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Psychosocial Pathways and Functional Consequences of Illness Behavior Trajectories Across Late Adulthood

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by

Brittany Lynn Bannon

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Dissertation Committee:
Dr. Chandra Reynolds, Chairperson
Dr. Kate Sweeny
Dr. Misaki Natsuaki

The Dissertation of Brittany Lynn Bannon is approved:

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Committee Chairperson

University of California, Riverside

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ABSTRACT OF THE DISSERTATION

Psychosocial Pathways and Functional Consequences of Illness Behavior Trajectories Across Late Adulthood

by

Brittany Lynn Bannon

Doctor of Philosophy, Graduate Program in Psychology
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Dr. Chandra Reynolds, Chairperson

The primary purpose of this dissertation was to examine age-related trajectories in illness behavior—or perceptions, evaluations, and responses to symptoms—across the late-life transition, the predictive role of perceived social support availability, and the extent to which illness behavior mediates the association of social support with subsequent functional decline. For Study 1, two large population-based samples were drawn from the Swedish Adoption/Twin Study of Aging (SATSA) and the Sex Differences in Health and Aging study (GENDER) to examine longitudinal measurement invariance in illness behavior. The extent to which social support availability from friends versus family members predicted reduced levels and change in illness behavior across 17 years from mid- to late-adulthood in SATSA was also examined. Confirmatory factor analyses supported strict factorial invariance in the illness behavior factor across four waves in SATSA, whereas partial, weak factorial invariance was supported in GENDER. Latent growth models suggested, a small, linear increase in illness behavior across age, and perceived support from friends and family both predicted reduced levels

of illness behavior—but not change—after controlling for sex, comorbidity, SES, marital status, and age at study entry.

For Study 2, age-related change in functional difficulty was evaluated across 23 years in SATSA, as well as the extent to which social support availability (again, from friends versus family) buffered decline. Additionally, longitudinal mediation models evaluated the extent to which illness behavior explained the association between social support availability and subsequent functional status and change. Overall, findings suggested piecewise growth in functional difficulty, with stability prior to age 75 and a linear increase afterward. Higher family support availability predicted faster decline, whereas friend support was not associated. Furthermore, illness behavior status mediated the association between social support availability and functional status; whereas intra-individual growth in illness behavior separately predicted a faster rate of functional decline. Collectively, these results suggest that illness behavior, as a unifying individual difference construct, exhibits systematic intra-individual change and significant variability during the transition to late adulthood, and represents a potentially important mediating and independent pathway linking perceived social support availability with subsequent physical functioning.

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The Psychosocial Development of Illness Behavior: An Introduction

Overview

Despite explosive advances in health care technology and treatments, Americans consistently report feeling worse than ever before (the "paradox of health and illness"; Barsky, 1988), with increases in subjective health complaints, medically unexplained symptoms, and worries about the health consequences of modern living (Eriksen, Hellesnes, Staff, & Ursin, 2004; Filipkowski et al., 2010). In fact, behavioral and emotional challenges are prevalent in primary care settings. For example, 40-60% of people with mental health challenges present almost exclusively in primary care, and they also utilize more medical services than those without mental health challenges (Kessler & Stafford, 2008). Non-adherence to medical treatment is another psychosocial issue that accounts for up to 300 billion dollars wasted in U.S. healthcare every year (DiMatteo, 2004) in a context where annual expenditures may reach approximately $3 trillion dollars (National Center for Health Statistics, 2011). Thus, although we continue to spend a lot on medical care, patients are not receiving or adhering to optimal treatment.

Existing epidemiological behavioral models of healthcare utilization (Andersen, 1968/1995) do not take the entire illness process into account—from its initial detection to treatment seeking—nor do they specify the interrelationships among the psychosocial antecedents at the individual level. For example, prior to seeking care, a patient must first notice and attend to a bodily symptom, develop a mental representation or "lay model" of the symptom (e.g., labels, attributions about severity or consequences), have an emotional

response (e.g., worry or distress), engage in strategies to cope, and subsequently evaluate their status as "well" or "ill" to form a decision about seeking medical care (Leventhal, Leventhal, & Contrada, 1998). Some people, however, either fail to notice or choose to dismiss their symptoms, and either delay care seeking or avoid it altogether. Individual variability in the perceptions, evaluations and behavioral responses to bodily symptoms that might signify illness is referred to as illness behavior (Mechanic, 1962; 1978). The construct of illness behavior encompasses a wide range of cognitions and responses, including symptom detection, appraisal and monitoring, information-seeking, sharing health concerns with others, and avoiding social or work-related obligations—to name a few. As such, it provides a useful target for research that seeks to clarify the behavioral pathways linking psychosocial processes to optimal health outcomes across the lifespan. The present study, by examining the psychosocial factors that underlie various aspects of illness behavior across late-life development—from somatic complaints and illness perceptions to use of non-prescription medications—aims to shed light on who is more likely to respond to bodily symptoms, and under what social circumstances. Specifically, this dissertation study will focus on the predictive value of proximal, social factors on the development of illness behavior patterns across the transition from mid- to late adulthood.

The overarching, conceptual model for the social processes in illness behavior development (i.e. the Social Processes in Illness Behavior/SPIB Model) is displayed in Figure 1.1. The SPIB model builds on previous models of health- and illness behavior (i.e. Behavioral Model; Andersen, 1968/1995/2014; Self-Regulatory Model; Leventhal,

1970; Integrated Change Model; de Vries, Mesters, van de Steeg, & Honing, 2005) by including potential direct and indirect mechanisms of the social environment's associations with illness behaviors and subsequent health outcomes, as well as specifying the interrelationships among social, emotional, and cognitive antecedents. For example, social support—the focus of the current dissertation—might relate to illness behaviors through direct pathways (e.g., behavioral norms, modeling, or access to resources) or indirect pathways (e.g., reductions in negative affect, promoting values for health promotion, or self-efficacy). Social support has also been shown to predict health directly via physiological pathways that are unmediated by health-relevant behaviors (e.g., immune functioning, pulmonary functioning, heart rate variability). In turn, the model proposes that illness behaviors during the late-life transition will have significant, prospective relationships with functional health. Moreover, the associations between social processes and illness behavior likely vary by individual characteristics such as age, gender, and disease or symptom severity, which are proposed as moderators. This model advances prior work in important ways. First, it emphasizes the range of symptom responses that occur prior to medical help-seeking to evaluate an understudied behavioral pathway to healthy aging, rather than emphasizing decisions about healthcare utilization or other single aspects of illness behavior (e.g., symptom reporting). Second, it explicitly considers social influences on illness behavior (apart from prior models' emphases on life stressors or communication) and it specifies the interrelationships among its psychosocial antecedents.

Illness behaviors encompass a wide range of actions and cognitions that could be characterized along a continuum of responsiveness to symptoms or health threats. For example, ignoring symptoms or delaying help seeking fall at the lower end of the responsiveness continuum; whereas excessive physical complaints, overt pain behaviors (e.g., grimacing, groaning), frequent care-seeking, and absenteeism or avoiding social obligations are characteristic of the higher end of the continuum. Importantly, highly responsive and consistent illness behaviors (i.e. excessive worry or monitoring of symptoms, frequent sick days and medical help-seeking; Whitehead, Winget, Fedoravicius, Wooley, & Blackwell, 1982) predict important outcomes such as exacerbated symptoms or increased pain levels in the short-term (Harkins, Price, & Braith, 1989), disability-related unemployment (Broadbent, Ellis, Thomas, Gamble, & Petrie, 2009), and even reduced emotional support from spousal caregivers over longer periods of time (Scharloo & Kaptein, 1997; Stephens, Martire, Cremeans-Smith, Druley, & Wojno, 2006). Among some patients, such extreme illness behaviors may become more disabling than the original physical problem and eventually require a treatment plan of their own (Waddell, Main, Morris, DiPaola, & Gray, 1984). For example, among patients with chronic low back pain, overt pain behaviors as rated by physicians (e.g., grimacing, abnormally rigid movements, vocalizations) were positively correlated with disability levels and work absences, yet were unrelated to objective physical impairments (Waddell & Richardson, 1992). In a separate study of primary care patients presenting with acute back pain, aspects of coping behavior (i.e. catastrophizing) and perceptions of low pain control at the initial visit were almost seven times more predictive of disability

at one-year follow-up than were clinical pain measures (Burton, Tillotson, Main, & Hollis, 1995).

Furthermore, when these illness behaviors are coupled with the increasingly widespread issue of medical overdiagnosis (Welch, Schwartz, & Woloshin, 2011)—or the uptake of screenings and treatments judged to be of minimal diagnostic value in most cases by the medical community (e.g., x-rays for low-back or knee pain, or MRIs for detecting a brain tumor; Scherer, Caverly, Burke, Zikmund-Fisher, Kullgren, Steinley,… & Fagerlin, 2016)—it can lead to unnecessary or overly aggressive medical intervention. These unnecessary interventions, in turn, may carry negative health consequences such as iatrogenic illness or iatrogenic disability, defined as accidental complications caused by medical diagnostic procedures or treatments. On the other end of the spectrum, however, stoicism (i.e. ignoring symptoms or suppressing one's reactions) can exacerbate symptoms, and in some cases of serious illness (e.g., cancer or myocardial infarction), it can predict life-threatening delays in diagnosis and treatment. Arguably, both extremes of the illness behavior continuum may negatively affect health outcomes through over- or under-treatment, and consequently also place an economic burden on the individual and on the healthcare system. Before targeting the appropriateness of such behaviors through policy and intervention, however, it is important to first examine the following: *how* and *when* certain patterns of illness behavior develop and change across the lifespan; the more proximal social antecedents of illness behavior trends and their mechanisms; and lastly, the relationship between these illness behavior trends and subsequent physical health and functioning.

Antecedents and mechanisms of illness behavior

One of the key empirical questions concerning illness behaviors is to what extent they reflect underlying pathology or the disease process itself (e.g., medical diagnoses, condition severity), the intrinsic characteristics of the patient (e.g., coping styles, attitudes), or the social environment (e.g., cultural norms, access to care, social support, and reinforcement). The answer to this question warrants further investigation, because illness behavior likely reflects a complicated interplay among biological, social, and psychological factors (see proposed conceptual model in Figure 1.1). In the extant literature, the predictive value of psychosocial factors on illness behavior outcomes is of similar magnitude to the widely-demonstrated associations of biological risks, or even patient demographics like gender (Vedsted & Christensen, 2005; Verbrugge, 1989), socioeconomic status (Phelan, Link, Diez-Roux, Kawachi, & Levin, 2004), or education (Al-Windi, Dag, & Kurt, 2002)—and across a variety of medical conditions and settings. One of the most widely used models of healthcare utilization, one key aspect of illness behavior, is Ronald Andersen's behavioral model (1968; 1995). This model posits that individuals' use of health services is dependent upon three categories of factors: *pre-disposing* factors, or characteristics that render some people more or less likely to utilize (e.g., age, gender, personality, health beliefs, social class); *enabling* factors, or those that remove key barriers to utilization (e.g., education, income, location of residence, health insurance); and *need-based* factors, that are most proximally related to utilization (e.g., self-rated health, medical diagnoses, functional status). This model has since been modified to a systems approach that includes broader influences of society (i.e., norms

for medical care, technology) and the healthcare system (i.e., resources, geographic distribution of these resources, and medical practices) (Andersen, Smedby, & Anderson, 1970; Andersen & Newman, 1973; Andersen, Davidson & Baumeister, 2014). It has also been criticized, however, for its exclusion of detailed social support measures as pre-disposing or enabling factors, and other aspects such as genetic influences have been added as pre-disposing factors in the model (Andersen, 1995; Andersen et al., 2014; True et al., 1997).

Other researchers have applied models of health behavior and behavior change to the prediction of illness behaviors like symptom reporting and adherence, also sometimes referred to as disease self-management behavior. Two such models are the Self-Regulatory Model (SRM; Leventhal, Leventhal, & Contrada, 1998) and Integrated Change (I-Change) Model (de Vries et al., 2005). At the heart of these two models is the assumption that emotional and cognitive processes (or in the I-Change Model, "pre-motivational" and "motivational" processes), together, predict people's responses to their symptoms, whether perceived firsthand or diagnosed by a health professional. The I-Change model further considers psychosocial influences, such that pre-motivational factors (e.g., personality, genetics, family socialization, sex, culture, cues to action) determine motivational factors (i.e., attitudes, self-efficacy, social and treatment expectations), which in turn, predict the illness behaviors engaged in (de Vries et al., 2005). Indeed, the key amplifiers (or reducers) of any bodily sensation—attention, mood, beliefs, and situational circumstances—are entirely psychosocial in nature (Barsky, 1988). The current study proposes that the amplifier of social circumstances may be more

broadly construed, above and beyond everyday situations, lifestyles, or culture, to include lifespan development. The sections that follow will highlight previous work on the key psychosocial predictors of illness behavior and their proposed mechanisms.

First, personality has been proposed as a predictor of primary, preventive health behaviors and illness behaviors alike (Harkins, Price & Braith, 1989; Jerant, Chapman, Duberstein, Robbins, & Franks, 2011). Generally defined as individuals' relatively stable tendencies of behaving, thinking, and feeling, personality theoretically encompasses the afore-mentioned symptom amplifiers of attention, mood, and beliefs. Indeed, it is posited that the link between personality and illness behaviors like visiting the doctor, for example, occurs through individuals' cognitive and emotional representations of their bodily symptoms, or their "lay models" of illness (Leventhal et al., 1998; Leventhal, Safer, & Panagis, 1983; Petrie, Weinman Sharpe, & Buckley, 1996). In particular, neuroticism, which is indexed by heightened emotional reactivity, especially in response to stressors, predicts a variety of illness behaviors. These include non-adherence to medical treatment in older adult samples (Jerant et al., 2011), excessive symptom reporting and increased fear response to health threats (Harvey, 2013), pain-related distress (Harkins, Price, & Braith, 1989), reporting of chronic conditions (Charles, Gatz, Kato, & Pedersen, 2008), attending to bodily symptoms (Costa & McCrae, 1987), and perceived vulnerability to age-relevant disease (Gerend, Aiken, West, & Erchull, 2004). Thus, one theory is that neuroticism indirectly influences illness responses through lay models of heightened vulnerability and worry or anxiety about symptoms; consistent with prior findings, such responses may have very little to do with physical disease, and

everything to do with individuals' subjective or emotional experiences. Apart from trait measures of negative affect like neuroticism, higher emotional reactivity to daily stressors is prospectively associated with the reporting of chronic health conditions (Piazza, Charles, Sliwinski, Mogle, & Almeida, 2013). Additionally, clinical depression is posited to influence coping behavior indirectly through the maladaptive thought patterns (e.g., reduced self-efficacy) associated with the disorder (Harvey, 2013). Thus, emotional processes at both the trait and state level contribute to illness behavior.

Health cognitions of particular importance for illness behavior include self-efficacy (belief in one's ability to carry out a behavior or achieve an outcome), treatment efficacy (belief in a prescribed treatment or management regimen's efficacy), health locus of control (belief that one's health outcomes are controlled by intrinsic or extrinsic factors), illness attributions (e.g., cause, timeline, treatability), perceived benefits and barriers, and lastly, perceived susceptibility to disease. When considering efficacy beliefs, the relative importance of self-efficacy as compared to treatment efficacy may vary according to the outcome in question. Specifically, treatment efficacy is a better predictor of clinic utilization or medication adherence, whereas self-efficacy may have stronger associations with other preventive or coping behaviors (Lawson, Bundy, Lyne, & Harvey, 2004). Furthermore, self-efficacy is suggested to moderate individuals' actively coping with the health threat itself or with their emotional reactions to the threat (e.g. fear, anger) (c.f., Self-Regulatory Model; Leventhal et al., 1983; Leventhal et al., 1998). Both of these coping processes likely encompass different behaviors, and someone facing a symptom or health threat with higher self-efficacy is more likely to cope actively

with the problem than with the emotion (Ruiter, Verplanken, & Werrij, 2003). In other words, self-efficacy may moderate associations between emotional processes, such as fear or anxiety, and illness behavior. Conversely, the availability of personal coping resources like social support, might encourage adaptive illness behaviors through reducing emotional distress and promoting self-efficacy, among other health cognitions.

Another cognitive construct related to self-efficacy, but distinct in its relative stability across time and in its having looser associations with previous successes or failures (Harvey, 2013), is Health Locus of Control (HLC). HLC indexes the degree to which individuals view their health as under their own personal control (internal locus), controlled by others (external locus), or determined by chance. An internal locus, when it does not foster negative emotional reactions and self-blame, is typically associated with more adaptive health and illness behaviors (Harvey, 2013). Another important factor to consider is individual differences in health values, or the degree to which people prioritize their health relative to other life domains, because this will influence decisions to respond to symptoms or adopt treatments (Harvey, 2013); in other words, individuals likely consider their own values when performing a cost-benefit analysis (e.g., concern for medication side effects or decreased social activities outweighing medication need). Thus far in the literature, this potentially important factor has not been fully examined. Third, perceptions of threat or susceptibility are predictive of screenings and other self-care behaviors; however, higher perceived vulnerability, when combined with low self-efficacy or fear, shows weak associations with desired health behavior, and may even predict maladaptive responses, such as denial (Harvey, 2013).

Social circumstances are another important source of variability in illness behavior. Both objective and subjective measures of individuals' social environments, roles, and relationships have been associated with dimensions of illness behavior. For example, being divorced, living alone, and unemployment status are important demographic predictors of frequent attendance in primary care (Vedsted & Christensen, 2005). Additionally, childhood experiences with personal illness or a family member's illness (e.g., chronic or terminal disease, hospitalizations) are associated with increased symptom reporting (Hotopf, 2002; Levy et al. 2004). In a similar vein, parental modeling and solicitousness to their children's symptoms predicts more symptom reporting, sick days, and clinic attendance in their children throughout adulthood, particularly for gastrointestinal symptoms (Levy et al., 2004; Walker & Zeman, 1992; Whitehead et al., 1982). Moreover, family conflict or poor psychosocial functioning predicts non-adherence to diabetes treatment regimens among children (Clayton, Stewart, Weibe, McConnel, Hughes, & White, 2013) as well as across a variety of other medical regimens and patient populations (DiMatteo, 2004). High family conflict is also associated with increased symptom reporting among adolescents (Horwitz & Neiderhiser, 2011; Mechanic & Hansell, 1989) and health utilization disproportionate to medical findings (Lee et al., 2013). Interpersonal conflict in general can also increase adoption of the "sick role" (Parsons, 1951), sometimes as a personal strategy to reconcile the relationship, avoid social responsibilities, or to seek attention (Mechanic, 1978; 1995).

Taken together, these findings indicate that the general family environment, cues to action (e.g., family health events), the socialization of norms for responding to

symptoms, and the quality of one's personal relationships are all associated with illness behavior tendencies. Precisely how pervasive and enduring these relationships are, however, is not yet known. Lastly, given the complex nature of social networks, with regards to their measurement (objective versus subjective, structural versus functional measures), their functions (norms, social control, comparison, and support), their forms (tangible, informational, or emotional support), their composition (family, friends, acquaintances), and their effects (moderators of other psychosocial predictors, or more direct influences), the current dissertation study will seek to understand precisely where social support availability from two different sources (friends versus family members) belongs in the proposed conceptual model of the social processes in illness behavior development (see Figure 1.1).

Lifespan development and individual variability in illness behavior

Associations between psychosocial factors and illness behavior may vary across individuals, and there is evidence to suggest that these individual differences can be partly accounted for by developmental factors such as lifespan stages or transitions, age-related shifts in symptom or disease characteristics, and gender. First, in research comparing adolescents to older adults, important differences in the health belief-treatment adherence relationship have been reported. For example, perceived benefits minus barriers predict adolescents' adherence to diabetes regimens, whereas perceived susceptibility is more strongly associated with older adults' adherence levels (Bond, Aiken, & Somerville, 1992; Brownlee-Duffeck, Peterson, Simonds, Goldstein, Kilo, & Hoette, 1987). Because barriers are stronger predictors of preventive health behavior in

diabetes management (e.g. screenings), whereas perceived vulnerability is more predictive of clinic attendance for treatment, it is suggested that adolescents' and young adults' views of illness—in this case, type 1 diabetes—are more geared toward prevention as opposed to disease management (Harvey, 2013). One possible explanation for this shift in personal models of illness is the better objective physical health experienced by adolescents and young adults relative to older adults, who are more likely to experience chronic, co-morbid conditions (Bradley & Hughes, 2013). Alternatively, differences in emotional processes, resource conservation strategies, and the self-knowledge that occur with aging may partially account for the differential associations of health cognitions with health behaviors in younger versus older adults (Egan & Beaton, 1987; Petrie et al., 1996).

Apart from age differences in the influence or relevance of health beliefs to behavior, the nature of symptoms or medical conditions also changes across the lifespan. Older adults (i.e. people 65 years of age and older) show increased variability in disease and disability levels, multi-morbidity of chronic conditions, as well as changes in how drugs are metabolized relative to their younger counterparts, all of which complicate their medical care (Bradley & Hughes, 2013). Such need-based factors account for individual differences in both illness behaviors and the health cognitions that are relevant for these behaviors. For example, acute, persistent, highly visible, or otherwise stigmatizing symptoms (e.g., chest pain, eye infection, or a skin rash) are much more likely to get noticed and treated, whereas symptoms that are ambiguous, chronic, or cyclical in nature (e.g., stomachache, abdominal or pelvic pain) are less likely to result in formal treatment-

seeking or treatment adherence (Harvey, 2013). Adding further complexity, some health conditions show few or no symptoms (e.g., hypertension). Furthermore, condition severity and co-morbidity are associated with more frequent healthcare utilization; however, when coupled with emotions like health worry and self-blame, or with low self-efficacy, these same characteristics can lead individuals to ignore symptoms or medical advice (DiMatteo, 2004). Moreover, the degree of controllability is another important disease characteristic that may predict illness behavior outcomes. For some conditions that can't be easily controlled with treatment or medication (e.g., cancer, stroke), self-efficacy may not be a relevant belief for predicting symptom responses, and in fact, may prove maladaptive (Hagger & Orbell, 2003).

Lastly, gender differences in illness behavior have been widely reported, with women consistently reporting symptoms and seeking out formal medical care more readily and more frequently than men (Vedsted & Christensen, 2005). Many explanations for this finding have been proposed, although none has received conclusive empirical support. For example, women might seek care more frequently than men due to obstetric and gynecological issues (Egan & Beaton, 1987). In particular, women are more likely to suffer from chronic pelvic pain and co-morbid depression as compared to men (Poleshuck & Woods, 2014), with approximately 15 % of women between the ages of 18 and 50 years experiencing chronic pelvic pain (Mathias, Kuppermann, Liberman, Lipschutz, & Steege, 1996). Women also experience elevated distress related to vulvar disorders that are frequently "normalized" or stigmatized in medical practice (Poleshuck & Woods, 2014; Poleshuck et al., 2014). A recent qualitative study of women with

dyspareunia (a condition involving pain during sexual intercourse) also underscored the problem of women seeking and often failing to obtain medical legitimacy for gynecological symptoms and pain (Braksmajer, 2017). The prevalent under-treatment or misdiagnosis that results from this failed legitimization or "normalization" in healthcare contributes to an increasing number of unnecessary emergency room visits, diagnostic procedures, and treatments. Illness behavior tendencies may also be socialized by gender, such that in childhood, females may elicit more solicitous parental responses to their symptoms than males, and even perceive parental responses differently (Walker & Zeman, 1992). Together, the observed differences by developmental stage, disease characteristics, and gender, suggest both within- and between-person heterogeneity in illness behavior patterns across late adulthood. The current study will seek to explain this heterogeneity by examining gender as another predictor of illness behavior outcomes.

Illness behavior scales and measurement

Extant measures of illness behavior have broken the construct down into several components. For example, the Illness Behavior Questionnaire (IBQ; Pilowsky & Spence, 1976) consists of the following subscales: hypochondriasis (i.e. anxious concern about health), symptom preoccupation, psychological versus somatic perceptions of illness, affective inhibition (i.e. inhibited expression of negative affect), affective disturbance (i.e. excessive anxiety), denial of life stresses, and irritability (i.e. interpersonal anger) (Keefe, Crisson, Maltbie, Bradley, & Gil, 1986). Interestingly, these dimensions include both cognitive and emotional coping processes that have also been incorporated into some of the existing models of health threat behavior (e.g., Protection Motivation

Theory/PMT; Rogers, 1975; Self-Regulatory Model/SRM; Leventhal & Cameron, 1987). Furthermore, Egan and Beaton (1987) factor-analyzed items from the Symptom Response Questionnaire (SRQ; 1987), in which participants indicated their responses to 13 standardized symptoms, and found evidence for three illness behavior factors: "self-help" (e.g., staying home from work or other sick role behaviors), "professional help" (e.g., health service utilization), and "think/talk (obsess)" (e.g., seeking information or support, causal attributions). Furthermore, illness behavior has been described as occurring in the following phases: initial symptom recognition or labeling, cost-benefit analyses, and treatment seeking (Shannon, 1977). Other self-report illness behavior questionnaires include the following: the Illness Cognition Questionnaire (ICQ: Evers, Kraaimaat, van Lankveld, Jongen, Jacobs, & Bijlsma, 2001), assessing specific thoughts of acceptance and helplessness in chronic illness; the Scale for the Assessment of Illness Behavior (SAIB: Rief, Ihle, & Pilger, 2003) with items on behavioral tendencies to express symptoms, monitor them, verify diagnoses or seek treatment; the Brief Illness Perception Questionnaire (Brief IPQ; Broadbent, Petrie, Main, & Weinman, 2006), measuring cognitions about illness; the Illness Attitudes Scale (IAS: Sirri, Grandi, & Fava, 2008), indexing hypochondriacal fears or beliefs; and the Illness Cognitions Scale (ICS: Berk et al., 2012) to assess investment in the sick role (c.f, Sirri, Fava, & Sonino, 2013 for a comprehensive theoretical review).

Many of these scales, however, have been primarily used to assess the "abnormal" illness behaviors of clinic-based patient samples (e.g., Berk et al., 2012; Egan & Beaton, 1987; Keefe et al., 1986; Pilowsky & Spence, 1976), ignoring the equally important

group of non-patients or non-utilizers of healthcare. One noteworthy exception is the Medical Maximizer-Minimizer Scale (Scherer, Caverly, Burke, Zikmund-Fisher, Kullgren, Steinley…& Fagerlin, 2016), which was developed to measure individual differences in people's treatment preferences in medical decision-making scenarios, ranging from preferring a "wait and see" approach to preferring aggressive treatments for minor physical symptoms (Scherer et al., 2016). However, this scale indexes individual preferences for medical treatment, which is a somewhat narrower construct than illness behavior, which encompasses individual differences in responding across the entire illness process (i.e. from initial symptom detection to treatment seeking). Because illness behavior describes not only how people respond to their symptoms, but also what they choose *not* to do, both utilizers and non-utilizers must be targeted for optimizing health care delivery. Lastly, most research to date has focused on examining single illness behavior aspects at a time, as opposed to evaluating a unified, multi-indicator construct of individuals' illness behavior tendencies (Sirri, Fava, & Sonino, 2013).

Thus, the current dissertation study includes measures of somatic complaints, frequency of over-the-counter medication use (to index a higher somatic orientation toward illness as opposed to psychological), pain-related disability, and perceived illness complications (i.e. difference between self-reported and physician-rated disability levels across a variety of medical conditions), to be aggregated into a latent illness behavior factor using multivariate statistical techniques. Because high values on these measures reflect the more responsive end of the illness behavior continuum, and would be expected to predict over-utilization of medical services, the emphasis for the current project will be

on the population-based illness behavior correlates of over-utilization as opposed to under-utilization. Study 1, however, will also evaluate convergence with self-reported primary (outpatient) healthcare utilization, which was directly measured in one representative sample of older adults.

Purpose and Research Aims

The primary purpose of this proposal is to examine the construct validity, longitudinal development, and psychosocial predictors of illness behavior (e.g., symptom reporting, pain-related disability, medication use, and perceived illness complications) with special emphasis on social measures; and to examine these relationships across a distinct developmental transition: from early- to mid- adulthood (a period of peak physical health and low service utilization; National Health Interview Survey; 2012) to late adulthood (a period of disproportionately heightened healthcare utilization; Population Reference Bureau, 2013). Therefore, this proposal will include two parts: The first will assess the longitudinal measurement and construct validity of a latent illness behavior construct, as well as the relative prediction of social support availability and other intrinsic factors (e.g., gender, comorbidity) on illness behavior trajectories from mid- to late- adulthood. The second study will examine the relation of psychosocial factors with illness behavior patterns and functional decline in older adulthood, as well as the long-term associations of older adults' illness behavior patterns with functional status. The dissertation study analyzes data from two population-based samples of aging adults: Swedish twin pairs from the Swedish Adoption/Twin Study of Aging (SATSA; Finkel & Pedersen, 2004; Pedersen et al., 2013), and opposite-sex, Swedish twin pairs ages 70 and

older from the Sex Differences in Health and Aging, or "GENDER" study (Gold, Malmberg, McClearn, Pedersen, & Berg, 2002).

Aims & Research Questions

Aim 1. Examine the factorial and construct validity of a latent, multi-indicator measure of illness behavior across time in two prospective, longitudinal samples.

Research Question 1a. Do the four observed indicators in SATSA (or three indicators in the GENDER study) load adequately onto a latent factor of illness behavior?

Research Question 1b. Can strong factorial measurement invariance in a latent illness behavior factor be established across time, in addition to its discriminant and convergent validity?

Aim 2. Examine the relative predictive value of social support availability from friends and family members on illness behavior trajectories across late adulthood.

Research Question 2a. What is the intra-individual, age-related pattern of change in illness behavior across four time-points from early- to late- adulthood (SATSA sample)?

Research Question 2b. What is the direct relationship between perceived support availability and illness behavior levels and change?

