quality. Additionally, there were interactive effects of frailty between spouses. Unlike my expectation that actor and partner interaction of health problems would have an amplifying effect, the interaction of frailty was conditional. When one has higher frailty, they do not experience additional decline in marital quality from spouse's frailty, but when one is healthy, their partners' frailty is linked to low marital quality. The multiplicative effects of frailty resonate with the previous study. In a longitudinal dyadic model, it was reported that one's frailty predicted grater frailty for the partner, and similar partner effect for depression (Monin et al., 2016).

What are the mechanisms that can explain the partner effects and actor-partner interaction effects? Series of observational studies that investigated depression in marriage outlined possible mechanisms, including cognitive and interpersonal processes (Joiner & Katz, 1999). For example, scholars have found a person with depression tends to have negative bias towards their own marital quality and increase in reassurance seeking behavior (Joiner & Katz, 1999; Rehman

et al., 2008). These behaviors contribute to interpersonal tension in marriage, which in turn, can further linked to increase in depression (i.e. bidirectional links; Kouros & Cummings, 2011; Whisman & Uebelacker, 2009). The cognitive and interpersonal model can explain the partner effect of depression found for marital quality. However, it may not be fully applicable to explain between-person effects of frailty. The phenotypic model of frailty lends insights to the how frailty is experienced by the person and their partner. Frailty is assessed with indicators including exhaustion, low physical activity, and weakness (Fried et al., 2004). Thus, the mechanisms linking frailty to actor and partner's lowered marital quality could be via physical and behavioral pathways, rather than cognitive or emotional pathways. First, frailty symptoms may be related to decline in sexual activity, resulting in decline in marital quality for both partners. It is documented that poor physical health (Karraker, DeLamater, & Schwartz, 2011; Galinsky & Waite, 2013) or multiple chronic conditions (Shen, 2019) can disrupt the ability or desire for sexual activity. It is highly likely that a person with higher frailty also experience sexual dysfunction, which has consequences to both partners. Indeed, it is reported that the level of sexual engagement played a mediating role for both one's own and partner's poor physical health to influence marital quality (Galinsky & Waite, 2013). Second, symptoms of frailty can change both partner's marital roles and expectations. Partner may need to accommodate the person's exhaustion, low energy level, and fatigue in daily manner, which can feel like spousal caregiving of illness. The couple may also need to adjust their social and recreational activities to accommodate the frailty. Especially with current study's finding that marital quality suffers only when healthy person has the partner with frailty, the increase demand for care provision may result in partner's lowered marital quality. Study using equity theory reports that while people benefit from both giving and receiving support with close relationships, the imbalance in support

received and provided resulted in poor psychological well-being for marital relationship (Wang & Gruenewald, 2017). With this theory, the healthy person could perceive that they are under-benefitting from the spousal relationship because they are providing more care to their spouse with frailty.

Finally, the spousal caregiving literature documents robust gender differences in marital functioning in context of health problems (Iveniuk, Waite, Laumann, McClintock, & Tiedt, 2014; Korporaal et al., 2013; Thomeer, Reczek, & Umberson, 2015; Thomeer, 2016). My finding agrees to the literature by finding a gendered partner effect of depression on marital strain, such that having a husband with higher frailty was associated with higher marital strain only for wives. The stronger partner effects experienced by wives can be explained that women are more likely to be emotionally distressed by caregiving (Pinquart & Sörensen, 2006) and engage more in emotion work for their spouse, regardless of their own health (Thomeer et al., 2015). It is explained that traditional gendered marital norms shapes how husband and wives respond and experience spousal care.

Limitations

A first limitation of this study is the selectivity of the sample. The sample was restricted couples in HRS where both partners completed the leave-behind questionnaire and were married to the each other for the duration of the study (2006-2014; 2008-2016). The findings are based on older adults and their partners where both persons did not drop out of the study due to health issues, or marital union was stable. The study's main target was to highlight the dynamic health context of married couples over eight years, so it was necessary to limit the sample to those who had the same spouse. However, given that marriage itself is known to be protective of frailty development (Etman et al., 2012; Woo et al., 2005), the sample may have lower frailty than the

overall population. Furthermore, the sample age ranged from 50-95 and the length of marriage ranging from 0.3 to 65.2 years at baseline, the sample's health and marital experience would be also diverse. The association of frailty and marital relationship could potentially differ for young-old and old-old, by marital length, or by marital history (i.e. long-term first marriage vs. coupling at later life). Second, the study used accumulated deficit model of frailty with creating continuous frailty index (FI) based on previous literature. The advantage of this model is that it can model more detailed levels of frailty, rather than phenotypic model of frailty with 3 categories (0-5 point scale; PF). However, majority of the clinical and population level studies on frailty only use PF, rendering it difficult to know if certain effects can be found robustly across various conceptualization of frailty. Third, the current study adopted the framework of health influencing marital quality as an outcome. However, extensive literature exists on the role of social relationship shaping our health. For example, results from Chapter 2 show that experiencing higher marital strain is associated with belonging to steep increase frailty trajectory. Further, most of the actor, partner, and interaction effects were stable over time, which may imply that bidirectional dynamic is in place. Future work can test the co-varying nature of frailty and social relationship by utilizing multiple waves for both measures. Lastly, current work theorized that existing level of frailty and depression jointly influence couple's marital quality, however, alternative model cannot be ruled out. Given that frailty and depression are also dynamic symptoms, and evidences suggest that physical health can influence marital quality via psychological distress (i.e. depressive symptoms; Galinsky & Waite, 2013). It is possible that there is sequential order between frailty and depression to influence social outcomes. The mechanisms of frailty and depression can be explored further in path analysis.

Conclusion

This study is the first to consider the dyadic model of health effects on marital outcomes, focusing on the symptoms of frailty and depression with large-scale population level data spanning eight years. It demonstrates that frailty and depression were independently linked to marital quality for older couples' marital support and marital strain. Frailty of either partner had consequences for marital strain for both partners and one's frailty was especially harmful to the healthier partner's marital quality, highlighting the importance of examining the dyadic context of health effects. Given that chronic health conditions such as frailty can take its toll on care burden of family and health care system, future research is needed to further investigate the various social costs of frailty. Further, as with increased longevity, more aging couples are confronted with dealing with multimorbidity of both partners. The study provides an evidence of spousal interaction of chronic health conditions, pointing future researchers to further explore potential mechanisms to explain and alleviate those health burdens in later life couples.

CHAPTER 4. CONCLUSION

This dissertation is focused on frailty syndrome, a growing public health concern for many developed countries with increasing longevity and multimorbidity among older population. The study utilized the large-scale national sample of older adults in the US (Health and Retirement Study) to address the increased scientific interest to understand and identify relevant factors related to prevention of frailty. The study filled an important gap in the literature by examining the social relationship context of the origins and consequences of frailty development.

First, the study found large heterogeneity in frailty progression trajectory in older US adults. This finding points to the fact that health symptoms in later life vary greatly by their onset and progression trend and contextual factors explaining such heterogeneity. The investigation of health outcomes should strive to adopt longitudinal data structure to study both concurrent levels as well as a diverse growth trajectory to understand the nature of the symptoms. As the literature of frailty is still evolving with issues regarding measurement and conceptualization, future work should test the results from the current study with multiple measures of frailty and diverse population sample.

Second, the first study highlighted the significance of social relationship as a contextual predictor for different trajectory of frailty progression. The size, frequency of contact, positive, and negative quality were linked differentially to frailty by relationship type. The frequent contact with friends had the positive effect on frailty, whereas for kin, the quality of the relationship (i.e. strain with spouse, children, and family) were more influential to frailty progression. The novel findings point to the importance of comprehensive examination of social experiences to identify “active ingredients” and modifiable risk factors for intervention and prevention programs.

Third, the second study addressed the reversed direction of how health conditions of an individual and their partner can shape their marital experiences. The level of frailty and depression were linked to perceived marital quality of both members of the older couple, especially in damaging manner when healthier spouse is faced with partner's health problems. With higher rate of multimorbity in older adulthood and the fact that majority of older adults maintain and seek romantic union, the complex and intertwined health context of couples require research attention from interdisciplinary perspectives. Further, the finding supports the need to assess patients' social context in clinical settings to understand the impact of the implemented intervention program.

Fourth, the dissertation provided examples of theoretically informed research design for studying a dynamic health condition. Grounded in the convoy model of social relations and the life course perspective, the theoretical framework provided the platform to generate important research questions that were lacking in the frailty studies in clinical and epidemiological literature. Moreover, the findings point toward the great potentials of bioecological theory (Bronfenbrenner & Morris, 2006) in understanding frailty and multimorbidity in later life. Future research can adopt the research framework of the Process-Person-Context-Time (PPCT) model to systematically examine the contextual factors that promote or protect the onset and rapid progression of frailty (Bronfenbrenner & Ceci, 1994; Bronfenbrenner & Morris, 2006; Rosa & Tudge, 2013; Schiamberg et al., 2011; Tudge, Mokrova, Hatfield, & Karnik, 2009). For example, the study can specify the individual with frailty as a central *person* who are influenced by and influence the environment with their intrapersonal factors such as age, gender, race, and personality traits. The PPCT model emphasize the ongoing, complex, and reciprocal interactions that the person has with other person, objects, and symbols (Tudge et al., 2009). The spouse, for

example, is a proximal figure that not only has their own personal characteristics to influence the focal person, but independently the interaction between the focal person and their spouse (i.e. marital conflict, spousal caregiving) are important *process* to shape the person's health (Schiamberg et al., 2011; Tudge et al., 2009). The person and the process exist within the multidimensional *context*. Recognizing the different types of context, the study can identify micro-, meso-, macro-, and exosystems that surrounds the focal processes. For example, to study the factors explaining the higher prevalence rate of frailty among nursing home residents (Kojima, 2015), the study can address the nursing home personnel, care and training procedures, administrative system, long-term care policy, and social and economic climate regarding elder care as a possible contexts of frailty progression (Schiamberg et al., 2011). Finally, the study should incorporate *time* by collecting data longitudinally and acknowledging the effect of historical time (Tudge et al., 2009).

In conclusion, frailty is a comprehensive syndrome representing a transitional phase between successful aging and disability (Cesari et al. 2014; Clegg et al., 2013). In preventing and managing age-related conditions, the researchers suggest that frailty may represent a novel and person-tailored way to conceptualize "biological age", replacing the concept of chronological age (Cesari et al. 2016). My dissertation contributes to the literature by identifying social relationship factors particularly relevant to the high level and rapid progression of frailty and how frailty and often accompanied depression can influence the marital wellbeing of older couples. The findings can be useful to practitioners and policy makers in understanding the intricate link between social context and frailty and in identifying modifiable factors for frailty prevention.

