The Influence of Behavioral and Cognitive Self-Regulation on Older Adults' Psychological, Biological, and Physical Health

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ABSTRACT

The Influence of Behavioral and Cognitive Self-Regulation on Older Adults' Psychological, Biological, and Physical Health

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As individuals age, they are at increased risk of experiencing health threats and loss of resources (Baltes et al., 1997; Heckhausen, Wrosch, & Schulz, 2010). Such age-related challenges can contribute to the experience of psychological distress and biological dysregulations, which in turn may instigate further deterioration of individuals' physical health (Schulz, Martire, Beach, & Scheier, 2000; Wrosch, Schulz, & Heckhausen, 2004). However, theory and research also suggest that normative losses in older adults' psychological, biological, and physical health can be circumvented if a person engages in adaptive self-regulation to address such age-related challenges (e.g., Baltes & Baltes, 1990; Carstensen et al., 1999; Hall, Chipperfield, Heckhausen, & Perry, 2010; Hamilton, Catley, & Karlson, 2007; Heckhausen & Schulz, 1995).

Using ideas from the Motivational Theory of Life-Span Development (Heckhausen & Schulz, 1995; Heckhausen, Wrosch, & Schulz, 2010), the three longitudinal studies of this dissertation extend prior lines of research by investigating whether the appropriate use of specific behavioral and cognitive self-regulation strategies can ameliorate age-associated losses among the elderly in general, and especially among "at-risk" individuals who confront adverse conditions (i.e., elevated stress or feelings of loneliness). The different studies are all based on the same multi-dimensional and multi-wave longitudinal dataset. While they address independent research questions, the nature of this dissertation creates some overlap in the reported procedures across studies.

Study 1 investigates the different roles of stress in the association between physical activity engagement and physical health among 157 older adults over four years of data collection. Results demonstrated that older adults' physical health symptoms increased across time. In addition, older adults' engagement in physical activity predicted a reduction of stress perceptions over two years and fewer physical health problems over four years among participants who perceived high levels of stress at baseline, but to a lesser extent among their counterparts who perceived low levels of stress at baseline. Finally, the long-term physical health benefits among highly stressed individuals were statistically mediated by a reduction of stress perceptions over two years. These findings suggest that physical activity has the potential to ameliorate chronically high perceptions of stress and thereby produce long-term benefits on older adults' physical health.

Study 2 examines the long-term associations between reports of sleep duration and diurnal cortisol secretion over four years among 157 older adults. The study's findings demonstrate that older adults experienced increases in diurnal cortisol secretion over time, and that this effect was forecasted by individual differences in sleep duration. Results from growth curve and cross-lagged panel analyses demonstrated that higher levels of and increases in sleep duration buffered long-term elevations of diurnal cortisol secretion. By contrast, lower levels of and declines in sleep duration predicted a steep increase in cortisol secretion over time. Reversed analyses indicated that diurnal cortisol secretion did not contribute to changes in sleep duration over time. These findings suggest that longer sleep exerts restorative functions and protects older adults from experiencing maladaptive increases in diurnal cortisol secretion over time.

Study 3 investigates in a sample of 122 older adults whether health-related self-

protection (e.g., avoiding self-blame for health problems or seeing the silver lining) can predict psychological and biological benefits among lonely (as compared to non-lonely) participants. Results demonstrated that baseline self-protection predicted a reduction of psychological stress and diurnal cortisol volume over two years, and lower CRP after 6 years, but only among lonely participants. In addition, mediation analyses demonstrated that the observed short-term reduction of cortisol secretion mediated the effects of self-protective control on lower long-term levels of systematic inflammation. These findings suggest that lonely older adults may ameliorate stress-related psychological and biological disturbances if they engage in self-protection to cope with emerging health threats.

Overall, these findings contribute to our knowledge about pathways to healthy aging. The results are discussed in light of life-span theories of successful aging and general psychological models of health and disease. In addition, implications for clinical practice and future research are addressed.

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GENERAL INTRODUCTION

The face of Canada and most other countries is being reshaped by demographic changes, given that the proportion of seniors has been increasing more rapidly over the last decades than all other age groups (Oeppen & Vaupel, 2002). This trend is expected to continue for the next decades mainly due to a decreased fertility rate (i.e., number of children by woman), an increase in life expectancy, and the effects of the baby boom. In 2010, 14 percent of the Canadian population was over the age of 65, a number that is expected to almost double over the next 25 years to reach 23 percent by 2031 (Statistics Canada, 2010). However, although health innovations and practices have contributed to extended life expectancies, older adults are not necessarily physically healthy (Rosenberg & Moore, 1997). In fact, the aging process is typically characterized by an inevitable accumulation of losses in a person's cognitive, psychological and physiological health status (Schulz & Heckhausen, 1996). As medical support and social services for addressing older adults' needs remain insufficient, the elderly are required to draw on their personal resources to effectively manage emerging age-related problems.

In this regard, several models have been developed over the past years, in an attempt to illuminate strategies that can help older individuals adapt to their changing circumstances (Baltes & Baltes, 1990; Brandtstädter & Renner, 1990; Carstensen, 1991; Heckhausen et al., 2010; Schulz & Heckhausen, 1996). An overarching assumption of these theories is that older adults can use self-regulatory strategies to attenuate the adverse consequences of age-related losses, while maintaining the capacity for developmental gains on many fronts. Further, the Motivational Theory of Life-Span Development specifies that individuals can create environments in which success is

possible, by adjusting goal engagement and goal disengagement strategies to changing contextual opportunities and constraints (Heckhausen et al., 2010). These theoretical propositions have been supported by a large body of empirical evidence demonstrating the differential benefits of behavioral engagement and cognitive disengagement strategies for managing stressors across the life-course (Heckhausen & Brim, 1997; Heckhausen & Schulz, 1995; Wrosch & Heckhausen, 1999; Wrosch, Heckhausen, & Lachman, 2000).