Aim 3. Test the prospective associations of social support availability from friends and family members with illness behavior trajectories and older adults' functional decline during the transition into late adulthood, as well as potential mediation through illness behavior.

Research Question 3a. What is the age-related pattern of change in functional status in the Swedish Adoption/Twin Study of Aging (SATSA)?

Research Question 3b. What are the direct relationships of social support availability measures with functional status (both levels and change)? Specifically, will the perceived availability of social support from friends versus family members predict older adults' functional outcomes (i.e. difficulties performing basic activities of daily living)?

Research Question 3c. To what extent are illness behavior levels and change in older adulthood associated with improved functional status outcomes over time?

Research Question 3d. Do illness behavior trends mediate the relationship between social support availability and functional status?

Original Study Descriptions

SATSA

The Swedish Adoption/Twin Study of Aging (SATSA; Pedersen, McClearn, Plomin, Nesselroade, Berg, & DeFaire, 1991; Finkel & Pedersen, 2004) is a longitudinal twin study drawn from the population-based Swedish Twin Registry that is maintained at the Karolinska Institute in Stockholm. SATSA began in 1984 and has focused on the genetic and environmental sources of individual differences in outcomes like health, personality, and cognition in aging. A subset of identical (i.e. monozygotic) and same-sex fraternal (i.e. dizygotic) twin pairs was recruited, some of whom had been separated before the age of 10 years and were raised apart (n = 346 pairs), and a matched sample of twins who were raised together (n = 404 pairs). Twins were matched with respect to age,

gender, and birth county. This created a natural adoption design as well as a twin design. Across 30 years of data collection between 1984 and 2014, nine mail-out questionnaires (Qs: 1984-2014) were sent, each spaced approximately three years apart, and ten in-person testing sessions (IPTs: 1986-2014) of twins older than 50 years-old, each spanning two years, were conducted. At the baseline questionnaire (Q1) in 1984, 2,018 twins (758 complete pairs, 502 single-responders; M_{age} = 60.14 years, *SD* = 14.02; 58 % female) out of an eligible 2,845 surviving twins in the registry had responded. All participants were Caucasian, and the average reported level of educational attainment was eight years (*SD* = 2.38; range = 6-16; Pedersen et al., 2013). Across all waves, longitudinal attrition in the sample was low, with approximately two-thirds of the sample completing three or more assessments. Across the Qs, up to 2,209 SATSA twins (ages 26 to 102 years) completed at least one questionnaire wave, and 65% completed three or more questionnaires. The current proposal includes data from up to six of the mail-out questionnaire waves, beginning with the second survey assessment (Q2) in 1987 (*N* = 1,310 twins from 915 pairs; ages 29 to 96 years; M_{age} = 60.51 years, *SD* = 13.34; 57 % female), with follow-up through the 2010 survey (Q7: M_{age} = 73.33 years, *SD* = 9.75).

GENDER

The GENDER study, with twins likewise drawn from the larger Swedish Twin Registry, sought to examine the genetic and environmental etiologies of emerging sex differences in morbidity and mortality among several cohorts of older adults. Participants from the baseline questionnaire assessment (Q1) were 605, opposite-sex (i.e. fraternal), Swedish twin pairs (*n*=1,210 individuals), ages 69 through 88 years, who were born

between 1906 and 1925 (Gold, Malmberg, McClearn, Pedersen, & Berg, 2002). There were five waves of data collection conducted between 1994 and 2007, which included two surveys and three intervening in-person testing assessments. Apart from demographics, the surveys included measures of health, health-related behavior, attitudes, and social factors (e.g., support availability), whereas the in-person testing sessions evaluated psychosocial factors (e.g., social support, network size, network quality) as well as health and physical functioning (both subjective and objective, as evaluated by a district nurse). The current proposal will include data from the two questionnaire waves in 1994 and in 2007, respectively (Q1: n=605 twin pairs, or 1,210 individuals; M_{age} = 74.43 years; SD = 4.28; 50 % female; and Q2: n= 279 twin pairs (77 complete and 202 incomplete), or 356 individuals; M_{age} = 85.58 years, SD = 2.87; 53.7 % female). The in-person testing sessions were excluded from the current analyses due to a lack of overlapping measures on key variables of interest. Across the five Q and IPT waves, 41 % of participants completed two waves, and 31 % completed three or more waves.

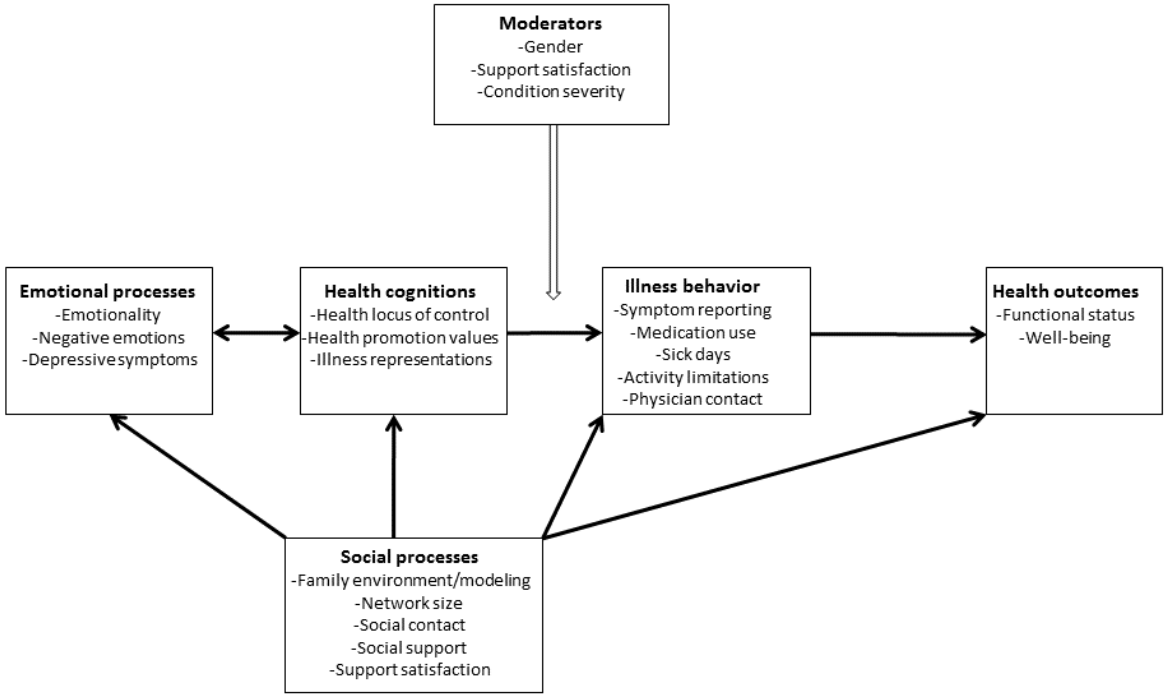

Figure 1.1. The proposed, conceptual Social Processes in Illness Behavior (SPIB) Model.

Chapter Two:

Illness Behavior Development and Change from Mid to Late Adulthood: The Predictive Role of Social Support Availability from Family and Friends

Introduction

The construct of illness behavior, first proposed by David Mechanic (1962/78), captures variability in people's perceptual, evaluative, and behavioral responses to physical symptoms that may signify illness. Illness behaviors predict a variety of health outcomes, including timely diagnosis and treatment for serious conditions like cancer (van Osch, Lechner, Reubsaet, de Nooijer, & de Vries, 2007), pain-related distress (Harkins, Price, & Braith, 1989), and return to work and daily functioning following acute health events, such as myocardial infarction (Broadbent, Ellis, Thomas, Gamble, & Petrie, 2009). Thus, illness behavior remains an important target of research on behavioral pathways to health and well-being. Since the inception of the study of illness behavior within medical sociology, a variety of scales have been developed to evaluate its cognitive, emotional, and behavioral dimensions. For example, the revised Illness Perception Questionnaire (IPQ-R; Moss-Morris, Weinman, Petrie, Horne, Cameron, & Buick, 2002) assesses several dimensions of illness representations based on Leventhal's Self-Regulatory Model of illness (e.g., identity, cause, timeline, consequences) and others (e.g., illness coherence, personal control, and treatment control), and it has been validated in a variety of clinical (Broadbent, Petrie, Main, & Weinman, 2006) and non-clinical (Figueiras & Alves, 2007) samples. The Behavioural Responses to Illness Questionnaire (BRIQ; Spence, Moss-Morris, & Chalder, 2005), rather than focusing on illness

cognitions, was developed to index behavioral responses to acute illness. These responses were characterized into four subscales of all-or-nothing behavior (i.e. powering through illness or not slowing down), limiting behavior (i.e. avoiding typical activities), practical support seeking (i.e. relying on help from others), and emotional support seeking (i.e. complaining to others for sympathy). Moreover, Rief and colleagues (2003) developed the Scale for the Assessment of Illness Behavior (SAIB; Rief, Ihle, & Pilger, 2003), which assesses behavioral tendencies to verify diagnoses, express symptoms, take medications, express illness consequences, and engage in scanning or monitoring.

Although the factor structure and psychometric properties of these scales have been subjected to close examination in some cases (Berk et al., 2012; Egan & Beaton, 1987; Hagger & Orbell, 2005; Moss-Morris, Weinman, Petrie, Horne, Cameron, & Buick, 2002), much of this research has employed cross-sectional methods and has assessed illness behavior within specific health domains (e.g., rheumatoid arthritis and multiple sclerosis; Evers, Kraaimaat, van Lankveld, Jongen, Jacobs, & Bijlsma, 2001; Type 1 diabetes; Lawson, Bundy, Lyne, & Harvey, 2004; chronic pain and psychiatry; Prior & Bond, 2008; acute infection; Spence, Moss-Morris, & Chalder, 2005). Furthermore, most illness behavior measures have been validated among clinic-based patient samples, which are biased toward the population of "utilizers" or individuals who have already sought treatment. Although the use of such clinic-based samples is useful for the development of targeted interventions for improving patient recovery within specific disease contexts and for potentially addressing issues of over-utilization in those contexts, this approach also omits adults from the general population who experience the

same symptoms as the "utilizers", but who tend to normalize them or even fail to notice them to begin with. In other words, little is known about the generalizability of an illness behavior measure in population-based adult samples, which should include both utilizers and non-utilizers of healthcare. In one recent exception, Scherer and colleagues (Scherer, Caverly, Burke, Zikmund-Fisher, Kullgren, Steinley…& Fagerlin, 2016) developed and validated the Medical Maximizer-Minimizer Scale in a large sample of adult participants on Amazon's Mechanical Turk (Mturk), as well as a separate sample of healthy adult males recruited from a hospital clinic (i.e. hospital guests and staff who had never been diagnosed with prostate cancer). The Medical Maximizer-Minimizer Scale measures individual differences in people's general approach to medicine across many health decision contexts, with some individuals preferring a watchful waiting approach across a range of symptoms (i.e. minimizers), and others preferring to seek out aggressive treatment for even minor physical complaints (i.e. maximizers; Scherer et al., 2016). The scale showed strong test-retest reliability and discriminant validity through its weak associations with theoretically similar constructs, such as hypochondriasis, medical distrust, and self-reported health status. In the clinic sample, the scale also showed convergent validity with participants' self-reported healthcare utilization and preferences for procedures of limited value in hypothetical medical scenarios (e.g., PSA testing for prostate cancer, and MRI screening for a brain tumor). Importantly, the authors also confirmed the fit of a single-factor structure for this scale, supporting the measurement of medical minimizing-maximizing preferences as a unifying construct, with a moderate degree of rank-order stability (Scherer et al., 2016). This scale represents an important

advancement in the measurement of individual differences in people's preferences for (or against) medical intervention. However, it does not fully capture individual differences in the detection of symptoms, the self-care strategies that people use to manage these symptoms apart from formal help seeking (e.g., over-the-counter medications, and limiting one's daily activities), nor perceptions of illness, all of which constitute illness behavior and also underlie issues of medical over- and underutilization.

No studies to date have applied longitudinal data to investigate the stability of illness behavior as a unifying, multi-indicator construct across time or development. Consequently, most of the extant epidemiological datasets of health and aging do not include previously established illness behavior scales in their assessments, although dimensions of the construct are often studied in isolation (e.g., symptom reports; Levy et al., 2004; Levy, 2011; Michel, 2006; healthcare utilization; Clayton, Stewart, Weibe, McConnel, Hughes, & White, 2013; Al-Windi, Dag, & Kurt, 2002; and illness perceptions; Broadbent, Ellis, Thomas, Gamble, & Petrie, 2009; Rutter & Rutter, 2007). Because illness behavior not only encompasses utilization decisions but also the lay responses that occur prior to professional help-seeking, trends in a multi-indicator construct of illness behavior across different lifespan periods, and variability in these trends, might inform prevention efforts aimed at improving individual health and healthcare efficiency through appropriate care-seeking.

With advances in structural equation modeling methods—namely, techniques like confirmatory factor analysis (CFA)—researchers can test explicit hypotheses about the relation of these disparate measures to an underlying latent factor. CFA is a powerful

approach for developing and validating measures, examining construct validity, and even evaluating the extent to which the same construct is being measured across time (i.e. factorial invariance) (Jackson, Gillaspy, Jr., & Purc-Stephenson, 2009)—a key assumption of growth modeling techniques (Ferrer, Balluerka, &Widaman, 2008). This latter application of CFA methods is an important first step in evaluating lifespan changes in psychological attributes or behaviors over time, because it ensures that estimates of change reflect true quantitative growth rather than different meanings of a construct over age, other forms of response shift (e.g., changes in individuals' values or frames of reference; Schwartz & Sprangers, 1999; Schwartz, 2010), or measurement error.

Although the rank-order stability of illness behavior aspects has received some attention, particularly within daily diary studies (e.g., Kasl, Gore, & Cobb, 1975; Michel, 2006; van Wijk, Huisman, & Kolk, 1999), the longitudinal follow-up is typically over a relatively short period of weeks or months. Regarding rank-order stability, an early study by Mechanic assessed the correlations of health and illness behaviors across two time-points spanning 16 years from childhood to adulthood. Results suggested little or no rank-order stability in illness behaviors like seeing a physician or communicating to others about symptoms, whereas stoicism, or the denial of pain, showed relative stability from childhood to adulthood (Mechanic, 1979). Another study by Stoller and colleagues applied daily diary methods to evaluate the variability in symptom experiences and self-care strategies (e.g., ignoring symptoms, using over-the-counter medications or home remedies) of community-dwelling older adults across 21 consecutive days. Results suggested significant within-person variability over time in the

self-care strategies used to manage the same symptoms, and the majority sought to manage their symptoms through medications and remedies rather than engaging in preventative lifestyle changes or seeking more information (Stoller, Forster, & Portugal, 1993). However, the long-term, mean-level stability of illness behavior as a latent construct across a key lifespan developmental transition—from mid- to late-adulthood—has yet to be explored.

With regards to healthcare utilization, older adults have fairly consistent physician utilization patterns over time relative to other age groups, and the proportion of consistently high utilizers is very low in comparison to other utilization categories, such as consistently low or inconsistently high utilization (Stump et al., 1995). Other related work has stratified individuals according to their utilization percentiles (e.g., the 25th percentile or less, greater than the 75th percentile, etc.) and found similarly stable patterns (McCall & Wai, 1983). Such findings help to counter the myth that older adults become hypochondriacs with age (Erber & Schuzman, 2014). Rather, these findings might suggest adaptively responding to aging-related increases in disease incidence and comorbidity, or conversely, they might reflect normalizing symptoms as signs of aging rather than of underlying disease processes.

Importantly, however, this research has not sought to explain such consistency, or lack thereof. Moreover, some researchers suggest that count-based or dichotomized units of analysis for health utilization outcomes may explain why secondary analyses have failed to find large effects of psychosocial predictors relative to need-based factors like health status (objective and self-rated) and prior utilization (Mechanic et al., 1992;

Wolinsky, 1994). For studies seeking to evaluate psychosocial predictors of over- or under-utilization, these individual-level estimates are posited to be strongest when examining utilization that is under the patient's control or patient-driven, such as initial physician contact or emergency room visits. Because hospitalizations and other referral services are comparatively under the control of a physician, patients' physical need (often as judged by a health professional) should be more predictive of this utilization type (Hansell, Sherman, & Mechanic, 1991). In support of this hypothesis, Hansell and colleagues (1991) found that higher body awareness, or the tendency to monitor one's bodily sensations, among older adults enrolled in a Health Maintenance Organization (HMO), predicted more emergency room visits and initial physician contacts, but was not significantly related to inpatient visits or physician referrals. Therefore, although the patient-driven nature of health service utilization was not directly assessed in this study, measures of self-reported contact with a primary care (outpatient) physician and frequency of sick days were included to evaluate the convergent validity of a latent illness behavior factor, whereas hospitalizations and contact with a district nurse were included as tests of discriminant validity. Although sick days is an important dimension of illness behavior, it was not included as an indicator in the present study. This was because many of the participants were of retirement age, and sick days have a different meaning pre- versus post-retirement. Furthermore, this variable's distributional properties were not ideal for inclusion in confirmatory factor analyses. In a similar vein, use of over-the-counter medications was included as an indicator of illness behavior, because

this type of symptom response often precedes formal medical help-seeking decisions, and is also under individual's volitional control.

Moreover, research has consistently shown that illness behaviors and medical help-seeking decisions are often based on factors independent of biological risk. For example, apart from the influences of patient demographics and disease characteristics, psychosocial factors such as beliefs (e.g., low perceived control, low treatment efficacy), emotional processes (e.g., anxiety, depression), and low-quality social support act to amplify any given physical symptom and predict more frequent use of medical services (Al-Windi, Dag, & Kurt, 2002; Barsky, 1988; Haug, Musil, Warner, & Morris, 1997). Indeed, treatment efficacy, or confidence in the medical system's treatment capabilities, is among the strongest cognitive predictors of seeking medical care (Lawson et al., 2004; Harvey, 2013), and among older adults, specific beliefs about medication have been associated with adherence above and beyond other clinical or demographic factors (Horne & Weinman, 1999). Perceptions of symptom severity and self-rated health are also strong determinants of illness behaviors such as providing an illness label for a given bodily change, self-care behaviors, like taking over-the-counter medications, and visiting the doctor (Haug et al., 1997). Apart from cognitions, emotional processes—namely, the experience of anxiety and other negative emotions—relate to higher levels of somatization, more frequent contact with medical services, and in some cases, non-adherence to medical treatment among older adults (Jerant, Chapman, Duberstein, Robbins, & Franks, 2011). However, the precise mechanisms and relative importance of these factors' associations with older adults' illness behaviors remain to be understood

and formally tested simultaneously, using a prospective, longitudinal design. Because illness behaviors are embedded within individuals' social networks, which serve as external cues for interpreting and responding to symptoms, this remains an important target for research in this area.

Social support and illness behavior

The experience of illness is a process that begins with the recognition and labeling of bodily sensations, and ends with a trajectory of treatment or recovery; thus, the various dimensions of illness behavior (perceptual, evaluative, behavioral) are likely to be predicted by many factors. For example, symptom reporting, symptom monitoring, and pain experiences have been associated with intrinsic factors (e.g., neuroticism, genetic sensitivity pathways; Charles, Gatz, Kato, & Pedersen, 2008), whereas behaviors like activity restriction, medication use, treatment non-adherence, and contact with the medical system are largely associated with fluctuations in the tangible help, advice, and emotional support received from one's social networks (Cameron et al., 1993; DiMatteo, 2004; Kasl, Gore, & Cobb, 1975).

In the transition to older adulthood, social networks tend to decrease in size and network members become increasingly involved in medical decision-making processes relative to early- and middle-adulthood (Cornwell, Schumm, Laumann, & Graber, 2009). Furthermore, aging-related shifts in self-regulation abilities (e.g., declines in certain executive functions, but enhancement in impulse control and emotion regulation; Carstensen & Hartel, 2006); increased variability in physical functioning (Buurman et al., 2011); and changes in personal models of illness (i.e. from a prevention-focused model to

a disease management model; Harvey & Lawson, 2008; Leventhal & Crouch, 1997), together, may render older adults more likely to rely on external motivators, such as family concerns, encouragement from others, and immediate situational demands or health threats, in their attempts to initiate health behavior change. In contrast, younger and middle-aged adults might be more likely to rely on internal motivators in their behavior change efforts, such as concerns about physical appearance or attaining personal goals (Carstensen & Hartel, 2006). Research suggests that social support and perceptions of belongingness from close relationships become especially important for outcomes of well-being (Ryan & Willits, 2007), morbidity, and mortality (Tomaka, Thompson, & Palacios, 2006) in late-life, although the type of support sought from family members versus friends and community members might differ. For example, friends are more likely to provide emotional support in late-life, whereas relatives are more likely to provide instrumental support (Antonucci, 1990). Furthermore, despite an increased risk for loneliness and social isolation among the oldest-old age groups (Cornwell et al., 2009), social psychological theories, such as the social convoy theory (Antonucci, Ajrouch, & Birditt, 2014; Kahn & Antonucci, 1980) and socio-emotional selectivity theory (Carstensen, 1992; Carstensen, 2006) posit a lot of stability in older adults' social networks regarding the individual members. This stability might be due—at least in part—to increased selection of close-knit relationships over superficial ones. Therefore, although the overall size of social networks might shrink during the transition into late adulthood, the quality of relationships in the network and the support they provide are

thought to be largely stable across the lifespan, and to be more important for health and well-being outcomes than the structural factors of network size or composition.

With regards to social influences on long-term health behavior change and maintenance, this stability in social relationships, combined with older adults' increasingly consistent daily routines (Martin & Park, 2003), can serve as important resources for reducing maladaptive behaviors in late-life, and as a positive reinforcer of maintenance efforts over longer periods of time (Carstensen & Hartel, 2006). However, this situational stability can be a double-edged sword, such that in situations of stable but strained family relationships, the reduced autonomy and increased reliance on these relationships for medical decision-making or caregiving, combined with the negative social control that may occur in this context (Rook, 2014), might result in conflict and interfere with older adults' health and behavior. For example, Stephens and colleagues found that among a community sample of older adults (ages 55 to 70 years), those who were entering retirement with either restricted or family-dependent networks (as compared to networks with a wider range of contacts and connections beyond the family) experienced poorer mental health outcomes (Stephens, Alpass, Towers, & Stevenson, 2011). Still, there is reason to believe that older adults who have more active and fulfilling social lives—and who perceive more varied sources of available support—might show reduced illness behaviors like somatization and perceptions of illness-related impairments over time. Furthermore, this association might operate at a variety of levels, from attentional and perceptual processes to exerting a more direct relationship with illness behavior. For example, Pennebaker and colleagues' symptom perception theory

(Pennebaker & Brittingham, 1982), posits that the more external stimuli are present within people's surroundings, the less likely they are to turn their attention inward and to attend to bodily sensations; and consequently, the less likely they will be to notice or report physical symptoms. Apart from enhancing the novelty in our surroundings and focusing our attention externally, supportive relationships also provide important norms and reinforcements for health behaviors, particularly when these relationships are valued (Gallant, 2013). Thus, the current study focused on examining availability of support from friends and family members as key predictors of illness behavior trajectories across the transition from early- to late- adulthood.

Aims & Hypotheses

The first aim of this study was to evaluate the factorial invariance and construct validity of illness behavior as a single latent outcome by applying CFA methods in two population-based samples of adults. A unidimensional, one-factor model of illness behavior was specified a priori with the same observed indicators at each assessment: four indicators (i.e. somatic complaints, over-the-counter medication use, activity limitations due to pain, and a composite of perceived illness complications) were included across four questionnaire waves in SATSA (1987-2004); and three indicators (i.e. somatic complaints, over-the-counter medication use, and perceived illness complications) were included across two questionnaire waves in GENDER (1994 and 2007). A single-factor solution was tested based on the construct originally proposed by Mechanic (1962/77), as well as previous findings from psychometric studies in support of a unidimensional illness behavior scale (e.g., Scherer et al., 2016, Sirri, Fava, & Sonino,

2013; Wyke, Adamson, Dixon, & Hunt, 2013). Four alternative factor-analytic models of longitudinal measurement invariance were tested using the approach advocated by Widaman and colleagues (Widaman, Ferrer, & Conger, 2010) and adapted from Anderson and Gerbing's two-step approach (1988).

The first of these models evaluated configural invariance, or the extent to which the overall pattern—but not the values—of the indicators and their factor loadings holds across time. Subsequent models were then tested which applied stepwise equality constraints to the factor loadings (M2), intercepts (M3), and residual variances (M4) of the same indicators across time to evaluate weak, strong, and strict factorial invariance, respectively. At minimum, establishing strong factorial invariance is necessary to ensure that the same latent construct is being modeled on the same metric over time and to justify subsequent growth modeling of the latent factor (Ferrer et al., 2008; Widaman et al., 2010). A single-factor structure of illness behavior was hypothesized to be invariant across time in both samples. More specifically, support for strong factorial invariance was expected. Furthermore, the discriminant validity of the illness behavior index was expected to be supported by weak-to-moderate zero-order correlations between the factors and theoretically related—but distinct—constructs, including comorbidity, self-rated health, and health promotion values (i.e. GENDER only; two items on participants' beliefs about the importance of getting regular physician exams and taking their medications). Finally, in the GENDER sample only, the convergent validity of the illness behavior factor was expected through significant associations of the factor with self-reported sick days and primary healthcare utilization.

The second aim of this study was to evaluate the pattern of age-related, intra-individual change in illness behavior across the transition from mid- to late-adulthood (SATSA only), as well as the relative predictive value of social support availability from friends versus family members on illness behavior levels and change. Hypotheses included that there would be systematic linear change in illness behavior over age, as well as significant between-person variability in both illness behavior levels and change. Additionally, higher availability of support from both friends and family members were hypothesized to predict reduced levels of illness behavior and a reduced rate of change, given the relative importance of both sources of social support for promoting behavior change and maintenance during the late-life transition.

Methods

Participants

SATSA sample. SATSA is a longitudinal twin study examining the genetic and environmental sources of individual differences in health, personality, and cognitive outcomes in aging. Twin pairs were recruited from the population-based Swedish Twin Registry, some of whom had been separated before the age of 10 years and raised apart, and a sample of twins who were raised together and matched on age, gender, and birth county (Pedersen, McClearn, Plomin, Nesselroade, Berg, & DeFaire, 1991). Across 30 years of data collection, 9 mail-out questionnaires and 10 in-person testing sessions were conducted, each about three years apart. At the baseline questionnaire (Q1) in 1984, 2,018 twins (758 complete pairs, 502 single-responders) out of an eligible 2,845 surviving twins in the registry had responded. Across all assessments, about two-thirds

(67 %) of the sample completed at least three measurement waves. The current study included data from four questionnaire waves (Q2-Q5) from 1987 to 2004, with the Q2 survey designated as the baseline assessment (N =1,637).

For the first set of confirmatory factor analyses, participants were included if they provided any illness behavior data across the four questionnaire waves. From a base sample of 1,898 SATSA twins who provided any predictor and outcome data across the four questionnaires, twelve participants had no data on any of the illness behavior indicators. Thus, participants in the final analysis sample included 1,886 twins (from 1,223 pairs) ages 29 to 102 years at the baseline (1987; Q2) questionnaire (M_{age} baseline = 62.32 years, SD = 13.69, 59 % female) from the Swedish Adoption/Twin Study of Aging (SATSA; Pedersen et al., 1991; Pedersen et al., 2013). Of this final analysis sample, 90 % of participants (n = 1,705) provided at least one complete wave of data on the illness behavior indicators.

For the second set of latent growth curve analyses, the base sample included 1,626 SATSA participants (M_{age} = 62.32 years, SD = 13.69, 59 % female) who provided any data on the baseline (Q2) predictors plus covariates. Of these, 1,362 participants (940 twin pairs, M_{age} baseline = 60.69 years, SD = 13.36, 57.8 % female) provided complete data on the Q2 predictors. However, 48 participants were missing outcome data on the illness behavior factor scores. Participants who had complete data on all predictors and covariates at baseline and who also provided at least one wave of illness behavior data across the four questionnaire assessments were included in the analysis. Thus, for the latent growth curve models, the final analysis sample included 1,314 SATSA twins from

within 910 pairs (404 complete, 506 single-responders), ages 29 to 95.88 years (M_{age} = 60.26 years, *SD* = 13.20, 57.46 % female) at the baseline (1987) survey. Illness behavior data were available across four questionnaire waves (Q2-Q5: 1987-2004), and predictors were included from the baseline (Q2) survey. Seventy-one percent of participants provided at least three waves of illness behavior data across the four questionnaires.

GENDER sample. For the first set of analyses—the CFA models—participants in the analysis sample were 1,208 twins (nested within 605 unlike-sex pairs; M_{age} = 74.43, *SD* = 4.29, 50.1 % female) from the Sex Differences in Health and Aging Study, or GENDER (Gold et al., 2002). The base sample of GENDER included 1,210 opposite-sex twins (605 complete pairs) recruited from the population-based Swedish Twin Registry; two participants, however, were missing data on illness behavior indicators at Q1. Participants who provided any data on illness behavior measures across the two questionnaire waves in 1994 (Q1) and 2007 (Q2) were included in the analyses. Approximately 29.5 % of the sample completed both questionnaire assessments.

Measures

SATSA Measures

Illness behavior. Indicators from SATSA included somatic complaints, frequency of over-the-counter medication use, the presence of activity limitations due to pain, and perceived illness complications (subjective ratings adjusted for physician panel ratings). These measures were chosen because they are posited to capture people's behavioral, affective, or cognitive responses to everyday symptoms of illness—responses considered to be relatively under individuals' volitional control and decision-making,

rather than being motivated by other factors (e.g., the instructions of a health provider, the life-threatening nature of a symptom, or a family member seeking medical care on a patient's behalf).