APPENDICES

APPENDIX A: Chapter 2 Tables

Table 2.1. Differences between Selected Analytic Sample and excluded sample in HRS.

	Selected		Excluded		$\chi^2(df)$
Characteristics	N	%	N	%	
Gender (Female)	5,118	57.6%	3,570	62.7%	38.56***(1)
Married	5,406	61.8%	4,293	76.9%	356.38***(1)
Race (White, non-Hispanic)	7,064	79.5%	4,145	72.8%	85.12***(1)
	M	SD	M	SD	*t*-test (*df*)
Age (years)	74.03	6.96	56.95	5.18	-169.39*** (14256.22)
Education (years)	12.26	3.16	13.13	2.91	16.83*** (14560)
Depressive symptoms	1.38	1.86	1.55	2.10	4.81*** (10636.31)
Cognitive functioning	14.56	4.36	16.86	4.06	31.83*** (12252.41)
N	8,892		5,691		

Note. The comparison is made between selected analytic sample and excluded sample in HRS 2006 wave data (N = 14,583). * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2.2. Descriptive Statistics of the study variables (N = 8,892)

	N	%	M	SD	Min	Max
Frailty Index						
Frailty T1 (2006)	8,751		0.24	0.12	0	0.82
Frailty T2 (2008)	8,495		0.24	0.13	0	0.92
Frailty T3 (2010)	7,422		0.27	0.14	0	0.87
Frailty T4 (2012)	6,642		0.26	0.14	0	0.83
Frailty T5 (2014)	5,711		0.29	0.14	0	0.81
Frailty T6 (2016)	4,628		0.30	0.15	0	0.89
Demographic and health characteristics (2006)						
Gender (Female)	5,118	57.6%				
Age (years)	8,892		74.03	6.96	65	104
Education (years)	8,891		12.26	3.16	0	17
Race/ethnicity						
White, non-Hispanic	7,064	79.4%				
Black, non-Hispanic	1,070	12.3%				
Hispanic	614	6.9%				
Other, non-Hispanic	143	1.6%				
Depressive symptoms	8,572		1.38	1.86	0	8
Cognitive functioning	8,575		14.56	4.36	0	27
Social network and relationship quality (2006/08)						
Lives with spouse/partner	5,238	58.9%				
Marital status						
Married	5,179	58.2%				
Separated/Divorced	900	10.1%				
Widowed	2,625	29.5%				
Never married	188	2.1%				
Number of close children	8,436		2.76	2.51	0	20
Number of close family members	7,853		3.85	3.98	0	20
Number of close friends	8,040		4.43	4.15	0	20
Frequency of contact with children	8,078		3.97	1.02	1	6
Frequency of contact with family members	8,095		3.37	1.12	1	6
Frequency of contact with friends	8,147		3.72	1.08	1	6
Spouse support	5,535		3.48	0.65	1	4
Children support	8,132		3.35	0.68	1	4
Family support	8,173		2.93	0.87	1	4
Friends support	8,135		3.03	0.75	1	4
Spouse strain	5,507		1.95	0.67	1	4
Children strain	8,108		1.62	0.60	1	4
Family strain	8,141		1.49	0.57	1	4
Friends strain	8,109		1.37	0.47	1	4
Loneliness (2006/08)	8,565		1.46	0.53	1	3

Table 2.3. Intercorrelations of the study variables.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.	24.	25.	26.	27.	28.
1. Frailty W1	1																											
2. Frailty W2	.83**	1																										
3. Frailty W3	.76**	.81**	1																									
4. Frailty W4	.71**	.77**	.83**	1																								
5. Frailty W5	.68**	.71**	.79**	.84**	1																							
6. Frailty W6	.65**	.69**	.72**	.77**	.82**	1																						
7. Gender (Female)	.11**	.10**	.10**	.09**	.10**	.12**	1																					
8. Age (years)	.15**	.16**	.20**	.21**	.21**	.19**	.04**	1																				
9. Education (years)	-.19**	-.19**	-.19**	-.19**	-.20**	-.20**	-.05**	-.06**	1																			
10. Race/ethnicity (White)	-.06**	-.06**	-.06**	-.08**	-.07**	-.08**	-.04**	.09**	.32**	1																		
11. Depressive symptoms W1	.49**	.43**	.40**	.37**	.36**	.37**	.13**	.07**	-.23**	-.11**	1																	
12. Cognitive functioning W1	-.23**	-.24**	-.24**	-.25**	-.25**	-.23**	.05**	-.28**	.42**	.28**	-.22**	1																
13. Lives with spouse/partner	-.15**	-.14**	-.15**	-.15**	-.13**	-.13**	-.33**	-.23**	.12**	.11**	-.21**	.13**	1															
14. # of close children	0.01	.03*	0.01	.03*	0.01	0.02	0.01	-0.01	-.11**	-.09**	-0.01	-.05**	.05**	1														
15. # of close family	0.01	0.02	0.02	0.02	0.00	-0.02	.06**	0.00	-.10**	-.10**	-0.01	-.04**	-0.02	.32**	1													
16. # of close friends	-.08**	-.07**	-.07**	-.06**	-.08**	-.05**	-0.02	0.00	.07**	.06**	-.11**	.06**	.07**	.16**	.34**	1												
17. Contact with children	-.08**	-.07**	-.06**	-.05**	-.04**	-.04**	.18**	0.00	.09**	.04**	-.07**	.12**	-.02*	.09**	.12**	.11**	1											
18. Contact with family	-.02*	-.03**	-0.01	-0.01	-0.02	-0.01	.15**	.03**	-0.02	-.06**	0.00	0.00	-.06**	.07**	.19**	.13**	.47**	1										
19. Contact with friends	-.10**	-.10**	-.09**	-.09**	-.09**	-.08**	.17**	-.05**	.18**	.09**	-.08**	.16**	-.09**	-.03*	.07**	.22**	.37**	.34**	1									
20. Spouse support	-.16**	-.16**	-.16**	-.15**	-.15**	-.16**	-.19**	0.00	.13**	.14**	-.26**	.09**	.23**	0.01	.03*	.11**	.08**	.04**	.07**	1								
21. Children support	-.09**	-.09**	-.09**	-.09**	-.08**	-.08**	.13**	.13**	-0.01	-0.01	-.13**	0.00	-.07**	.13**	.18**	.14**	.38**	.22**	.09**	.22**	1							
22. Family support	-.02*	-.04**	-0.02	-0.02	-0.03	-0.01	.11**	.06**	-.10**	-.14**	-.03**	-.07**	-.10**	.09**	.21**	.14**	.18**	.49**	.10**	.12**	.42**							
23. Friends support	-0.02	-.03*	-.03*	-.04**	-0.03	-0.02	.21**	-.03**	.03**	-0.01	-0.01	.04**	-.12**	0.00	.07**	.20**	.13**	.15**	.37**	.10**	.26**	.31**						
24. Spouse strain	.12**	.12**	.12**	.11**	.12**	.12**	.08**	-.05**	-.06**	-.11**	.18**	-.04**	.03*	0.00	-.03*	-.08**	-0.02	-0.02	-.03*	-.46**	-.16**	-.09**	-.06**					
25. Children strain	.15**	.15**	.14**	.14**	.14**	.14**	0.01	-.12**	-.05**	-.10**	.19**	-.05**	-0.01	-.06**	-.06**	-.07**	-.09**	-.05**	-.03*	-.16**	-.39**	-.18**	-.06**	.40**				
26. Family strain	.13**	.15**	.13**	.13**	.13**	.12**	.03**	-.10**	-.07**	-.12**	.16**	-.08**	-0.02	-.03**	-.03*	-.05**	-.05**	.02*	-0.02	-.15**	-.23**	-.16**	-.03*	.31**	.56**			
27. Friends strain	.07**	.09**	.07**	.08**	.09**	.10**	-.04**	-.08**	-.06**	-.12**	.10**	-.08**	-.03**	-.04**	-0.02	-0.01	-.04**	0.01	.04**	-.13**	-.15**	-.06**	-.08**	.27**	.41**	.47**		
28. Loneliness	.28**	.28**	.27**	.25**	.24**	.24**	.07**	.05**	-.13**	-.07**	.41**	-.16**	-.24**	-.06**	-.08**	-.18**	-.12**	-.08**	-.16**	-.44**	-.23**	-.14**	-.14**	.38**	.29**	.26**	.23**	1

Note. * p < .05. ** p < .01.

Table 2.4. Model fit indices for unconditional latent growth curve model (non-mixture) of frailty progression

Fit index	Intercept	Linear	Quadratic	Latent basis
Number of parameters	8	11	15	15
χ^2	8049.665	1253.621	724.688	749.363
df	19	16	12	12
$\chi 2/df$	423.67	78.35	60.39	62.45
p value	0.000	0.000	0.000	0.000
AIC	-76208.911	-85034.660	-85771.346	-85847.601
CFI	0.605	0.939	0.965	0.964
TLI	0.688	0.943	0.956	0.955
RMSEA	0.218	0.093	0.082	0.083
(RMSEA 90% C.I.)	(0.214 - 0.222)	(0.089 - 0.098)	(0.077 - 0.087)	(0.078 - 0.088)

Note. AIC = Akaike information criterion; CFI = Comparative Fit Index, TLI = Tucker-Lewis index, RMSEA = Root Mean Square Error of Approximation.

Table 2.5. Model fit indices for growth mixture modeling of frailty progression

Fit index	1 class	2 classes	3 classes	4 classes	5 classes	6 classes
Unconditional GMM						
Number of parameters	10	13	16	19	22	25
Log-likelihood	42805.509	43285.213	44113.78	44322.914	44436.429	44113.78
BIC	-85520.09	-86452.217	-88082.07	-88473.06	-88672.814	-88000.237
ABIC	-85520.09	-86493.529	-88132.92	-88533.44	-88742.727	-88079.683
Entropy	N/A	0.788	0.797	0.779	0.779	0.875
LMR *p* Value	N/A	0.000	0.000	0.011	0.070	0.240
Proportions of classes	N/A	87-13	82-12-6	78-11-8-2	77-9-8-5-2	82-12-6-0-0-0
Conditional GMM						
Number of parameters	15	18	21	24	27	30
Log-likelihood	42938.801	43815.791	44292.447	44480.716	44595.416	44685.58
BIC	-85741.21	-87467.91	-88393.94	-88743.2	-88945.324	-89098.374
ABIC	-85788.88	-87525.111	-88460.68	-88819.47	-89031.125	-89193.708
Entropy	N/A	0.836	0.798	0.783	0.785	0.77
LMR *p* Value	N/A	0	0	0.003	0.089	0.0001
Proportions of classes	N/A	93-7	81-12-7	78-11-8-2	77-10-8-3-1	70-15-6-4-4-1

Note. Unconditional growth mixture model is based on latent basis growth curve model of frailty; Conditional model added gender (woman, man), race (white, non-white), age (years, standardized), and education (years, standardized) as covariates in growth mixture modeling; BIC = Bayesian information criterion; ABIC = Sample size adjusted BIC; LMR = Lo-Mendel-Rubin test.