To illuminate pathways to successful aging, this dissertation expands a previously proposed model (Wrosch et al., 2004; Wrosch, Dunne, Scheier, & Schulz, 2006) to explain how older adults can manage normative declines in psychological, biological, and physical health outcomes. This model suggests that the adverse psychological consequences of age-related challenges can elicit biological dysregulations, and thereby contribute to physical health decline. Inspired by ideas from the Motivational Theory of Life-Span Development (Heckhausen et al., 2010), the model also proposes that older adults' engagement in adaptive behavioral and cognitive strategies may avoid the adverse effects of age-related challenges on psychological distress and through this mechanism benefit older adults' biological and physical health (Wrosch et al., 2004, 2006).

This dissertation is based on the theoretical tenet that older adults' engagement in proactive behaviors may be particularly well-suited to serve adaptive functions if opportunities for actively overcoming an adverse challenge are favourable (Wrosch et al., 2004). In this regard, it examines specific health-related engagement strategies that include energy expenditure (i.e., physical activity) and regeneration (i.e., restorative sleep) not only because of their widespread effects on emotional, biological, and physical functioning (Balbo, Leproult, & Van Cauter, 2010; Hamilton, Nelson, Stevens, &

Kitzman, 2007; Warburton, Nicol, & Bredin, 2006), but also because they cover a wide range of active and passive health behaviors within the 24 hour sleep-wake cycle. In contrast, in circumstances in which a challenge can hardly be addressed by a person's active coping efforts (e.g., socio-emotional problems or chronic health conditions), older adults' may be better off if they employ cognitive efforts that foster goal disengagement, such as self-protective attributions of failure experiences.

In preliminary support of the proposed model, studies have shown that aging can be associated with maladaptive changes in psychological distress, biological processes (e.g., cortisol patterns and immune function), and physical health outcomes (McEwen & Stellar, 1993; Sapolsky, 1992; Segerstrom & Miller, 2004). Further, research suggests that older adults can counteract these problems if they engage in health-related behavioral (e.g., physical activity and restorative sleep) and cognitive strategies (e.g., self-protective attributions of failure experiences). In this respect, it has been found that the adaptive value of these self-regulation strategies can vary with regard to the individual's opportunities for goal attainament (e.g., Wrosch et al., 2000). In addition, there is evidence showing that psychological distress can play a mediating role in the associations between age-related challenges and health-related outcomes (e.g., Wrosch, Schulz, Miller, Lupien, & Dunne, 2007).

Although these findings are consistent with the idea that adaptive self-regulation can contribute to psychological, biological, and physical health benefits, they are also associated with several limitations. These limitations are primarily due to the cross-sectional nature of the available research, the limited scope of self-regulation strategies and health-related variables that are currently being researched, and the lack of studies

examining changes in individuals' self-regulation processes over time. Moreover, the extant research provides evidence only for bits and pieces of the proposed model. For instance, it remains still unclear whether adaptive self-regulation processes can mediate physical health changes through its effects on psychological distress and biological dysregulation. As a consequence of these limitations, conclusions regarding the effectiveness of specific behavioral and cognitive strategies for overcoming age-related challenges remain limited. Thus, additional longitudinal research is needed to document the complete process linking age-related challenges and adaptive self-regulation with distress, biological processes, and physical health.

Research Objectives

The present studies contribute to the growing body of literature and empirical research on the topics of successful aging and self-regulation. The objective of these three interrelated studies is to investigate the adaptive value of behavioral and cognitive strategies for counteracting adverse changes in psychological distress, biological dysregulations, and physical health problems among older adults dealing with and without additional risk factors for health. To this end, the first two studies focus on the adaptive value of behavioral engagement strategies related to sleep and physical activity, whereas Study 3 investigates cognitive disengagement strategies related to self-protective attributions.

Study 1: The Different Roles of Perceived Stress in the Association Between Older Adults' Physical Activity and Physical Health

Study 1 attempts to investigate whether engagement in physical activity could play an important role in maintaining older adults' long-term physical health. While

previous studies have demonstrated that health benefits of physical activity may occur particularly among individuals who perceive high levels of stress (Brown, 1991; Brown & Siegel, 1988; Carmack, Boudreaux, Amaral-Melendez, Brantley, & Moor, 1999), and that physical activity can reduce stress levels in vulnerable populations that confront problematic life circumstances (for a review see Salmon, 2001), there is no research to date that has documented the complete process linking physical activity, perceived stress, and physical health outcomes over time. In this regard, the present study extends prior lines of research, by examining whether the beneficial effects of physical activity on the prevention of chronically high levels of stress can mediate subsequent levels of physical health. In line with these objectives, this study addresses the following main research questions:

> ***Question 1****: Can baseline levels of physical activity prevent 4-year increases in physical health problems particularly among participants who perceive high baseline levels of stress, but to a lesser extent among participants who perceive low baseline levels of stress?*
>
> ***Question 2****: Can baseline levels of physical activity contribute to 2-year declines in perceived stress particularly among participants who perceive high baseline levels of stress, but to a lesser extent among participants who perceive low baseline levels of stress?*
>
> ***Question 3****: Can 2-year changes in perceived stress statistically mediate the association between baseline levels of physical activity and improvements in physical health over four years among highly stressed (but not among low stressed) older adults?*

Study 2: Sleep Duration Buffers Diurnal Cortisol Increases in Older Adulthood

The objective of Study 2 is to illuminate the long-term associations between the engagement in adaptive sleep patterns and diurnal cortisol secretion in older adulthood. Research suggests that the restorative functions of long sleep may have the potential to down-regulate high cortisol levels (e.g., Balbo et al., 2010; Kumari, Badrick, Ferrie, Perski, Marmot, & Chandola, 2009), and that these effects can be especially adaptive in older adulthood when age-related challenges contribute to psychological and biological disturbances (Wrosch et al., 2008; Wrosch, Miller, Scheier, & Brun de Pontet, 2007). In addition, there is some evidence that cortisol can also be associated with changes in sleep patterns, although such effects have not been found consistently across studies (for a review see Steiger, 2003). In an attempt to overcome limitations associated with past research on the role of sleep in older adulthood, this study is set out to examine the directional relation between older adults' sleep duration and diurnal cortisol secretion, by tracking changes in these variables over a prolonged period of time. In line with these objectives, this study addresses the following main research questions:

> ***Question 1****: Can longer (as compared to shorter) sleep protect older adults from experiencing 2-year increases in diurnal cortisol secretion over time?*
>
> ***Question 3****: Can diurnal cortisol patterns also contribute to changes in a person's sleep duration over time?*

Study 3: Health-Related Self-Protection Predicts Psychological and Biological Benefits among Lonely Older Adults

Study 3 seeks to elucidate the effects of self-protective cognitive strategies (i.e., avoiding self-blame for health problems or positive reappraisals) on psychological

distress and biological dysregulations (i.e., elevated cortisol secretion and inflammation) among lonely (as compared to non-lonely) older adults. The extant evidence suggests that feelings of loneliness can increase people's risk of a variety of physical health problems (Cacioppo et al., 2002b; Kiecolt-Glaser et al., 1984; Russell, Cutrona, de la Mora, & Wallace, 1997; Seeman, 2000), and such adverse health consequences may be related to lonely individuals' heightened vulnerability to psychological stress and its biological sequelae (Hawkely & Cacioppo, 2010). While these health-compromising effects may be especially profound in older adulthood (Hawkley & Cacioppo, 2007), theory and research also suggest that older adults can cope with health threats and emotional turmoil by engaging in internal self-protection (e.g., Heckhausen et al., 2010; Wrosch, Miller, & Schulz, 2009). To address the pressing need for understanding how older adults can deal with feelings of loneliness, this study investigates whether the use of self-protective cognitive strategies may have the capacity to counteract adverse psychological and health-related processes among lonely older adults. In addition, Study 3 seeks to contribute to knowledge about the effects of psychological and biological variables on a persons' immune system, by investigating the mediating roles of psychological distress and cortisol secretion in the association between self-protective coping and inflammatory cytokines among lonely older individuals. In line with these objectives, this study addresses the following main research questions:

> ***Question 1****: Can self-protective control strategies for managing health threats (i.e., positive reappraisal and external attributions) prevent 2-year increases in psychological stress and diurnal cortisol secretion, as well as higher 6-year levels*

of the inflammatory biomarker C-reactive protein (CRP) among lonely (as compared to non-lonely) older adults?

Question 2*: Can longitudinal changes in perceived stress and cortisol secretion mediate the effects of self-protective control strategies on 6-year levels of CRP among lonely (as compared to non-lonely) older adults?*

REVIEW OF THE LITERATURE

Age-related Challenges, Psychological Distress, and Health

The aging process is typically characterized by an accumulation of significant and often unavoidable challenges, such as increased exposure to death and bereavement, loss of independence and autonomy, as well as cognitive and physical health declines (e.g. Baltes, 1987; Heckhausen, 1999). The experience of such age-related challenges can compromise older adults' quality of life and elicit negative affective states, such as depressive mood and anxiety (for a review see Wrosch et al., 2006). For example, older adults who exhibit specific chronic health conditions that are likely to emerge during the aging process, such as knee osteoarthritis or rheumatoid arthritis, have been found to report increased levels of depressive symptoms (Penninx et al., 1996). In addition, societal life changes (e.g., entry into care) and hardships originating from aging (e.g., widowhood, poor functional status, or loss of hearing) may be likely to elicit feelings of social isolation (e.g., Savikko, Routasalo, Tilvis, Strandberg, & Pitkälä, 2005). For example, older people who lose their spouse or live in residential homes may be vulnerable to feelings of existential loneliness because of their loss of identity-salient roles.

Further, the adverse psychological consequences of age-related challenges on the experience of distress may instigate biological problems in older adulthood, thereby leading to additional health problems (Cohen, Janicki-Deverts, & Miller, 2007; Heim, Ehlert, & Hellhammer, 2000; McEwen, 1998). Such adverse effects of psychological distress on biological and physical health have further been shown to increase with age in a way that accelerates normative declines in a persons' physiological resilience (Hawkley

& Cacioppo, 2007; Hawkley, Masi, Berry, & Cacioppo, 2006; Segerstrom & Miller, 2004).

Together, research suggests that with advancing age individuals become more likely to encounter life circumstances that can instigate psychological problems, such as perceived stress or feelings of loneliness. This emotional vulnerability stemming from age-related decline may be profound enough to trigger maladaptive changes in biological processes and thereby contribute to further physical health problems. This association between psychological and physical health appears to be reciprocal, suggesting that older adults dealing with age-related health threats are not only at increased risk of experiencing emotional problems, but also of developing further health problems as a result of their emotional problems (for a review see Lenze et al., 2001; Wrosch et al., 2004).

Psychological Distress, Biological Dysregulation, and Physical Health in Older Adulthood

One key biological mechanism through which a psychological threat (e.g., perceived stress or feelings of loneliness) may influence physical health is associated with the hypothalamus–pituitary–adrenal (HPA) axis (Cohen et al., 2007; Hawkely & Cacioppo, 2010; McEwen, 2003). This neuroendocrine system helps the organism adapt to increased demands and maintain homeostasis after challenge. The hypothalamus secretes corticotropin-releasing hormone (CRH) in response to physiological or psychological distress, and this process provokes the release of adrenocorticotropic hormones (ACTH) from the pituitary. ACTH triggers the secretion of glucocorticoids, such as cortisol, from the adrenal cortex. The HPA axis is a regulatory system that is

controlled by several negative feedback loops to ensure that sufficient amounts of cortisol are released to adapt to a stressor (Tsigos & Chrousos, 2002; Watts, 2000). After sufficient levels of cortisol have been secreted to help the organism deal with the threat, circulating cortisol feed back on glucocorticoid receptors located in the anterior pituitary hypothalamus, and hippocampus to inhibit ACTH and CRH synthesis and release. Cortisol has widespread regulatory influences on the body which include the mobilization of glucose, the regulation of fluid volume, the modulation of immune function, and various central nervous system effects on behaviors related to feeding, sleeping, and learning (Sapolsky, Romero, & Munck, 2000). Short-term cortisol release in response to an acute stressor is considered to be an adaptive mechanism aimed at mobilizing energy and cardiovascular functions to enhance survival during an emergency situation (Chrousos & Gold, 1992). By contrast, exposure to chronically high levels of cortisol secretion can trigger dysregulations in a person's immune, metabolic, and central nervous systems (Heim et al., 2000; Lupien et al., 1998; Weiner, 1992) and contribute to the onset of various diseases, including cardiovascular disease, diabetes, hypertension, and cancer (Bjorntorp & Rosmond, 1999; Lupien, McEwen, Gunnar, & Heim, 2009; McEwen & Stellar, 1993; Sapolsky, 1992).