Somatic complaints (past week). The psychomotor retardation or somatic subscale of the Center for Epidemiological Studies Depression scale (CES-D; Radloff, 1977) was used to index somatic complaints across the four questionnaires (1987-2004). This subscale was a simple composite of seven items, for which participants reported how frequently they experienced a series of common physical complaints (e.g., restless sleep, poor appetite, and fatigue) during the past week prior to testing, each on a four-point scale (from 0 = *Never*, to 3 = *Always/Almost Always*). These symptoms are not associated with any specific, underlying disease or pathology, but rather are more commonly experienced by the general population and often associated with depressive symptomatology. Possible scores ranged from 0 to 21, with higher scores reflecting higher somatization. In the SATSA project, Cronbach's alpha for the CES-D composite was .89 at each wave (Fiske et al., 2003), and measurement invariance has been established across participant age groups and validated across different countries (Gatz, Johansson, Pedersen, Berg, & Reynolds, 1993). If participants did not respond to one or more of the seven items, their composite scores were set to missing.

Over-the-counter medication use (past month). Medication use was indexed by a simple composite of nine dichotomous items (0 = *No*; 1= *Yes*) on participants' use of non-prescription medications during the previous month. Medication items included cough medicine/nose drops, skin ointments, laxatives, hemorrhoid medicine, iron

medicine, vitamins/other strengthening medicine, over-the-counter analgesics, over-the-counter gastritis medicine, and herbal or alternative medicines. If participants were missing more than one of the medication checklist items, their composite scores were set to missing. Otherwise, participants' composite scores were prorated based on the number of missing items (0 or 1 at most), and possible scores ranged from 0 to 9. Because less than 2 % of participants endorsed taking more than five medications at any wave, the composite was collapsed into six response categories for subsequent analyses, with possible scores ranging from 0 (*no medication use*) to 5 (*use of five or more medications*).

Pain-related disability. Self-reported activity limitations due to pain was assessed using a simple composite of three dichotomous items asking participants to report the presence of neck, back, or shoulder pain so severe it prevented them from performing their daily tasks or activities (0= *No*; 1 = *Yes*). If participants responded to at least one of the items, they received a score. Because of the composite's positive skew and low number of response categories, it was treated as an ordinal indicator in subsequent confirmatory factor analyses.

Perceived illness complications. This was included as an observed indicator given the importance of illness perceptions of severity and consequences in theoretical models of illness coping and self-regulation (Leventhal, Meyer, & Nerenz, 1980; Leventhal, Leventhal, & Contrada, 1998). Perceived illness disability was a composite of difference scores, calculated from subtracting a physician panel's objective ratings of expected disability for each of 35 medical conditions (each on a 3-point scale from 1= *Little or no disability*; to 3 = *Severe disability*; Fiske, Gatz, & Pedersen, 2003) from participants'

self-reports of how much each of the same (endorsed) medical conditions complicated their daily lives (on a parallel 3-point scale: 1 = *Not at all*; 3 = *A lot*). At least five out of seven physicians on the panel agreed on their disability ratings for most of the items (Fiske et al., 2003). Possible scores ranged from - 9 to 13 across all four of the measurement occasions, and the composite was also normally distributed. Positive composite scores reflected higher perceived illness complications relative to what the healthcare providers expected, whereas negative composite scores reflected less perceived disability than would be expected from the objective ratings. A score of zero on the composite reflected "accuracy"—or rather, it reflected agreement—in perceptions of illness complications. Participants also received a score of zero if they: a) completed the illness checklist but did not endorse any medical conditions (i.e. they did not provide any illness complication ratings), or b) responded to an illness item in the checklist indicating they did not have that condition, but then responded to the associated complications item for the condition (see appendix for the list of health conditions included in the composite). Approximately 40 participants received a composite score of 0 based on this response pattern. If one or more items from the illness checklist were missing, but a participant responded to the associated complications item, or conversely, if they reported having a condition but did not respond to the complications item, the composite score was set to missing (n = 10 participants at Q2).

Age. Participant age (in years) was included as the time predictor in analyses of age-related change in illness behavior. Participant age data was available from all four of

the measurement occasions and grand-mean centered at age 60 years (i.e. the mean age at Q2).

Social support availability (friends and family). Perceptions regarding the availability of social support from family and from friends or acquaintances were included as separate measures, or two standardized composites, created from four and eight items, respectively (see Appendix 1 for the full list of items). These items were adapted from the Interview Schedule for Social Interaction (ISSI; Henderson, Duncan-Jones, & Byrne, 1980; Eklund, Bengtsson-Tops, & Lindstedt, 2007). Specifically, items were included from two of the adapted ISSI subscales—the availability of social integration subscale (e.g., having friends or family members who share the same interests, or who participants can meet and talk to) and the availability of attachment subscale (e.g., having friends or family who participants can share inner thoughts with, and who they can confide in); items from the other two subscales on the perceived adequacy of social integration and attachment were not included.

Covariates

Age at study entry (baseline). Because of the wide range of participant ages (from 29 to 102 years) at the baseline survey, the possible confounding of age at study entry was adjusted for. This age covariate was included from the Q2 (1987) survey and centered at 70 years. The majority (74 %) of participants were below the age of 70 when they completed the baseline questionnaire.

Illness comorbidity (baseline). Illness comorbidity was a simple composite adapted from the Cumulative Illness Rating Scale (CIRS; Linn, Linn, & Gurel, 1968).

Participants completed a checklist including 35 medical conditions, endorsing whether a diagnosis was present for each item (0= *No*; 1= *Yes*). All endorsed health conditions were subsequently grouped into categories reflecting the organ system affected (e.g., cardiovascular, musculoskeletal, respiratory, endocrine). In the original scale, the composite is calculated from the sum of the number of impaired organ systems (out of a possible 14) and weighted by physician ratings of severity of impairment (on a five-point scale from 1 = *None/No impairment to that organ/system* to 5 = *Extremely severe/Impairment is life-threatening*). In cases where multiple diseases are endorsed within a single organ system, only the most severe illness is rated for severity. This scale has demonstrated reliability (ICCs = .80 to .83; de Groot, Beckerman, Lankhorst, & Bouter, 2003; Miller et al., 1992; Rochon et al., 1996) and validity across a variety of clinical samples (e.g., older adults, Miller et al., 1992; victims of completed suicide; Conwell, Forbes, Cox, & Caine, 1993) and in different medical settings (e.g., primary care or family practice, Hudon, Fortin, & Vanasse, 2005; and acute hospitalization settings, Salvi et al., 2008). In SATSA, an adapted composite was calculated from a simple sum of impaired organ systems out of a possible 12 (actual scores ranged from 0 to 11). Corresponding physician severity (i.e. life-threatening) ratings were not available, however, so composite scores were not adjusted for severity. In the present study, the composite was also centered at one, such that the interpretation of the variable's effect on illness behavior was the change resulting from illness comorbidity as opposed to the change in illness behavior from endorsing any one medical condition. Eighty-six percent of participants reported having at least one affected bodily system at baseline.

Sex. Participants' self-reported sex was included from baseline and coded dichotomously (0 = *male*; 1 = *female*).

Socioeconomic status (baseline). Participants' objective socioeconomic status in adulthood was measured using six self-report items, which were standardized ($M = 0$; $SD = 1$) and summed into a composite ($M = 0.35$, $SD = 2.67$; range = - 8.67 to 6.00). Participants reported on their income, home ownership, whether they received a rent subsidy, and the number of cars they owned.

Marital status (baseline). Participants' self-reported marital status was included from the Q2 survey and coded dichotomously (0 = *never married/divorced/widowed*; 1 = *married*). Sixty-nine percent of the sample reported being married.

Demographic Variables

Comorbidity. The description of this measure is outlined in the previous section.

Self-rated health. Self-rated health was a standardized composite of four items on participants' general health status; health status now compared to 5 years ago; health status as compared to others; and the extent to which health prevents them from engaging in activities. Each item was coded on a three-point scale (1 to 3), and reverse-scored such that higher scores indicated better health status. The items were then standardized (weighted by the mean and standard deviation from Q1) and summed into a composite ($N = 1{,}288$; $M = 0.34$; $SD = 2.73$; range = -9.06 to 5.22).

GENDER Measures

Illness behavior. Illness behavior indicators included the somatic complaints subscale of the CES-D, a composite of over-the-counter medication use (past three

months), and a composite of perceived illness complications, which, again, was adjusted for physician panel ratings of the expected disability resulting from each endorsed medical condition. Items were included from the two questionnaire waves in 1994 and in 2007, respectively.

Somatic complaints. The somatic subscale of the Center for Epidemiological Studies Depression scale (CES-D; Radloff, 1977) was used to index somatic complaints across the two questionnaires (1994-2007). This subscale was a simple composite of seven items, for which participants reported how frequently they experienced a series of common physical complaints (e.g., restless sleep, poor appetite, and fatigue) during the past week prior to testing, each on a four-point scale (from 0 = *Never*, to 3 = *Always/Almost Always*). Possible scores ranged from 0 to 21, with higher scores reflecting higher somatization. Measurement invariance has been established across age groups and across different countries (Gatz, Johansson, Pedersen, Berg, & Reynolds, 1993). If participants did not respond to one or more of the seven items, their composite scores were set to missing.

Over-the-counter medication use (past three months). Medication use was indexed by a simple composite of thirteen dichotomous items (0 = *No*; 1= *Yes*) on participants' use of non-prescription medications during the previous three months. Medication items included cough medicine/nose drops, skin ointments, laxatives, hemorrhoid medicine, iron medicine, vitamins/other strengthening medicine, over-the-counter analgesics, over-the-counter gastritis medicine, and herbal or alternative medicines. If participants were missing more than two of the medication checklist items,

their composite scores were set to missing. Otherwise, participants' composite scores were prorated based on the number of missing items (0 to 2), and possible scores ranged from 0 to 9. The composite was subsequently collapsed into seven response categories for CFA analyses, with possible scores ranging from 0 (*no medication use*) to 6 (*use of six or more medications*).

Perceived illness complications. This indicator was a composite of difference scores, calculated from subtracting a physician panel's objective ratings of expected disability for each of 24 medical conditions (each on a 3-point scale from 1= *Little or no disability*; to 3 = *Severe disability*; Fiske, Gatz, & Pedersen, 2003) from participants' self-reports of how much each of the same (endorsed) medical conditions complicated their daily lives (on a parallel 3-point scale: 1 = *Not at all*; 3 = *A lot*). At least five out of seven physicians on the panel agreed on their disability ratings for most of the items (Fiske et al., 2003). Possible scores ranged from -6 to 8 across the two measurement occasions, and the composite was also normally distributed. Positive composite scores reflected higher perceived illness complications relative to what the healthcare providers expected, whereas negative composite scores reflected less perceived disability than would be expected from the objective ratings. A score of zero on the composite reflected "accuracy"—or rather, it reflected agreement between the participant and physicians—in perceptions of illness complications. Participants also received a score of zero if they: a) completed the illness checklist but did not endorse any medical conditions (i.e. they did not provide any illness complication ratings), or b) responded to an illness item in the checklist indicating they did not have that condition, but then responded to the associated

complications item for the condition (see appendix for the list of health conditions included in the composite). If one or more items from the illness checklist were missing, but a participant responded to the associated complications item, or conversely, if they reported having a condition but did not respond to the complications item, the composite score was set to missing.

Demographics

Self-rated health. One item from Q1 asked participants to indicate how they would rate their current general health status. Participants responded on a three-point scale (1 = *good*; 2 = *about average*; 3 = *bad*), such that higher scores reflected poorer health ratings (N = 1,204; M = 1.52; SD = 0.58; range = 1 to 3).

Comorbidity. A simple composite of the number of medical conditions endorsed from a dichotomous (0 = *No*; 1 = *Yes*) checklist of 33 medical condition items from the Q1 survey was used to index illness comorbidity (N = 1,206; M = 3.67; SD = 2.85; range = 0 to 19).

Health promotion values. Two items were selected from a larger 13-item scale (developed specifically for GENDER) assessing the extent to which participants believe in the importance of various health promotion behaviors for mind and body (e.g., to exercise regularly, take vitamins, and to have good eating habits). Each item was on a three-point rating scale from 1 (*Not at all important*) to 3 (*Very important*). The two items included from Q1 asked about the importance of "getting physical exams regularly" (N = 1,133; M = 1.29; SD = 0.69) and "taking your medicine" (N = 1,118; M = 1.73; SD = 0.52).

Health utilization. To evaluate the convergent and discriminant validity of the latent illness behavior factors, self-report measures of the frequency of sick days and healthcare utilization were included from the baseline questionnaire (Q1).

Sick days. Participants indicated how many days within the prior six months they had been so ill that they couldn't perform their usual chores. This item was scored on a five-point scale from *None* (0) to *4-6 months* (4) (N = 1,192; M = 0.34; SD = 0.85; range = 0 to 4).

Hospitalizations. Participants indicated how many days within the prior six months they had stayed in a hospital, convalescent home, or rest home. This item was scored on a five-point scale from *None* (0) to *4-6 months* (4) (N = 1,195; M = 0.23; SD = 0.64; range = 0 to 4).

Physician contact. Participants indicated if they had visited the doctor in the last six months. This item was scored dichotomously 0=*No*, 1=*Yes*, and if participants responded yes, they then indicated how many times they had contact (N = 1,176; M = 1.65; SD = 2.18; range = 0 to 21).

District nurse contact. Participants indicated if they had contact with a district nurse in the last six months. This item was scored dichotomously 0=*No*, 1=*Yes*, and if participants responded yes, they then indicated how many times they had contact (N = 1,181; M = 1.24; SD = 6.23; range = 0 to 150).

Statistical Analysis

The first set of models were longitudinal confirmatory factor analysis models which evaluated the relationship between the four manifest variables in SATSA (i.e.,

somatic complaints, over-the-counter medication use, pain-related activity limitations, and perceived illness complications)—or three manifest variables (i.e. somatic complaints, over-the-counter medication use, and perceived illness complications) in GENDER—and a latent, underlying illness behavior factor across measurement occasions. In the first and least constrained model (*M1a*), configural invariance was evaluated by constraining the factor loadings and intercepts of the reference indicator (i.e. somatic complaints) to equal, and by allowing for covariance among the observed indicators across the measurement occasions. In addition, the first latent factor was fixed to have a mean of 0 and unit variance to place it on a standardized metric. In the next submodel (*M1b*), the thresholds of the categorical indicator (i.e. pain-related disability in SATSA, and over-the-counter medication use in GENDER), were equated across the assessments. The next model (*M2*) evaluated weak factorial invariance by equating the factor loadings of each respective indicator across time. The strong factorial invariance model (*M3*) equated the intercepts of similar continuous indicators across time. The final model (*M4*) evaluated strict factorial invariance by constraining the unique variances of the indicators to be equal, as well as equating the covariances among the unique variances of similar indicators over time.

All longitudinal CFA analyses were run in the M*plus* program version 7.4 (Muthén & Muthén, 2012) using the weighted least square mean variance (WLSMV) estimator and accounting for participant clustering within twin pairs. The missing data option was specified and full-information modeling of all data was applied to make use of all available observations in the dataset and to reduce any possible attrition bias; no

pairwise or listwise deletion or imputation methods were applied to the missing data. Most of the indicators were treated as continuous manifest variables, except for the pain-related activity limitations composite in SATSA and the over-the-counter medication use composite in GENDER, which were specified as ordinal. The goodness-of-fit of all nested measurement models was evaluated using the likelihood ratio chi-square difference test (i.e. the DIFFTEST option for the WLSMV estimator in M*plus*) and practical fit indices including the comparative fit index (CFI; Bentler, 1990), Tucker-Lewis Index (TFI; Tucker & Lewis, 1973), and the root-mean-square error of approximation (RMSEA; Browne & Cudeck, 1993) with its associated 90-percent confidence interval. For the CFI and TLI, values above .90 and .95 indicated acceptable and good model fit, respectively, whereas for RMSEA, a value less than .05 and .08 with a confidence interval that includes zero (or comes close) was considered an excellent and acceptable model fit, respectively (Makikangas & Feldt, Kinnunen, Tolvanen, Kinnunen, & Pulkkinen, 2006; Marsh et al., 2009).

For the second set of analyses, latent growth curve (LGC) models were fitted to the SATSA data to evaluate age-related linear change in the latent illness behavior factor (output from the final, best-fitting CFA model) from mid- to late- adulthood. First, an unconditional means model (*Model 1*) was fitted to estimate the amount of variability in the illness behavior factor to be explained at the between-person and between-pair levels (Singer & Willett, 2003). Second, an unconditional linear change model (*Model 2*) was fitted that added the time predictor of centered age (i.e. centered at 60 years, or the average age at baseline) to estimate within-person change in the illness behavior factor

scores across the four questionnaire waves. Next, an intercept-as-outcome model (*Model 3*) was fitted to test the predictive value of family and friend social support availability on the intercept (i.e. illness behavior levels at age 60), adjusted for covariates. Predictors were entered in the following order: comorbidity, sex, SES, marital status, study entry age (i.e. centered at 70 years), friend support availability, and family support availability. Finally, an intercept-and-slope as outcome model (*Model 4*) was fitted that evaluated the prediction of both illness behavior intercept and slope.

All growth models were run in M*plus* version 7.4 (Muthén & Muthén, 2012), using the robust maximum likelihood estimator (MLR), specifying the missing data option and accounting for clustering within twin pairs. Participants were included in the analysis if they had complete data on the baseline predictors and at least one wave of the illness behavior outcome. Goodness of fit of the nested models was evaluated using the Satorra-Bentler chi-square difference test statistic (Bryant & Satorra, 2012; Satorra & Bentler, 2010) with the formula specified by Muthén and Muthén (2010) (http://www.statmodel.com/chidiff.html) for robust maximum likelihood estimation. Grand-mean centering was used for the age predictor, whereas illness comorbidity was centered at the value of one (i.e. one medical condition or rather, one organ system affected by illness), and sex and marital status were dummy-coded. Social support availability and the SES variable were standardized composites with meaningful zero-points.

Results

Descriptive Information on the Illness Behavior Indicators

SATSA. The descriptive results (means and standard deviations) and zero-order correlations of the observed indicators of illness behavior across each measurement occasion are presented in Tables 2.1 and 2.2, respectively. Patterns of mean values for each indicator generally suggested higher somatic complaints (*N*s ranged from 742 to 1,478), increased non-prescription medication use (*N*s ranged from 750 to 1,425), and except for Q4, greater perceived illness complications (*N*s ranged from 786 to 1,572) across the waves. Endorsements of disabling back, neck, or shoulder pain across the waves were suggestive of stability in overall response frequencies (*N*s ranged from 771 to 1,593). Intercorrelations among the same illness behavior indicators revealed moderate-to-strong rank-order stability across the waves. The somatic composites were positively correlated from $r = .40$ to $r = .58$; the medication composites were positively correlated from $r = .43$ to $r = .59$; the pain items were correlated from $r = .53$ to $r = .71$; and perceived illness complications composites were correlated from $r = .20$ to $r = .55$ (all $p < .0001$).

GENDER. The descriptive results and zero-order correlations among the illness behavior indicators across the two questionnaire waves suggested higher somatic complaints (*N*s ranged from 317 to 927) but slightly lower perceived illness complications (*N*s ranged from 354 to 1,206) at Q2 relative to Q1 (see Tables 2.5 and 2.6 for descriptive results and correlations, respectively). Endorsements for non-prescription medication use also suggested a small increase in the overall frequencies for the upper

response categories at Q2 relative to Q1, whereas the proportion of participants who endorsed taking one medication or no medications decreased across the two waves (*N*s ranged from 338 to 1,183). Within each questionnaire wave, the illness behavior indicators showed weak-to-moderate positive associations. For example, at Q1, intercorrelations among the indicators ranged from $r = .13$ (somatic complaints with perceived illness complications) to $r = .24$ (somatic complaints with medication use), while at Q2, the indicator correlations ranged from $r = .05$ (medication use with perceived illness complications) to $r = .36$ (somatic complaints with medication use). Intercorrelations among the same illness behavior indicators across the two questionnaires revealed weak-to-moderate rank-order stability. The somatic complaints subscales were positively correlated at $r = .44$ across the questionnaire waves; the medication use composites were positively correlated at $r = .54$; and the perceived illness complications composites were positively correlated at $r = .31$ (all $p < .0001$).

Longitudinal CFA Models

SATSA. The goodness-of-fit results for the models of factorial invariance across time are presented in Table 2.3. For the SATSA sample, the first single-factor models of configural invariance (*M1a* and *M1b*) had good fit to the data, as indicated by the practical fit indices (*M1a*: CFI = 0.99, TLI = 1.00, RMSEA = 0.02, 90% CI: [0.014, 0.025]); *M1b*: CFI = 0.99, TLI = 0.98, RMSEA = 0.02, 90% CI: [0.018, 0.028]). The use of the WLSMV estimator, however, precluded tests of relative fit against a saturated model. The next model of weak factorial invariance (*M2*) equated the indicator factor loadings across time; this model was restricted by 9 degrees of freedom and resulted in a

significantly reduced model fit relative to *M1b* ($\Delta \chi^2 / \Delta$ df = 28.15/9, $p = .001$), but there was no change in fit per the CFI, TLI, and RMSEA. The next model of strong factorial invariance (*M3*), which set the indicator intercepts to be invariant, restricted the model by 6 degrees of freedom, and again significantly reduced model fit per the χ^2 difference statistic ($\Delta \chi^2 / \Delta$ df = 38.27/6, $p < .0001$), but there was no change in the practical indices. Finally, the likelihood ratio chi-square ($\chi^2 = 410.77$, df = 107, $p < .0001$) and χ^2 difference test suggested the model of strict factorial invariance (*M4*) resulted in significantly poorer fit relative to the strong factorial invariance model ($\Delta \chi^2 / \Delta$ df = 240.53/9, $p < .001$), whereas the incremental fit statistics were slightly reduced but still suggested good fit to the data (CFI = 0.96, TLI = 0.95, RMSEA = 0.04, 90% CI: [0.035, 0.043]). Although the chi-square difference tests revealed a significant worsening of model fit with added constraints for measurement invariance, the practical fit indices were consistent across these constraints and revealed acceptable model fit. Previous reviews suggest that with larger sample sizes the chi-square value is likely to reach significance due to increased power of the test, whereas practical fit indices are independent of sample size and violations of multivariate normality assumptions (Marsh et al., 2009). Therefore, in the interest of model interpretation and efficiency, and because there was no substantial loss of fit in the practical fit indices, the most restricted model was chosen as the best-fitting model (c.f., Widaman et al., 2010).

The final, best-fitting CFA model of strict factorial invariance with the unstandardized solution is shown in Figure 2.1, and the standardized indicator factor loadings, their standard errors, and latent factor intercorrelations are presented in Table

2.4. All indicators had moderate-to-strong standardized factor loadings ranging from 0.43 to 0.66, and all were significant ($p < .0001$).

Properties of the factors and associations with demographic variables. The latent illness behavior factors had high rank-order stability across the four measurement occasions, with zero-order correlations (Table 2.4) ranging from $r = .60$ (the association between the second and fourth wave) to $r = .92$ (the association between the first and second wave; all $p < .0001$). Finally, the latent illness behavior factor showed acceptable discriminant validity, evidenced by moderate zero-order correlations between the illness behavior factor scores (saved out from M*plus*) and the other constructs from the baseline (Q2) assessment. Specifically, better self-rated health status was correlated moderately and negatively with the illness behavior factor scores across time (average $r = -.49$, $p < .0001$; *N*s across the waves ranged from 619 to 1,273). Greater illness comorbidity was also moderately and positively associated with the illness behavior factors (average $r = .56$, $p < .0001$; *N*s across the waves ranged from 625 to 1,276).

GENDER. The goodness-of-fit results for the models of factorial invariance across time fitted to GENDER data are presented in Table 2.7. The first model of configural invariance (*M1a*) was supported by the chi-square goodness-of-fit statistic ($\chi^2 (5) = 6.76$, $p = .24$) and by the practical fit indices (RMSEA= .017; 90 % CI = [.000, .046], $p > .05$; CFI = .996; TLI = .987). The next submodel (*M1b*) equated the six thresholds of the ordinal medication use indicator across the two waves. This model showed even better fit to the data relative to the first model ($\Delta \chi^2 / \Delta$ df = 4.15 / 6, $p = .66$; RMSEA = .000; 90 % CI = [.000, .030], $p > .05$; CFI = 1.00; TLI = 1.00). The

second model of weak factorial invariance (*M2a*) did not improve fit to the data relative to the configural invariance model, *M1b*, according to the chi-square difference test statistic ($\Delta \chi^2 / \Delta$ df = 16.62 / 2, $p < .001$). However, the practical fit indices suggested acceptable fit for weak factorial invariance in illness behavior (RMSEA = .034; 90 % CI = [.019, .050], $p > .05$; CFI = .955; TLI = .948), with standardized factor loadings of the indicators ranging from 0.27 to 0.55 (all $p < .0001$). The subsequent models of strong (*M3a*) and strict factorial invariance (*M4a*) were not supported by the data (RMSEAs > .05; CFIs < .90; TLIs < .90; and $\Delta \chi^2 / \Delta$ df = 31.30 / 1, $p < .0001$; and 10.86 / 2, $p < .01$, respectively).

Specifically, the perceived illness complications indicator did not load strongly onto the proposed latent factor across the two questionnaire waves. Across all CFA models, the standardized factor loadings for this indicator ranged from 0.20 to 0.34 at the first questionnaire wave ($p < .0001$), and from 0.12 to 0.27 at the second wave (most non-significant at $p > .05$). Therefore, a sequence of partial measurement invariance models was also evaluated, which freed the factor loadings of the perceived illness complications composite across Q1 and Q2. The original model of weak factorial invariance (*M2a*), which imposed an equality constraint on these two factor loadings, significantly reduced model fit relative to the freer model of partial weak factorial invariance (*M2b*) ($\Delta \chi^2 / \Delta$ df = 9.35 / 1, $p = .002$). A model of partial, strong factorial invariance (*M3b*) reduced model fit relative to *M2b* ($\Delta \chi^2 / \Delta$ df = 9.86 / 1, $p = .002$), whereas the practical fit indices suggested almost acceptable fit (RMSEA = .034; 90 % CI = [.019, .050], $p > .05$; CFI = .954; TLI = .947). Once again, a model of partial strict factorial variance (*M4b*) in

illness behavior was not supported ($\Delta \chi^2$ / Δ df = 15.75 / 2, $p < .001$; RMSEA = .04, 90 % CI = [.029, .06], $p > .05$; CFI = .92, TLI = .92). Therefore, a model of partial weak factorial invariance, in which the loadings of the perceived illness complications indicator were free to vary across the questionnaires, was selected as the best-fitting model. The unstandardized solution from this model is shown in Figure 2.2, and the standardized indicator factor loadings with their standard errors are presented in Table 2.8, along with the latent factor intercorrelation.

Furthermore, because the perceived illness complications indicator did not load as well onto the proposed latent factor at Q2, a CFA model of illness behavior was fitted that included the indicators from Q1 only. The standardized factor loadings of somatic complaints, non-prescription medication use, and perceived illness complications from this model were 0.37 (*SE* = .05), 0.65 (*SE* = .08), and 0.35 (*SE* = .05), respectively (all $p < .0001$). The illness behavior factor scores from this Q1-only model were output from M*plus* (and all imputed scores were set to missing) for use in subsequent tests of discriminant and convergent validity (*N* = 921; *M* = 0.02, *SD* = 0.68; range = - 1.81 to 2.64).

Properties of the factor and associations with demographic variables. The latent illness behavior factor from Q1 showed good discriminant validity, evidenced by weak to moderate zero-order correlations between the illness behavior factor scores (saved out from *Mplus*) and other overlapping—but distinct—constructs from the same assessment wave. Specifically, poorer self-rated health status and higher comorbidity were both correlated moderately and positively with the illness behavior factor scores (*r*

(920) = 0.36, $p < .0001$; and r (921) = 0.43, $p < .0001$, respectively). Furthermore, greater health promotion values—that is, placing greater importance on getting regular physical exams and taking one's medications—was also weakly and positively associated with higher illness behavior factor scores (r (898) = 0.11, $p < .001$; and r (886) = 0.11, $p < .01$, respectively). The convergent validity of the illness behavior factor was also supported, based on its significant associations with self-reported health utilization variables. Specifically, higher illness behavior factor scores were positively correlated with a greater number of reported sick days (r (918) = 0.25, $p < .0001$) and more frequent contact with a physician in the past six months (r (909) = 0.23, $p < .0001$). Illness behavior factor scores were also weakly, but positively, associated with self-reported hospitalizations (r (917) = 0.10, $p < .01$) and frequency of contact with a district nurse (r (914) = 0.09, $p < .01$), suggesting that illness behavior is relatively independent from these forms of need-based health utilization.

Descriptive Information on Predictors and Illness Behavior Outcome

SATSA. The descriptive results (i.e. means, standard deviations, range, and skewness) for all observed predictors and the latent illness behavior outcomes are presented in Table 2.9. Zero-order correlations among the predictors and illness behavior factors are shown in Table 2.10. Overall, the descriptive results for the (T-score scaled) illness behavior factors suggested that, on average, illness behaviors increased over time (except at the Q4 assessment), as did their variability (except at the Q5 assessment). Furthermore, the zero-order correlations suggested that greater comorbidity at baseline was moderately and positively associated with higher illness behavior factor scores

across the waves (*r*s ranged from .52 to .56, $p < .0001$); whereas higher SES, friend support availability, and family support availability were weakly and negatively correlated with illness behavior levels (SES: *r*s ranged from -.15 to -.18, $p < .0001$; Friend support: *r*s ranged from -.16 to -.19, $p < .001$; Family support: *r*s ranged from -.06 to -.09, $p < .05$). Older participants and females were more likely to report higher illness behaviors across the waves (*r*s ranged from .10 to .14, $p < .05$; and .22 to .25, $p < .0001$, respectively); whereas being married—versus being never married, widowed, or divorced—was weakly and negatively associated with illness behavior scores (*r*s ranged from -.05, *ns*, to -.11, $p < .0001$). Regarding intercorrelations among the predictors, friend and family support were moderately and positively correlated ($r = .40$, $p < .0001$). Older participants and females reported less availability of support from friends ($r = -.18$, $p < .0001$; $r = -.11$, $p < .0001$) but higher availability of support from family members ($r = .13$, $p < .0001$, $r = .09$, $p < .001$) as compared to younger participants and males. Higher SES and marital status (i.e. being married) were weakly but positively correlated with the availability of friend and family support (SES: $r = .22$, $p < .0001$, and $r = .06$, $p < .05$, respectively; Marital: both $r = .12$, $p < .0001$). Finally, comorbidity was weakly and negatively correlated with friend support availability ($r = -.09$, $p < .001$), but it was not associated with family support availability.