Table 2.6. Parameter estimates for the chosen three class model of frailty progression

	Class 1 Average frailty	Class 2 High frailty	Class 3 Steep Increase frailty
Sample size (N)	7,161	1,062	667
Proportion (%)	80.6%	11.9%	7.5%
Average probability of class membership	0.929	0.816	0.823
Growth factor means			
Intercept mean (S.E.)	0.20 (0.002)	0.45 (0.01)	0.23 (0.01)
Slope mean (S.E.)	0.09 (0.003)	0.07 (0.01)	0.49 (0.02)
Slope loadings (S.E.)			
Time 1		0.00 (0.00)	
Time 2		0.04 (0.01)	
Time 3		0.34 (0.01)	
Time 4		0.44 (0.01)	
Time 5		0.74 (0.02)	
Time 6		1.00 (0.00)	
Growth factor variances			
Intercept variance (S.E.)		0.007 (0.00)	
Slope variance (S.E.)		0.004 (0.00)	
Intercept-slope covariance (S.E.)		0.001 (0.00)	
Residual variances (S.E.)		0.003 (0.00)	
Intercept factor on covariates		b (S.E.)	
Gender (Woman =1, Man = 0)		0.006 (0.001)	
Race (Minority = 1, White = 0)		0.000 (0.002)	
Education		-0.012 (0.001)	
Age		0.015 (0.002)	

Table 2.6. (cont'd)

Slope factor on covariates	
Gender (Woman =1, Man = 0)	-0.001 (0.001)
Race (Minority = 1, White = 0)	-0.003 (0.002)
Education	-0.004 (0.002)
Age	0.018 (0.002)

Note. Conditional growth mixture model is based on latent basis growth curve model of frailty with covariates (gender, race, age, and education)

Table 2.7. Demographic, health, and social network, relationship quality, and loneliness of the three frailty progression groups

	Average frailty		**High frailty**		**Steep increase frailty**		Group comparisons		
	Mean (*SD*) or %		Mean (*SD*) or %		Mean (*SD*) or %		F(df) or χ^2(df)	*p*	Post hoc comparisons[b]
Demographic and health (T1)									
Gender (% Female)	56.0%		67.5%		58.6%		50.66 (2)	p < .001	
Age (years)	73.4	(6.5)	74.4	(7.2)	80.2	(8.1)	309.24 (2, 8887)	p < .001	g1 < g2 < g3
Education (years)	12.5	(3.0)	11.1	(3.4)	11.4	(3.5)	123.04 (2, 8887)	p < .001	g1> g2 = g3
Race/ethnicity									
White, non-Hispanic	80.6%		73.0%		77.2%		43.67 (6)	p < .001	
Black, non-Hispanic	11.2%		16.7%		13.5%				
Hispanic	6.5%		8.9%		8.4%				
Other, non-Hispanic	1.7%		1.5%		0.9%				
Depressive symptoms	1.1	(1.6)	3.3	(2.3)	1.5	(1.8)	708.25 (2, 8569)	p < .001	g2 > g3 > g1
Cognitive functioning	15.0	(4.2)	12.8	(4.5)	12.1	(4.7)	237.12 (2, 8572)	p < .001	g1> g2> g3
Social network (T1)									
Lives with spouse/partner	63.7%		48.7%		48.9%		124.889 (2)	p < .001	
Marital status									
Married	61.0%		47.6%		45.7%		158.33 (6)	p < .001	
Separated/Divorced	10.0%		12.8%		7.6%				
Widowed	26.9%		37.6%		45.3%				
Never married	2.2%		2.1%		1.3%				
Overall network size[a]	10.2	(7.6)	9.8	(8.3)	9.7	(8.0)	2.24 (2, 8805)	0.107	

Table 2.7. (cont'd)

	Average frailty		**High frailty**		**Steep increase frailty**		Group comparisons		
	Mean (*SD*) or %		Mean (*SD*) or %		Mean (*SD*) or %		F(df) or $\chi 2$(df)	*p*	Post hoc comparisons[b]
Number of close children	2.8	(2.4)	2.9	(2.9)	2.7	(2.5)	1.63 (2, 8431)	0.196	
Number of close family	3.8	(3.9)	4.1	(4.4)	3.9	(4.4)	2.09 (2, 7848)	0.124	
Number of close friends	4.5	(4.2)	3.8	(4.2)	4.3	(4.0)	11.85 (2, 8035)	p < .001	g1>g2, g2=g3, g1=g3
Contact with children	4.0	(1.0)	3.8	(1.1)	3.9	(1.1)	21.35 (2, 8073)	p < .001	g1 = g3 > g2
Contact with family	3.4	(1.1)	3.3	(1.2)	3.4	(1.2)	5.07 (2, 8091)	0.006	g1>g2, g1=g3, g2=g3
Contact with friends	3.8	(1.1)	3.5	(1.1)	3.5	(1.1)	43.55 (2, 8143)	p < .001	g1 > g2 = g3
Social relationship quality (T1)									
Spouse support	3.5	(0.6)	3.2	(0.8)	3.4	(0.7)	53.56 (2, 5531)	p < .001	g1 > g3 > g2
Children support	3.4	(0.7)	3.2	(0.8)	3.4	(0.7)	32.45 (2, 8127)	p < .001	g1 = g3 > g2
Family support	2.9	(0.9)	2.9	(0.9)	3.0	(0.9)	4.51 (2, 8169)	0.011	g1 = g3 > g2
Friends support	3.0	(0.7)	3.0	(0.8)	3.0	(0.8)	0.42 (2, 8132)	0.656	
Spouse strain	1.9	(0.7)	2.1	(0.7)	2.0	(0.7)	23.13 (2, 5503)	p < .001	g2 = g3 > g1
Children strain	1.6	(0.6)	1.8	(0.7)	1.6	(0.6)	78.26 (2, 8103)	p < .001	g2 > g1 = g3
Family strain	1.5	(0.6)	1.7	(0.7)	1.5	(0.6)	60.97 (2, 8137)	p < .001	g2 > g1 = g3
Friends strain	1.4	(0.5)	1.5	(0.5)	1.4	(0.5)	13.28 (2, 8106)	p < .001	g2 > g1 = g3
Loneliness (T1)	1.4	(0.5)	1.8	(0.6)	1.6	(0.6)	235.27 (2, 8562)	p < .001	g2> g3> g1

Note. [a]Overall network size is computed as sum of the number of close children, family, and friends; [b]Post hoc comparisons are based on Tamhane's T2 (when the assumption of equal variances was not met) and Tukey HSD (when the assumption of homogeneity of variances were met based on Levene statistic).

Table 2.8. Multinomial logistic regression results of social network and loneliness predictors of frailty group

	Model 1: Social network									Model 2: Adding loneliness								
	High frailty (ref= Ave frailty)			Steep increase frailty (ref= Ave frailty)			Steep increase frailty (ref= High frailty)			High frailty (ref= Ave frailty)			Steep increase frailty (ref= Ave frailty)			Steep increase frailty (ref= High frailty)		
	b	*(s.e.)*	*p*	*b*	*(s.e.)*	*p*	*b*	*(s.e.)*	*p*	*b*	*(s.e.)*	*p*	*b*	*(s.e.)*	*p*	*b*	*(s.e.)*	*p*
Demographic (T1)																		
Female	0.38	(0.08)	0.000	0.05	(0.10)	0.616	-0.34	(0.12)	0.005	0.39	(0.08)	0.000	0.05	(0.10)	0.587	-0.33	(0.12)	0.005
Age	0.00	(0.01)	0.796	0.11	(0.01)	0.000	0.11	(0.01)	0.000	0.00	(0.01)	0.609	0.11	(0.01)	0.000	0.11	(0.01)	0.000
Education	-0.03	(0.01)	0.038	-0.04	(0.02)	0.019	-0.01	(0.02)	0.738	-0.03	(0.01)	0.036	-0.04	(0.02)	0.019	-0.01	(0.02)	0.746
Race (ref=White)																		
Black	-0.03	(0.11)	0.798	0.05	(0.14)	0.709	0.08	(0.17)	0.628	-0.02	(0.11)	0.892	0.05	(0.14)	0.705	0.07	(0.17)	0.683
Hispanic	-0.45	(0.16)	0.005	0.02	(0.18)	0.921	0.46	(0.22)	0.033	-0.42	(0.16)	0.007	0.02	(0.18)	0.911	0.44	(0.22)	0.043
Other	-0.36	(0.31)	0.240	-0.69	(0.40)	0.084	-0.34	(0.48)	0.487	-0.35	(0.31)	0.251	-0.68	(0.40)	0.089	-0.33	(0.48)	0.495
Depressive symptoms	0.44	(0.02)	0.000	0.04	(0.03)	0.170	-0.40	(0.03)	0.000	0.40	(0.02)	0.000	0.01	(0.03)	0.756	-0.39	(0.03)	0.000
Cognitive functioning	-0.07	(0.01)	0.000	-0.09	(0.01)	0.000	-0.02	(0.02)	0.239	-0.07	(0.01)	0.000	-0.09	(0.01)	0.000	-0.02	(0.02)	0.255
Social network (T1)																		
Lives with spouse/partner	-0.07	(0.08)	0.423	-0.06	(0.10)	0.552	0.01	(0.12)	0.956	0.02	(0.09)	0.842	0.00	(0.10)	0.980	-0.02	(0.12)	0.876
Number of close children	0.01	(0.02)	0.540	-0.02	(0.02)	0.292	-0.03	(0.02)	0.190	0.01	(0.02)	0.450	-0.02	(0.02)	0.330	-0.03	(0.02)	0.177
Number of close family	0.02	(0.01)	0.043	0.00	(0.01)	0.814	-0.02	(0.02)	0.231	0.02	(0.01)	0.032	0.00	(0.01)	0.747	-0.02	(0.02)	0.224
Number of close friends	0.00	(0.01)	0.879	0.01	(0.01)	0.366	0.01	(0.02)	0.418	0.00	(0.01)	0.761	0.01	(0.01)	0.235	0.01	(0.02)	0.493
Contact with children	-0.07	(0.05)	0.111	0.01	(0.05)	0.913	0.08	(0.07)	0.228	-0.06	(0.05)	0.170	0.01	(0.05)	0.868	0.07	(0.07)	0.273
Contact with family	-0.06	(0.04)	0.173	0.00	(0.05)	0.960	0.05	(0.06)	0.365	-0.05	(0.04)	0.200	0.00	(0.05)	0.991	0.05	(0.06)	0.366
Contact with friends	-0.18	(0.04)	0.000	-0.14	(0.05)	0.005	0.05	(0.06)	0.428	-0.16	(0.04)	0.000	-0.12	(0.05)	0.012	0.04	(0.06)	0.523
Loneliness (T1)										0.40	(0.07)	0.000	0.30	(0.09)	0.001	-0.10	(0.11)	0.372