In fact, it is widely recognized that the biological stress response interacts with other bodily systems, such as the immune system, in affecting health (Segerstrom & Miller, 2004). In this regard, research suggests that cortisol secretion can have different consequences for a person's immune function. For instance, normative cortisol release can suppress immune funtion and thereby exert anti-inflammatory effects (Chrousos & Gold, 1992; Sapolsky, Romero, & Munck, 2000). By contrast, dysregulated cortisol

release (i.e., chronically elevated or blunted cortisol release) can have the reversed effects and may render innate immune cells partially resistant to glucocorticoid inhibition, allowing inflammation to escape normal regulatory controls (Kiecolt-Glaser, Mcguire, Robles, & Glaser, 2002; Miller et al., 2008; Raison & Miller, 2003). For example, using functional genomic methodology, Miller and colleagues (2008) have found evidence suggesting that psychological distress was associated with an underlying resistance to glucocorticoid signaling, which may stimulate the production of pro-inflammatory markers. In support of this idea, chronically stressed individuals have been found to exhibit increased systematic inflammatory activity, delayed healing of experimentally administered wounds, and blunted antibody responses to vaccination (e.g., Irwin et al., 1998; Miller, Cohen, & Ritchey, 2002; Miller, Freedland, Duntley, & Carney, 2005; Raison, Capuron, & Miller, 2006). Given that inflammatory cytokines may play a pathogenic role in various infectious, cardiovascular, physical and neoplastic diseases (Coussens & Werb, 2002; Eccles, 2005; Libby & Theroux, 2005; Perkins, 2007), it has been suggested that the endocrine consequences of psychological distress on a person's physical health could be conferred through inflammatory pathways (Segerstrom & Miller, 2004). However, empirical evidence for this idea remains insufficient. While some studies have found that atypical patterns of cortisol can forecast increases in inflammatory biomarkers, other studies have failed to find such associations (Miller et al., 2002).

Further, it has been outlined that the capacity of the organism to adapt to a challenge and to maintain or regain homeostatic balance may decrease with age (McEwen & Stellar, 1993; Sapolsky, 1992). In fact, accumulating evidence suggests that

individuals may experience increases in their basal levels of diurnal cortisol secretion with advancing age (Almeida, Piazza, & Stawski, 2009; Kern, Dodt, Born, & Fehm, 1996; Van Cauter, Leproult, & Kupfer, 1996, Van Cauter, Leproult, & Plat, 2000). In this regard, a meta-analysis conducted by Otte et al. (2005) demonstrated that older adults tend to exhibit elevated cortisol secretion in response to psychological and physical challenges. In addition, older adulthood has been associated with dysregulations in many aspects of adaptive immune function (Castle, Uyemura, Fulop, & Makinodan, 2007; McLachlan, Serkin, Morrey, & Bakouche, 1995; Pawelec & Solana, 1997). Indeed, a substantial body of evidence documents age-related increases in plasma levels and stimulated production of the inflammatory biomarkers C-reactive protein (CRP) and interleukin-6 (IL-6), which signal a chronic and systematic state of mild inflammation; Ershler & Keller, 2000; Kiecolt-Glaser, Mcguire, Robles, & Glaser, 2002; Miller et al., 2002, 2008; Roubenoff et al., 1998). Moreover, recent evidence suggests that increased plasma levels of CRP also actively contribute to a wide range of diseases associated with aging, including incident cardiovascular disease (Ridker, Rifai, Rose, Buring, & Cook, 2002), functional disability (Kuo, Bean, Yen, & Leveille, 2006), type 2 diabetes (Pradhan, Manson, Rifai, Buring, & Ridker, 2001), and dementia (Engelhart et al., 2004). Further, elevated CRP has been associated with increased short- and long-term risk of death in prospective cohort studies involving initially healthy middle-aged and older adults (e.g., Jenny, Yanez, Psaty, Kuller, Hirsch, & Tracey, 2007; Ridker & Silvertown, 2008; Strandberg & Tilvis, 2000). Accordingly, it seems to be likely that age-related dysregulations in the endocrine and immune system can accelerate the rate of physiological decline in older adulthood (Straub, Cutolo, Zietz, & Scholmerich, 2001).

In sum, the discussed research indicates that many older adults are at elevated risk of encountering a cascade of emotional, biological, and physical health problems. However, a clear pattern linking these processes has not yet been established. For instance, psychological distress has not always been shown to forecast biological dysregulations (Burke, Davis, Otte, & Mohr, 2005; McEwen, 2000; Miller et al., 2002), and research is struggling with demonstrating a clear relationship between biological functioning and physical health (Miller et al., 2004; Wrosch, Miller, Lupien, & Pruessner, 2008). Similarly, research demonstrates considerable individual differences in age-associated changes of cortisol patterns (Lupien et al., 1998; 2005; Seeman, McEwen, Singer, Albert, & Rowe, 1997) and immune markers (Beharka, Meydani, Wu, Leka, Meydani, & Meydani, 2001; Butcher et al., 2001). This variability in older adults' emotional, biological, and physical health trajectories suggests that there may be protective factors that could help some older adults maintain or restore adequate biological and physical functioning despite normative decline.