Latent Growth Models

Model estimates and expected trajectories for the latent growth curve models of illness behavior factor scores fitted to SATSA data are presented in Table 2.11 and in Figure 2.3, respectively. Results from the unconditional change model, Model 2,

suggested that an age-based, linear model of change in illness behavior had a good fit to the data as compared to Model 1, the means-only model ($\Delta \chi^2 / \Delta df = 477.71 / 3$, $p < .0001$). Specifically, it showed a gradual, yearly increase in illness behavior scores across age ($b_1 = .13$, $p < .0001$). Model 3, a conditional intercept-as-outcome model, suggested that perceived availability of support from friends or acquaintances negatively predicted illness behavior levels at age 60 years ($b_{06} = -.18$, $p < .0001$) above and beyond the other covariates, wherein each unit increase above the average level of friend support predicted lower levels of somatic complaints, medication use, pain-related disability, and self-reported illness complications. Family support also negatively predicted illness behavior levels, but its effect was non-significant ($b_{07} = -.14$, $p > .05$).

Model 4 added random variation around the slope as well as the intercept. In Model 4a, a DEFINE statement in M*plus* was used to equate the scales of the friend and family support composite variables ($M = 0$; $SD = 1$) and regressed the intercept and slope on all predictors. In this model, perceived friend and family support again negatively predicted illness behavior levels at age 60 ($B_{06} = -.99$, $p < .0001$; $B_{07} = -.44$, $p > .05$; respectively), such that a one-unit standard deviation increase in each variable was associated with a small decrease (Cohen's $d = -.10$ and -.04, respectively) in somatic complaints, medication use, and perceived illness complications at age 60. Neither support measure, however, was significantly associated with linear change in illness behavior over time. In a subsequent, post-hoc model (Model 4b), the effects of friend and family support on illness behavior level and slope were constrained to be equal to evaluate whether their associations with illness behavior were significantly different.

Equating their effects fit trivially but non-significantly worse than the model in which both effects were freely estimated, suggesting that Model 4b was a more parsimonious model relative to Model 4a ($\Delta \chi^2 / \Delta df = 1.60 / 1$, $p > .05$), and supporting the notion that friend and family support have the same negative association with the illness behavior intercept. In this model, friend and family support variables negatively predicted illness behavior levels (*B*s = - .71, $p < .0001$), such that a one-unit standard deviation increase in friend or family support beyond the average was associated with a significant, albeit small, decrease in somatic complaints, medication use, pain-related disability, and illness complications equivalent to a Cohen's *d* effect size of .07; however, neither support variable predicted linear change.

Regarding the covariates, comorbidity positively predicted illness behavior levels at age 60, such that a one-unit increase in comorbidity (i.e. each additional diagnosis beyond one health condition) was associated with a 2.50 unit (i.e. one-fourth of a standard deviation) increase in illness behavior levels ($p < .0001$). Greater comorbidity also predicted a slight increase in the annual, linear rate of illness behavior change ($b_{11} = .02$, $p < .0001$). Females also reported significantly higher levels of illness behavior at age 60 relative to males ($b_{02} = 2.74$, $p < .0001$), and a higher age at study entry (i.e. each unit-increase in years beyond age 70) negatively predicted illness behavior levels ($b_{05} = -.19$, $p < .0001$). In sum, although the perceived availability of support from friends and family members did not predict change in the rate of growth in illness behavior, both sources of support appeared to similarly predict illness behavior levels at entry into the older adulthood transition. Thus, although older adults' social connectedness with friends

and family members might not play a significant role in affecting behavior change, these results do seem to support their roles in shaping the behavioral trajectory across time, given the higher variability in illness behavior levels relative to its rates of change.

Discussion

The present study investigated the longitudinal measurement and construct validity of a multi-indicator illness behavior factor in two population-based samples of older adults (i.e. the Swedish Adoption/Twin Study of Aging and GENDER). The illness behavior factors included indicators of somatic complaints, non-prescription medication use, pain-related disability, and perceptions of illness complications (adjusted for physician panel ratings of expected complications). Previous work has applied cross-sectional and longitudinal methods to examine the psychosocial processes underlying these aspects of illness behavior and others (e.g., primary healthcare utilization; Stump et al., 1995; symptom monitoring; van Osch et al., 2007) in isolation. Illness behavior as a unifying construct (Sirri et al., 2013), however, represents a promising framework for better understanding individual differences in responsiveness to symptoms across many levels—from perception, to cognition, to behavior. Thus, a series of longitudinal CFA models were fitted to SATSA and GENDER data to evaluate whether variation on these observed indicators could be accounted for by a single, latent illness behavior factor, as well as the extent to which the quantitative and qualitative meaning of this factor was invariant across a span of seventeen years in SATSA (1987-2004) and thirteen years in GENDER (1994-2007). Based on the theoretical dimensions of illness behavior originally proposed by David Mechanic (1962/1977) and more recent work on the

development of a unifying illness behavior scale (Berk et al., 2012; Scherer et al., 2016; Sirri et al., 2013), at minimum, strong factorial invariance in the latent factor was hypothesized—in other words, results were expected to support measurement of the same construct on the same scale across all assessment waves. Illness behavior factors were expected to show acceptable discriminant validity, through weak-to-moderate associations with theoretically similar—but distinct—constructs including self-rated health, comorbidity, health promotion values (GENDER only), hospitalizations and district nurse contact (GENDER only). In the GENDER sample, the factors' convergent validity was expected to be supported through significant associations with healthcare utilization (i.e. sick days and doctor visits).

Another focus of the current study was examining age-related trajectories in illness behavior from mid-to-late adulthood, and the relative predictive value of perceived social support availability from two separate sources—friends versus family members—on illness behavior levels and intra-individual change (or maintenance) during this developmental transition. Prior cross-sectional work has compared age cohorts on health promotion behaviors and finds that older adults often outperform younger and middle-aged adults in their disease management. For example, older adults consistently seek out formal medical services, with relatively stable primary care utilization patterns (Stump et al., 1995), and are less likely than middle-aged adults to delay seeking medical care for new symptoms, particularly when the symptoms are perceived as potentially serious versus either mild or severe (Leventhal, Leventhal, Schaefer, & Easterling, 1993). However, the longitudinal patterns of stability or change in less formal types of help-

seeking or "lay care strategies" (Stoller, Forster, & Portugal, 1993; p. 24)—vis a vis a latent construct of illness behavior—across the transition into late-adulthood, and how such trajectories might be altered by perceptions of social resources, is not yet understood. Based on the literature's suggestion that older adults show increasing self-regulation capabilities for coping with health problems (Leventhal, Leventhal, & Contrada, 1998) and shifting personal models of illness geared toward disease management and more adaptive self-care (Harvey & Lawson, 2008), a significant, age-related linear increase in illness behaviors was hypothesized. With regards to social support, however, both sources of support from friends and family members were hypothesized to predict reduced levels of illness behavior and greater behavioral stability (i.e. reduced linear change) over time.

Results from the longitudinal confirmatory factor analyses partially supported the hypothesis of strong factorial invariance in a latent illness behavior factor over time. In SATSA, results supported strict factorial invariance in the illness behavior factor scores across the four questionnaire waves, with moderate-to-strong standardized loadings for somatic complaints, non-prescription medication use, pain-related disability, and perceived illness complications. Consequently, we could be confident that the same construct was on the same metric across the waves, and further exploration of quantitative growth in the illness behavior factor was justified. In the GENDER sample, however, this hypothesis was not supported. A model of partial, weak factorial invariance was the best-fitting model, suggesting that the overall meaning of the illness behavior

factor was similar across the two waves (i.e. the same pattern of three indicators held across time, two of which had the same loadings); however, it was not on the same scale.

Consequently, quantitative change in the illness behavior factor would be confounded with response shift and measurement error, and latent differences between the two waves could not be assessed. The model misfit was attributable to the indicator of perceived illness complications, which had a substantially lower, and non-significant, factor loading at the second questionnaire wave and could not be constrained to be equal across the two waves. One possible explanation for this discrepancy is the older and narrower age ranges of the GENDER participants (ages 70 to 88 years at baseline) as compared to the SATSA participants (ages 29 to 96 years at baseline, but with an average age of 66 years across all of the assessment waves). Among the older participants in GENDER, there might have been a shift in the meaning and relevance of perceived illness complications to the underlying illness behavior construct in late-life. Whereas this measure might serve as an indicator of individual differences in perceptions and responses to symptoms in midlife adults or the young-old adults, the internal frame of reference for endorsing complications might exhibit a response shift in late-life. For example, the oldest-old age groups might be less likely to endorse complications from illness due to a shift in personal definitions of what constitutes a complication.

In both samples, the construct validity of the illness behavior factors was supported (i.e. the four factor scores output from SATSA Q2-Q5, and the one factor output from GENDER Q1 only). In SATSA, higher illness behavior scores were moderately and positively correlated with comorbidity, but were moderately and

negatively associated with better self-rated health status. In GENDER, the illness behavior factor was moderately and positively correlated with comorbidity and poorer self-rated health. It also had weak, positive associations with the two health promotion value items (i.e. the importance of getting regular physical exams and taking one's medicine), as well as with self-reports of hospitalizations and frequency of contact with a district nurse. In contrast, the factor was more positively, albeit still weakly, correlated with self-reported sick days and primary physician visits, supporting its convergent validity. Together, these results suggest that the present study's illness behavior construct is relatively independent from participants' general self-rated health status, objective health status, personal beliefs about health promotion, and less volitional forms of healthcare utilization as compared to sick days and primary care visits (consistent with prior work on associations of body awareness with emergency room visits or initial physician contact, but not with referrals or hospitalizations; Hansell, Sherman, & Mechanic, 1991).

In the second set of analyses, a series of longitudinal growth curve models were fitted to evaluate the age-related pattern of change in illness behavior (i.e. factor scores including somatic complaints, non-prescription medication use, pain-related disability, and perceived illness complications) across seventeen years in SATSA (Q2-Q5: 1987-2004), and the long-term predictive role of social support availability from friends versus family members in shaping these behavioral trajectories. Analyses adjusted for clustering in twin pairs, as well as the effects of baseline comorbidity, sex, SES, marital status, and entry age (at Q2). Consistent with this study's hypothesis, there was a significant, annual

linear increase in the illness behavior outcome after age 60. This finding is in line with other literature suggesting that older adults take increasing responsibility for their health and engage in more health-promoting behaviors relative to young and middle-aged adults (Walker, Volkan, Sechrist, & Pender, 1988), and are less likely to be avoidant about their health risks or delay medical help-seeking in the case of ambiguous but potentially serious symptoms (Leventhal, Leventhal, & Contrada, 1998). They may also engage in more self-care and self-management of symptoms outside of the doctor's office (e.g., through use of over-the-counter medications) than young or middle-aged adults (Stoller et al., 1993). Moreover, consistent with prior work on the demographic predictors of illness behavior, females in this study had higher illness behavior scores than males, as did unmarried individuals or those with lower SES (Vedsted & Christensen, 2005; Verbrugge, 1989). Greater comorbidity was also associated with higher illness behavior scores at age 60, and a steeper, linear increase per year in illness behaviors afterward, underscoring the important role of disease processes and disease severity in illness behavior development.

In partial support of the second hypothesis, friend and family support availability predicted reduced illness behavior levels, but neither variable was associated with the rate of intra-individual change. Furthermore, the effects of friend and family support availability on illness behavior status could be equated, which was also unexpected. However, in consideration of the vast literature on the links of both friend and family social ties with a wide range of preventive health behaviors in older adults (e.g., use of screenings and exercise; Seeman, 2000), and recent work on friend and family support's

protective roles for biophysiology (e.g., reducing inflammation risk; Yang, Scorpp, & Mullan Harris, 2014), our finding that both sources of available support had the same association with decreased levels of somatic complaints, non-prescription medication use, pain-related disability, and perceived illness complications, is not entirely unexpected. Although the research on illness behavior development and maintenance is limited, perceived social connections and support have been associated with behavior change (and maintenance) among older adults for disease management behaviors, such as hypertension detection and control (Cornwell & Waite, 2012). Although the current study did not find effects of social support on intra-individual change or maintenance in illness behavior, its association with the intercept suggested that it did play a role in shaping the overall trajectory.

Strengths of the current study included the application of prospective, longitudinal methods to examine the relationships among the indicators, social predictors, and subsequent illness behavior outcomes. The structural equation-modeling framework also provided more flexibility for handling missing data across the assessment waves, robustness to issues of non-normality, the inclusion of both continuous and ordinal variables, and the ability to account for both measurement error and structural relationships. Furthermore, the large, representative samples of adults increased statistical power for detecting the effects of interest, as well as the generalizability of the results. In particular, the inclusion of twin pairs is beneficial for investigating the role of social contexts in health-relevant behavioral processes, while adjusting for biological or familial factors the might confound the relationship.

Study limitations were in part related to the availability and overlap of illness behavior measures across the assessment waves in SATSA and GENDER. Some potentially useful indicators (e.g., a broader symptom checklist in SATSA, and pain response and attribution items in GENDER) did not overlap across the assessments and therefore were not included. Another limitation was the inclusion of all self-report and retrospective measures from the same respondents across the waves. Because all data were included from the questionnaire assessments, the present study's results could not account for potential biases due to same-method covariance. Finally, the treatment of all study predictors and covariates as time-invariant might be a limitation. Although the present study took advantage of the temporal relationships between baseline measures of social support with subsequent illness behavior trajectories, modeling the concurrent age-related shifts in social support and increasing comorbidity might better elucidate parallel social and biological processes that underlie illness behavior development.

The current study suggests that a multi-indicator measure of illness behavior is a valid construct with useful psychometric properties for examining intra-individual behavior change (and between-individual differences in that change) during the late-life transition. It suggests that, on average, there is a small, linear increase with age in somatic complaints, non-prescription medication use, pain-related disability, and perceptions of illness complications across age, but that perceptions of higher social support availability from friends and family members predict lower levels of these behaviors at the entry into older adulthood. Although the effects of social support on illness behavior status was small, the associations remained after adjusting for the

substantive effects of objective health status (i.e. comorbidity), sex, marital status, and SES. Ideally, future research would explore the causal mechanisms of this association (e.g., potential mediation or moderation by health cognitions or emotional processes), as well as investigate the long-term consequences of illness behavior trends for outcomes of mental and physical functioning.

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Table 2.1

Means, Standard Deviations, and Response Rate Percentages of Observed Illness Behavior Indicators Across Time (SATSA sample)

	Wave							
	Q2		Q3		Q4		Q5	
Indicator	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*n*	*M* (*SD*)	*N*	*M* (*SD*)
Somatic	1,478	3.19 (3.07)	1,362	3.36 (3.04)	1,333	3.43 (3.19)	742	3.79 (3.22)
Medication use[a]	1,425	1.07 (1.25)	1,346	1.15 (1.25)	1,411	1.32 (1.34)	750	1.44 (1.34)
Illness complications	1,572	.20 (1.44)	1,460	.21 (1.58)	1,432	.16 (1.74)	786	.24 (1.67)
	Wave							
	Q2		Q3		Q4		Q5	
Pain item response categories[b]	*n* (Total= 1,593)	%	*n* (Total= 1,471	%	*n* (Total= 1,436)	%	*n* (Total= 771)	%
0	999	62.70	894	60.80	905	63.00	489	63.40
1	323	20.30	299	20.30	254	17.70	169	21.90
2	135	8.50	126	8.60	99	6.90	56	7.30
3	136	8.50	152	10.30	178	12.40	57	7.40

Note. All values are WLSMV estimates from M*plus*. Pain item response categories: 0 = *no disabling neck, back, or shoulder pain*; 1 = *endorsed at least one pain item*; 2 = *endorsed at least two pain items*; 3 = *endorsed all three pain items (i.e. the presence of disabling neck, back, and shoulder pain)*.

[a] Collapsed into five response categories. [b] Treated as an ordinal indicator.

Table 2.2

Inter-correlations of Observed Illness Behavior Indicators Over Time (SATSA sample)

Variable	Somatic				Medication use				Pain				Illness disability			
	Wave															
	1	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*	*1*	*2*	*3*	*4*
Somatic1	1.00	.58	.52	.40	.22	.16	.23	.19	.26	.22	.23	.18**	.25	.23	.16	.20
Somatic2		1.00	.57	.41	.20	.21	.23	.16	.23	.28	.22	.18	.18	.27	.16	.14**
Somatic3			1.00	.45	.20	.17	.23	.17	.22	.24	.25	.14**	.18	.24	.24	.23
Somatic4				1.00	.17	.12**	.16	.25	.21	.14**	.18**	.18	.12*	.09^{ns}	.13**	.22
Med1					1.00	.59	.54	.44	.32	.24	.25	.21	.20	.16	.15	.09*
Med2						1.00	.53	.49	.29	.25	.25	.22	.17	.17	.14	.05^{ns}
Med3							1.00	.43	.33	.28	.31	.24	.20	.23	.17	.12**
Med4								1.00	.34	.27	.22	.29	.15**	.12*	.11*	.22
Pain1									1.00	.71	.63	.53	.29	.27	.24	.24
Pain2										1.00	.72	.56	.20	.25	.26	.20

Pain3	1.00	.60	.18	.25	.27	.24
Pain4		1.00	.12*	.13**	.17**	.19
Disability1			1.00	.44	.31	.20
Disability2				1.00	.55	.47
Disability3					1.00	.42
Disability4						1.00

*Note. N*s ranged from 632 to 1,542. All values are WLSMV estimates from M*plus*.

[ns] Non-significant at an alpha level of .05. * $p < .05$. ** $p < .01$. For all other estimates, $p < .0001$.

Table 2.3

Goodness-of-fit Indices for the Confirmatory Factor Analysis Models of Factorial Invariance in Illness Behavior Across Time (SATSA sample)

Factor model[a]	χ^2	*df*	*P*	$\Delta\chi^2/\Delta$ *df*	*p*	CFI	TLI	RMSEA	(90 % CI)
M1a	126.891	74	.000	---	---	.992	.998	.019	(.014 - .025)
M1b	167.535	83	.000	50.537 / 9[b]	.000	.988	.983	.023	(.018 - .028)
M2	196.284	92	.000	28.151 / 9[c]	.001	.985	.981	.025	(.020 - .029)
M3	226.547	98	.000	38.267 / 6[d]	.000	.982	.978	.026	(.022 - .031)
M4	410.771	107	.000	240.530 / 9[e]	.000	.957	.951	.031	(.035 - .043)

Note. N = 1,886. CI = confidence interval. Model 1a (M1a) tested configural invariance, with constrained factor loadings of the reference indicator (i.e., somatic complaints) across time, M1b included an additional constraint of equated thresholds for the categorical indicator (i.e. pain-related disability) across time, M2 tested weak factorial invariance, with equated factor loadings of the same indicators across time, M3 tested strong factorial invariance, with equated intercepts of the same indicators across time, and M4 tested strict factorial invariance, with equated variances and residual covariances of the same indicators across time. CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root-mean-square error of approximation.

[a] Unidimensional, single-factor. [b] M1a versus M1b. [c] M1b versus M2. [d] M2 versus M3. [e] M3 versus M4.

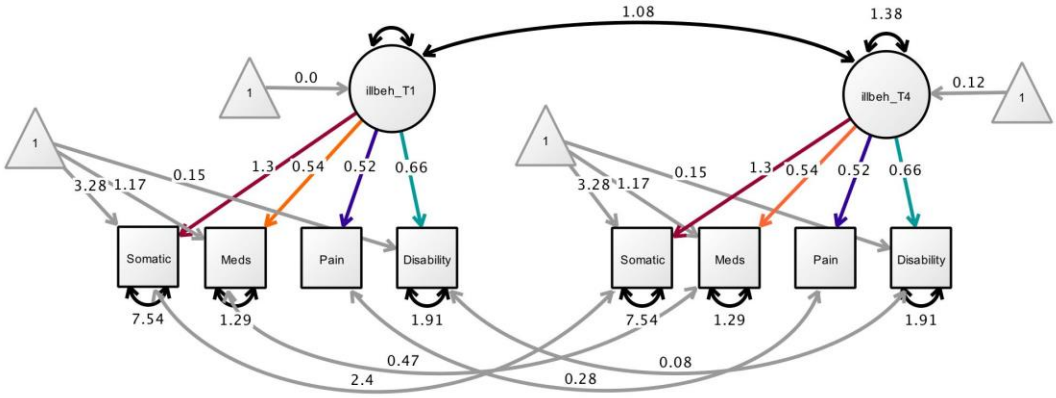


Figure 2.1. Final longitudinal confirmatory factor model of strict factorial invariance in illness behavior (SATSA sample), consisting of four manifest variables assessed at each of four times of measurement (only the first and last waves are shown here for simplicity). Completely unstandardized mean- and variance-adjusted weighted least square (WLSMV) parameter estimates. Paths for the equated factor loadings are depicted with the same colors. Intercepts and covariances are shown in grey.

Table 2.4

Standardized Factor Loadings and Latent Factor Intercorrelations Across Time from Confirmatory Factor Analysis Model of Strict Factorial Invariance (SATSA sample)

Factor	Wave			
Indicators	*Q2*	*Q3*	*Q4*	*Q5*
Illness behavior				
Somatic	.43 (.03)	.47 (.03)	.52 (.03)	.49 (.03)
Medication use	.43 (.02)	.46 (.03)	.51 (.03)	.49 (.03)
Pain	.52 (.03)	.58 (.03)	.66 (.03)	.61 (.04)
Illness complications	.43 (.02)	.47 (.02)	.52 (.02)	.49 (.02)
	Latent Factor Correlations			
	F1	*F2*	*F3*	*F4*
F1	1.00	---	---	---
F2	.92 (.03)	1.00	---	---
F3	.85 (.04)	.83 (.03)	1.00	---
F4	.78 (.07)	.60 (.06)	.66 (.05)	1.00

*Note. N*s ranged from 697 to 1,292. Values in parentheses indicate standard errors. All values are WLSMV estimates from M*plus*. For all estimates, $p < .0001$. Somatic = somatic complaints composite from the CES-D; Medication use = composite of non-prescription medication use (past 30 days); Pain = ordinal item on the presence of

disabling neck, back, and shoulder pain; Illness complications = difference score composite of perceived illness complications, adjusted for physician panel ratings. *F1* = illness behavior factor at wave 1 (Q2); *F2* = illness behavior factor at wave 2 (Q3); *F3* = illness behavior factor at wave 3 (Q4); *F4* = illness behavior factor at wave 4 (Q5).

Table 2.5

Means, Standard Deviations, and Response Rate Percentages of Observed Illness Behavior Indicators Across Time (GENDER Sample)

	Waves			
	Q1		Q2	
Indicator	*n*	*M* (*SD*)	*n*	*M* (*SD*)
Somatic complaints	927	2.97 (3.03)	317	4.47 (3.67)
Illness complications	1,206	0.05 (1.47)	354	-0.17 (1.55)
	Q1		Q2	
Medication use item response categories[a,b]	*n* (Total= 1,183)	%	*n* (Total= 338)	%
0	379	32.00	70	20.70
1	314	26.50	62	18.30
2	218	18.40	75	22.20
3	136	11.50	59	17.50
4	74	6.30	38	11.20
5	39	3.30	19	5.60
6	23	1.90	15	4.40

Note. All values are WLSMV estimates from M*plus*. Medication item response categories: 0 = *no use of non-prescription medications in the past 30 days*; 1 = *endorsed one of the medication items*; 2 = *endorsed two medication items*; 3 = *endorsed three medication items*; 4 = *endorsed four medication items*; 5 = *endorsed five medication items*; 6 = *endorsed taking six or more non-prescription medications in the past 30 days (out of a possible 13).*

[a] Collapsed into seven response categories. [b] Treated as an ordinal indicator.

Table 2.6

Intercorrelations of Observed Illness Behavior Indicators Over Time (GENDER sample)

Variable	Somatic		Medication use		Illness disability	
	Q1	*Q2*	*Q1*	*Q2*	*Q1*	*Q2*
Somatic1	1.00	.44***	.24***	.22**	.13***	.13*
Somatic2		1.00	.26***	.36***	.11†	.07
Med1			1.00	.54***	.23***	.04
Med2				1.00	.10†	.05
Disability1					1.00	.31***
Disability2						1.00

Note. *N*s ranged from 273 to 1,183. All values are WLSMV estimates from M*plus*.

† Trending significant at $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .0001$.

Table 2.7

Goodness-of-fit Indices for the Confirmatory Factor Analysis Models of Factorial Invariance in Illness Behavior Across Time (GENDER sample)

Factor model[a]	χ^2	*df*	*p*	$\Delta\chi^2/\Delta$ *df*	*p*	CFI	TLI	RMSEA	(90 % CI)
M1a	6.764	5	.239	---	---	.996	.987	.017	(.000 - .046)
M1b	10.920	11	.450	4.146 / 6[b]	.657	1.00	1.00	.000	(.000 - .030)
M2a	31.412	13	.003	16.623 / 2[c]	.000	.955	.948	.034	(.019 - .050)
M3a	63.803	14	.000	31.302 / 1[d]	.000	.879	.870	.054	(.041 - .068)
M4a	73.574	16	.000	10.857 / 2[e]	.004	.860	.868	.055	(.042 - .068)
M2b	19.001	12	.089	7.45 / 1[f]	.006	.983	.979	.022	(.000 - .040)
M3b	31.662	13	.003	9.86 / 1[g]	.002	.954	.947	.034	(.019 - .050
M4b	47.259	15	.000	15.747 / 2[h]	.000	.921	.921	.042	(.029 - .056)

Note. N = 1,208. CI = confidence interval. Model 1a (M1a) tested configural invariance, with constrained factor loadings of the reference indicator (i.e. somatic complaints) across time, M1b included an additional constraint of equated thresholds for the categorical indicator (i.e. non-prescription medication use) across time, M2 tested weak factorial invariance, with equated factor loadings of the same indicators across time, M3 tested strong factorial invariance, with equated intercepts of the same indicators across

time, and M4 tested strict factorial invariance, with equated variances and residual covariances of the same indicators across time, M2b tested partial weak factorial invariance by removing the equality constraint on the two perceived illness complications loadings across time, M3b tested partial strong factorial invariance with the same two loadings freed, M4b tested partial strict factorial invariance with the same two loadings freed. CFI = comparative fit index; TLI = Tucker-Lewis index; RMSEA = root-mean-square error of approximation.

[a] Unidimensional, single-factor. [b] M1a versus M1b. [c] M1b versus M2a. [d] M2a versus M3a. [e] M3a versus M4a. [f] M1b versus M2b. [g] M2b versus M3b. [h] M3b versus M4b.

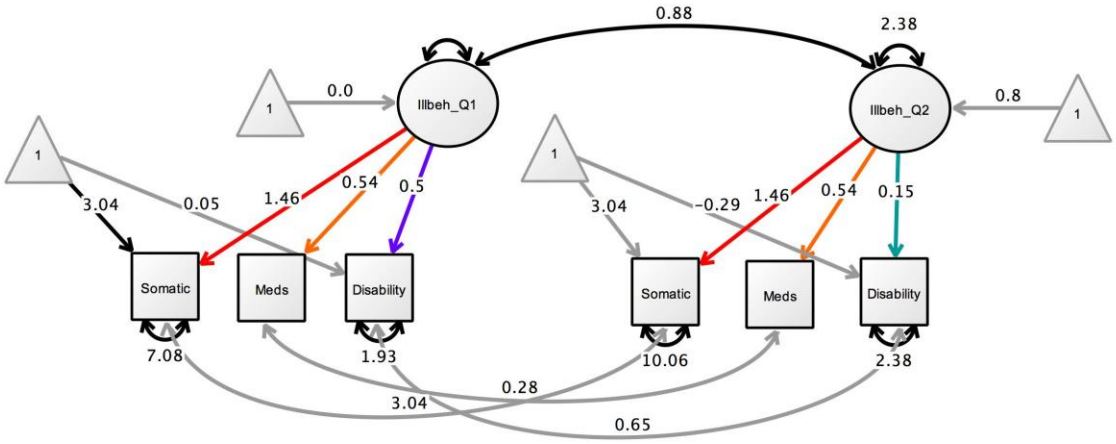


Figure 2.2. Final longitudinal confirmatory factor model of (partial) weak factorial invariance in illness behavior (GENDER sample), consisting of three manifest variables assessed at two measurement waves (Q1 and Q2). Completely unstandardized mean- and variance-adjusted weighted least square (WLSMV) parameter estimates. Paths for equated factor loadings are depicted with the same colors, and paths for the free loadings are depicted in different colors. Intercepts and covariances are in grey.

Table 2.8

Standardized Factor Loadings and Latent Factor Intercorrelation Across Time from Confirmatory Factor Analysis Model of Partial Weak Factorial Invariance (GENDER sample)

Factor	Wave	
Indicators	*Q1*	*Q2*
Illness behavior		
Somatic	.48 (.05)	.50 (.05)
Medication use	.54 (.05)	.69 (.08)
Illness complications	.34 (.04)	.12 (.07) [ns]

	F1	*F2*
F1	1.00	---
F2	.69*** (.10)	1.00

*Note. N*s ranged from 317 to 1,206. Values in parentheses indicate standard errors. All values are WLSMV estimates from M*plus*. For all estimates, except illness complications at Q2, $p < .0001$. Somatic = somatic complaints composite from the CES-D; Medication use = composite of non-prescription medication use; Illness complications = difference score composite of perceived illness complications, adjusted for physician panel ratings. *F1* = illness behavior factor at Q1; *F2* = illness behavior factor at Q2.