Table 2.9. Multinomial logistic regression results of social relationship quality and loneliness predictors of frailty group

	Model 1: Social network + Relationship quality									Model 2: Adding loneliness								
	High frailty (ref= Ave frailty)			Steep increase frailty (ref= Ave frailty)			Steep increase frailty (ref= High frailty)			High frailty (ref= Ave frailty)			Steep increase frailty (ref= Ave frailty)			Steep increase frailty (ref= High frailty)		
	b	*(s.e.)*	*p*	*b*	*(s.e.)*	*p*	*b*	*(s.e.)*	*p*	*b*	*(s.e.)*	*p*	*b*	*(s.e.)*	*p*	*b*	*(s.e.)*	*p*
Demographic (T1)																		
Female	0.35	(0.09)	0.000	-0.01	(0.10)	0.890	-0.36	(0.13)	0.004	0.36	(0.09)	0.000	0.01	(0.10)	0.937	-0.47	(0.13)	0.000
Age	0.01	(0.01)	0.164	0.11	(0.01)	0.000	0.10	(0.01)	0.000	0.01	(0.01)	0.184	0.11	(0.01)	0.000	0.10	(0.01)	0.000
Education	-0.03	(0.01)	0.028	-0.04	(0.02)	0.018	-0.01	(0.02)	0.815	-0.03	(0.01)	0.031	-0.04	(0.02)	0.017	0.00	(0.02)	0.898
Black (ref=White)	-0.07	(0.11)	0.530	0.01	(0.14)	0.947	0.08	(0.17)	0.633	-0.05	(0.11)	0.663	0.03	(0.14)	0.842	0.04	(0.18)	0.833
Hispanic (ref=White)	-0.43	(0.16)	0.007	-0.02	(0.18)	0.915	0.41	(0.22)	0.063	-0.40	(0.16)	0.012	0.00	(0.18)	0.997	0.39	(0.24)	0.110
Other (ref=White)	-0.42	(0.30)	0.162	-0.78	(0.41)	0.057	-0.35	(0.48)	0.464	-0.39	(0.30)	0.203	-0.74	(0.41)	0.069	-0.62	(0.54)	0.256
Depressive symptoms	0.41	(0.02)	0.000	0.01	(0.03)	0.646	-0.40	(0.03)	0.000	0.40	(0.02)	0.000	0.00	(0.03)	0.964	-0.46	(0.04)	0.000
Cognitive functioning	-0.07	(0.01)	0.000	-0.09	(0.01)	0.000	-0.02	(0.02)	0.218	-0.07	(0.01)	0.000	-0.09	(0.01)	0.000	-0.02	(0.02)	0.132
Social network (T1)																		
Lives with spouse/partner	0.05	(0.11)	0.631	-0.08	(0.12)	0.506	-0.13	(0.15)	0.380	0.11	(0.11)	0.329	-0.07	(0.12)	0.576	-0.19	(0.17)	0.273
Network total size	0.01	(0.01)	0.005	0.00	(0.01)	0.877	-0.01	(0.01)	0.079	0.02	(0.01)	0.001	0.00	(0.01)	0.772	-0.01	(0.01)	0.090
Contact with children	-0.02	(0.05)	0.651	0.00	(0.06)	0.994	0.02	(0.07)	0.743	-0.02	(0.05)	0.661	0.00	(0.06)	0.967	-0.01	(0.07)	0.903
Contact with family	-0.04	(0.05)	0.386	0.05	(0.06)	0.366	0.09	(0.07)	0.177	-0.04	(0.05)	0.366	0.05	(0.06)	0.396	0.11	(0.07)	0.136
Contact with friends	-0.23	(0.05)	0.000	-0.17	(0.05)	0.001	0.06	(0.07)	0.403	-0.21	(0.05)	0.000	-0.16	(0.05)	0.003	0.08	(0.07)	0.300
Social relationship quality (T1)																		
Spouse support	-0.15	(0.09)	0.076	-0.09	(0.10)	0.369	0.06	(0.12)	0.608	-0.10	(0.09)	0.270	-0.02	(0.10)	0.814	0.02	(0.14)	0.871
Children support	-0.09	(0.08)	0.220	0.04	(0.09)	0.654	0.13	(0.11)	0.224	-0.08	(0.08)	0.301	0.04	(0.09)	0.655	0.13	(0.11)	0.244
Family support	-0.01	(0.06)	0.882	-0.12	(0.07)	0.090	-0.11	(0.09)	0.197	0.00	(0.06)	0.977	-0.12	(0.07)	0.094	-0.11	(0.09)	0.247
Friends support	0.10	(0.06)	0.107	0.15	(0.08)	0.047	0.05	(0.09)	0.595	0.11	(0.06)	0.083	0.15	(0.08)	0.039	0.03	(0.10)	0.732
Spouse strain	-0.14	(0.09)	0.129	0.22	(0.10)	0.034	0.36	(0.13)	0.006	-0.18	(0.10)	0.063	0.19	(0.10)	0.073	0.37	(0.14)	0.010
Children strain	0.29	(0.09)	0.001	0.06	(0.11)	0.580	-0.23	(0.13)	0.070	0.26	(0.09)	0.002	0.04	(0.11)	0.695	-0.21	(0.14)	0.129
Family strain	0.22	(0.08)	0.007	-0.07	(0.11)	0.542	-0.28	(0.13)	0.026	0.20	(0.08)	0.015	-0.08	(0.11)	0.500	-0.21	(0.14)	0.123
Friends strain	-0.07	(0.09)	0.451	0.06	(0.12)	0.600	0.13	(0.14)	0.341	-0.09	(0.09)	0.311	0.05	(0.12)	0.682	0.06	(0.16)	0.692
Loneliness (T1)										0.33	(0.09)	0.000	0.19	(0.11)	0.075	-0.14	(0.14)	0.308

APPENDIX B: Chapter 3 Tables

Table 3.1. Sample selection criteria for Chapter 3.

Selection criteria	2006 cohort N	2008 cohort N	Selection logic
1. Total sample at W1	18,469	17,217	total sample in RAND longitudinal file
2. Randomly selected and completed SAQ at W1	7,603	6,946	KLBELIG=1, LLBELIG=1, SAQcomp06=1, SAQcomp08=1 1 'Self completion; returned by mail', 2 'Self completion; completed by phone with interviewer'
3. Age older than 50 at W1	7,387	6,821	R8AGEY_B>49, R9AGEY_B>49
4. Alive at W3	5,484	4,235	alive14=1, alive16=1.
5. Married/partnered at W1	3,920	2,889	R8MSTAT, R9MSTAT 1 'Married' OR 2 'Married, spouse absent' OR 3 'Partnered'
6. Partnered to same spouse during study period (W1-W3)	2,858	2,240	spchange0614=1, spchange0816=1
7. Selecting members of the household where both spouses meet criteria above and have data on SAQ	2,476	1,942	Matching cases by HHID and Gender Excluding same-sex couples
Merged final sample N	4,418 (2,209 couples)		

Note. SAQ = Self-administered Questionnaire. W1 reflects the first wave of both cohorts (2006/08); W2 reflects the second wave of both cohorts (2010/12); W3 reflects the third wave of both cohorts (2014/16).

Table 3.2. Descriptive Statistics of the study variables.

	Husbands		Wives		Paired *t*-test (*df*)	Pearson's r	Sample Range
Characteristics	*M*	*SD*	*M*	*SD*			
Age (years)	66.49	7.81	63.62	7.63	29.693*** (2205)	0.83***	50-89
Education (years)	13.22	3.13	13.08	2.78	2.460* (2202)	0.59***	0-17
Length of marriage (years)	36.63	13.74	36.59	13.78	0.805 (2094)	0.99***	0.3-65.2
Racial minority (Non-White)	17.7%		17.7%				
Cognitive functioning W1	15.91	3.67	17.03	3.84	-11.314*** (2134)	0.26***	7-35
Frailty W1	0.18	0.10	0.19	0.11	-4.237*** (2208)	0.25***	0-.74
Depressive symptoms W1	0.81	1.38	1.02	1.62	-4.850*** (2133)	0.13***	0-8
Marital support W1	3.56	0.55	3.55	0.55	0.734 (2173)	0.30***	1-4
Marital support W2	3.57	0.55	3.54	0.57	1.242 (1892)	0.29***	1-4
Marital support W3	3.55	0.57	3.51	0.60	2.806** (1786)	0.28***	1-4
Marital strain W1	1.89	0.61	1.97	0.66	-5.631*** (2172)	0.36***	1-4
Marital strain W2	1.84	0.61	1.94	0.65	-5.637*** (1890)	0.34***	1-4
Marital strain W3	1.87	0.62	1.94	0.67	-4.273*** (1783)	0.36***	1-4
N	2,209		2,209				

Note. N = 4,418 (2,209 couples). Racial minority (Non-White) includes those who identified as Non-Hispanic Black, Hispanic, and Other. W1 reflects the first wave of both cohorts (2006/08); W2 reflects the second wave of both cohorts (2010/12); W3 reflects the third wave of both cohorts (2014/16). * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 3.3. Correlations of the study variables for husbands and wives.