How to Manage Age-related Challenges in the Elderly

Several life-span models have been developed over the last decades, in an attempt to explain how individuals can influence their development and regulate or prevent the negative consequences of age-related challenges (Baltes & Baltes, 1990; Carstensen, Isaacowitz, & Charles, 1999; Heckhausen & Schulz, 1995). In general, these models portray individuals as active agents of their development (Lerner & Busch-Rossnagel, 1981) and postulate that the elderly can cope with the emergence of age-related losses and continue to actively develop themselves on numerous fronts by engaging in adaptive goal-related and emotion-regulation processes (Baltes & Baltes, 1990; Carstensen et al.,

1999; Heckhausen & Schulz, 1995). As such, successful aging is not only marked by the compensation for loss, but also involves the potential for gains (Baltes, 1987).

One of the most influential models of successful aging is the life-span model of Selective Optimization with Compensation (SOC), which proposes that older adults can remain satisfied with their lives and experience advances in selected domains if they engage in selection, optimization, and compensation (Baltes & Baltes, 1990). From the view-point of this model, individuals *select* goals that are important to them, *optimize* the internal resources and external aids that facilitate goal attainment, and *compensate* for losses (via the acquisition and use of alternative means) in order to attain personal goals and maintain quality of life despite age-related losses. While the three components of successful development are assumed to be operating throughout life and in all domains of functioning, their use may become particularly important in the later stages of life by enabling individuals to restore prior levels of functioning and promoting further development in selected areas (Baltes & Baltes, 1990; Baltes & Carstensen, 1996; Freund & Baltes, 2000; Marsiske, Lang, Baltes, & Baltes, 1995).

Heckhausen and colleagues extended and elaborated the idea of selective optimization with compensation in their Motivational Theory of Life-Span Development (Heckhausen et al., 2010; Heckhausen & Schulz, 1995; Schulz & Heckhausen, 1996, White, 1959). The authors take a different approach to successful aging based on the premise that human motivation is guided by a fundamental and universal need associated with striving for control. According to their theory, successful development depends on the individual's ability to adjust goal engagement and goal disengagement strategies to changing opportunities and constraints over the life course in order to maximize their

long-term capacity for primary control (i.e., having an active influence on one's development, Heckhausen et al., 2010). When control opportunities are present, such as in the face of manageable health problems, the capacity for primary control is maintained or even expanded by engagement strategies aimed at proactively changing the environment (e.g., persistence, goal commitment). By contrast, when opportunities are limited, such as in the face of irreversible health problems, disengagement strategies (e.g., positive reappraisal and external attributions of losses) become increasingly important in order to minimize failure experiences and maintain the potential for pursuing and achieving important goals (Heckhausen et al., 2010; Heckhausen & Brim, 1997; Heckhausen & Schulz, 1995; Wrosch et al., 2000; Wrosch & Heckhausen, 1999). Hence, the adaptive value of goal engagement and disengagement processes depends on whether they will help optimize an individual's long-term capacity for primary control.

According to a taxonomy originally proposed by Heckhausen and Schulz (1993), goal engagement and disengagement has been categorized in terms of four different control strategies. *Selective primary control* refers to the investment of personal resources (e.g., effort and time) to achieve one's goal. *Compensatory primary control* entails the recruitment of external resources (e.g., technical aids or other people's help) when one's own capabilities are insufficient. *Selective secondary control* strengthens the volitional commitment to a chosen goal. Whereas the former three types of control represent goal engagement, the function of *compensatory secondary control* serves to protect the motivational and emotional resources via goal disengagement when one's personal control is threatened and when goal pursuit becomes maladaptive (Heckhausen et al., 2010).

Although control processes related to engaging in and disengaging from goals can involve both action and cognition, goal engagement is more frequently characterized in terms of behaviors engaging the external world (e.g., investing effort and time in the pursuit of a goal and using external aids). By contrast, goal disengagement is typically characterized in terms of internal cognitive processes, such as adjusting expectancies and making self-protective attributions, aimed at preventing the futile investment of motivational resources and redirecting them toward productive endeavours (Hall et al., 2010; Heckhausen & Schulz, 1995, 1999).

While the reviewed models of successful aging suggest that older individuals can adapt to their changing life conditions by engaging in adaptive behavioral and cognitive processes, they do not indicate the specific goal domains that will be favoured by older adults dealing with age-related threats. Carstensen and her colleagues (Carstensen, 1992; Carstensen et al., 1999; Carstensen, Fung, & Charles, 2003) addressed this issue by examining the specific processes of adaptive functioning in the domains of social relationships and emotions. The Socioemotional Selectivity Theory (SST; Carstensen, 1991; Carstensen et al., 1999) claims that individuals select and pursue social goals in congruence with their perceptions of remaining lifetime. When time is perceived as open-ended, such as in younger adulthood, future-oriented social goals that are related to the pursuit of knowledge and information gathering are prioritized. In contrast, when time horizons are constrained, such as in older adulthood, individuals shift their focus to present-oriented social goals, which prioritize emotional meaning. As a consequence, older adults narrow their social networks to devote more emotional resources to fewer relationships with close friends and family (Carstensen et al., 1999; Lang & Carstensen,

2002). In support of these assumptions, empirical evidence indicates that older (as compared to younger) individuals prioritize familiar social partners (Carstensen et al., 1999; Lang & Carstensen, 2002; Fredrickson & Carstensen, 1990) and smaller social networks, with a higher percentage of emotionally close partners (Lang, 2001). These motivational shifts in the face of constrained life time contribute to maximizing gains and minimizing risks in social and emotional domains (Löckenhoff & Carstensen, 2004).

Together, the reviewed literature acknowledges losses as an inevitable part of the aging process. In addition they also suggest that older adults are able to manage losses and continue to generate positive outcomes. In order to maximize the potential for a meaningful and satisfying existence in the later stages of life, individuals engage in action (e.g., health-related behaviors or emotionally satisfying social interactions) and/or cognition (e.g., self-protective attributions) to create an environment, which makes success possible while effectively dealing with age-related decline. In this regard, the different aging models, and in particular the Motivational Theory of Life-Span Development, provide a conceptual framework for examining protective cognitive and behavioral mechanisms that may help older adults deal with, or even prevent, the adverse consequences of encountering age-related challenges.