Table 2.9

Means, Standard Deviations, Range, and Skewness of Baseline Predictors and Illness Behavior Outcome (SATSA sample)

Variable	*n*	*M*	*SD*	Range		Skewness
				Potential	Actual	
Predictors[a]						
Entry age (years)	1,314	60.26	13.20	29-102	29.1-95.9	-0.28
SES	1,314	0.38	2.66	---	-8.7-6.0	-0.79
CIRS	1,314	2.51	2.99	0-12	0.0-11.0	1.03
Friend	1,314	0.34	5.71	---	-14.6-14.1	-0.02
Family	1,314	0.01	2.99	---	-8.5-6.4	-0.68
Illness behavior factors						
Q2	1,184	49.42	8.80	0-100	31.0-87.4	1.00
Q3	1,037	50.53	9.98	0-100	30.1-93.8	1.02
Q4	1,023	51.03	11.18	0-100	27.8-100.4	1.00
Q5	593	50.94	9.60	0-100	30.4-85.3	0.80

Note. SES = standardized composite of objective socioeconomic status; CIRS = composite of illness comorbidity, adapted from the Cumulative Illness Rating Scale (CIRS) categories; Friend = perceived availability of social support from friends (adapted ISSI subscale); and Family = perceived availability of social support from relatives

(adapted ISSI subscale). Entry age was subsequently centered at 70 years, and illness comorbidity was centered at one (i.e. one bodily system affected by illness). All other predictors were standardized composites ($M = 0$; $SD = 1$). [a] Descriptive information for dichotomously-coded covariates of sex and marital status are not shown. [b] Illness behavior factors were *T*-score scaled to a mean of 50 and a standard deviation of 10.

Table 2.10

Zero-Order Correlations Among Baseline Predictors and Illness Behavior Factors (SATSA sample)

Variable	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*
1. CIRS	1.00	**.14**	**-.18**	-.09**	**.23**	-.09**	-.00	**.56**	**.56**	**.55**	**.52**
2. Sex [a]		1.00	**-.14**	**-.11**	.05*	**-.11**	.09**	**.24**	**.24**	**.25**	**.22**
3. SES			1.00	**.39**	**-.45**	**.22**	.06*	**-.15**	**-.16**	**-.17**	**-.18**
4. Marital[a]				1.00	**-.19**	**.12**	**.12**	**-.11**	-.09*	- .08*	- .05
5. Age					1.00	**-.18**	**.13**	.10**	.10**	.11**	.14*
6. Friend						1.00	**.40**	**-.18**	**-.19**	**-.19**	- .16**
7. Family							1.00	-.09*	-.08*	-.08*	-.06
8. Illbeh1								1.00	**.99**	**.98**	**.94**
9. Illbeh2									1.00	**.98**	**.89**
10. Illbeh3										1.00	**.90**
11. Illbeh4											1.00

Note. *N*s ranged from 593 to 1,314. All values are robust maximum likelihood estimates from M*plus*. Values in bold are significant at $p < .0001$. Illbeh1 = illness behavior factor scores from Q2; Illbeh2 = illness behavior factor scores from Q3; Illbeh3 = illness behavior factor scores from Q4; Illbeh4 = illness behavior factor scores from Q5.

[a] Spearman correlation. *$p < .05$. **$p < .001$.

Table 2.11

Results of Unconditional and Conditional Latent Growth Curve Models of Illness Behavior in SATSA (N = 1,314)

Parameter	Model 1	Model 2	Model 3	Model 4	Model 5
	Means-Only	Unconditional	Random intercept[a]	Random intercept & slope[b]	Equated friend & family[c]
Means or intercepts					
i (level)	50.68 (.29) ***	49.82 (.29) ***	43.01 (.53) ***	42.19 (.56) ***	42.15 (.56) ***
s (slope)	----	0.13 (.01) ***	0.14 (.01) ***	0.15 (.02) ***	0.16 (.02) ***
Covariate regressions					
i on					
CIRS	----	----	2.54 (.14) ***	2.50 (.14) ***	2.50 (.14) ***
Sex	----	----	2.62 (.46) ***	2.63 (.46) ***	2.74 (.46) ***
SES	----	----	-0.11 (.10)	-0.04 (.10)	-0.04 (.10)
Marital	----	----	-0.43 (.53)	-0.32 (.53)	-0.27 (.53)
Entry age	----	----	-0.19 (.02) ***	-0.20 (.02) ***	-0.19 (.02) ***

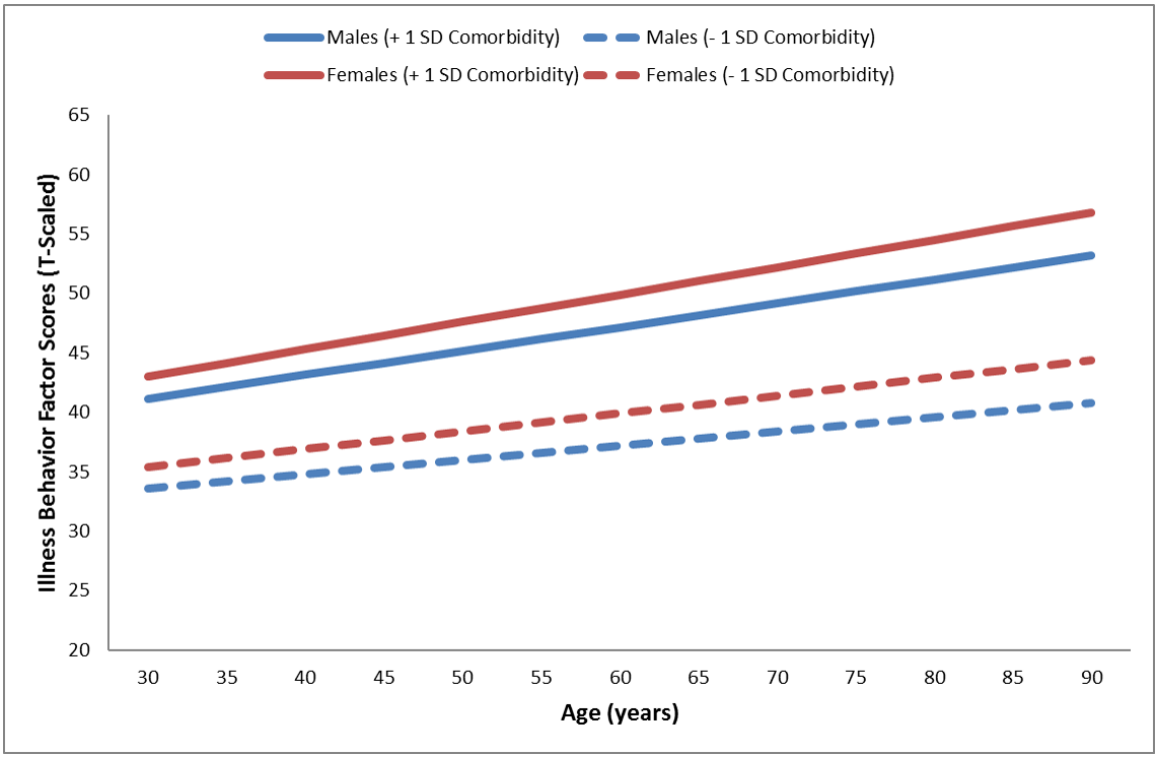
Friend	----	----	-0.18 (.04) ***	-0.99 (.25) ***	-0.71 (.13) ***
Family	----	----	-0.14 (.09)	-0.44 (.26)	-0.71 (.13) ***
s on					
CIRS	----	----	___	0.02 (.01) *	0.02 (.01) *
Sex	----	----	----	0.03 (.02)	0.03 (.02)
SES	----	----	----	-0.01 (.01)	-0.01 (.01)
Marital	----	----	----	0.01 (.02)	0.01 (.02)
Entry age	----	----	----	0.00 (.00) ***	0.00 (.00) ***
Friend	----	----	----	-0.02 (0.01)	-0.02 (0.01)
Family	----	----	----	0.02 (0.01)	0.02 (0.01)
Variances, residual variances, and covariance					
i (level)	95.19 (5.12) ***	83.20 (4.61) ***	53.21 (2.86) ***	53.66 (2.87) ***	53.72 (2.87) ***
s (slope)	----	0.05 (.01) ***	0.04 (.00) ***	0.04 (.00) ***	0.04 (0.00) ***
Covariance of *s* and *i*	----	0.55 (.11) ***	0.38 (.07) ***	0.34 (.07) ***	0.35 (.07) ***
Model fit indices					

Log-Likelihood	-11310.64	-11006.44	-10739.04	-10706.80	-10707.66
Parameters	3	6	13	20	19
Scaling correction	1.87	1.57	1.28	1.24	1.25
AIC	22627.29	22024.87	21504.09	21453.60	21453.32
BIC	22642.83	22055.96	21571.44	21557.22	21551.75
$\Delta\chi^2$ / Δ *df*	----	477.71 / 3***	548.27 / 14***	55.68 / 7***	1.60 / 1^{ns}

Note. Values in parentheses indicate standard errors. All values are robust maximum likelihood estimates from M*plus*.

[a] Conditional intercept-as-outcome model with the slope estimated as a fixed effect. [b] Conditional intercept- and slope-as-outcome model, using a DEFINE statement to equate the scales of the friend and family support predictors. [c] Conditional, intercept- and slope-as-outcome model constraining the effects of friend and family support variables to equal.

*$p < .05$. **$p < .01$. ***$p < .0001$.


Males (+ 1 SD Comorbidity)
Males (- 1 SD Comorbidity)
Females (+ 1 SD Comorbidity)
Females (- 1 SD Comorbidity)
Illness Behavior Factor Scores (T-Scaled)
65
60
55
50
45
40
35
30
25
20
30
35
40
45
50
55
60
65
70
75
80
85
90
Age (years)

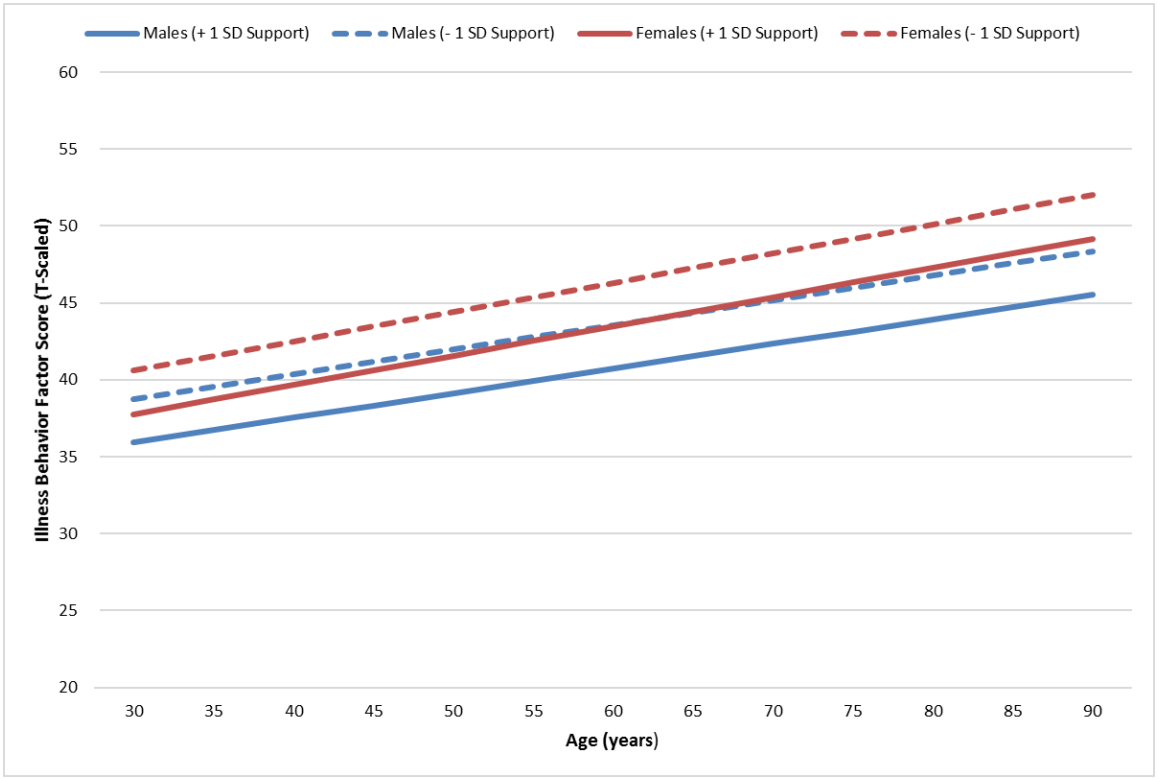

Males (+ 1 SD Support)
Males (- 1 SD Support)
Females (+ 1 SD Support)
Females (- 1 SD Support)
Illness Behavior Factor Score (T-Scaled)
60
55
50
45
40
35
30
25
20
30
35
40
45
50
55
60
65
70
75
80
85
90
Age (years)

Figure 2.3. a) Expected mean trajectories of illness behavior (T-scaled factor scores) across age for males and females, per one-unit standard deviation increase or decrease in illness comorbidity (for unmarried individuals of average SES and average levels of social support, who began the study at age 70); b) Expected mean trajectories of illness behavior (T-scaled factor scores) across age for males and females, per one-unit standard deviation increase or decrease in support availability from friends and family members (for unmarried individuals of average SES and one chronic health condition who began the study at age 70).

Chapter Three:

Psychosocial Antecedents and Functional Consequences of Illness Behavior Trajectories Among Older Adults

Introduction

The world population is aging, with older adults making up 16% of the population in developed countries (an increase from 8% in 1950). Older adults account for a disproportionate percentage of contact with the healthcare system, and one-third of all pharmaceutical drug prescriptions in the United States (Bradley & Hughes, 2013; Population Reference Bureau, 2013). It is estimated that by the year 2030, one out of five Americans will be age 65 years or older, owing in part to widespread increases in longevity (U.S. Census Bureau, 2000; cited in Rice & Fineman, 2004). Furthermore, within this group, the proportion of the oldest-old (defined as age 85 and above) is increasing; because health expenditures are often concentrated within the final years of individuals' lives, this demographic shift carries tremendous implications for the future of U.S. healthcare (Rice & Fineman, 2004). Moreover, although reduced mortality from conditions like stroke and heart disease can be attributed to improvements in both lifestyle factors and medical treatment, older adults experience more chronic health conditions and disability than other age groups, despite slowing rates of disability in recent years (Rice & Fineman, 2004). In the recent "Aging in America" survey report, U.S. adults ages 65 years and older rated loss of independence as their greatest fear, whereas relatively few people mentioned a fear of death (26 % versus 3 % of the sample; Prince & Butler, 2007). Therefore, with respect to public health interventions and policies

aimed at increasing the efficiency of health care delivery, reducing health expenditures due to over- and under-utilization, and improving individual health in late-life, addressing functional health in this particularly vulnerable age demographic is crucial.

Functional decline, defined as limitations in physical functioning or a restriction of one's daily activities (McKusker, Kakuma, & Abrahamowicz, 2002), is one of many indicators of healthy aging, as it predicts higher mortality risk, permanent disability, and institutionalization (Buurman et al., 2011). Although disability tends to increase with advanced age, there is also a high degree of variability in the onset and rate of decline (Buurman et al., 2011). Functional status and decline is measured objectively, via tasks evaluating grip strength, walking, balance, or chair stands (Finkel, Ernsth-Bravell, & Pedersen, 2015), or subjectively, via questionnaires regarding difficulties with basic or instrumental activities of daily living (ADLs and IADLs, respectively).

The more frequent and timely use of medical services observed among older adults as compared to young and middle-aged adults is posited to stem from a natural shift toward resource conservation strategies across development (Baltes & Baltes, 1990), whereby older adults integrate their physical limitations and acknowledge—perhaps subconsciously—a reduced ability for self-care (Egan & Beaton, 1987; Leventhal & Crouch, 1997). Indeed, one study evaluating recent trends in functional decline among a nationally representative, community-dwelling sample of older adults in the United States reported that, across a 15-year period, improvements were exclusive to routine limitations in instrumental activities of daily living (IADLs; e.g., shopping, using the phone), whereas the more severe limitations in basic activities of daily living (ADLs; e.g.,

bathing, eating, dressing) remained relatively stable (Schoeni, Freedman, & Wallace, 2000). Other research with the U.S. Health and Retirement Study (HRS), however, has reported the opposite trend, such that the IADL trajectories of older adults ages 75 years and above remained relatively flat across a nine-year follow-up period from 1995 to 2004, whereas the trajectories for ADL difficulty showed some decline, as did the onset of these limitations (Freedman, Martin, Schoeni, & Cornman, 2008). However, regardless of where such functional improvements lie, the concept of "successful aging" that was introduced into the field by Rowe and Kahn (1987) has challenged disease-oriented views of the aging process. Once described as a negative, deterministic process resulting in inevitable cognitive, physical and functional declines up until death, researchers now acknowledge the potential of various psychosocial factors (e.g., improved education, lifestyle behaviors) to buffer or prevent negative aging outcomes, as well as normative trends of older adults leading longer, independent lives in their communities (Rice & Fineman, 2004). For example, Freedman and colleagues found that early- and mid-life factors, such as lifetime occupation and changes in educational attainment, predicted reduced onset of functional difficulty (Freedman, Martin, Schoeni, & Cornman, 2008).

Furthermore, with regards to initiating and maintaining health behavior change, in some ways older adults might have an easier time maintaining health promotion behaviors over longer periods of time as compared to middle-aged and younger adults, despite facing potentially greater challenges to initiating the change effort in the first place (Carstensen & Hartel, 2006). Although researchers have evaluated disease (i.e. history of chronic conditions and comorbidities) and functional status as need-based

determinants of older adults' healthcare utilization (Andersen's behavioral model; cf. Andersen, Stedmy, & Anderson, 1970; Andersen, 1995; Andersen et al., 2014) the role of change or maintenance in the perceptions, evaluations, and responses to symptoms that precedes health utilization—or illness behaviors—in predicting subsequent rates of functional decline (or improvements) into late adulthood is unclear.

Findings from health psychology suggest that the simple act of visiting the doctor can provide patients with the temporary psychological coping resource of perceived personal control (Martin, Haskard-Zolnierek, & DiMatteo, 2010). This response might be particularly motivating in older adulthood, when health threats are more likely to occur. However, prospective research has yet to explore the long-term health consequences of high- or low-responsiveness to symptoms, particularly in the later years of life, when aggressive medical treatments can sometimes pose a greater risk to health and longevity than receiving no treatment at all (Wallis, 2015). If older adults, on average, are in fact experiencing slower rates of functional decline, distinguishing whether these improvements can be attributed to lifestyle (e.g., health- and illness- behaviors, or self-care) and social factors (e.g., social support or engagement), as compared to increased access and use of medical services, will inform both targeted interventions for older adults at-risk and health policy (Schoeni et al., 2000). Thus, the current study examined the predictive value of social support and illness behavior trajectories on subsequent functional status and change in late adulthood, with an emphasis on illness behavior as a possible mediating pathway.

Social support and functional health

In particular, social support provides a promising target for intervention. Decades of research examining relationships and health in late-life has consistently reported that older adults who are socially "connected" or "integrated" experience better physical and mental health outcomes, including a stronger immune system, increased longevity, and reduced depression risk. Social connectedness, however, is a broad construct, encompassing a wide range of social network dimensions, each with potentially varying relationships to health outcomes. For example, interpersonal network size (i.e. total number of close confidantes or relationships) and network density (i.e. how close network members are with one another) are posited to increase the likelihood that an individual will access useful information and other valuable resources relevant to disease management and health maintenance (Berkman, Glass, Brissette, & Seeman, 2000; Berkman & Syme, 1979); whereas the perceived availability or *quality* of such relationships is suggested to influence health through increased well-being, feelings of belongingness, reductions in negative affect or depressive symptoms, and even normative pressures for performing certain health behaviors that are especially strong within these highly valued relationships (Gallant, 2013). Additionally, previous research shows that older adults' social networks become increasingly family-centered over time (McPherson, Smith-Lovin, & Brashears, 2006); consequently, older adults are especially likely to involve close others, particularly children or other family relationships, in medical decision-making and conversations about their health (Cornwell, Schumm, Laumann, & Garber, 2009). This shift, in turn, may lead to better self-care practices or

even improved medical treatment. In other words, some social network dimensions may influence health behaviors or outcomes indirectly, through affective (i.e. reductions in depressive symptoms) or cognitive (i.e. decision-making, self-efficacy) processes, whereas others may have more direct associations.

With regards to more direct influences on disease management behaviors, emotional support, which includes interpersonal behaviors like the expression of empathy, predicts both timely diagnosis and better control of chronic conditions like hypertension in late adulthood (Cornwell & Waite, 2012). Additionally, tangible social support, or the provision of practical assistance, is suggested to have direct associations with disease management behaviors like medication adherence or clinic visits (DiMatteo, 2004); someone with more tangible support might utilize health services more frequently simply because a network member has offered transportation, money, or another form of direct assistance. In light of this promising hypothesis, the current proposal will examine direct associations between the availability of emotional and tangible support from friends and family members and illness behavior trends.

Although both structural and interpersonal elements of social networks are associated with health outcomes and behaviors across the lifespan, most prior work has examined one or two aspects at a time, rather than examining their potentially synergistic pathways toward health and well-being (Cornwell, Laumann, & Schumm, 2008; Gallant, 2013). Applying a broader approach to examine the mechanisms of these various aspects has only recently been a focus of aging research (Cornwell et al., 2008). Furthermore, the literature has included structural (e.g., size, density, type or composition) and

functional (e.g., support availability) measures to assess both interpersonal and community-based social engagement. Interpersonal social engagement includes older adults' dyadic relationships with primary network members (e.g., family, spouses, friends), whereas community engagement refers to the extent of social engagement with the broader community (e.g., religious attendance, interactions with neighbors, volunteerism). In recent work, the social engagement trends most strongly associated with increasing age (through ages 70 to 80 years) are decreased network size, reduced feelings of closeness, and more frequent religious attendance and volunteerism (Cornwell et al., 2008). Importantly, these age effects were independent from other age-related transitions, such as bereavement, retirement, and physical health, both self-rated and functional status (Cornwell et al., 2008). Moreover, although some work has suggested that older adults' social engagement might be dependent on physical activity and other health indices, at least one study has shown that social interactions in late-life are not confounded by such factors; physical activity and social contact have both independent and interacting effects on rates of functional decline (Unger, Johnson, & Marks, 1997). In fact, in some cases, declining health and other later-life transitions (e.g., bereavement) may serve to increase older adults' social contact with family or other primary network members (Cornwell et al., 2008). Together, these findings point to the benefits of structural, longitudinal research approaches that allow for the inclusion of multiple social support measures and their simultaneous pathways to illness behavior and late-life functioning.

Finally, it is clear that men and women experience differences not only in their social network characteristics, but also in associations between these network facets and health outcomes. For example, women tend to have larger, more emotionally connected support networks, and report having a person to confide in, whereas males tend to have smaller, less emotionally supportive, networks (Taylor, 2011). There are also gender differences in the relative importance of various sources of support, such that friends may have a greater influence on women for enacting certain types of health behavior change, like dieting, for example (Kelsey et al., 1996). Of course, such gender differences may depend on the behavior in question. Lastly, there are widely reported gender differences in the frequency of health utilization and other illness behaviors, with women consistently seeking out more medical care and reporting more symptoms as compared to men (Gold et al., 2002; Mechanic, 1977; Vedsted & Christensen, 2005; Verbrugge, 1989). Thus, the current study will evaluate gender as a covariate in hypothesized associations between social support, illness behaviors, and functional status.

Apart from gender differences in social relationships' associations with behavioral pathways to health, the way social relationships influence health outcomes directly may also differ for men and women. For example, one study found that frequent social interactions buffered the effects of widowhood on functional declines among women only (Unger, Johnson, & Marks, 1997). Additionally, a recent meta-analysis on the social relationship-mortality link found the mortality risk of weak social connections was comparable to that of smoking 15 cigarettes per day, and twice the risk of obesity (Holt-Lunstad, Smith, & Layton, 2010). This toxic effect is posited to stem from chronic

feelings of threat, which weakens the immune system over time (Cole, Hawkley, Arevalo, & Cacioppo, 2011). The negative implications of loneliness, combined with the looser nature of male's social networks, may partially explain this gender difference in the buffering effects of social contacts.

Aims & Hypotheses

The first aim of the current study was to examine the prospective associations between social support availability from friends and family members and subsequent functional status, adjusting for baseline health status and other demographic variables. Based on prior work, functional status was expected to follow a discontinuous trend across age, with relative stability prior to age 75 years, and a steeper rate of decline afterward. Furthermore, perceived availability of both sources of social support (i.e. friend and family) were hypothesized to predict higher levels of physical functioning and to buffer the rate of functional decline after age 75.

The second aim of this study was to evaluate illness behavior status and trajectories as mediators of the association between the aforementioned social support variables and functional status outcomes (both levels and change). This hypothesis is underscored by recent work that identified older adults' healthcare utilization, but not other types of health behavior, as a partial mediator of the relationships of social network dimensions (i.e. larger networks and emotional support) with the timely diagnosis of hypertension and blood pressure control (Cornwell & Waite, 2012). In another study, physician utilization was significantly associated with subsequent functional status, such that higher levels of physician utilization predicted lower levels of functional ability

across a six-year assessment period in the Longitudinal Study of Aging (LSOA) (Wolinsky, Stump, Callahan, & Johnson, 1995). Together, these findings suggest that older adults' illness behaviors might represent a unique pathway, distinct from other preventive health or self-care behaviors, to maintaining functional health across late adulthood.

Methods

Participants

Participants in the present study's first set of latent growth analyses were 1,310 twins (915 pairs; M_{age} at baseline = 60.51 years, *SD* = 13.34; 57 % female), ages 29 to 96 years, from the population-based Swedish Adoption/Twin Study of Aging (SATSA), drawn from the Swedish Twin Registry (Finkel & Pedersen, 2004; Pedersen et al., 2013). Analyses included available data on functional status across six questionnaire waves (Q2-Q7: 1987-2010), as well as participant sex, SES, comorbidity, and depressed mood. Participants were included in the analysis if they had at least one wave of functional status data across the six questionnaires, and if they provided complete data on the baseline (Q2) predictors. For the second set of analyses on longitudinal mediation, participants were 1,314 twins (910 pairs; M_{age} at baseline = 60.26 years, *SD* = 13.20, 57.46 % female). These analyses included available data on illness behavior (i.e. a factor score created from confirmatory factor analysis with four indicators; see Study 1a) across four questionnaire waves (Q2-Q5: 1987-2004); functional status data (i.e. physical activities of daily living) across six questionnaire waves (Q2-Q7: 1987-2010); and participant sex, SES, and comorbidity. Participants were included in the analysis if they

had at least one wave of functional status data across the six questionnaires, at least one wave of illness behavior data across the four questionnaires, and if they provided complete data on the baseline (Q2) predictors. Psychosocial predictors and covariates were included from the baseline assessment, which, for these analyses, was the 1987 questionnaire (Q2). This was done because 1987 questionnaire was the first assessment wave that included separate items pertaining to friend and family support availability; the SATSA intake questionnaire in 1984 (Q1) did not differentiate these two sources of support.

Measures

Outcomes

Functional Status. The functional status outcome was a composite of seven items on difficulty performing the following physical activities of daily living (adapted from the ADL Index; Katz, Ford, Moskowitz, Jackson, & Jaffe, 1963): eating, transfer in and out of bed, bathing, dressing, grooming, walking, and toileting. Participants reported on their degree of difficulty performing each task on a three-point scale (0 = *No difficulty*; 1 = *Some difficulty*; 2 = *A lot of difficulty*). The composite was calculated across six questionnaire waves (1987-2010), with higher scores reflecting greater difficulty with these tasks, or worse physical functioning. The toileting item was missing from the 2007 questionnaire (Q6) and was therefore imputed with the score from the previous questionnaire (Q5: 2004). Participants who were missing more than one ADL item did not receive a composite score at that wave. To address the positive skew and increase model stability, the composite was square-root transformed and multiplied by 10, such

that a score of 10 on the transformed composite (M_{1987} (n = 1,304) = 10.52, SD = 2.06; M_{2010} (n = 436) = 11.47, SD = 3.56; range = 10.0 to 37.4) was equivalent to a 0 (i.e. no difficulty) on the raw scale.

Illness behavior. Illness behavior measures were factor scores output from a prior confirmatory factor analysis of strict factorial measurement invariance across the four questionnaire waves (Q2-Q5; see Study 1a). The illness behavior factor scores were based on four observed indicators at each wave, including the somatic complaints subscale of the CES-D, non-prescription medication use, pain-related disability, and perceived illness complications. The factor scores were included from each of four questionnaire waves (Q2-Q5: 1987-2004), and were subsequently T-score scaled to have a mean of 50 and a standard deviation of 10.

Predictors

Age (years). Participants' ages at each wave (in year units) were included as the time predictor of functional status. Age was centered at 75 at each wave, based on prior work in the SATSA sample which examined change in functional capacity from the in-person testing measures and other work on the ADL questionnaire items. This work found very little change in all functional status measures (i.e. flexibility, balance, fine motor skills, and activities of daily living) prior to the ages of 70 and 75 years, respectively, with steeper increases in performance difficulty and variability afterward (Finkel, Ernsth-Bravell, & Pedersen, 2015; Foebel et al., 2015).