Intercorrelations													
	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Frailty W1	1	.437**	-.140**	-.109**	-.122**	.134**	.128**	.148**	.162**	-.233**	.109**	.065**	-.222**
2. Depression W1	.398**	1	-.210**	-.117**	-.159**	.225**	.155**	.192**	-.085**	-.157**	-.04	.090**	-.137**
3. Marital support W1	-.096**	-.155**	1	.654**	.563**	-.433**	-.363**	-.327**	.068**	.085**	.059**	-.089**	.043*
4. Marital support W2	-.091**	-.142**	.632**	1	.638**	-.356**	-.454**	-.357**	.051*	.054*	.054*	-.085**	.067**
5. Marital support W3	-.116**	-.139**	.583**	.645**	1	-.342**	-.396**	-.450**	-.004	.085**	.011	-.077**	.087**
6. Marital strain W1	.111**	.197**	-.356**	-.278**	-.257**	1	.640**	.573**	-.066**	-.088**	-.021	.113**	-.051*
7. Marital strain W2	.093**	.181**	-.319**	-.398**	-.335**	.613**	1	.614**	-.064**	-.050*	-.036	.097**	-.056*
8. Marital strain W3	.131**	.184**	-.288**	-.327**	-.383**	.551**	.636**	1	-.053*	-.098**	-.015	.116**	-.088**
9. Age (years)	.185**	-.085**	.034	.036	-.004	-.053*	-.046*	-.021	1	-.091**	.551**	-.083**	-.198**
10. Education (years)	-.212**	-.161**	.117**	.128**	.136**	-.039	-.065**	-.092**	-.106**	1	-.114**	-.297**	.435**
11. Length of marriage (years)	.062**	-.076**	.039	.053*	.034	-.024	-.052*	-.034	.450**	-.069**	1	-.057**	-.122**
12. Racial minority (Non-White)	.021	.109**	-.134**	-.096**	-.083**	.126**	.127**	.142**	-.082**	-.324**	-.050*	1	-.319**
13. Cognitive functioning W1	-.207**	-.154**	.116**	.116**	.118**	-.059**	-.081**	-.108**	-.238**	.449**	-.108**	-.252**	1

Note. N = 4,418 (2,209 couples). Statistics for husbands are presented below the diagonal and wives are presented above the diagonal.
* p < .01; ** p < .001.

Table 3.4. Effects of Own and Partner Frailty and Depression on Marital Support and Strain Including Time Moderation

	Marital Support						**Marital Strain**					
				95% CI						95% CI		
	Estimates (b)	*SE*	*p*	*LB*	*UB*	*r*	*Estimates (b)*	*SE*	*p*	*LB*	*UB*	*r*
Intercept	3.517	0.012	0.000	3.4929	3.5408	0.99	1.965	0.014	0.000	1.9379	1.9921	0.94
Gender	-0.011	0.007	0.105	-0.0243	0.0023	0.03	0.034	0.007	0.000	0.0200	0.0473	0.10
Time (linear)	-0.022	0.005	0.000	-0.0321	-0.0119	0.10	-0.021	0.006	0.000	-0.0325	-0.0095	0.08
Gender × Time	-0.008	0.004	0.051	-0.0154	0.0000	0.05	-0.007	0.005	0.157	-0.0156	0.0025	0.03
Frailty and depression main and interaction effects												
Actor frailty	-0.295	0.082	0.000	-0.4554	-0.1349	0.06	0.445	0.089	0.000	0.2709	0.6191	0.08
Partner frailty	-0.129	0.081	0.111	-0.2877	0.0295	0.03	0.337	0.088	0.000	0.1647	0.5092	0.06
Actor depression	-0.040	0.006	0.000	-0.0525	-0.0277	0.10	0.064	0.007	0.000	0.0503	0.0774	0.15
Partner depression	-0.036	0.006	0.000	-0.0483	-0.0236	0.09	0.042	0.007	0.000	0.0285	0.0554	0.10
Actor Frailty × Actor Depression	-0.007	0.04	0.870	-0.0852	0.0721	0.00	-0.086	0.044	0.052	-0.1728	0.0006	0.03
Actor Frailty × Partner Frailty	2.248	0.796	0.005	0.6876	3.8086	0.06	-2.488	0.907	0.006	-4.2655	-0.7097	0.06
Actor Frailty × Partner Depression	-0.005	0.055	0.926	-0.1136	0.1034	0.00	-0.029	0.061	0.642	-0.1489	0.0918	0.01
Actor Depression × Partner Frailty	-0.046	0.056	0.408	-0.155	0.063	0.01	0.027	0.061	0.657	-0.0932	0.1478	0.01
Actor Depression × Partner Depression	0.003	0.004	0.456	-0.005	0.0111	0.02	0.003	0.005	0.460	-0.0057	0.0126	0.02
Partner Frailty × Partner Depression	0.027	0.04	0.492	-0.0504	0.1049	0.01	-0.069	0.043	0.112	-0.1539	0.0161	0.03
Time interaction												
Actor frailty × Time	-0.084	0.045	0.065	-0.1723	0.0051	0.03	0.086	0.052	0.100	-0.0165	0.1880	0.03
Partner frailty × Time	-0.036	0.045	0.426	-0.1243	0.0525	0.01	0.065	0.052	0.212	-0.0372	0.1673	0.02
Actor depression × Time	0.002	0.004	0.528	-0.0047	0.0091	0.01	-0.008	0.004	0.052	-0.0160	0.0001	0.03
Partner depression × Time	0.006	0.004	0.118	-0.0014	0.0126	0.03	-0.012	0.004	0.003	-0.0200	-0.0040	0.05
Actor Frailty × Actor Depression × Time	0.055	0.023	0.017	0.0099	0.1007	0.04	0.014	0.027	0.592	-0.0382	0.067	0.01
Actor Frailty × Partner Frailty × Time	0.058	0.431	0.892	-0.7867	0.9034	0.00	0.753	0.488	0.123	-0.2041	1.7111	0.03
Actor Frailty × Partner Depression × Time	-0.03	0.031	0.332	-0.0914	0.0309	0.02	0.003	0.036	0.945	-0.0684	0.0734	0.00

Table 3.4. (cont'd)

	Marital Support						Marital Strain					
				95% CI						95% CI		
	Estimates (b)	*SE*	*p*	*LB*	*UB*	*r*	*Estimates (b)*	*SE*	*p*	*LB*	*UB*	*r*
Actor Depression × Partner Frailty × Time	-0.014	0.031	0.657	-0.0756	0.0476	0.01	-0.081	0.036	0.025	-0.1511	-0.0104	0.04
Actor Depression × Partner Depression × Time	-0.004	0.002	0.048	-0.0089	0.0000	0.04	0.004	0.003	0.157	-0.0014	0.0087	0.03
Partner Frailty × Partner Depression × Time	0.069	0.023	0.002	0.0249	0.1138	0.05	0.041	0.026	0.109	-0.0092	0.092	0.03
Control variables (W1)												
Age (years)	0.001	0.001	0.274	-0.0011	0.0039	0.02	-0.004	0.001	0.011	-0.0063	-0.0008	0.04
Education (years)	0.007	0.003	0.017	0.0013	0.0134	0.04	0.005	0.003	0.127	-0.0015	0.0117	0.02
Marital duration (years)	0.002	0.001	0.027	0.0002	0.0029	0.05	0.000	0.001	0.571	-0.0020	0.0011	0.01
Racial minority (Non-White)	-0.038	0.012	0.001	-0.0611	-0.0153	0.06	0.064	0.013	0.000	0.0383	0.0896	0.09
Cognitive functioning	0.002	0.002	0.306	-0.0021	0.0067	0.02	0.000	0.002	0.904	-0.0050	0.0044	0.00
Random effects												
Var. intercept male	0.179	0.007	0.000	0.1660	0.1934		0.208	0.008	0.000	0.1921	0.2245	
Var. intercept female	0.200	0.008	0.000	0.1857	0.2158		0.237	0.009	0.000	0.2193	0.2562	
Var. slope male	0.011	0.003	0.000	0.0068	0.0182		0.018	0.004	0.000	0.0118	0.0264	
Var. slope female	0.020	0.003	0.000	0.0144	0.0265		0.018	0.004	0.000	0.0118	0.0287	
Cov. intercept male, intercept female	0.058	0.005	0.000	0.0473	0.0686		0.100	0.007	0.000	0.0871	0.1134	
Cov. intercept male, slope male	0.007	0.003	0.011	0.0016	0.0122		0.004	0.003	0.209	-0.0024	0.0109	
Cov. intercept female, slope female	0.015	0.003	0.000	0.0087	0.0208		0.000	0.004	0.898	-0.0070	0.0080	
Cov. intercept male, slope female	0.000	0.003	0.875	-0.0053	0.0063		0.001	0.004	0.787	-0.0062	0.0081	
Cov. slope male, intercept female	0.003	0.003	0.248	-0.0024	0.0092		-0.002	0.004	0.578	-0.0092	0.0051	
Cov. slope male, slope female	0.003	0.002	0.108	-0.0008	0.0077		0.004	0.003	0.110	-0.0010	0.0100	
Residual male	0.103	0.004	0.000	0.0960	0.1098		0.132	0.004	0.000	0.1231	0.1407	
Residual female	0.104	0.004	0.000	0.0976	0.1118		0.152	0.005	0.000	0.1425	0.1627	
Cov. residual male, female	0.148	0.025	0.000	0.0986	0.1959		0.100	0.025	0.000	0.0521	0.1483	

Table 3.4. (cont'd)

	Marital Support						Marital Strain					
				95% CI						95% CI		
	Estimates (b)	*SE*	*p*	*LB*	*UB*	*r*	*Estimates (b)*	*SE*	*p*	*LB*	*UB*	*r*
Model fit indices												
− 2 Log likelihood			14641.276						17487.999			
AIC			14667.276						17513.999			

Table 3.5. Effects of Own and Partner Frailty and Depression on Marital Support and Strain Including Gender Moderation