Drawing upon the previously discussed research and theory, this dissertation expands a previously proposed model (Wrosch et al., 2004, 2006) to explain how older adults can manage age-related challenges. This model is illustrated in Figure 1 and suggests that the adverse psychological consequences of age-related challenges (Fig. 1, path *a*-*b*) can elicit biological dysregulations (Fig. 1, path *b*-*c*), and thereby contribute to physical health decline (Fig. 1, path *c*-*d*). For example, age-normative losses may

instigate psychological distress, which in turn may trigger patterns of biological dysregulation in the endocrine and immune systems (e.g., cortisol or C-reactive protein) and increase an older person's likelihood of developing physical disease (Cohen et al., 2007; Heim et al., 2000; Kiecolt-Glaser et al., 2002; McEwen, 2003). To account for the observation that psychological and physical health variables can reciprocally influence each other (Lenze et al., 2001; Wrosch et al., 2004, 2008), the model also incorporates several feedback loops. For instance, the experience of physical health complaints may trigger psychological distress either directly or indirectly through the interplay with biological processes (Fig. 1, path *d*-*b*). In addition, psychological distress may accelerate the occurrence of certain age-related challenges, such as cardiovascular disease (Rozanski, Blumenthal, & Kaplan, 1999; Fig. 1, path *b*-*a*). Of importance, the model also proposes that a downward spiral characterized by age-related challenges, psychological distress, biological dysregulation, and further health decline may be disrupted if older adults engage in adaptive self-regulation, including adaptive health behaviors and cognitions (Fig. 1, effect of adaptive self-regulation on association *a*-*d*).

According to the Motivational Theory of Life-Span Development (Heckhausen et al., 2010), the adaptive effects of older adults' engagement in proactive control behaviors can be particularly appropriate in circumstances in which opportunities for overcoming adverse challenges are present, such as in the light of manageable health problems (Wrosch et al., 2004). In this regard, it may be useful to examine health behaviors that include energy expenditure (i.e., physical activity) and regeneration (i.e., restorative sleep). Investigating these behaviors may not only be important because of their widespread effects on emotional, biological, and physical functioning (Balbo et al., 2010;

Faulkner, Green, & White, 1994; Hamilton, et al., 2007; Warburton et al., 2006), but also because they cover a wide range of active and passive health behaviors within the 24 hour sleep-wake cycle. While the engagement in physical activity requires motivation as well as an adequate level of physical functioning, restorative sleep patterns can be maintained mainly independent of an individuals' initiative or level of functioning. Thus, restorative sleep may represent a useful and alternative strategy, which may be particularly well-suited to protect quality of life among older adults who experience serious age-related declines of resources and are unable to engage in more active health behaviors. This argument is consistent with theories suggesting that the adaptive value of a specific behavioral pattern may depend on older adults' particular circumstances and opportunities for influencing their development (e.g., Heckhausen et al., 2010).

Given that aging is a heterogeneous process, older adults are likely to experience a wide range of age-related challenges, which require the use of different coping strategies in order to adequately address the controllability of various specific problems. Although a substantial amount of older adults may maintain the capacity to improve their emotional, biological, and physical health by engaging in adaptive health behaviors (Rowe & Kahn, 1998), it has been found that control opportunities generally decline with advancing age due to the accumulation of irreversible problems and developmental constraints (Heckhausen, Dixon, & Baltes, 1989; Heckhausen & Schulz, 1995).

In this regard, the Motivational Theory of Life-Span Development argues that the adaptive effects of self-protective cognitions (e.g., positive reappraisals or external attributions for health threats) on psychological, biological, and physical health may become particularly pronounced in older adulthood when opportunities for goal

attainment decrease. Such adaptive effects are likely to be observed if self-protective processes foster acceptance of, and disengagement from, problems that cannot be overcome through a person's active coping behaviors (Heckhausen et al., 2010; Heckhausen & Schulz, 1995). Given that emotional, biological, and physical processes may interact with each other, it is further plausible that the emotional benefits derived from cognitive disengagement strategies could protect older adults against biological dysregulations and thereby provide physical health benefits. For example, a lonely older person may be able to maintain high levels of psychological well-being, biological functioning, and consequently physical health if he or she engages in internal self-protection (e.g., seeing the silver lining or avoiding self-blame for problems) to cope with socio-emotional problems. Conversely, a lonely older adult who fails to endorse such internal self-protective processes may be at increased vulnerability for experiencing the adverse consequences of socio-emotional problems on psychological, biological, and physical health. Based on these considerations, it seems likely that self-protective coping may become paramount in circumstances in which active coping is insufficient or even fruitless for dealing with a problem, such as in the context of certain emotional problems or chronic health problems.

In sum, older adults may be able to counteract the adverse consequences of age-related challenges on their emotional, biological, and physical health by engaging in appropriate (opportunity-adapted) self-regulation strategies. As illustrated in Figure 1, such adaptive strategies may be adaptive at any stage of the proposed downward spiral. This implies that older adults are not generally at risk of experiencing an adverse cascade of health-related problems per se, but that such an adverse process may be observed only

when adaptive self-regulation are unable to adequately address some of the health-compromising processes associated with increasing age-related challenges.