Social support availability (baseline). Two standardized composites adapted from the Interview Schedule for Social Interaction (ISSI; Henderson, Duncan-Jones, &

Byrne, 1980; Eklund, Bengtsson-Tops, & Lindstedt, 2007) were included from the 1987 questionnaire (Q2) to index perceived availability of contact, shared interests, intimacy, and support received from friends and relatives.

Friend support. The availability of support from friends and acquaintances (e.g., "individuals in the neighborhood") was a standardized sum of eight items (scores ranged from – 14.59 to 14.10) on participants' reports of the number of individuals or friends who meet or talk on the phone with them in an ordinary week; who share their interests, feelings, and joy; who could drop in on them at any time; who they could ask for things; and who can provide support.

Family support. The availability of support from relatives was a standardized composite of four items (scores ranged from - 8.51 to 6.41) on participants' reports of the number of relatives who meet or talk on the phone with them in an ordinary week, who they can share their innermost feelings and joy with, and who can provide support.

Covariates

Sex. Participants' self-reported sex was included from baseline and coded dichotomously (0 = *male*; 1 = *female*).

Socioeconomic status (baseline). Participants' objective socioeconomic status was measured using six self-report items, which were each standardized ($M = 0$; $SD = 1$) and summed into a composite ($M = 0.35$, $SD = 2.67$; range = - 8.67 to 6.00). Participants reported on their income, home ownership, whether or not they received a rent subsidy, and the number of cars they owned.

Depressed mood (baseline). This measure was a simple composite of six items from the depressed mood subscale of the Center for Epidemiological Studies-Depression scale (CES-D; Radloff, 1977), the most commonly used, and cross-nationally validated, measure of self-reported depressive symptomatology in adults (Gatz, Johansson, Berg, Pedersen, & Reynolds, 1993). For the depressed mood subscale, participants indicated the frequency of experiencing feelings of sadness and worthlessness (i.e. felt depressed, afraid, lonely, sad, and had crying spells) in the past week prior to testing, with each item on a four-point scale (0 = *Never*; 3= *Always/Almost always*). Possible scores on this subscale ranged from 0 to 18.

Illness comorbidity (baseline). Illness comorbidity was a simple composite adapted from the Cumulative Illness Rating Scale (CIRS; Linn, Linn, & Gurel, 1968). Participants completed a checklist including 35 medical conditions, endorsing whether a diagnosis was present for each item (0= *No*; 1= *Yes*). All endorsed health conditions were subsequently grouped into categories reflecting the organ system affected (e.g., cardiovascular, musculoskeletal, respiratory, endocrine). In the original scale, the composite is calculated from the sum of the number of impaired organ systems (out of a possible 14) and weighted by physician ratings of severity of impairment (on a five-point scale from 1 = *None/No impairment to that organ/system* to 5 = *Extremely Severe/Impairment is life-threatening*). In cases where multiple diseases are endorsed within a single organ system, only the most severe illness is rated for severity. This scale has demonstrated reliability (ICCs = .80 to .83; de Groot, Beckerman, Lankhorst, & Bouter, 2003; Miller et al., 1992; Rochon et al., 1996) and validity (Conwell, Forbes,

Cox, & Caine, 1993; Hudon, Fortin, & Vanasse, 2005; Miller et al., 1992; Salvi et al., 2008). In SATSA, an adapted composite was calculated from a simple sum of impaired organ systems out of a possible 12 (actual scores ranged from 0 to 11). Corresponding physician severity (i.e. life-threatening) ratings were not available, however, so composite scores were not adjusted for severity. In the present study, the composite was also centered at one, such that the interpretation of the variable's effect on illness behavior was the change resulting from illness comorbidity as opposed to the change in illness behavior from endorsing any one medical condition. Eighty-six percent of participants reported having at least one medical condition at baseline.

Statistical Analysis

First, bivariate relationships among the predictors, covariates, and outcomes were examined. For the first set of analyses, longitudinal growth curve models in M*plus* version 7.4 (Muthén & Muthén, 2012) were fitted to evaluate the average age-related trajectory of change in functional difficulty (i.e. activities of daily living) across the six SATSA questionnaires waves considered (1987-2010), as well as the relative predictive value of friend and family support availability. A path diagram of the hypothesized growth model is displayed in Figure 3.1. All analyses adjusted for nesting within twin pairs. The robust maximum likelihood (MLR) estimator was used to account for violations of normality among the outcome measures, and the TSCORE option was also included to account for the variability in participants' ages at each assessment wave. Furthermore, the missing data option was specified to make use of all available data; no pairwise or listwise deletion was applied to missing data. In the first model (LGM1), a

means-only model, the degree of between-individual variability in functional status was evaluated. The second model (LGM2) of unconditional growth added a within-person time predictor to evaluate the annual rate of linear change in physical ADL difficulty as a function of participants' age at each questionnaire wave, which was centered at 75 years. This decision was made a priori, based on prior work in the SATSA sample that examined change in physical functional status measures from the in-person testing assessments (Finkel et al., 2015) and from the questionnaires (Foebel, Zavala, Ernsth Bravell, Reynolds, & Pedersen, 2015). Finkel and colleagues found little change in three objectively measured factors of balance, flexibility, or fine motor skills prior to age 70 years, with steeper declines seen afterwards (Finkel et al., 2015). In contrast, the questionnaire measures showed a faster rate of impairment after age 75 (Foebel et al., 2015). This pattern was also confirmed in exploratory ordinary least squares (OLS) trajectories of the self-reported functional difficulty composite included across the questionnaire waves.

In consideration of this finding, another unconditional model of piecewise growth in ADL difficulty was tested (i.e. a spline model; LGM3). This model included two latent slopes: one to capture linear change in ADL difficulty prior to the age of 75 years (slope 1), and another to capture a different rate of linear change after 75 (slope 2). Once a best-fitting unconditional change model was established, two conditional models evaluated social support availability from friends and family members as predictors of functional difficulty status at age 75 (i.e. intercept-as-outcome model, or LGM4), and the extent to which family and friend support availability predicted both functional status at age 75

and change in functional status over time (i.e. random intercept and slope model, or LGM5; see Figure 3.1). In both conditional models, predictors and covariates from the Q2 (baseline) assessment were entered in the following order: participant sex, SES, depressed mood, illness comorbidity (i.e. the adapted CIRS measure), friend support availability, and family support availability. No centering method was applied to the predictors of friend and family support availability, SES, or depressed mood, because these composites already had meaningful zero-points. Participant age was centered at 75 across the six questionnaires, and disease comorbidity was centered at the value of one (i.e. one reported organ system affected by illness). Sex was dummy-coded.

For the second set of analyses, longitudinal mediation models in a latent growth curve framework were fitted, again in M*plus* version 7.4 with robust maximum likelihood (MLR) estimation. These models evaluated the extent to which illness behavior trends predicted subsequent functional status at age 75 and rates of functional difficulty; and whether illness behavior trends mediated the association between support availability and ADL difficulty (i.e. level and change after age 75). The application of longitudinal mediation methods within latent growth curve models allows for individuals' latent intercepts and slopes to serve as predictors, mediators, or outcomes, and it also enables the researcher to simultaneously evaluate the joint, structural relationships among multiple predictors, mediators, and outcomes (Gunzler, Chen, & Zhang, 2013; Preacher, 2015; Selig & Preacher, 2009). In other words, it allows for both the average rate of intra-individual change and inter-individual differences in that change to be included in theories and tests of mediation (Nesselroade, 1991). In the full mediation model (see

Figure 3.3), a latent intercept and linear slope were regressed onto the four repeated-measures of observed illness behavior factor scores (from across Q2-Q5 in SATSA). The illness behavior latent intercept (centered at age 60 years) and slope were both evaluated as mediating variables in the structural paths from friend and family support availability to the latent intercept and slope of ADL difficulty. Cohen's *d* effect sizes for the slope effects were calculated by multiplying the change in the average growth rate by time, and dividing by the standard deviation of raw scores (c.f., Feingold, 2009, Equation 7, p. 47), although there is lack of clarity in the literature on effect size calculations in latent growth modeling contexts.

All analyses were adjusted for dependency of the data and demographic variables. The missing data option was specified to make use of all available data on illness behavior and ADLs, and the TSCORE option was also used to accommodate participants' varying ages at each assessment wave. The latent intercept and second slope of ADL difficulty, and the latent intercept and slope of illness behavior, were all adjusted for participant sex, SES, and illness comorbidity. Grand-mean centering was used for the age predictor (at age 60 years) in the estimation of illness behavior intercept and slope, whereas the age predictor in the ADL difficulty intercept and slope estimation was centered at 75 based on prior empirical findings and theory. Comorbidity was centered at one (i.e. one organ system affected by illness), and sex was dummy-coded. Social support availability measures and SES were standardized composites with meaningful zero-points representing the average or grand mean. Therefore, no centering method was used.

For both sets of analyses, nested model comparisons were conducted to evaluate fit to the observed data. Goodness-of-fit indices for these model comparisons included the Satorra-Bentler chi-square difference test statistic (Bryant & Satorra, 2012; Satorra & Bentler, 2010) with the formula specified by Muthén and Muthén (2010) (http://www.statmodel.com/chidiff.html) for robust maximum likelihood estimation, as well as the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC). For the longitudinal mediation analysis, the first model comparisons were conducted between the full mediation model and two nested models dropping the indirect (*a* and *b*) paths—from friend and family support availability to the mediators (i.e. illness behavior latent intercept and slope), and from illness behavior to the outcomes (i.e. functional status and change after age 75), respectively (c.f., Cole & Maxwell, 2003). In the next model comparison, the full mediation model was compared to a model omitting the direct (c') path from friend and family support availability to functional status and change after age 75, and adjusted for illness behavior's indirect effect. Model misfit was evaluated with the Satorra-Bentler chi-square difference test statistic (Bryant & Satorra, 2012; Satorra & Bentler, 2010), AIC, and BIC. In addition, the MODEL CONSTRAINT command in M*plus* was used to estimate the specific indirect, direct, and total effects (i.e. separating the mediating paths that predict ADL status versus the mediating paths that predict ADL change), as well as the overall indirect, direct, and total effects. For statistical inference of the indirect effect, the following measures of effect size were also calculated: the ratio of the indirect to the total effect to index the proportion mediated

(P_M), and the ratio of the indirect effect to the standard deviation in ADL difficulty, or the partially standardized indirect effect (ab_{ps}; Preacher & Kelley, 2011).

Results

Descriptive Information on Predictors and Functional Status Outcomes

The means and standard deviations for the predictors (see Table 3.1) suggested that most participants reported being of average SES. Most of the sample also reported having at least two CIRS-based organ systems affected by illness (M = 2.54, SD = 3.01; out of a possible 12) and relatively few depressive symptoms (M = 2.30, SD = 3.05; out of a possible score of 18). The means and standard deviations of the ADL difficulty composites (the raw composites were square-root transformed and multiplied by 10) across the six questionnaire waves suggested an incremental increase in reported levels of functional difficulty over time (M_{Q2} = 10.52 to M_{Q7} = 11.47) as well as increasing variability (SD_{Q2} = 2.06 to SD_{Q7} = 3.56), except for at the Q6 assessment. Univariate tests of skewness revealed the ADL difficulty composites were still positively skewed after performing the transformation, with all skewness values exceeding 3.0, albeit decreasing slightly across the waves. Comorbidity and depressed mood variables were also slightly positively skewed, but not problematically so (with values of 1.02 and 1.61, respectively). Growth models were estimated using robust maximum likelihood (MLR), which is more robust to issues of non-normality than maximum likelihood (ML) estimation.

Zero-order correlations among the predictors and outcomes are displayed in Table 3.2. Pearson correlations among the ADL composites suggested a high degree of rank-

order stability in functional difficulty across the waves, with moderate-to-strong positive associations ranging from $r = .36$ ($p < .0001$; Q2 with Q6) to $r = .94$ ($p < .0001$; Q5 with Q6). Friend support availability at baseline (Q2) was weakly and negatively correlated with ADL difficulty across the assessments, with values ranging from $r = -.10$ (ns) to $r = -.18$ ($p < .01$), whereas family support availability was less consistently associated (*r*s ranged from $r = -.02$, *ns*; to $r = .08$, $p < .05$). Additionally, friend and family support availability were moderately and positively correlated ($r = .40$, $p < .0001$). Greater comorbidity was positively correlated with ADL difficulty across the waves (*r*s ranged from $r = .25$, $p < .0001$; to $r = .33$, $p < .01$), as was higher depressed mood (*rs* ranged from $r = .11$, *ns*; to $r = .18$, $p < .0001$). SES had weak-to-moderate negative associations with ADL difficulty (*r*s ranged from $r = -.16$, $p < .0001$; to $r = -.33$, $p < .05$). Females reported slightly more ADL difficulty than males (*r*s ranged from $r = .06$, $p < .05$; to $r = .11$, $p < .0001$), whereas married individuals were less likely to report ADL limitations than those who reported being never married, divorced, or widowed (*r*s ranged from $r = -.09$, $p < .01$; to $r = -.18$, $p < .01$).

Latent Growth Model

Model estimates and expected trajectories for the final latent growth curve models of ADL difficulty fitted to SATSA data are presented in Table 3.3 and in Figure 3.2, respectively. Results from the unconditional linear change model (LGM2), suggested an age-based, linear model of change in functional difficulty had a better fit to the data as compared to the means-only model (LGM1) ($\Delta \chi^2 / \Delta df = 173.28 / 3$, $p < .0001$). The linear model revealed a small, yearly increase in self-reported functional difficulty after

age 75 years (b_1 = .05, p < .0001) from a low (i.e. near-zero) level of reported ADL difficulty at age 75 (b_0 = 11.18, p < .0001). The next unconditional growth model (LGM3) evaluated piecewise linear growth in functional difficulty with two slopes (i.e. one fixed to stability prior to age 75 years, and another estimating the rate of change after age 75). This model had a better fit to the data as compared to the means-only model ($\Delta \chi^2 / \Delta df$ = 313.60 / 3, p < .0001), and it also had lower AIC and BIC values relative to the unconditional linear growth model. Therefore, the spline model was chosen as the best-fitting unconditional model of change. In this model, the estimate for the latent intercept again reflected almost no reported ADL difficulty at age 75 (b_0 = 10.45, p < .0001), with a small, yearly linear increase in ADL difficulty afterward (b_2 = .30, p < .0001).

Next, a conditional intercept-as-outcome model (LGM4) was fitted, which regressed the latent ADL intercept (b_0 = 9.98, p < .0001) onto the observed predictors and covariates while treating the latent slopes as fixed effects. This model improved fit relative to the unconditional spline model ($\Delta \chi^2 / \Delta df$ = 71.13 / 6, p < .0001), and it suggested that neither perceived availability of support from friends nor support availability from family members predicted functional difficulty levels at age 75 (b_{05} = -.01, p > .05; and b_{06} = -.01, p > .05; respectively). The next conditional model (LGM5) added random variation around the second latent slope (i.e. after age 75) as well as the intercept. This model had the best fit with the data and improved fit relative to the previous, more constrained, intercept-as-outcome model ($\Delta \chi^2 / \Delta df$ = 17.93 / 6, p < .01). Furthermore, it suggested that friend support availability predicted neither the intercept nor the second slope of ADL difficulty (b_{05} = -.01, p > .05; and b_{25} = -.01, p > .05;

respectively). Family support availability, however, positively predicted the ADL difficulty slope after age 75 (b_{26} = .03, p < .05), such that a one-unit increase in perceived availability of support from family members relative to the average was associated with a Cohen's *d* of .07, or a mean difference of .07 standard deviation units in the annual linear rate of ADL difficulty after age 75, across a 5-year period (i.e. age 75 to 80 years). Family support availability did not significantly predict the ADL intercept, however (b_{06} = -.01, p > .05).

Regarding the covariates, females reported slightly higher ADL difficulty at age 75 than males (b_{01} = .18, p < .10) and higher SES also positively predicted the intercept (b_{02} = .04, p < .10). Both effects were at trend significance, however. Higher depressed mood predicted higher ADL difficulty status (b_{03} = .05, p < .01), such that for every one-unit increase in depressive symptoms, reported ADL difficulty could be expected to increase by .05 units (a Cohen's *d* effect size equivalent of .02). Greater comorbidity predicted an increased level of ADL difficulty at age 75 (b_{04} = .17, p < .0001), such that for every additional reported organ system affected by illness, reported ADL difficulty at age 75 would be expected to increase by .17 units, or a Cohen's *d* of .08. Comorbidity was also the only covariate to significantly predict ADL slope after age 75 (b_{24} = .04, p < .01). With each additional organ system affected by illness beyond the first at baseline, the average annual linear increase in ADL difficulty after age 75 would be expected to steepen by .10 standard deviation units (Cohen's *d* = .10) across a 5-year period (i.e. age 75 to 80 years).

In a subsequent, post-hoc model (LGM5b), a DEFINE statement in M*plus* was used to equate the scales of the friend and family support composite variables ($M = 0$; $SD = 1$), and constrained the effects of friend and family support on ADL difficulty level and slope 2 to be equal. This constrained model was then compared to the freer growth model (LGM5) to evaluate the degree of misfit and whether their associations with functional status and change were significantly different. In this equated model, neither friend nor family support availability predicted ADL difficulty status or change (*B*s = -.03 and .01, respectively; $p > .10$). Equating their effects, however, resulted in significantly worse fit than the model in which both effects were freely estimated, suggesting that the more parsimonious model was not a good fit to the data ($\Delta \chi^2 / \Delta df = 6.04 / 2$, $p < .05$; AIC = 20881.77, BIC = 20964.61), although it verged on being trivially non-significant. Therefore, the notion that friend and family support have the same association with functional status and change was not supported.

In sum, although perceived availability of support did not predict functional difficulty at age 75, the availability of support from family members, but not from friends, did appear to have a very small, positive association with an increasing rate of functional difficulty after age 75 (see Figure 3.2). In other words, although older adults' perceptions of support from friends and family members might not play a significant role in the onset of ADL difficulty, these results do seem to suggest the role of family support in experiencing change once the onset of limitations begins.

Longitudinal Mediation Model

Descriptive information on the relationships among predictors, mediator, and outcome. Pearson correlations among the illness behavior factor scores and ADL composites are presented in Table 3.4. The illness behavior factors showed weak, positive associations with reported ADL difficulty across the waves, such that higher illness behavior levels were associated with greater reported functional difficulty. Correlations ranged from $r = .20$ (i.e. the first ADL wave with the fourth illness behavior wave, $p < .0001$) to $r = .32$ (i.e. the third ADL wave with the concurrent, third illness behavior wave; $p < .0001$). Furthermore, both friend and family support availability at baseline (Q2) were negatively correlated with illness behavior across the four questionnaire waves (Q2-Q5). For friend support, correlations with illness behavior ranged from $r = -.16$ ($p < .001$) to $r = -.18$ ($p < .0001$); whereas family support associations ranged from $r = -.06$ (*ns*) to $r = -0.09$ ($p < .05$). For friend support, correlations with ADL difficulty ranged from $r = -.10$ (*ns*) at the third ADL wave, to $r = -.18$ ($p < .01$) at the fourth wave; whereas family support associations were weaker and ranged from $r = -.02$ (*ns*) at the first ADL wave to $r = .08$ ($p < .05$) at the sixth wave.

Full growth model regression results and model comparisons. Based on previous findings from the illness behavior growth analyses (Study 1b) and the current study's piecewise growth models of ADL difficulty, a full mediation model was fitted that regressed a latent ADL difficulty intercept and slope 2 (i.e. linear change after 75) onto both predictors of friend and family support (with their effects unconstrained), as well as a latent illness behavior intercept and linear slope regressed onto both social

support predictors (with their effects constrained to be equal). The latent intercept and slope of illness behavior were both evaluated for longitudinal mediation of the relationship between social support availability and ADL difficulty (see Figure 3.3; AIC = 50077.63, BIC = 50274.50). All model outcomes were adjusted for the demographic covariates of participant sex, SES, and comorbidity.

In the first model comparison, the first mediating paths (*a* paths) from social support to the illness behavior intercept and slope were dropped. This model was reduced by 2 parameters, and it showed significantly reduced fit relative to the full mediation model ($\Delta \chi^2 / \Delta df = 34.70 / 2$, $p < .0001$; AIC = 50108.32, BIC = 50294.83). The next model dropped only the second mediating paths (*b* paths) from the illness behavior intercept to the ADL difficulty intercept and slope; and from the illness behavior slope to the second slope of ADL difficulty. This model was constrained by 3 parameters and showed significantly worse model fit relative to the full mediation model ($\Delta \chi^2 / \Delta df = 41.08 / 3$, $p < .0001$; AIC = 50119.55, BIC = 50300.87). The third nested model, however, which dropped the four direct paths (*c'* paths) from friend and family support availability to the latent intercept and slope of ADL difficulty, did not significantly reduce model fit compared to the full mediation model ($\Delta \chi^2 / \Delta df = 6.51 / 4$, $p = .16$; AIC = 50077.01, BIC = 50253.16). This suggested possible full mediation of social support availability on ADL status and change via illness behavior, because the direct effects were no longer significant when the indirect effects were included in the model (i.e. they could be constrained to zero without significant misfit). The significance of the indirect effect was underscored by the final model comparison, in which all mediating paths (both

a and *b* paths) were simultaneously dropped. This model was constrained by 5 parameters and showed significantly reduced model fit relative to the full model, suggesting that the indirect effects could not be excluded ($\Delta \chi^2 / \Delta df = 74.65 / 5$, $p < .0001$; AIC = 50149.88, BIC = 50320.85). Finally, a reduced model without mediators (i.e. a "*c* path only" model; AIC = 28562.63, BIC = 28671.42) was compared to a model in which the effects of social support on ADL status and change were constrained to be zero. This model had significantly worse fit ($\Delta \chi^2 / \Delta df = 9.80 / 4$, $p = .04$; AIC = 28566.34, BIC = 28654.41), suggesting that the total effect of social support availability on ADL status and change could not be excluded from the model.

All unstandardized parameter results, standard errors, and corresponding 95 % confidence intervals from the final longitudinal mediation model are displayed in Figure 3.3 and Table 3.5, respectively (covariates and their regression paths are not shown for simplicity). From the final model, social support availability (i.e. friend and family support availability with effects constrained to be equal) significantly predicted a reduced illness behavior intercept (*B*s = -.80, $p < .0001$), but again, showed no association with the illness behavior slope. In practical terms, a one-unit standard deviation increase in social support availability (from both friends and family members) corresponded with a .80-unit decrease in somatic complaints, non-prescription medication use, pain-related disability, and perceived illness complications at age 60, which was equivalent to a Cohen's *d* effect size of .08. In turn, the illness behavior intercept positively predicted ADL difficulty status at age 75 ($b = .04$, $p < .0001$), but it did not predict change in ADL difficulty afterward. Specifically, a one-unit increase in illness behavior scores at age 60

predicted a small, .04-unit increase in ADL difficulty status at age 75 (a Cohen's *d* equivalent of .02). Neither friend nor family support availability predicted ADL status after adjusting for illness behavior, although family support availability remained a positive predictor of an increased rate of ADL difficulty after age 75 ($b = .07$, $p = .024$). Across a 5-year period (i.e., from age 75 to 80 years), the mean difference in the rate of functional difficulty as a function of a one-unit increase in family support beyond the average would be equivalent to .17 standard deviation units (Cohen's $d = .17$). Thus, the mediation of social support availability appears to be occurring through the illness behavior intercept rather than the slope, and the only significant indirect association was with ADL status rather than change. Furthermore, there was a separate, unmediated path from the illness behavior slope to the second slope of ADL difficulty ($b = .51$, $p = .011$), such that a one-unit increase in illness behavior's linear rate of change across a 5-year period (i.e. from 75 to 80 years) was associated with an increase in the rate of ADL difficulty of 1.25 standard deviation-units (Cohen's $d = 1.25$)

Regarding the covariates, being female predicted a higher illness behavior intercept ($b = 2.98$, $p < .0001$; Cohen's $d = .30$) and trended toward a significant association with increased rate of change across age ($b = 0.03$, $p = .067$). Specifically, the cumulative mean difference in illness behavior change across a 5-year period (e.g., 60 to 65 years), as a function of being female, was slightly less than .02 standard deviation-units. However, sex was not a significant predictor of functional status at age 75 nor linear change afterward. Higher SES predicted slightly increased illness behavior levels ($b = 0.24$, $p = .014$; Cohen's $d = .02$), and a small reduction in the linear rate of change (*b*

= -0.01, p = .037), such that a one-unit increase in SES above the average was associated with a reduction of .01 standard deviation-units in the linear rate of change, across a 5-year period (Cohen's d = - .01). Lastly, greater comorbidity positively predicted illness behavior levels at age 60 (b = 2.31, p < .0001; Cohen's d = .23) and an increased rate of change afterward (b = 0.02, p < .01). Comorbidity was also the only covariate to predict functional status and change. Specifically, greater comorbidity predicted higher reported ADL difficulty at age 75 (b = .09, p < .01; Cohen's d = .04), as well as a steeper increase in ADL difficulty afterward, although its effect on slope was at trend significance (b = .03, p = .059; Cohen's d = .07, across a 5-year period).

Statistical inference for the indirect effect. The results from the specific and overall tests of indirect, direct, and total effects (from the M*plus* MODEL CONSTRAINT command) are reported in Table 3.5. The overall indirect effect was statistically significant (p = .001), as was the specific indirect effect on the intercept of ADL difficulty (p < .0001). The specific indirect effect via the illness behavior intercept and slope to the slope of ADL difficulty, however, did not reach significance. Furthermore, all direct and total effects were non-significant. Although some researchers using Baron and Kenny's causal-steps approach to mediation have suggested that a failure to obtain statistical significance for the total effect implies that mediation analyses should not proceed; other researchers have suggested that it is justifiable to examine indirect effects even in the absence of a statistically significant overall effect, as mediation can still take place (MacKinnon & Fairchild, 2009). The ratio of the indirect and total effects suggested that 60 % of social support availability's relationship with

functional status was mediated by illness behavior status (P_M = .60). Although this result implies a strong and significant mediating effect, the total effect of social support availability on ADL difficulty was small to begin with; thus, the proportion mediated is a slightly misleading effect size when estimating the practical importance of mediational processes (Preacher & Kelley, 2011). In contrast, the partially standardized indirect effect (ab_{ps} = -0.01) suggested that ADL difficulty at age 75 years was expected to decrease by only .01 standard deviations for every one-unit standard deviation increase in social support availability from friends and relatives, indirectly via levels of illness behavior.

Discussion

The current study investigated age-related intra-individual change and between-individual differences in functional status across twenty-three years (1987-2010) among older adults from the Swedish Adoption/Twin Study of Aging (SATSA). Specifically, the joint predictive value of social support availability from friends and family members on both functional status and change after age 75 was evaluated, above and beyond other baseline demographic and psychological risk measures including sex, SES, depression, and comorbidity. Social support, a key function of social networks throughout the adult lifespan, predicts important outcomes of morbidity and mortality (Holt-Lundstad, Smith, & Layton, 2010) and has been associated with other indices of mental and physical health (Seeman, 2000). Thus, it was hypothesized that there would be relative stability in physical functioning prior to age 75, with a steeper increase in the rate of difficulty afterward. Moreover, both sources of available support—from friends and relatives—

were hypothesized to predict reduced onset of ADL difficulty and serve to buffer the rate of functional decline (i.e. the rate of difficulty) after age 75.

Another focus of the present study was on testing a potential behavioral mechanism underlying the relationships of social support availability with functional status and decline. Social support is posited to influence health outcomes through many pathways, including direct, biophysiological pathways (e.g., improved immune response, pulmonary functioning, and C-reactive protein), as well as indirect pathways, such as encouraging (or deterring) health-relevant behaviors, providing norms for behavior, affecting emotional processes (e.g., reducing depressive symptoms) and buffering stress, to name a few. Although preventive health behaviors (e.g., diet, exercise, and screenings) have been investigated as key intervening pathways in the existing literature, the mediating role of illness behavior has not been explored. Illness behaviors are associated with health outcomes such as functional recovery (Broadbent, Ellis, Thomas, Gamble, & Petrie, 2009), distress or symptom exacerbation (Harkins, Price, & Braith, 1989), timely detection and diagnosis (van Osch, Lechner, Reubsaet, de Nooijer, & de Vries, 2007), and changes in emotional support from caregivers (Stephens et al., 2006) in clinical settings, and might represent a unique behavioral pathway linking social support with late-life physical functioning. Specifically, illness behavior levels and trajectories were hypothesized to partially mediate the effects of social support availability on functional status and change, wherein reduced levels of illness behavior at age 60 and more stable patterns would predict lower ADL difficulty status at age 75, and a reduced rate of reported difficulty afterward.

Initial analyses examining correlations between the functional difficulty composites and predictors suggested protective relationships among social support measures and reduced illness behavior scores with functional status. Both friend and family support availability were negatively correlated with levels of ADL difficulty across the waves, although family support was not significantly associated (except for at the final assessment). A similar pattern of results emerged from the correlations of support availability with the illness behavior factors. Friend and family support were weakly and negatively correlated with illness behavior scores across the four questionnaires (Q2-Q5), but again, family support had weaker associations. Finally, the illness behavior factor scores were positively correlated with ADL difficulty across the six assessments. Thus, all key variables in the hypothesized mediational process were related at the bivariate level.

Growth models evaluated the age-related pattern of change in functional difficulty in SATSA, and the extent to which social support availability from friends and family members predicted functional status and buffered subsequent decline. Consistent with the current study's hypotheses, growth models suggested very low levels of difficulty performing basic activities of daily living at age 75, with piecewise change over time. Specifically, there was almost no change in ADL difficulty observed prior to age 75, with a small, annual linear increase in difficulty afterward. This is consistent with prior SATSA work on objective measures of functional status, which found significant change after age 70 (Finkel et al., 2015), and with prior work on the questionnaire measures, which found significant change after age 75 (Foebel et al., 2015). The current study's

centering age of 75 was chosen based on the work by Foebel and colleagues, as well as the exploratory, ordinary least-squares (OLS) trajectory analyses, which suggested initial decline after age 75. This small discrepancy between the objective and subjective measures might be due to this study's inclusion of self-report functional status measures, which were positively skewed relative to the measures from the in-person testing sessions.