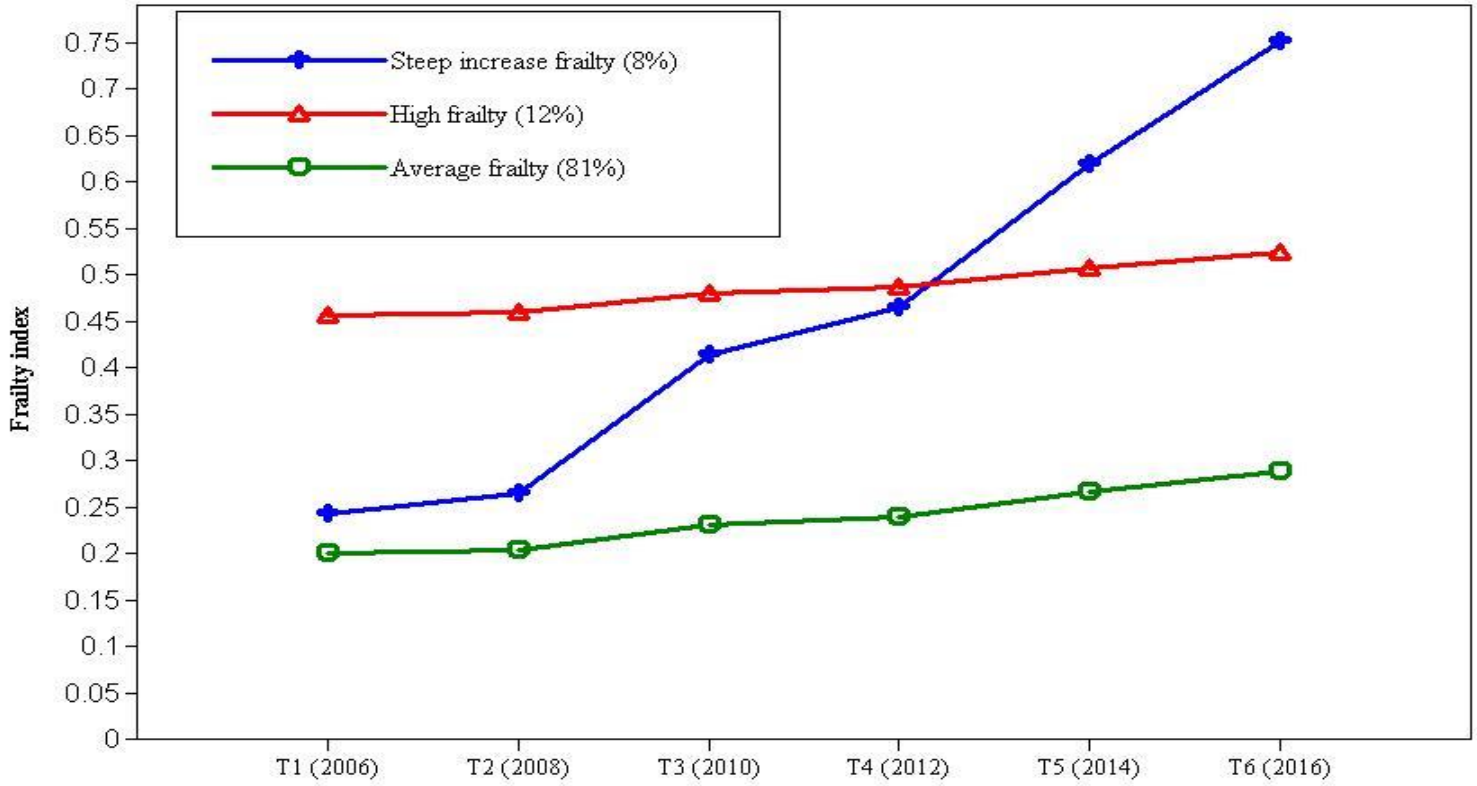
	Marital Support						**Marital Strain**					
				95% CI						95% CI		
	Estimates (b)	*SE*	*p*	*LB*	*UB*	*r*	*Estimates (b)*	*SE*	*p*	*LB*	*UB*	*r*
Intercept	3.516	0.012	0.000	3.4915	3.5396	0.99	1.968	0.014	0.000	1.9412	1.9956	0.94
Gender	-0.012	0.008	0.128	-0.0264	0.0033	0.03	0.032	0.008	0.000	0.0169	0.0473	0.09
Time (linear)	-0.016	0.004	0.000	-0.0241	-0.0072	0.06	-0.015	0.005	0.002	-0.0248	-0.0056	0.05
Gender × Time	-0.009	0.004	0.020	-0.0158	-0.0013	0.04	-0.006	0.004	0.185	-0.0144	0.0028	0.02
Frailty and depression main and interaction effects												
Actor frailty	-0.275	0.082	0.001	-0.4354	-0.1148	0.05	0.419	0.089	0.000	0.2443	0.5929	0.08
Partner frailty	-0.128	0.081	0.115	-0.2865	0.0313	0.02	0.349	0.088	0.000	0.1762	0.5222	0.06
Actor depression	-0.041	0.006	0.000	-0.0532	-0.0282	0.10	0.065	0.007	0.000	0.0511	0.0785	0.15
Partner depression	-0.039	0.006	0.000	-0.0514	-0.0262	0.10	0.045	0.007	0.000	0.0315	0.0591	0.11
Actor Frailty × Actor Depression	-0.011	0.040	0.781	-0.0903	0.0679	0.00	-0.083	0.044	0.063	-0.1698	0.0044	0.03
Actor Frailty × Partner Frailty	2.28	0.799	0.004	0.7138	3.8459	0.06	-2.508	0.911	0.006	-4.2948	-0.7205	0.06
Actor Frailty × Partner Depression	0.014	0.056	0.804	-0.0957	0.1235	0.00	-0.048	0.062	0.438	-0.1699	0.0735	0.01
Actor Depression × Partner Frailty	-0.037	0.056	0.506	-0.1469	0.0725	0.01	0.02	0.062	0.748	-0.1017	0.1415	0.01
Actor Depression × Partner Depression	0.003	0.004	0.534	-0.0055	0.0106	0.01	0.002	0.005	0.618	-0.0069	0.0116	0.01
Partner Frailty × Partner Depression	0.021	0.040	0.605	-0.0584	0.1002	0.01	-0.062	0.045	0.161	-0.1498	0.0249	0.02
Gender interaction												
Actor frailty × Gender	0.068	0.083	0.414	-0.0951	0.2310	0.01	-0.124	0.09223	0.178	-0.3050	0.0566	0.02
Partner frailty × Gender	-0.153	0.083	0.067	-0.3160	0.0107	0.03	0.199	0.09247	0.031	0.0178	0.3804	0.04
Actor depression × Gender	0.001	0.006	0.876	-0.0116	0.0136	0.00	-0.010	0.00706	0.175	-0.0234	0.0043	0.02
Partner depression × Gender	-0.003	0.007	0.684	-0.0154	0.0101	0.01	0.014	0.00717	0.058	-0.0004	0.0277	0.03
Actor Frailty × Actor Depression × Gender	-0.032	0.04	0.423	-0.1102	0.0463	0.01	0.086	0.04382	0.050	0.000	0.1718	0.03
Actor Frailty × Partner Frailty × Gender	0.037	0.60	0.951	-1.139	1.213	0.00	-0.44	0.61144	0.472	-1.6386	0.7596	0.02
Actor Frailty × Partner Depression × Gender	0.086	0.053	0.105	-0.018	0.1897	0.03	0.04	0.05767	0.489	-0.0732	0.1530	0.01

Table 3.5. (cont'd)

	Marital Support						Marital Strain					
				95% CI						95% CI		
	Estimates (b)	*SE*	*p*	*LB*	*UB*	*r*	*Estimates (b)*	*SE*	*p*	*LB*	*UB*	*r*
Actor Depression × Partner Frailty × Gender	0.003	0.053	0.953	-0.101	0.1072	0.00	-0.053	0.05767	0.362	-0.1656	0.0605	0.01
Actor Depression × Partner Depression × Gender	-0.002	0.003	0.453	-0.0084	0.0038	0.02	-0.002	0.00318	0.615	-0.0078	0.0046	0.01
Partner Frailty × Partner Depression × Gender	0.016	0.040	0.682	-0.0621	0.0949	0.01	-0.029	0.04395	0.508	-0.1152	0.0571	0.01
Control variables (W1)												
Age (years)	0.001	0.001	0.349	-0.0013	0.0037	0.00	-0.004	0.001	0.011	-0.0063	-0.0008	0.04
Education (years)	0.008	0.003	0.013	0.0016	0.0138	0.01	0.005	0.003	0.152	-0.0018	0.0114	0.02
Marital duration (years)	0.002	0.001	0.029	0.0002	0.0029	0.00	0.000	0.001	0.699	-0.0018	0.0012	0.01
Racial minority (Non-White)	-0.038	0.012	0.001	-0.0611	-0.0151	-0.02	0.065	0.013	0.000	0.0395	0.0908	0.09
Cognitive functioning	0.002	0.002	0.273	-0.0020	0.0069	0.01	0.000	0.002	0.906	-0.0050	0.0044	0.00
Random effects												
Var. intercept male	0.175	0.007	0.000	0.1615	0.1886		0.201	0.008	0.000	0.1853	0.2173	0.201
Var. intercept female	0.192	0.008	0.000	0.1774	0.2071		0.230	0.009	0.000	0.2123	0.2488	0.230
Cov. intercept male, intercept female	0.056	0.005	0.000	0.0458	0.0669		0.098	0.007	0.000	0.0848	0.1109	0.098
Residual male	0.114	0.003	0.000	0.1090	0.1197		0.149	0.004	0.000	0.1425	0.1563	0.149
Residual female	0.125	0.003	0.000	0.1190	0.1306		0.171	0.004	0.000	0.1637	0.1795	0.171
Cov. residual male, female	0.163	0.017	0.000	0.1283	0.1965		0.120	0.017	0.000	0.0864	0.1527	0.120
Model fit indices												
– 2 Log likelihood			14749.662						17532.094			
AIC			14761.662						17544.094			