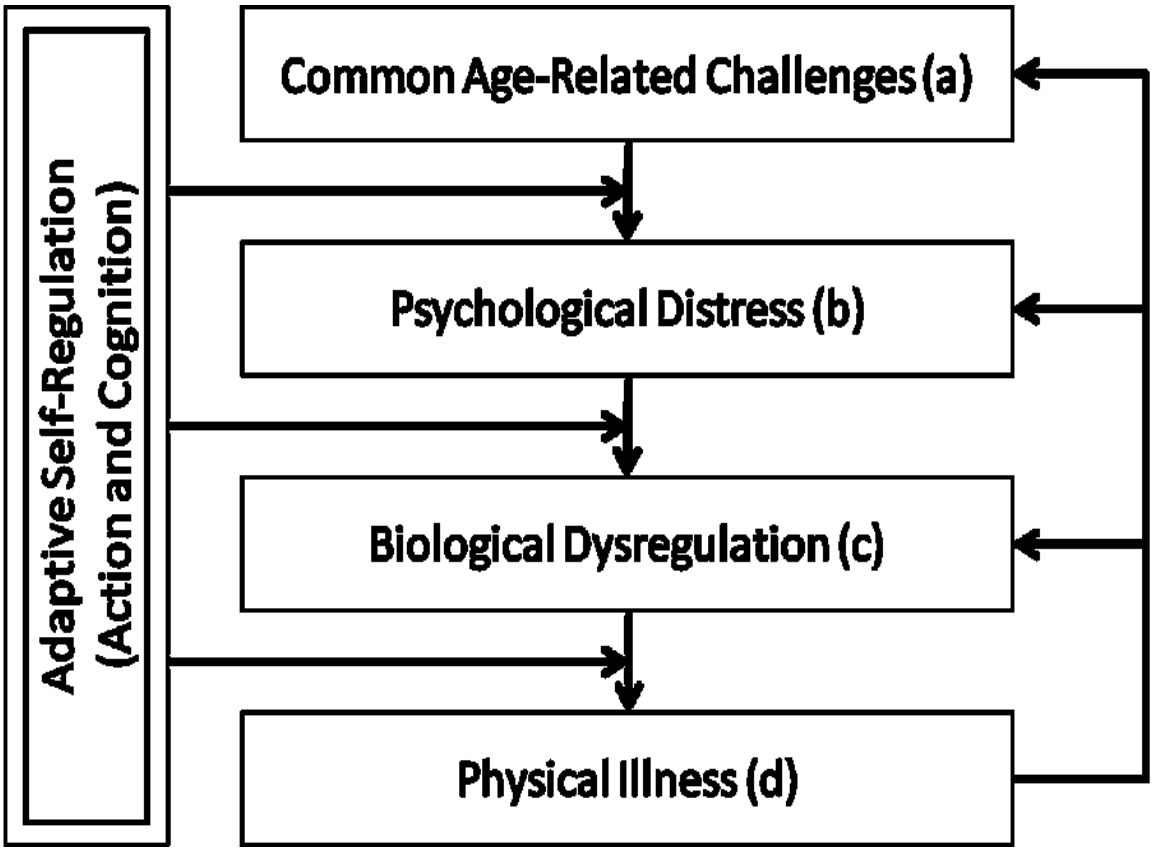

Fig. 1. Theoretical model illustrating the importance of adaptive self-regulation in the associations between common age-related challenges (a), emotional distress (b), biological dysregulation (c), and physical illness (d) in older adults.

Evidence for the Effectiveness of Adaptive Self-regulation in Older Adulthood

Cognitive Strategies

Preliminary support for the proposed model stems from a number of studies documenting that cognitive strategies that foster acceptance of, and disengagement from, uncontrollable problems can attenuate the psychological consequences of specific age-related challenges (Heckhausen, Wrosch, & Fleeson, 2001; Wrosch & Heckhausen, 1999, 2002; Wrosch et al., 2000; Wrosch, Schulz, & Heckhausen, 2002). For example, Wrosch and colleagues examined the adaptive value of internal self-protection (e.g., seeing the silver lining or avoiding self-blame) for coping with numerous adverse physical and psychological challenges, including physical illness, unattainable goals, and life regrets (for review see Wrosch et al., 2006). This body of research has shown across different areas of life that self-protective processes are associated with greater psychological well-being among older individuals who experience age-related financial and health stressors (Wrosch et al., 2000), confront a partnership separation (Wrosch & Heckhausen, 1999), or women who had passed the biological clock for having their own children (Heckhausen et al., 2001). For instance, in a six-year longitudinal study of 135 community-dwelling older adults, Dunne and colleagues (2011) have shown that functional disability forecasted increases in depressive symptomatology, but only among older adults who had difficulty disengaging from unattainable goals. By contrast, high goal disengagement capacities were found to attenuate the adverse effect of functional disability on depressive symptoms. These findings indicate that the capacity to withdraw effort and commitment from unattainable goals can protect older adults from experiencing elevated levels of depressive symptomatology when they are confronted

with increases in functional disability.

While these studies provide strong evidence for the theoretical idea that internal self-protective strategies that foster goal disengagement may become increasingly important for maintaining older adults' psychological well-being if active coping proves to be fruitless, there are also some studies indicating that the same strategies can positively influence biological processes and health outcomes in the face of uncontrollable problems (Affleck, Tennen, Croog, & Levine, 1987; Hall et al., 2010; Wrosch, Miller, Scheier, & Brun de Pontet, 2007). For instance, Wrosch et al. (2007) have shown that disengagement from important goals that have become unattainable was associated with better self-reported physical health and more normative patterns of diurnal cortisol secretion. In addition, the results from this research indicated that the association between goal disengagement and better physical health was mediated through adaptive changes in well-being. In support of the proposed model, these findings demonstrate not only that the use of cognitive self-protection can buffer the adverse effects of unattainable goals on biological and physical health, but also that psychological distress can mediate the associations between age-related challenges (i.e., unattainable goals) and health-related outcomes (i.e, cortisol dysregulations and physical problems).

Behavioral Strategies

While the reviewed studies were predominantly concerned with self-protective cognitions and behavioral tendencies related to goal disengagement, they did not provide evidence for the effectiveness of specific behavioral strategies related to goal engagement. Therefore, the following section focuses on research that provides evidence

for the potential adaptive value of older adults' engagement in concrete health behaviors, including physical activity and restorative sleep.