The hypothesis that friend and family support would predict reduced functional difficulty and buffer decline was not supported. Neither friend nor family support predicted reduced ADL difficulty, and an unexpected result emerged regarding their effects on change, wherein higher social support availability from family members predicted an increased rate of difficulty after age 75. Friend support availability, however, was not predictive. Although the effect of family support on change in ADL difficulty was small, this relationship was evaluated across a span of more than twenty years, and therefore had the potential to significantly alter the trajectories through late-life. Although this result is inconsistent with other findings on associations of social integration and close family relationships with mental and physical functioning in late-life (Crittenden, Pressman, Cohen, Janicki-Deverts, & Smith, 2011; Ryan & Willits, 2007), there are a few possible explanations. First, family support's prediction of accelerated ADL difficulty might simply reflect declining health among the participants at the later assessment waves, rather than being an antecedent. Older adults who experience multi-morbidity and face other losses, such as bereavement, through the late-life transition might naturally elicit more support from their family members, as they play

an increasing role in medical decision-making processes and provide practical assistance or care. One alternative explanation is that, along with an increase in the availability of support from family members, there might have been an unmeasured element of strain in these relationships, to the extent that participants were not satisfied with the higher levels of support that they perceived. Although unlikely, it is possible that family support did lead to worse physical functioning through reduced feelings of autonomy or negative social control, which this study's measure of support availability did not sufficiently capture. Regarding the covariates, higher depressed mood and greater comorbidity significantly predicted higher ADL difficulty status, but comorbidity was the only covariate to also predict an accelerated rate of difficulty. Significant sex differences in the rate of ADL difficulty were not observed, which contradicts prior work suggesting that women experience faster declines than men in some functional domains (Finkel et al., 2015). Sex effects on ADL status at age 75 were, however, trending toward significance, with females reporting more difficulty.

Finally, the results from the longitudinal mediation analyses partially supported the hypothesis that illness behavior intercept and slope would mediate the relationships between social support availability and functional status and change. First, the nested model comparisons of the full mediation model suggested a significant total effect of social support availability on functional status and change, as well as a significant indirect effect of social support availability on functional difficulty via illness behavior; however, when the mediating paths of illness behavior were included in the model, the direct effect of support on functional status and change was no longer significant. Further

tests of statistical inference for the indirect effects suggested that although the total indirect effect parameter was significant, when this parameter was further split into specific indirect effects paths contributing to the ADL intercept versus the slope, only the effect on ADL status was significant. Therefore, the mediational process played a role in predicting functional status at age 75, but it did not play a role in intra-individual change. Furthermore, the significant, negative effect of social support availability on the illness behavior intercept and its non-significant association with the slope, together, suggested illness behavior status—rather than behavior change or maintenance—played a role in the mediational process. Although the proportion mediated was large, the partially standardized indirect effect suggested a small (one-percent of a standard-deviation) decrease in functional difficulty per one-unit standard deviation increase in social support, indirectly via illness behavior.

Also notable was the finding that, above and beyond illness behavior's indirect effect, family support availability remained a positive predictor of an accelerated linear increase in functional difficulty after age 75. Moreover, there was a substantive and separate (i.e. unmediated) path from the illness behavior slope to accelerated growth in functional difficulty. This suggested that illness behavior trends were significantly associated with functional decline—albeit a distinct process, not linked with social support—such that increasing levels of somatic complaints, non-prescription medication use, pain-related disability, and perceived illness contributions across age predicted an increased rate of difficulty in performing physical activities of daily living during the late-life transition. Although this provided some initial support for the hypothesis that

illness behavior trends may carry consequences for healthy aging, future research should explore the causal mechanisms of this association, as well as its generalizability to other health outcomes, like objective measures of physical functioning and mortality.

Applying a longitudinal research design was valuable in examining the long-term social underpinnings of older adults' functional status, controlling for biological risk factors (e.g., comorbidity and familial factors), as well as in evaluating the association of illness behavior with subsequent physical functioning. To test part of my conceptual model of the social development of illness behavior (Figure 1.1), this study analyzed secondary data from the Swedish Adoption/Twin Study of Aging (SATSA), a twin design study of all eligible twins from the Swedish Twin Registry from early- to late-adulthood (i.e. ages 29 to 96 years). This study also took advantage of SATSA's twin design to adjust for the possible confounding of familial factors (reflecting both biological relatedness and a shared rearing environment) on illness behavior trends and functional status outcomes. The unique ability to consider the social context using twin pairs provides an advantage, because participants are matched as closely as possible on genetic and childhood environmental factors.

The current study's theoretical groundings and statistical methods contribute to a better understanding of the social—rather than straightforwardly physiological—predictors of illness behavior in this age group, as well as the possible consequences of illness behaviors for healthy aging (defined here as functional ability). Testing such illness behavior pathways might begin to elucidate widely observed demographic disparities in older adults' medical utilization (e.g., based on gender, SES, education, and

marital status; Vedsted & Christensen, 2005). Such research might also inform some areas of intervention for promoting healthy aging through appropriate self-care strategies, and through building or buffering interpersonal resources before the transition into late adulthood.

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Table 3.1

Means, Standard Deviations, Range, and Skewness of Baseline Predictors and Functional Status Outcome (SATSA sample)

Variable	*N*	*M*	*SD*	Range		Skewness
				Potential	Actual	
Predictors[a]						
SES	1,310	0.35	2.67	---	-8.7-6.0	-0.76
Depress	1,310	2.30	3.05	0-18	0.0-18.0	1.61
CIRS	1,310	1.54	2.01	-1-11	-1.0-10.0	1.02
Friend	1,310	0.35	5.73	---	-14.6-14.1	-0.04
Family	1,310	0.04	2.99	---	-8.5-6.4	-0.69
ADL difficulty composite[b]						
Q2	1,304	10.52	2.06	10-38.73	10.0-34.6	5.51
Q3	1,121	10.58	2.03	10-38.73	10.0-30.0	5.02
Q4	1,046	10.72	2.28	10-38.73	10.0-37.4	4.99
Q5	633	11.13	3.25	10-38.73	10.0-38.7	4.26
Q6	520	11.09	2.90	10-38.73	10.0-30.0	3.57
Q7	436	11.47	3.56	10-38.73	10.0-37.4	3.56

Note. SES = standardized composite of objective socioeconomic status; Depress= depressed mood subscale of the CES-D; CIRS = composite of illness comorbidity, adapted from the Cumulative Illness Rating Scale (CIRS) categories; Friend = perceived availability of social support from friends (ISSI subscale); Family = perceived

availability of social support from relatives (ISSI subscale); and ADL = physical activities of daily living composite created across six questionnaire waves (Q2-Q7). Illness comorbidity was centered at one (i.e. one chronic health condition). All other predictors were standardized composites ($M = 0$; $SD = 1$). [a]Descriptive information for dichotomously-coded covariate of sex (1 = *female*) is not shown. [b]The raw ADL composites at each wave were square-root transformed and multiplied by 10 to increase model stability, such that a score of 10 on the composite was equivalent to a 0 on the raw scale (i.e. no reported ADL difficulty).

Table 3.2

Zero-Order Correlations Among the Baseline Predictors and Functional Status (i.e. ADL Difficulty) Across the Six Questionnaires (SATSA sample)

Variable	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*
1. Sex (1 = *female*)	1.00	**-.14**	**.14**	**-.12**	**.13**	**-.11**	**.09**	**.11**	.07**	**.10**	.10**	.08**	.06*
2. SES		1.00	**-.22**	**.38**	**-.20**	**.23**	.08**	**-.16**	**-.19**	**-.24**	**-.30**	-.31**	-.33*
3. Depress			1.00	**-.26**	**.23**	**-.28**	**-.18**	**.18**	**.13**	**.13**	.11	.13**	.13
4. Marital[a]				1.00	-.10**	**.13**	**.11**	-.09**	-.10**	-.10**	-.13	-.14*	-.18**
5. CIRS					1.00	-.10**	-.01	**.27**	**.25**	**.28**	**.31**	.31**	.33**
6. Friend						1.00	**.40**	**-.14**	-.13**	-.10	-.18**	-.16	-.18*

7. Family	1.00	-.02	.02	.04	.04	.04	.08*
8. ADL1		1.00	**.69**	**.66**	**.45**	**.36**	.44**
9. ADL2			1.00	**.83**	**.53**	**.47**	**.55**
10. ADL3				1.00	**.59**	**.52**	**.54**
11. ADL4					1.00	**.94**	**.78**
12. ADL5						1.00	**.79**
13. ADL6							1.00

Note. *N*s ranged from 392 to 1,310. All values are robust maximum likelihood estimates from M*plus*. Values in bold are significant at $p < .0001$. ADL1 = composite from Q2; ADL2 = composite from Q3; ADL3 = composite from Q4; ADL4 = composite from Q5; ADL5 = composite from Q6; and ADL6 = composite from Q7.

[a] Predictor not included in final growth model.

*$p < .05$. **$p < .01$.

Table 3.3

Results of Unconditional and Conditional Latent Growth Curve Models of ADL Difficulty in SATSA (N = 1,310)

Parameter	LGM1	LGM2	LGM3	LGM4	LGM5
	Means-Only	Unconditional linear	Unconditional spline[a]	Random intercept[b]	Random intercept & slope[c]
Means or intercepts					
i (level)	10.87 (.06)***	11.18 (.08)***	10.45 (.05)***	9.98 (.08)***	9.98 (.08)***
s1 (slope A)	----	0.05 (.01)***	0.0 (.00) *ns*	0.0 (.00) *ns*	0.0 (.00) *ns*
s2 (slope B)	----	----	0.30 (.03)***	0.29 (.03)***	0.22 (.05)***
Covariate regressions					
i on					
Sex	----	----	----	0.17 (.10) ǂ	0.18 (.10) ǂ
SES	----	----	----	0.04 (.02) *ns*	0.04 (.02) ǂ
Depress	----	----	----	0.05 (.02)**	0.05 (.02)**
CIRS	----	----	----	0.18(.04)***	0.17(.04)***
Friend	----	----	----	-0.01 (.01) *ns*	-0.01 (.01) *ns*
Family	----	----	----	-0.01 (.02) *ns*	-0.01 (.02) *ns*
s2 on					
Sex	----	----	----	----	-0.01 (.05) *ns*

SES	----	----	----	----	-0.01 (.01) ^{ns}
Depress	----	----	----	----	-0.0 (.01) ^{ns}
CIRS	----	----	----	----	0.04 (.01)**
Friend	----	----	----	----	-0.01(.01) ^{ns}
Family	----	----	----	----	0.03 (.01)*
Variances, residual variances, and covariance					
i (level)	2.43 (.50)***	5.13 (.92)***	1.89 (.56)***	1.73 (.51)**	1.72(.51)**
s1 (slope A)	----	.01 (.00)***	0.0 (.00) ^{ns}	0.0 (.00) ^{ns}	0.0 (.00) ^{ns}
s2 (slope B)	----	----	0.28 (.07)***	0.28 (.07)***	0.26 (.06)***
Covariance of *s2* and *i*	----	0.23 (.04)***	0.15 (.09) ^{ns}	0.11 (.09) ^{ns}	0.12 (.09) ^{ns}
Model fit indices					
Log-Likelihood	-10664.44	-10989.65	-10474.63	-10432.32	-10421.12
Parameters	3	6	6	12	18
Scaling correction	5.95	8.97	8.92	5.05	3.78
AIC	23301.93	21991.30	20960.93	20888.63	20878.25
BIC	23317.46	22022.37	20991.99	20950.77	20971.45
$\Delta\chi^2$ / Δ *df*	----	173.28 / 3***	313.60 / 3***	71.13 / 6***	17.93 / 6**

Note. Values in parentheses indicate standard errors. All values are robust maximum likelihood estimates from M*plus*.

[a] Unconditional piecewise growth model with two slopes: one capturing ADL change prior to age 75 (fixed to be stable) and one estimating linear ADL change after age 75. [b] Conditional intercept--as-outcome model, with the ADL intercept regressed onto social support predictors and covariates, and both slopes estimated as fixed effects. [c] Conditional intercept- and slope-as-outcome model, with random variation around both the intercept and slope B.

*$p < .05$. **$p < .01$. ***$p < .0001$.

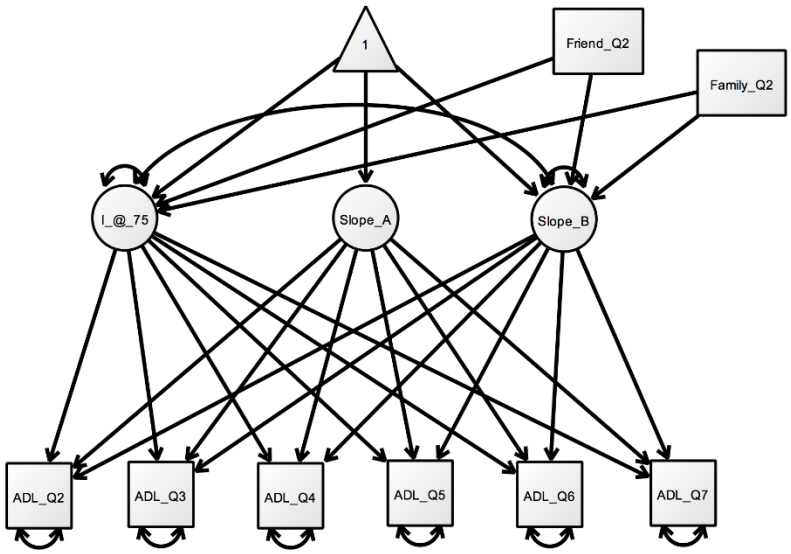

Figure 3.1. Path diagram of the hypothesized conditional, latent growth model of age-related change in ADL difficulty with six predictors. This is an intercept and slope model including two separate slopes: one to account for the rate of change in ADL difficulty prior to age 75 (Slope A), and one accounting for the different rate of change in ADL difficulty after the age of 75 (Slope B). Individual growth models were fitted to the observed, repeated measures of ADLs across the six assessment waves (Q2-Q7). The paths from the latent slopes to the observed scores are age-basis coefficients, or each person's observed age minus the centering age of 75. The random errors or uniquenesses represent the variability not accounted for by the growth model. The mean constants at the top are the group estimates of intercept and slopes for the entire sample, again, adjusted for the centering age and other predictors. Covariates are not shown.

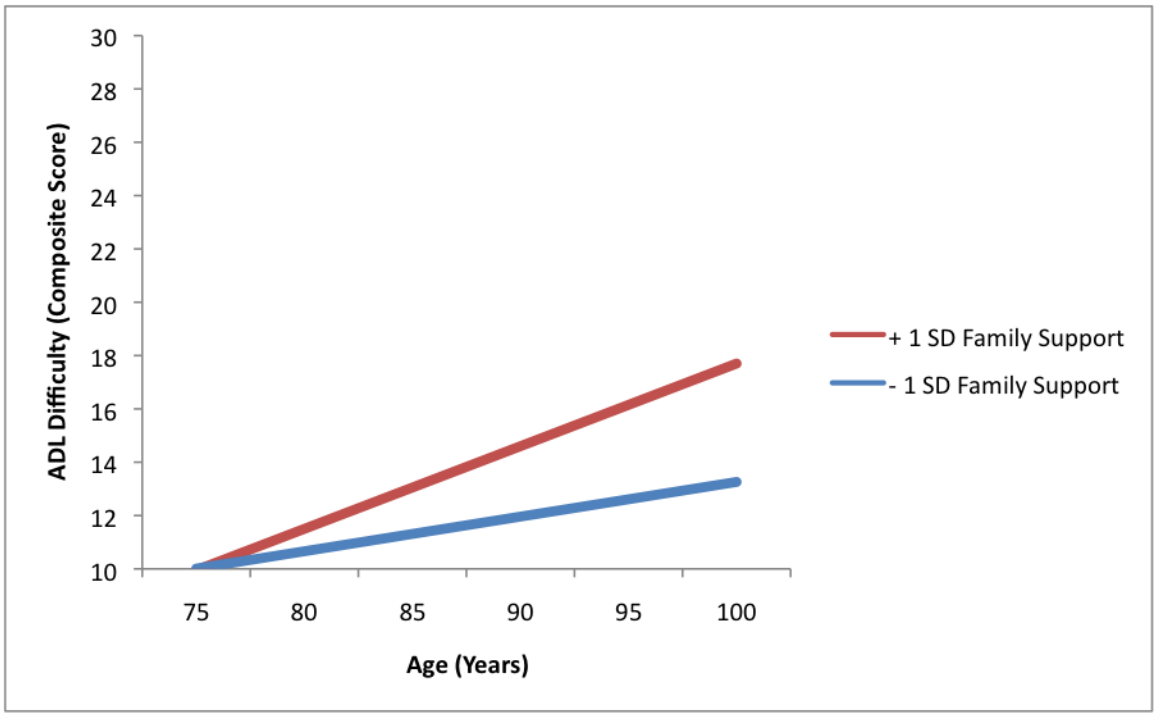

Figure 3.2. Expected mean trajectories of ADL difficulty after age 75 years per one-unit standard deviation increase and per one-unit standard deviation decrease in perceived support availability from family members.

Table 3.4

Zero-order Correlations of Illness Behavior (T-Scaled Factor Scores) with ADL Difficulty Across Assessment Waves (SATSA sample)

Factor	ADL Wave					
	Q2	*Q3*	*Q4*	*Q5*	*Q6*	*Q7*
Illbeh1	.28	.26	.27	.23	.24	.26**
Illbeh2	.29	.28	.29	.23*	.23**	.26
Illbeh3	.31	.31	.32	.26**	.26*	.29**
Illbeh4	.20	.21**	.23	.25	.26	.26*

*Note. N*s ranged from 386 to 1,165. All values are robust maximum likelihood (MLR) estimates from M*plus*.

* $p < .01$. **$p < .001$. Otherwise, all $p < .0001$.

Table 3.5

Latent Growth Model Regressions for Mediation of the Effect of Social Support Availability on ADL Difficulty Status and Change by Illness Behavior

Model	Estimate	*SE*	*p*	CI (lower)	CI (upper)
Model without mediator					
ADL Intercept	10.05	0.07	< .0001	9.92	10.18
FRN → ADL (*c1*)	-0.05	0.05	.28	-0.15	0.04
REL → ADL (*c2*)	-0.05	0.05	.26	-0.15	0.04
ADL Slope 2	0.22	0.04	< .0001	0.13	0.30
FRN → ADL (*c3*)	-0.05	0.04	.21	-0.13	0.03
REL → ADL (*c4*)	0.08	0.03	.01	0.02	0.14
Model with mediator					
ADL Intercept	8.40	0.37	< .0001	7.67	9.13
SUPP → ILLB_i (*a1*)	-0.80	0.14	< .0001	-1.07	-0.53

ILLB_i → ADL (*b1*)	0.04	0.01	< .0001	0.02	0.05
FRN → ADL (*cp1*)	-0.03	0.05	.59	-0.12	0.07
REL → ADL (*cp2*)	-0.02	0.05	.67	-0.12	0.08
Indirect effect (*a1*b1*)	-0.03	0.01	< .0001	-0.05	-0.01
Total effect (*a1*b1 + cp1*)	-0.08	0.06	.18	-0.19	0.04
ADL Slope 2	0.15	0.15	.32	-0.14	0.43
SUPP → ILLB_s (*a2*)	0.00	0.01	.70	-0.01	0.01
ILLB_i → ADL (*b2*)	0.00	0.00	.85	-0.01	0.01
ILLB_s → ADL (*b3*)	0.51	0.20	.01	0.12	0.91
FRN → ADL (*cp3*)	-0.04	0.04	.26	-0.12	0.03
REL → ADL (*cp4*)	0.07	0.03	.02	0.01	0.14
Indirect effect (*a2*b2 + a2*b3*)	0.00	0.00	.89	-0.01	0.01

Total effect (*a2*b2 + a2*b3*) + *cp2*	0.03	0.03	.37	-0.04	0.09
Overall effects					
Indirect	-0.03	0.01	.001	-0.05	-0.01
Direct	-0.02	0.06	.77	-0.14	0.10
Total	-0.05	0.06	.44	-0.16	0.07

Note. In this model, FRN (composite of friend support availability), REL (composite of family support availability), and SUPP (composites of social support availability from family and friends, constrained to be equal) were the independent variables (*X*), ILLB_i and ILLB_s (illness behavior latent intercept and slope, respectively) were the mediators (*M*), and Intercept and Slope 2 of ADL (i.e. functional difficulty and change) were the outcomes (*Y*). Participant sex, SES, and illness comorbidity were included as covariates in both models. CI (lower) = lower bound of a 95 % confidence interval; CI (upper) = upper bound of a 95 % confidence interval; → = regression path.

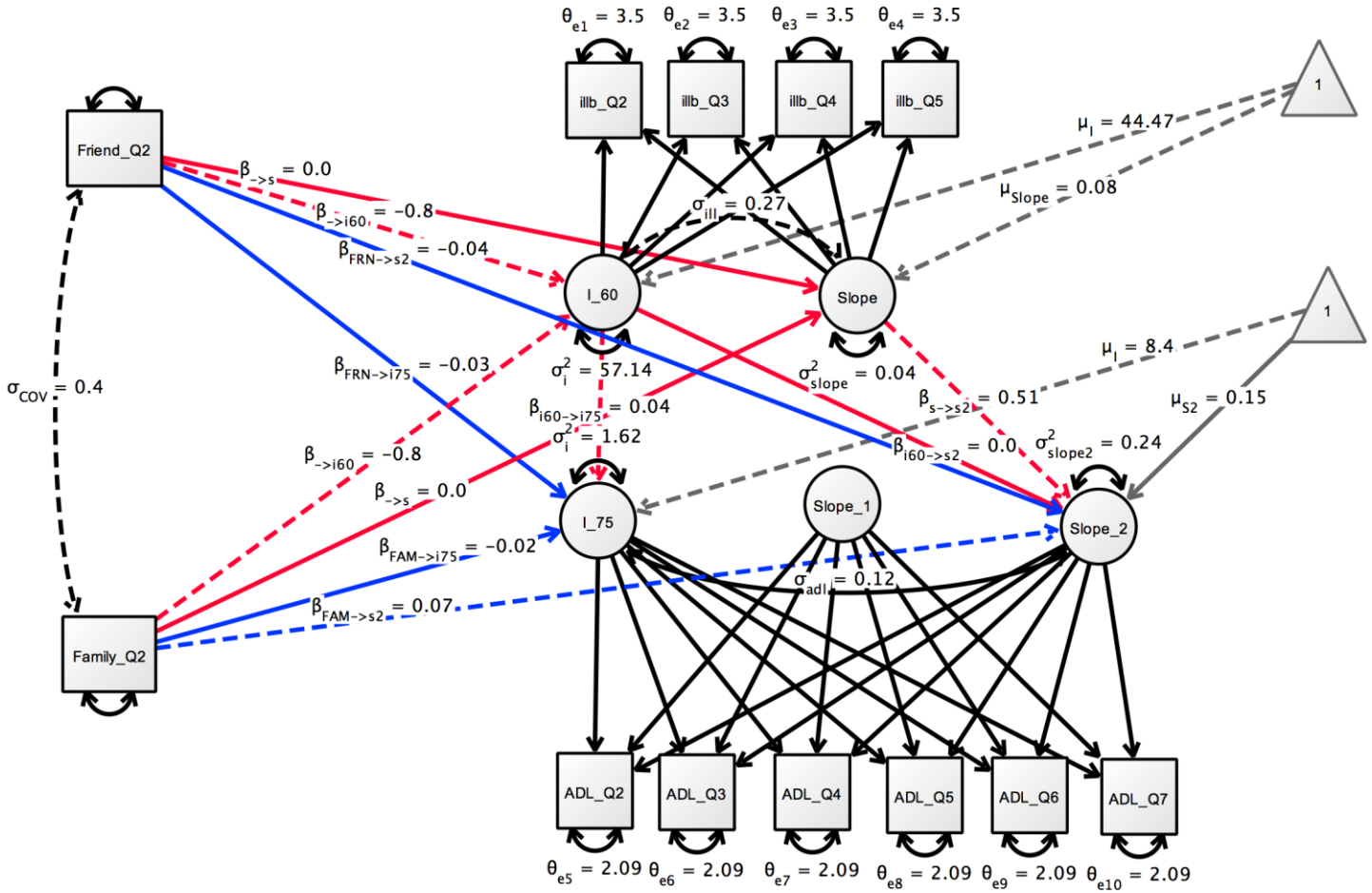
θe1 = 3.5
θe2 = 3.5
θe3 = 3.5
θe4 = 3.5
illb_Q2
illb_Q3
illb_Q4
illb_Q5
1
μI = 44.47
μSlope = 0.08
σ²illb = 0.27
Friend_Q2
βFRN->s = 0.0
βFRN->i60 = −0.8
βFRN->s2 = −0.04
I_60
Slope
σ²I = 57.14
σ²slope = 0.04
σCOV = 0.4
βFRN->i75 = −0.03
βi60->i75 = 0.04
σ²I = 1.62
μI = 8.4
βs->s2 = 0.51
μS2 = 0.15
βi60->s2 = 0.0
σ²slope2 = 0.24
βFRN->i60 = −0.8
βFRN->s = 0.0
I_75
Slope_1
Slope_2
βFAM->i75 = −0.02
σ²adh = 0.12
βFAM->s2 = 0.07
Family_Q2
ADL_Q2
ADL_Q3
ADL_Q4
ADL_Q5
ADL_Q6
ADL_Q7
θe5 = 2.09
θe6 = 2.09
θe7 = 2.09
θe8 = 2.09
θe9 = 2.09
θe10 = 2.09

Figure 3.3. Final latent growth curve model for longitudinal mediation of the relationship between perceived social support availability and ADL difficulty (latent intercept and slope after age 75) by illness behavior (latent intercept and slope). Completely unstandardized robust maximum likelihood (MLR) estimates from M*plus*. illb = observed illness behavior factor scores (T-scaled); ADL = observed functional difficulty composites (i.e. square-root transformed and multiplied by 10); Friend = friend support availability composite; and Family = family support availability composite. Friend and family support were standardized ($M = 0$, $SD = 1$) and their effects on illness behavior were constrained to be equal. Regression paths from latent slopes to observed illness behavior factor scores across the four assessment waves (Q2-Q5) and to observed ADL difficulty composites across the six assessment waves (Q2-Q7) were random age-basis coefficients (not shown for simplicity). Paths for the direct effects (*c'*) are shown in blue, and all indirect or mediating (*a* and *b*) paths are shown in red. Dashed lines were used to represent significant paths. Covariates of sex, SES, and illness comorbidity are not shown for simplicity.

GENERAL DISCUSSION

Decisions to seek medical care are often based on factors independent of biological risk. Apart from the influences of patient demographics and disease characteristics, psychosocial factors such as health beliefs, emotional processes, and social circumstances (e.g., the quality of social support) may act to amplify any given physical symptom and predict more frequent use of medical services (c.f., Al-Windi, Dag, & Kurt, 2002; Barsky, 1988; Haug, Musil, Warner, & Morris, 1997). However, the relative importance of social factors' associations with older adults' illness behaviors—perceptions, evaluations, and responses to symptoms that often precede formal medical help seeking—are not well understood, nor have they been formally tested using a longitudinal design, until now. The primary purpose of this dissertation was to examine the longitudinal measurement, social predictors, and functional consequences of intra-individual trajectories of illness behavior across the transition from mid-to-late adulthood, as well as illness behavior's potential value as a mediating behavioral pathway linking the availability of social support (from friends and family) to late-life physical functioning. Across two studies, the current dissertation project investigated the following research questions:

Research Question 1. Will the observed indicators load adequately onto a single, latent construct of illness behavior, and can strong factorial measurement in the factors be established across the assessment waves, in addition to their discriminant and convergent validity?

Research Question 2. Is there a systematic pattern of age-related, intra-individual change in illness behavior from mid- to late-adulthood, and to what extent does social support availability from friends versus family members account for between-person differences in illness behavior status and change?

Research Question 3. What is the age-related pattern of intra-individual change in functional difficulty from mid- to late- adulthood? To what extent does the availability of social support (from family members versus friends), illness behavior levels, and illness behavior change predict better functional status and serve to buffer functional decline across the late-life transition? Specifically, do illness behavior trends partially mediate the associations between social support and functional health?

The guiding framework and conceptual model for the social antecedents of illness behavior is the Social Processes in Illness Behavior (SPIB) Model proposed in Chapter 1 (see Figure 1.1), in which direct and indirect pathways were proposed linking social processes (e.g., social support, social contact, network size, and family environments) to illness behaviors and subsequent health outcomes. For example, social processes might exert more direct effects on illness behaviors, through behavioral norms, modeling, reinforcement, or the provision of practical or enabling resources (Berkman, Glass, Brissette, & Seeman, 2000; Gallant, 2013). Conversely, they might indirectly predict these behaviors via emotional processes (e.g., reduced negative emotions like worry and fear), or health cognitions (e.g., self-efficacy or values regarding health promotion). Moreover, these social processes can have direct associations with health outcomes through physiological pathways that do not involve health- and illness- behaviors

(Seeman, 2000). This model is consistent with existing theoretical models of health behavior (e.g., Behavioral Model; Andersen, 1995/2014; Common Sense Model; Leventhal, 1970; I-Change Model; de Vries, 1998) and with conceptual models of social integration and health (Berkman et al., 2000; Seeman, 2000), as it considers the joint effects of emotional, cognitive, and social processes in behavioral outcomes and acknowledges the possibility of distinct pathways for structural and functional aspects of individuals' social environments.