APPENDIX C: Chapter 2 Figures

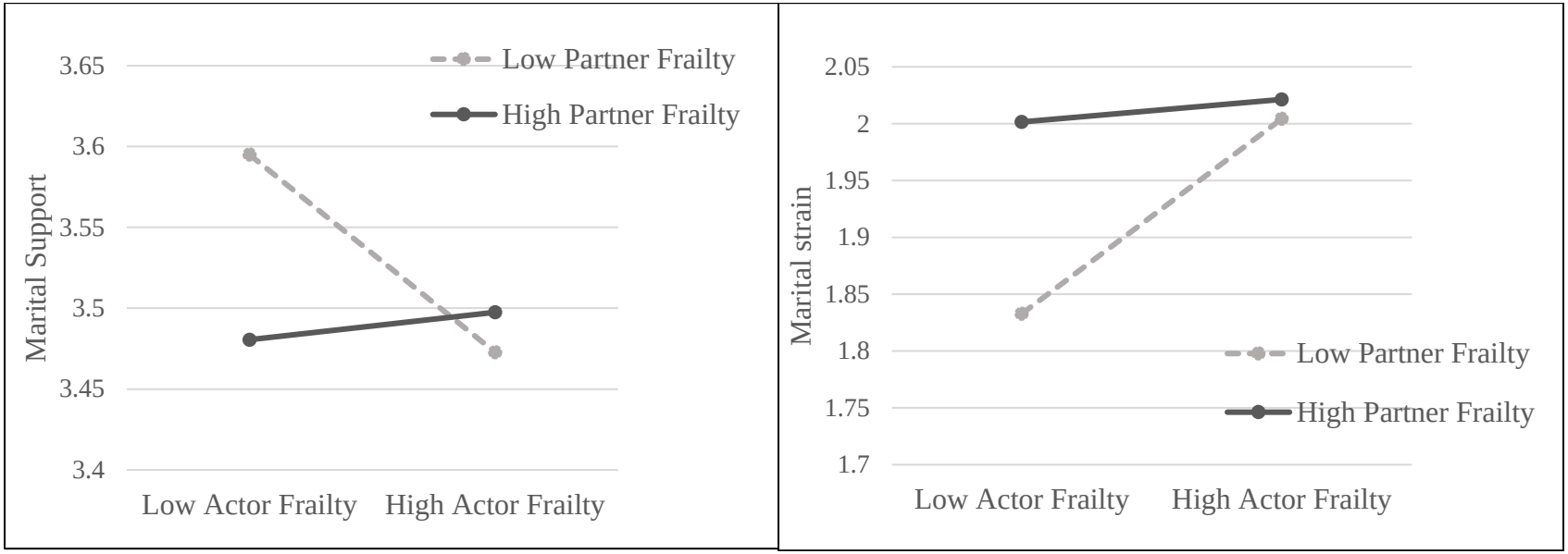
Figure 2.1. Frailty progression group trajectories



The largest group was "Average frailty" group with low initial frailty level and least steep slope (circles), the second largest group was "High frailty" group with higher baseline frailty level and small slope over time (triangles), and the smallest group was "Steep increase frailty" group with low baseline frailty level with steep increase over time (cross).

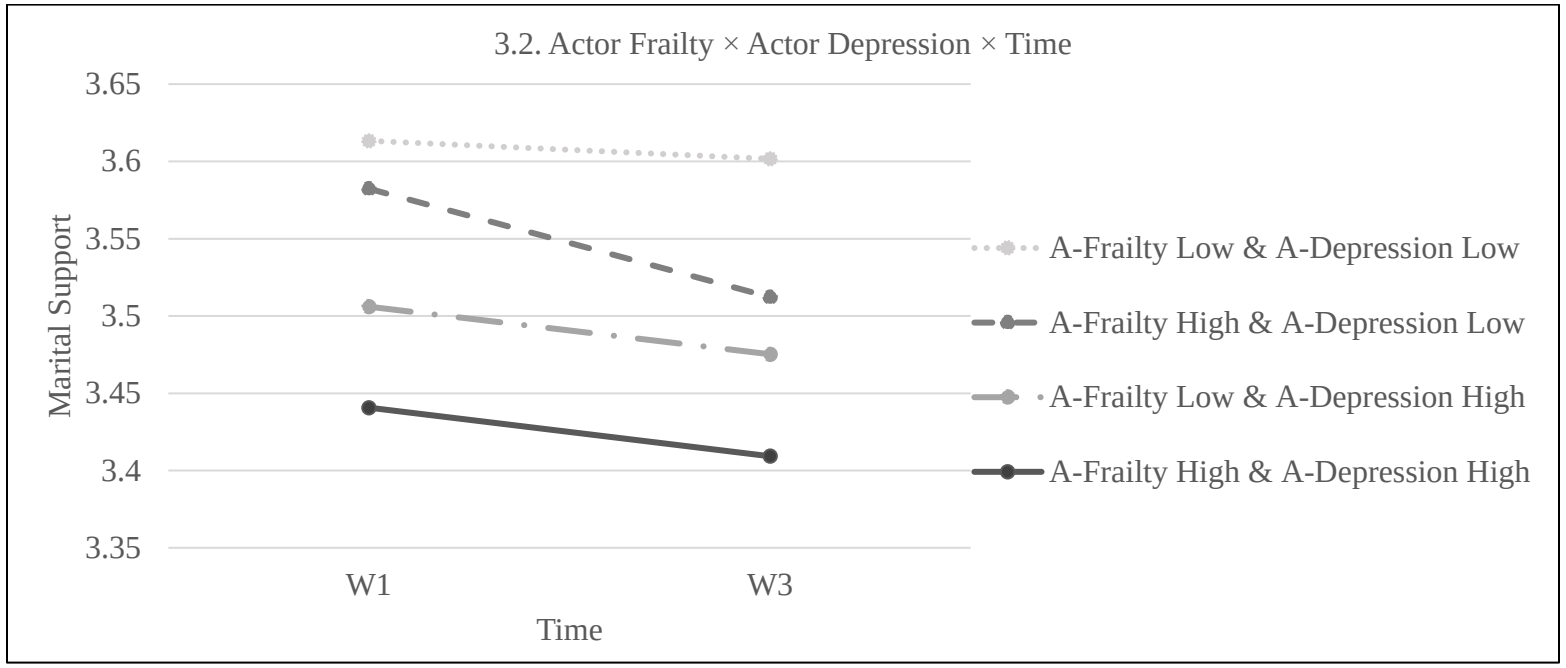
APPENDIX D: Chapter 3 Figures

Figure 3.1. Actor × Partner frailty interactions predicting marital support and marital strain



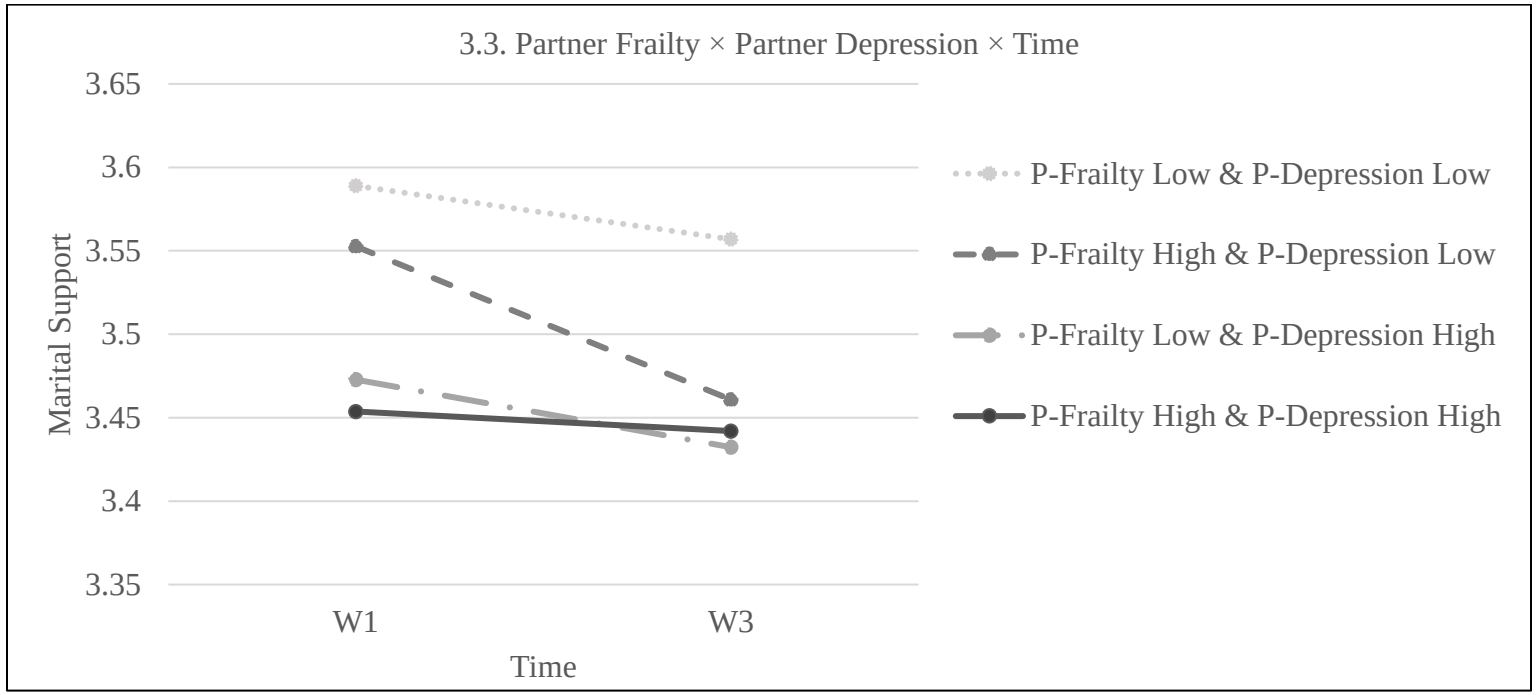
Actor × Partner frailty interactions predicting marital support (left) and marital strain (right), plotted at one standard deviation above and below the mean of actor frailty and partner frailty.

Figure 3.2. Time moderation in multiplicative effects of actor's frailty and depression on their marital support