Regarding illness behavior, individuals' perceptions, evaluations, and responses to symptoms have been found to predict regular medical help seeking (e.g., Lawson, Bundy, Lyne, & Harvey 2004), functional recovery (Broadbent, Ellis, Thomas, Gamble, & Petrie, 2009), and they are also posited to account for the costliest aspects of illness (Rief et al., 2003). In turn, high-quality social support predicts improvements in both preventive health behaviors and disease management behaviors like treatment adherence (DiMatteo, 2004). This relationship is posited to be particularly salient in older adulthood, when personal responsibility for health maintenance is high, symptoms and health threats are more commonly experienced, and social networks tend to decrease in size but increase in quality. Therefore, the model proposes that individual differences in a latent construct of illness behavior represents a unique, behavioral mediator of the relationship between social support and functional health in late-life.

Summary of General Findings

Study 1. Two large, population-based samples of older adults were drawn from the Swedish Adoption/Twin Study of Aging (SATSA) and the Sex Differences in Health

and Aging study (GENDER) to examine the factorial measurement and construct validity of a latent illness behavior factor. Longitudinal CFA models evaluated factorial invariance across four questionnaire waves spanning seventeen years in SATSA (1987-2004) and across two questionnaire waves spanning thirteen years in GENDER (1994-2007). In SATSA, strict factorial invariance in the illness behavior factor (indicated by somatic complaints, non-prescription medication use, pain-related disability, and perceived illness complications) was supported. In GENDER only partial, weak factorial invariance in the illness behavior factors (indicated by somatic complaints, non-prescription medication use, and perceived illness complications) could be established, wherein the loadings of the perceived illness complications composite were unconstrained across the two questionnaires. Across both samples, however, the construct validity of the illness behavior factor was supported. The factors were correlated with, but relatively independent from, participants' self-rated health status, objective health status (i.e. comorbidity), health promotion values, and forms of health utilization that were more need-based and not typically under participants' volitional control (i.e. frequency of contact with a district nurse and hospitalizations); whereas in GENDER, the illness behavior factor was also related to the self-reported frequency of sick days and primary care physician visits. In SATSA, the longitudinal trajectories of the illness behavior factors were also examined, as well as the predictive value of social support availability on status and intra-individual change. Longitudinal growth curve analyses suggested a small, annual linear increase in illness behaviors after entry into old adulthood (i.e. age 60). They also suggested higher availability of support from both

friends and family members had a small, negative effect on illness behavior scores at age 60. Furthermore, the protective associations of friend and family support availability with illness behavior levels were the same, despite initial analyses suggesting a significant association for friend support, but not for family support. Neither source of available support predicted illness behavior change across age; however, their effect on the intercept at age 60 impacted the height of the overall trajectories across many remaining years of the lifespan (see Figure 2.3), and consequently might still translate into cost savings and improvements in quality of life at both individual and population levels.

Study 2. Longitudinal growth in functional status (i.e. difficulty performing basic activities of daily living), the predictive value of social support availability from friends versus family on functional status and change, and the mediating role of illness behavior were examined across six questionnaire waves spanning twenty-three years (1987-2010) in the SATSA sample. The results suggested a discontinuous pattern of change in functional difficulty, with no reported difficulty prior to age 75, and a small, annual linear increase afterward. Neither friend nor family support predicted reduced ADL difficulty, nor did they buffer the rate of decline after age 75. However, unexpectedly, higher availability of family support emerged as a significant predictor of an increased rate of difficulty after age 75. This lends support to the theoretical SPIB Model's proposed direct association between social support and functional health, although the physiobiological pathways were not directly tested. A series of mediation models in a longitudinal growth modeling framework evaluated mediation of the social support availability associations with functional status and change via illness behavior levels and change. Overall, the

results suggested a significant, albeit small, indirect effect of social support's association with ADL difficulty status at age 75, but not with change, via illness behavior status at age 60. Although the illness behavior intercept accounted for more than half of the total relationship, the effect was notably small to begin with. Thus, it is somewhat difficult to gauge the practical importance of the indirect effect. Additionally, findings suggested that family support availability remained a positive predictor of accelerated ADL difficulty after age 75, and the illness behavior slope also uniquely predicted an accelerated rate of ADL difficulty after age 75. Importantly, this path was distinct and unmediated. Thus, even though intra-individual change in illness behavior did not play a significant role in the mediational process, it did play a role in shaping trajectories of functional decline. Together, these findings underscored the SPIB Model's proposed association between the availability of social support and illness behavior, as well as the direct role of illness behavior in predicting subsequent functional decline, and finally, the indirect role of illness behavior as a mediating pathway between social processes and health.

Implications

The world population is aging, and the rising incidence of disabling chronic conditions among community-dwelling adults ages 65 years and older will carry tremendous implications for individual health as well as for healthcare costs and efficiency. As chronic disease is the leading cause of death and disability worldwide (World Health Organization, 2014), psychosocial or behavioral issues in the illness process are of central importance to public health promotion efforts aimed at extending the healthy lifespan, or the "healthspan" (Nikolich-Žugich et al., 2015). Illness behaviors

and social connections represent two promising targets for accomplishing this goal. Although the literature on social relationships and health outcomes is abundant, research on the behavioral pathways linking the two have largely focused on primary, preventive health behaviors like diet, exercise, or medical screenings. Furthermore, the research on illness behavior is disparate, with an emphasis on predicting single decisions to utilize health services, or other isolated dimensions. Illness behavior, however, is better understood as a process of symptom perceptions, evaluations, and behavioral responses that may or may not lead to formal help-seeking decisions, or an "integrated symptom-response framework" (Wyke et al., 2013, p. 83). Furthermore, although demographic variables like female gender and increased age are associated with increased symptom reporting and frequent attendance in primary care (Sirri et al., 2013), the extant research is largely correlational and cross-sectional. As such, the longitudinal trajectories and systematic, within- and between-person differences in illness behavior across development are not yet understood.

The current dissertation begins its theoretical contributions with the validation of a latent, unifying construct of illness behavior, and with the first attempt to quantify and predict intra-individual change across adult development, rather than across a single illness episode. Predominant models of health- and illness- behavior (e.g., Behavioral Model; Andersen, 1995/2014; Common Sense Model; Leventhal et al., 1998; Integrated/I-Change Model; de Vries et al., 2005) suggest the important predictive roles of symptom characteristics (e.g., intensity, persistence, or severity), motivational factors (e.g., personality, emotions, health beliefs, existing knowledge about disease), and

external cues (e.g., norms, values, support, health communication, and resources, embedded in social networks or culture) to patterns of symptom responding. Despite their acknowledgement of the important role of social networks as enabling (Andersen, 1995/2014) or motivational (de Vries et al., 2005) factors in illness behavior, most of these models emphasize cognitive processes (Wyke et al., 2013) and therefore do not explicitly address the mechanisms of social influences on behavior, nor their interrelationships with other motivating factors. And although developmental factors such as age and gender are viewed as important precursors, or "pre-disposing factors" (Andersen, 1995) in some of these models, a life-course framework is necessary for examining the within-person change or stability in these patterns during the transition into older adulthood, a period of increasing adaptation to changes in health and social roles (Hansson & Carpenter, 1994). Furthermore, existing theories of social relationships and health (e.g., the social convoy model; Kahn & Antonucci, 1980; socio-emotional selectivity theory; Carstensen, 1992; Carstensen, 2006), suggest that although there is variability within social networks across development, there are also cumulative effects that may become increasingly significant with time (Antonucci et al., 2014). Thus, an initial hypothesis was that a latent, multi-indicator measure of illness behavior would exhibit systematic change from mid- to late-adulthood. The results of Study 1 have implications for the proposed association between social support availability and illness behavior development during the late-life transition.

For study 1, the confirmatory factor analyses suggested that a latent factor of illness behavior could be established across four assessment waves in SATSA, with

consistent measurement and interpretation over time. In the GENDER sample, however, this hypothesis was not supported. Specifically, the perceived illness complications composite did not load well at the second assessment wave. Although the GENDER participants were older and less variable in age across the assessments (ages ranged from 69 to 88 years at Q1, and from 82 to 96 years at Q2), the implications of this result are that careful consideration of age and possible sources of response shift should be considered in the development and validation of illness behavior measures. Illness perceptions might also be a difficult indicator to measure and to assess longitudinally. Although dimensions of illness representations are found to have high levels of short-term stability, most of the research has examined several dimensions in particular disease contexts (e.g., diabetes; Lawson, Bundy, & Harvey, 2008; irritable bowel syndrome; Rutter & Rutter, 2007). Nevertheless, the strict factorial invariance of an illness behavior factor within the SATSA sample is promising in that it suggests that illness behavior can be measured as a unifying individual difference construct across many years of the lifespan. In the longitudinal analyses, the hypothesis that illness behavior would exhibit systematic change with age was supported. Specifically, there was a small, linear increase after the entry into older adulthood, and importantly, with evidence of significant variability. This result is consistent with prior work suggesting that older adulthood is a period of increasing responsibility for health and a shifting model of illness, from that of prevention to disease management (Harvey, 2013). Importantly, this growth was only partly accounted for by objective health status.

Additionally, both studies speak to how perceptions of support from both friends and family members may serve a unique protective role in reducing illness behavior levels at entry into older adulthood, above and beyond the effects of gender, comorbidity, objective SES, and marital status. Conversely, support availability from family, but not from friends, predicted an accelerated rate of functional decline after age 75, whereas neither played a protective role in the onset of functional difficulty. The theoretical implications of this finding are not yet clear, but it might suggest that a higher level of family support serves as an indicator of functional decline rather than a precursor, such that loss of functioning and other negative events in late-life may serve to bring one's relatives closer and increase support. Conversely, these relationships might bring increased strain and conflict over medical decisions, and subsequently impact older adults' physical functioning through other pathways (e.g., reduced autonomy, reduced well-being, or physiological markers of stress).

Finally, study 2's longitudinal mediation analyses suggested that illness behavior status represented a significant mediating, behavioral pathway linking social support availability to improved functional status in older adulthood (i.e. reduced ADL difficulty at age 75). This study's results advance existing theories on the links between social relationship and health, suggesting illness behavior as a mechanism distinct from the literature's current emphasis on preventive health behaviors. This finding is also consistent with existing life-course models of the social determinants of health. Specifically, this mediational process might reflect a social trajectory model (Berkman, 2009), wherein early-life or adulthood social conditions (i.e. perceptions of supportive

relationships) predict subsequent social contexts in adulthood, which in turn, are more proximally related to outcomes of health and disease. Moreover, illness behavior trends (i.e., annual, linear increase in symptom reporting, non-prescription medication use, pain-related disability, and perceived illness complications) played a significant, predictive role in a hastened rate of functional decline after age 75. This association was separate from the effects of social support availability and illness behavior status at age 60, suggesting that intra-individual change in illness behavior has functional health consequences that are independent from and not mitigated by the perceived availability of social support. In other words, change in illness behavior and change in physical functioning were associated independently from social support paths. One potential implication is that changes in illness behavior after entry into older adulthood may be indicative of other primary and secondary objective aging processes, such as increasing frailty, a precursor to disability. For example, one study by Gill and colleagues found that the cause of death differed based on the number of months of disability prior to death, such that death from organ failure and frailty predicted low disability with an upward spike a few months before death, while dementia was associated with consistently high disability levels through the entire year before death (Gill, Gahbauer, Han, & Allore, 2010). Furthermore, chronic pain conditions (e.g., arthritis), respiratory conditions (e.g., chronic bronchitis, asthma, and emphysema), heart conditions (e.g., angina), and diabetes mellitis are leading causes of death, morbidity, and disability in older adults in the United States (Weiss, Boyd, & Yu, 2007) and tend to have onset in mid- or late-life (Verbrugge & Patrick, 1995). Thus, an alternative implication is that this relationship reflects

qualitative shifts in disease processes into late adulthood that also simultaneously impact functional decline and responses to symptoms.

Strengths and Limitations

Strengths of the current dissertation study included the application of longitudinal, multi-variate techniques within a structural equation modeling framework, which allowed the temporal relationships among key predictors and outcomes of interest to be established, in addition to the examination of health-relevant processes that unfold across many years of the adult lifespan. The inclusion of multiple time-points for functional status outcomes also permitted tests of complex, non-linear patterns. Additionally, the use of large, population-based, and representative (i.e. non-clinical) adult samples allowed for the exploration of illness behavior as an individual difference measure across a broad range of background characteristics and disease contexts. The use of twin pair clustering was also valuable for examining the unique predictive value of the social context, while accounting for genetic or familial factors.

Study limitations included the inclusion of self-report measures from the same respondents across the assessment waves. Thus, it was not feasible to account for possible biases due to same-method covariance among the predictors and outcomes. In future work, it would be useful to incorporate objective measures of physical functioning (e.g., grip strength, balance, and flexibility) and other respondents (e.g., nurse reports from the in-person testing sessions), as well as consideration of other outcomes like mortality. Although measures of basic activities of daily living capture individuals at the extremes of functional impairment, analytic approaches that also include instrumental

activities of daily living (e.g., shopping, handling medications) and that consider the sequential or hierarchical loss of activities in late-life (Jagger, Arthur, Spiers, & Clark, 2001) would provide a more complete picture. Such analyses might include a second-order growth modeling approach to examine growth curves and structural relationships, while simultaneously evaluating the measurement of latent factors of functional status (indicated by observed ADL and IADL items) that can account for different item weightings within each assessment.

Another limitation concerned the lack of specificity in the measures of social support availability, which made it difficult to distinguish who the participants regarded as friends or family members. For example, it is unclear to what extent co-twins, spouses, and children were included in participants' family definitions when they were responding to these items (although separate items asked about twin contact and contact with children), and each of these sources might have unique and important associations with behavior and health. However, the separation of friend and family sources of support at the Q2 assessment represents a strength. Furthermore, both measures of social support availability combined items on emotional and tangible support, and the current study did not examine their potentially differing pathways to illness behavior and physical functioning. For example, instrumental support might be more directly associated with functional health to the extent that sufficient coping resources are provided, whereas emotional support might act indirectly through reductions in psychological distress or maladaptive illness behavior patterns. Moreover, the items asked about the perceived availability of support from each source, rather than the enacted support that was

received. More important than the receipt of support, however, are considerations of the quality or participants' satisfaction with the support they're receiving. There are important individual differences in people's desire for social interaction or tendency to seek support from others; thus, even if a person reports high levels of available support from friends and family, they may desire more or less support than is available.

Furthermore, the focus on ethnically and culturally homogeneous samples from Sweden limited the ability to generalize these results to populations of older adults in the United States, which has a very different healthcare system and different cultural norms for help-seeking. However, as older adults in the United States do have access to a form of universal health care (i.e. Medicare) the current study's findings could still be informative. This also limits the ability to generalize to other racial or ethnic groups who do not have the same access to or experiences with healthcare. Furthermore, the inclusion of time-varying predictors might have allowed for a clearer understanding of the links between social processes and illness behavior development. For example, because older adults are posited to be more sensitive to losses of psychosocial resources (Leventhal et al., 1998), reductions in perceived availability of social support with age might be more potent predictors of illness behavior change and functional declines than distal levels of support from baseline. Also, regarding disease processes in late-life, there are important systematic shifts in multi-morbidity that could be considered in parallel with illness behavior development. The effects of social support or morbidity on behavior change and physical functioning is not likely to be constant throughout the late-life transition.

Therefore, incorporating time-varying predictors would provide useful tests of study hypotheses.

Finally, the current study's illness behavior indicators of somatic complaints, non-prescription medication use, and perceptions of pain and illness complications were intended to capture the correlates of over-utilization of formal health services. Consequently, the latent factor might not have captured the full range of individual differences in illness behavior—namely, the tendency to ignore or deny symptoms. Rather than occurring along an illness behavior continuum, it is possible that these types of avoidant responses are better represented as a distinct individual difference factor. This possibility is underscored by recent research on the Medical Minimizer-Maximizer Scale (Scherer et al., 2016), in which specific items on avoidance of medications did not load with the rest of the items in the scale. The antecedents of these responses might differ as well. For example, psychiatric comorbidity of anxiety and depression is a strong predictor of frequent attendance in primary care, whereas fear and a lack of perceived coping resources are more related to medical delay and other types of avoidant behavior (Sirri et al., 2013). Consequently, the current study's results can only speak to predictors and functional consequences of the behavioral tendency to over-respond to symptoms, rather than the tendency for denial or avoidance.

Future Directions

In the current investigation of the social predictors of illness behavior development and physical functioning, the perceived availability of support from friends and family was emphasized, which is only one of many potential indicators of social ties

and relationship quality. This focus on support availability was largely based on the literature's suggestion that the functional aspects of social networks—namely perceived social support—are more consequential for health than structural aspects, such as network size, density, composition, or even objective measures of enacted support (Antonnuci, Ajrouch, & Birditt, 2014; Due et al., 1999). However, the exclusion of other relevant social factors might have also led to a somewhat conservative estimate of the relationships of social support with illness behavior and functional status. The larger, sociocultural context may serve as a macro-level or "upstream" influence on health by shaping the structural aspects of social networks, which in turn, shape the micro-level or "downstream" functional aspects of social networks that are more likely to affect behavior (e.g., support, social influence, and resources) (Berkman et al., 2000). Thus, in future studies, it would be useful to incorporate other social network measures to examine their joint, interacting, or unique effects with perceptions of social support on illness behavior trends and functional health.

Furthermore, the negative functions of social networks, such as negative social control or relational strain, should be further examined in association with illness behavior trends, as they have been shown to predict increased risk for depression and physiological profiles associated with disease processes in older adults (e.g., increased cortisol, cardiovascular reactivity, and suppressed immune responses; Seeman, 2000). The current work suggests that family support availability at baseline, but not support from friends, uniquely predicts a hastened rate functional decline after age 75. Although this was an unexpected result and causal conclusions are not yet warranted, it might

suggest that the presence of family members in late-life does reduce older adults' physical functioning, to the extent that this support is viewed as overbearing or discourages autonomy and expression. For example, in a recent cross-sectional study of women experiencing pelvic or urogenital pain recruited from a urology clinic, perceptions of social constraints (i.e. feeling that close others inhibit or discourage disclosures of one's feelings or problems) predicted elevated distress, pain severity, and reduced physical functioning; this predictive association was also independent from the effect of higher pain catastrophizing (Tomakowsky, Carty, Lumley, & Peters, 2016). Examining individuals who diverge in their perceptions of available social support and satisfaction with that support (i.e. individuals with high levels of support, but low satisfaction/high conflict, or individuals with lower levels of support, but high satisfaction/low conflict), might help to elucidate the role of opposing social forces in shaping behavioral trajectories and physical functioning. Although generally, relational strain or conflict has been found to decline with increasing age (Due, Holstein, Lund, Modvig, Avlund, 1999), heterogeneity in this trend could be consequential for late-life health. For example, Sneed and Cohen analyzed longitudinal data from the Health and Retirement Study (HRS) and found that negative social interactions with partners, children, family, and friends among a community sample of older adults (ages 50 years and older) predicted an increased risk for hypertension, above and beyond the effects of positive interactions (Sneed & Cohen, 2014). Of note, this risk was present for women in the sample but not for the men. Indeed, the consequences of negative aspects of

relationships on physical health might outweigh the effects of positive contexts in late-life (Yang, Schorpp, & Mullan Harris, 2014).

The current work also suggests that both sex and comorbidity (i.e. the total number of bodily systems affected by one or more illnesses), are reliable demographic predictors of illness behaviors, such that women and individuals with multi-morbidity report higher levels of somatic complaints, over-the-counter medication use, pain-related disability and higher perceived illness complications than men and those who endorsed fewer medical diagnoses. Comorbidity, however, uniquely predicted higher functional difficulty status and hastened the increase in difficulty after age 75, whereas no significant gender differences emerged in functional status or the rate of change. The current investigation's measure of comorbidity, however, was a simple count of the number of affected bodily systems, and was not weighted to account for differences in disease severity, as is typically done in the original CIRS. The nature of symptoms has important implications for illness behavior, such that undoubtedly severe symptoms or medical conditions (e.g., chest pain, stroke, cancer) are more likely to result in formal help-seeking among young and older adults alike, and mild symptoms are more likely to result in self-care (Leventhal et al., 1998). However, to the extent that symptoms are more ambiguous, or judged to be potentially serious, the availability of psychosocial resources might be especially relevant for reducing health anxiety and engaging in adaptive illness behavior responding.

Moreover, age and gender differences exist in the predictive value of social relationships on health and behavior. For example, Sneed and Cohen found that women's

incident hypertension risk was more negatively affected by negative social interactions in late-life than men's, but this effect was limited to young-old adults (ages 51 to 64) and was not observed for individuals ages 65 and older (Sneed & Cohen, 2014). Conversely, another longitudinal study on the effects of perceived social support and social strain on immune functioning among midlife and older adults from the Midlife Development in the United States (MIDUS) found no moderation by age or sex (Yang et al., 2014). In a study by Avlund and colleagues, structural (e.g., phone contact and club membership) and functional aspects (e.g., social support and caregiving) of social relations were evaluated as predictors of functional decline across a 5-year follow-up period in older adults ages 75 and older. Results suggested that both structural and functional aspects predicted functional decline for women, whereas only lack of weekly phone contact was predictive for men (Avlund, Lund, Holstein, Due, Sakari-Rantala, & Heikkinen, 2004). Future research should further examine gender, age, and disease severity as moderators of the associations of social support with illness behavior development and functional decline.

The current work also suggests that illness behavior trends uniquely predict subsequent rates of functional decline in late-life. Importantly, this relationship remained above and beyond the mediating effect of illness behavior status on the relation between social support and the onset of functional difficulty at age 75. Although analyses were adjusted for baseline comorbidity, future studies might further disentangle illness behavior trends from underlying disease processes using advanced, dynamic statistical techniques, such as dual parallel process models. Alternatively, future research should strive to identify the timing of the association between illness behavior and functional

status, given the wide age ranges within the SATSA sample. For example, by combining a cross-lag panel model of illness behavior and growth curves of functional decline, it might be possible to identify at which time-point, or for which age cohorts, the predictive value of illness behavior on health is the strongest. Finally, growth mixture modeling or latent class growth analyses could be used to identify different classes of individuals with particular illness behavior profiles or patterns (adjusting for background variables like age and gender), and the subsequent prediction of functional decline from class membership. Such person-centered approaches to evaluating illness behavior might more readily inform applications within clinical settings.

Conclusion

The current dissertation study sheds light on the longitudinal measurement of illness behavior as a unifying, individual difference construct, as well as the perceived availability of social support as a unique predictor of age-related development in illness behavior across a distinct lifespan transition—from mid-to late adulthood. Moreover, it establishes illness behavior's unique contributions to subsequent functional health and its mediating role in the link between perceptions of social support with functional status. Collectively, these results contribute to the health literature by considering the social predictors of an understudied behavioral pathway with implications for health promotion and the efficiency of healthcare.

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Appendices

Appendix 1. *Social Support Availability Questionnaire Items (SATSA): Study 1 & 2*

Appendix 2. *Perceived Illness Complications Items & Fiske Scale Physician Panel Ratings for SATSA and GENDER*

Appendix 1. ***Social Support Availability Questionnaire Items (SATSA): Study 1 & 2***

Items on the perceived availability of support from **relatives**:

- How many individuals (relatives) do you meet or talk on the phone with in an ordinary week? (1 = *Nobody*; 5 = *11 or more*)
- Is there anybody (relatives) you can go to for support? (1 = *No*; 2 = *Yes*)
- How many individuals to you know in your family with whom you can share your innermost feelings? (1 = *Nobody*; 2 = *1-2*; 3 = *3 or more*)
- How many individuals (relatives) do you know who could share your joy? (1 = *Nobody*; 4 = *6 or more*)

Items on the perceived availability of support from **friends or acquaintances**:

- How many individuals do you know who have the same interests as you? (1 = *Nobody*; 5 = *11 or more)*
- How many friends do you have who could drop in on you at any time? (1 = *Nobody*; 5 = *11 or more)*
- How many individuals in your neighborhood do you know so well you could ask them for things? (1 = *Nobody*; 5 = *11 or more)*
- How many individuals do you know to whom you can turn if you are in trouble? (1 = *Nobody*; 5 = *11 or more)*
- How many individuals (friends) do you meet or talk on the phone with in an ordinary week? (1 = *Nobody*; 5 = *11 or more)*
- Is there anybody special you can go to for support? (1 = *No*; 2 = *Yes*)

- How many individuals (friends) do you know with whom you can share your innermost feelings? (1 = *Nobody*; 2 = *1-2*; 3 = *3 or more*)
- How many individuals (friends) do you know who could share your joy? (1 = *Nobody*; 4 = *6 or more*)

Appendix 2. ***Perceived Illness Complications Items & Fiske Scale Physician Panel Ratings for SATSA and GENDER***

The original Fiske illness scale (Fiske, Gatz, & Pedersen, 2003) included a checklist of 38 possible health conditions, which represented a subset of the original SATSA checklist items that were not gender-specific (e.g., prostate cancer, hysterectomy) or confounded with mental health disorders like depression. For each item, participants indicated if they did (1) or did not (0) have that condition. All items were then separately weighted by the median physician panel's rating of how disabling that condition was expected to be (in the average adult, within the first three years of onset) on a 3-point scale from *little or no disability* (1) to *severe disability* (3), and subsequently summed into a composite (Cronbach's alpha = .61). Inter-rater agreement among the seven physicians on the panel was acceptable, reaching 70 % for most of the items. In addition to the physician panel disability ratings, participants indicated the extent to which each of the health conditions they endorsed complicated their daily lives, on a similar 3-point scale from *not at all* (1) to *a lot* (3). If a participant did not endorse a particular condition, they did not respond to the associated complications item for that condition. The participant-generated disability ratings were posited to be too subjective, however, and were therefore excluded from the Fiske illness scale, which aimed to create a more objective illness composite weighted by disability.

In the current dissertation study, *both* the physician ratings and the participant-generated disability ratings were seen as ideal to consider with respect to constructing an index reflective of Illness Behavior construct, and hence both ratings were leveraged. Specifically, the illness behavior indicator of perceived illness complications was a composite calculated from a subset of these same health condition items across SATSA

and GENDER. Illness items were only included in the composite if they were available across all of the assessment waves, which resulted in a smaller number of items than was included in the Fiske scale (34 items in SATSA, and 24 items in GENDER; respectively). Because participant and physician ratings of disability were both on a parallel, 3-point scale, the perceived illness complications composite summed participants' subjective ratings of illness disability for all endorsed items, while adjusting for the more "objective" physician disability ratings.

To create the composites, a series of difference scores was calculated for each health condition item, which first multiplied the participant's item response (0 = *no* or 1 = *yes*) by the participant's self-reported disability rating for that condition (from 1 to 3, if they endorsed the item), and then subtracted the median physician disability rating for that same item (from 1 to 3). For all checklist items that were not endorsed, the difference scores were calculated to be zero. These difference scores were then summed into a composite. For example, a participant who endorsed diagnoses of hypertension and chronic bronchitis at a particular wave, and who also reported a lot of complications related to both illnesses (a disability score of 3), but no other illness events, would receive the following composite score:

a) Difference score equation:

Δ = (*illness endorsement*self-rating*) – (*illness endorsement*physician rating*)

$\Delta_{\text{hypertension}} = (1*3) - (1*1) = 2$

$\Delta_{\text{chronic bronchitis}} = (1*3) - (1*2) = 1$

b) Composite score equation:

$$\text{Total score} = \sum (\textit{difference scores across all completed items})$$

$$= \sum (\Delta_{\text{hypertension}} + \Delta_{\text{chronic bronchitis}}) = 2 + 1 = 3$$

This person's composite score of + 3 suggests that, across all endorsed health conditions, they reported more complications than would have been expected by a physician. On the other hand, if they had received a total composite score of 0, this would indicate that they were generally in agreement with the physician's expected levels of disability, while a negative score would indicate that they reported fewer complications than would have been expected by a physician. For information on the illness items, median physician disability ratings, and item coverage across the SATSA and GENDER samples, refer to Table A1 below.

Table A1

Fiske Illness Scale Items and Coverage in the SATSA and GENDER samples

Illness scale items (Fiske disability ratings)	**SATSA items**	**GENDER items**
Cardiovascular disorders		
Heart insufficiency (3)	X	X
Angina (2)	X	X
Myocardial infarction (3)	X	X
Phlebitis (1)	X	---
Claudication (2)	X	---
Thrombosis (2)	X	X
Stroke (3)	X	X
Hypertension* (1)	X	X
Respiratory disorders		
Emphysema* (3)	X	---
Chronic bronchitis (2)	X	---
Tuberculosis* (1)	X	---
Asthma* (3)	---	---
Musculoskeletal disorders		
Rheumatoid arthritis* (3)	X	X
Sciatica (2)	X	X
Osteoporosis (1)	X	X
Hip problems (2)	X	X
Neurological disorders		
Migraine (2)	X	X
Seizures (2)	X	---
Epilepsy* (2)	X	X
Parkinson's* (3)	X	X
Multiple sclerosis* (3)	X	---
Speech disorder (1)	X	X
Polio (3)	X	---
Eye disorders		
Blind* (3)	---	---
Ear disorders		
Deaf* (2)	---	---
Metabolic disorders		
Diabetes* (2)	X	X
Goiter (1)	X	X
Anemia (1)	---	---
Gout (1)	X	X
GI disorders		
Ulcer (2)	X	X

Intestinal disorder (2)	X	---
Gall disorder (1)	X	X
Liver disease* (2)	X	X
Urologic disorders		
Kidney disease* (2)	X	---
Urinary tract disorder (1)	X	X
Skin disorders		
Shingles (2)	X	X
Psoriasis (1)	X	X
Cancer		
Cancer* (non-specific) (3)	X	X

Note. Values in parentheses are the median physician disability ratings. 1 = *little or no disability*; 2 = *some disability*; 3 = *severe disability*.

* Condition onset was judged as likely to occur no more than once during the study period (Fiske et al., 2003).