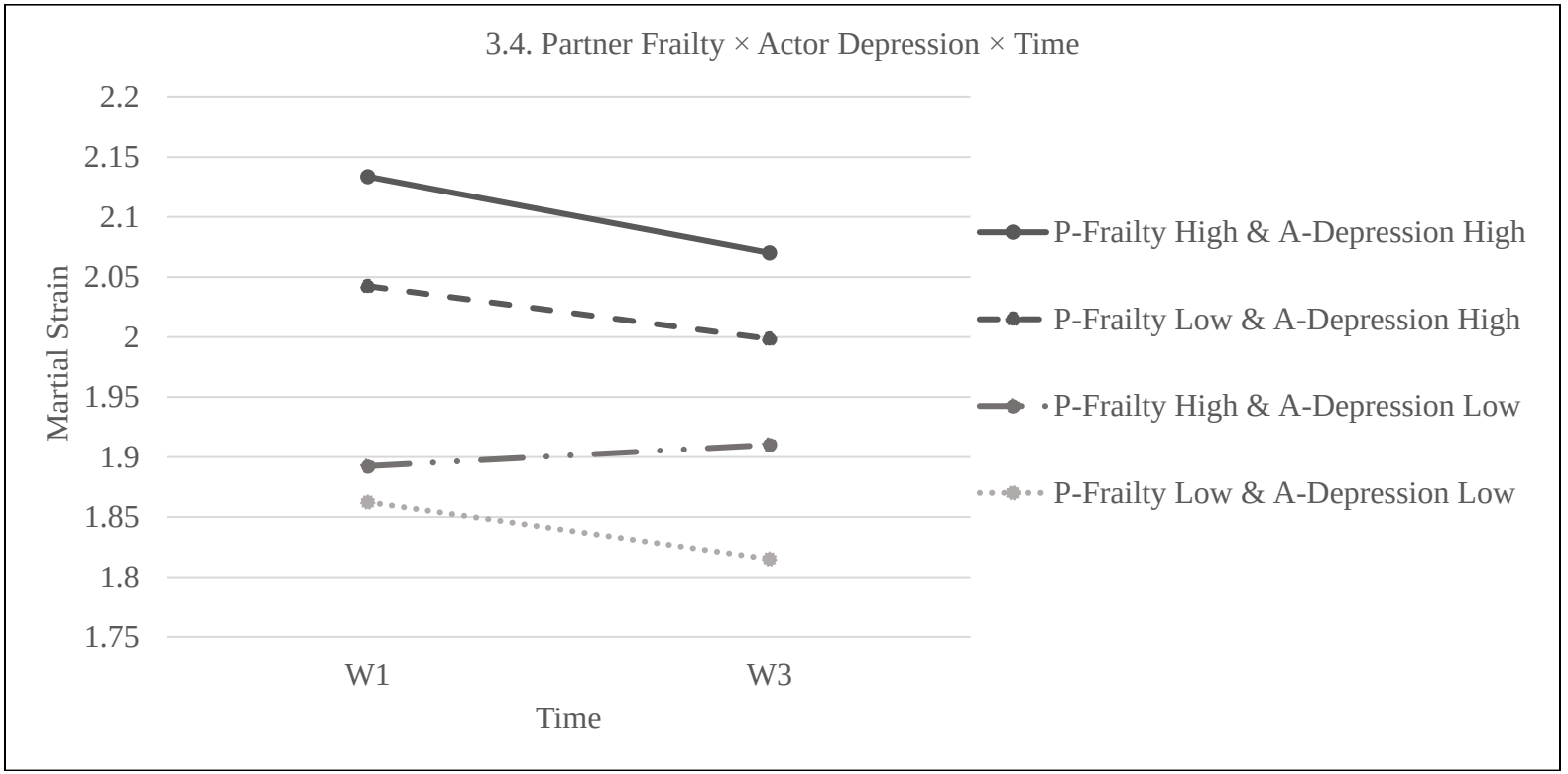
Time moderation in multiplicative effects of actor's frailty and depression on their marital support**,** plotted at one standard deviation above (high) and below (low) the mean of actor frailty and actor depression; except for when actor had low frailty and depression (light gray dotted), other three interaction conditions showed slight decrease in marital support for the person.

Figure 3.3. Time moderation in multiplicative effects of partner's frailty and depression on the person's marital support



Time moderation in multiplicative effects of partner's frailty and depression on the person's marital support**,** plotted at one standard deviation above (high) and below (low) the mean of partner frailty and partner depression; all four interaction conditions' effect on marital support decreased from W1 to W3, but the decrease in marital support was most drastic for the person when their partner had high frailty with low depression (gray-dashes); the person's marital support was similarly low when their partner had low frailty with high depression (gray-dash-dot) and when partner had high frailty and depression (black-line).

Figure 3.4. Time moderation in multiplicative effects of partner's frailty and actor's depression on the person's marital strain



Time moderation in multiplicative effects of partner's frailty and actor's depression on the person's marital strain, plotted at one standard deviation above (high) and below (low) the mean of partner frailty and actor depression; contrary to prediction, the interaction effects of partner frailty and actor depression on marital strain decreased over time for three conditions; only when actor's depression is low and their partner's frailty was high there was an increase in marital strain (gray line-dot-dot).

APPENDIX E: Supplementary Table

Table A1. List of items and coding schemes for creating the frailty index in HRS 2006-2016 waves

No.	Category	Operationalization in HRS (Current study)	Questionnaire item	Available waves	Final coding scheme
1	ADL	Difficulty getting in and out of bed	(Because of a health or memory problem do you have any difficulty with) getting in or out of bed?	All six	1= some difficulty, 0= no difficulty
2	ADL	Difficulty bathing or showering	(Because of a health or memory problem do you have any difficulty with) bathing or showering?	All six	1= some difficulty, 0= no difficulty
3	ADL	Difficulty with using toilet	(Because of a health or memory problem do you have any difficulty with) using the toilet, including getting up and down?	All six	1= some difficulty, 0= no difficulty
4	ADL	<Change> in everyday activities (6 items)	Change in activities of daily living (RwADLA - RpADLA), ADL= difficulty with walking across a room, getting in and out of bed, dressing, bathing, eating, using the toilet	All six	1=worsened ADL condition (current wave ADL score > previous wave ADL score), 0=same or improved ADL condition
5	IADL	Difficulty preparing hot meals	(Because of a health or memory problem do you have any difficulty) preparing a hot meal?	All six	1= some difficulty, 0= no difficulty
6	IADL	Difficulty shopping for groceries	(Because of a health or memory problem do you have any difficulty with) with shopping for groceries?	All six	1= some difficulty, 0= no difficulty
7	IADL	Difficulty taking medications	(Because of a health or memory problem do you have any difficulty with) with shopping for groceries?	All six	1= some difficulty, 0= no difficulty

Table A1. (cont'd)

No.	Category	Operationalization in HRS (Current study)	Questionnaire item	Available waves	Final coding scheme
8	Functional Limitations	Mobility (RwMOBILA): The five tasks included in the mobility index	Walking several blocks (RwWAKSA), walking one block (RwWALK1A), walking across the room (RwWALKRA), climbing several flights of stairs (RwCLIMSA) and climbing one flight of stairs (RwCLIM1A)	All six	RwMOBILA/5
9	Functional Limitations	Large Muscle (RwLGMUSA): The four tasks included in the large muscle index.	Sitting for two hours, getting up from a chair, stooping or kneeling or crouching, and pushing or pulling a large object.	All six	RwLGMUSA/4
10	Functional Limitations	Fine Motor Skills (RwFINEA): The three tasks included in this index are: picking up a dime, eating, and dressing.	RwDIMEA, RwEATA, RwDRESSA	All six	RwFINEA/3
11	Cardiovascular	High blood pressure	Has a doctor told you that you have high blood pressure or hypertension?	All six	1=yes, 0=no
12	Cardiovascular	Heart attack, coronary heart disease, angina, congestive heart failure, or other heart problems	Has a doctor told you that you had a heart attack, coronary heart disease, angina, congestive heart failure, or other heart problems?	All six	1=yes, 0=no
13	Cardiovascular	Stroke	Has a doctor told you that you had a stroke?	All six	1=yes, 0=no
14	Cognitive/psychiatric	Emotional, nervous, or psychiatric problems other than depression or GAD	Have you had or has a doctor told you that you have any emotional, nervous, or psychiatric problems?	All six	1=yes, 0=no

Table A1. (cont'd)

No.	Category	Operationalization in HRS (Current study)	Questionnaire item	Available waves	Final coding scheme
15	Cognitive/psychiatric	Long-term memory impairment	RwMEMRYE indicates whether or not a doctor has ever told the Respondent s/he had this condition in this or any previous wave. (R8,9). RwALZHEE and RwDEMENE indicate whether or not a doctor has ever told the Respondent s/he had this condition in this or any previous wave. (R10,11,12)	All six	0=none, 1=having either AD or dementia, 1=having both AD and dementia (R10, R11, R12)
16	Cognitive/psychiatric	<Change> in memory compared to two years ago	Compared to [the last two years/two years ago], would you say your memory is better now, about the same, or worse now than it was then?	All six	1= worse (3), 0=same (2) or better (1).
17	Metabolic	Diabetes or high blood sugar	Has a doctor told you that you have diabetes or high blood sugar?	All six	1=yes, 0=no
18	Cancer	Cancer or a malignant tumor of any kind except skin cancer	Has a doctor told you that you have cancer or a malignant tumor, excluding minor skin cancer?	All six	1=yes, 0=no
19	Respiratory	Chronic bronchitis or emphysema	Has a doctor told you that you have chronic lung disease such as chronic bronchitis or emphysema?	All six	1=yes, 0=no
20	Pain	Arthritis or rheumatism	Have you had, or has a doctor told you that you have arthritis or rheumatism?	All six	1=yes, 0=no
21	Incontinence	Incontinence past 12 months	During the last 12 months, have you lost any amount of urine beyond your control?	All six	1=yes, 0=no
22	Pain	Trouble with pain	Are you often troubled with pain?	All six	1=yes, 0=no
23	Respiratory	Persistent couch/wheeze/phlegm	Persistent wheezing, cough, or bringing up phlegm?	2008, 12, 16	1=yes, 0=no
24	Pain	Persistent headache	Have you had persistent headaches?	2008, 12, 16	1=yes, 0=no

Table A1. (cont'd)

No.	Category	Operationalization in HRS (Current study)	Questionnaire item	Available waves	Final coding scheme
25	Fatigue	Persistent or troublesome fatigue or exhaustion	Have you had any of the following persistent or troublesome problems? Severe fatigue or exhaustion?	2008, 12, 16	1=yes, 0=no
26	Pain	Back pain or problems	Have you had any of the following persistent or troublesome problems; Back pain or problems?	2008, 12, 16	1=yes, 0=no
27	Sensory	Poor self-rated hearing despite hearing aid	Is your hearing excellent, very good, good, fair, or poor using a hearing aid as usual?	All six	(5 level) 0= Poor, 0.25 = Fair, 0.5 = Good, 0.75 = Very Good, 1= Excellent
28	Sensory	Poor self-rated vision despite corrective lenses	Is your eyesight excellent, very good, good, fair, or poor using glasses or corrective lenses as usual?	All six	(5 level) 0= Legally blind or Poor, 0.25 = Fair, 0.5 = Good, 0.75 = Very Good, 1= Excellent
29	Self-report of health	Poor Self-report of health	Would you say your health is excellent, very good, good, fair, or poor?	All six	(5 level) 0= Poor, 0.25 = Fair, 0.5 = Good, 0.75 = Very Good, 1= Excellent
30	Body Mass Index (BMI)	low or high BMI: weight (kg)/square height (m2)	Underweight (BMI<18.5) OR Obesity (BMI $\geq$ 30) as a deficit	All six	1= (BMI<18.5) OR (BMI $\geq$ 30), 0=normal weight
31	Sleep	Trouble falling asleep	How often do you have trouble falling asleep?	2006, 10, 14	1= Most of the time, 0.5=Sometimes, 0=Rarely or never.
32	Sleep	Waking up during night	How often do you have trouble with waking up during the night?	2006, 10, 14	1= Most of the time, 0.5=Sometimes, 0=Rarely or never.

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