As mentioned earlier, physical activity plays an important role in maintaining physical health in older adulthood (Centers for Disease Control and Prevention, 2010; Haskell et al., 2007; Pate et al., 1995; Unger, Johnson, & Marks, 1997; Warburton et al., 2006). For instance, physical activity has been shown to minimize several risk factors of mortality in old age, such as cardiovascular incidents, high blood pressure, or coronary heart disease (Kelley & Goodpaster, 2001; Taylor et al., 2004). Such effects may in part be due to biological processes, including glucose metabolism, cardiopulmonary fitness, musculoskeletal strength, optimal lipid metabolism, as well as high density lipoprotein concentrations (Dunn, Marcus, Kampert, Garcia, Kohl, & Blair, 1997; Faulkner et al., 1994).

However, there are also reasons to suggest that a physically active lifestyle may contribute to good physical health via emotional pathways. Indeed, it has been documented that physical activity can improve psychological distress (Cramer, Nieman, & Lee, 1991; Hughes, Leung, & Naus, 2008; for a review see Salmon, 2001). In addition lower (as compared to higher) levels of psychological distress have been shown to play a protective role in maintaining good health (Cohen et al., 2007; Cohen, Kessler, & Underwood, 1997, McEwen, 1998). Although direct support for the mediating role of distress in the physical activity-health link has not yet been documented, these findings suggest that physical activity may improve psychological distress and thereby provide physical health benefits. Further, physical activity may exert its adaptive effects on manageable health issues particularly among individuals who are vulnerable to

developing a disease, if it attenuates or counteracts some of the underlying risk factors or pathogenic mechanisms (e.g., high levels of stress). In preliminary support of such a process, cross-sectional research has documented that the deleterious effects of perceived stress on physical health problems can be more pronounced among individuals who engaged in low, as compared to high, levels of physical activities (Brown, 1991; Brown & Siegel, 1988; Carmack et al., 1999).

In addition to physical activity, the adaptive effects of restorative sleep patterns could represent another important behavioral mechanism through which older adults may be able to preserve emotional, biological, and physical functioning. In fact, studies have shown that the beneficial effects derived from adequate sleep can foster psychological well-being, prevent dysregulation of biological processes, and restore health and vigor (Balbo et al., 2010; Hamilton et al., 2007; McEwen, 1998; Wrosch et al., 2008).

Several studies investigating the psychological effects derived from good sleep have shown that restorative sleep is related to reduced stress-reactivity and improved mental health (Baltes, Viken, Alexander, Beyers, & Stockton, 2002; Fuligni & Hardway, 2006; Hamilton et al., 2007; Manber, Bootzin, Acebo, & Carskadon, 1996; Zohar, Tzischinsky, Epstein, & Lavie, 2005). For instance, using cut-offs suggested by epidemiological research, Hamilton et al. (2007) have found in a large sample of around 500 community residents that optimal sleepers (those reporting an average of 6 hours or less than 8.5 hours per night) reported fewer symptoms of depression, and anxiety, and higher levels of psychological well-being as compared to suboptimal sleepers (those sleeping less than 6 hours or 8.5 or more hours per night). Further, there is evidence showing that adaptive sleep patterns can ameliorate affective responses to stress and pain

among individuals with fibromyalgia or rheumatoid arthritis (Hamilton et al., 2007). These findings raise the possibility that sleep could also play an important role in adapting to psychological and biological distress. In support of this stress-buffering hypothesis, studies have documented that sleep patterns can influence biological stress hormones, such as cortisol rhythms, and influence physical health outcomes. For instance, it has been demonstrated that reduced sleep duration and sleep quality may be tightly linked to HPA hyperactivity (for a review see Balbo et al., 2010). More specifically, cross-sectional research has demonstrated that shorter, as compared to longer, sleep duration was associated with higher cortisol levels across day in a cohort of adults (Kumari et al., 2009). Finally, there are several studies suggesting that maladaptive sleep contributes to susceptibility to the common cold (e.g., Cohen, Doyle, Alper, Janicki-Deverts, & Turner, 2009), compromises physical health outcomes (Briones et al., 1996; Reid et al., 2006), and predicts mortality (Dew et al., 2003; Kripke, Garfinkel, Wingard, Klauber, & Marler 2002). For instance, Cohen et al. (2009) have shown that poorer sleep efficiency and shorter sleep duration in the weeks preceding exposure to a rhinovirus were associated with lower resistance to illness. While the discussed research suggests that sleep can influence emotional, biological, and physical functioning in the general population, restorative sleep may also benefit emotional, biological, and physical functioning among older adults who confront age-related challenges. Some initial support for such a pathway has been provided by Wrosch and colleagues (2008) who have shown that the adverse effects of elevated cortisol levels on older adults' physical health can be attenuated by restorative sleep patterns. These health benefits have been explained by the possibility that the restorative function of sleep can down-regulate high levels of cortisol

over time (Wrosch et al., 2008), although direct evidence for such a process has not yet been reported.

Together, the discussed evidence is consistent with the theoretical model that common age-related challenges can forecast emotional distress (Fig. 1, supporting an effect for path *a*-*b*), diurnal cortisol (Fig. 1, supporting an effect for path *a*-*c*), and physical health problems (Fig. 1, supporting an effect for path *a*-*d*). Further, it shows empirical support for the idea that adaptive self-regulation (i.e., health behaviors and cognitions) can exert benefits on psychological, biological, and physical health (Fig. 1, supporting an effect of adaptive self-regulation on *b*, *c*, *d*). More specifically, the reviewed evidence suggests that cognitive self-regulation (i.e., self-protective attributions) may be particularly well-suited to attenuate the adverse consequences of adverse challenges on psychological distress (Fig. 1, supporting an effect of self-protective coping on the association *a*-*b*), biological dysregulation (Fig. 1, supporting an effect of self-protective coping on the association *a*-*c*), and physical health problems (Fig. 1, supporting an effect of self-protective coping on the association *a*-*d*) in the face of problems that can hardly be controlled through a person's active efforts. By contrast, if the opportunities for engaging in active coping are favorable, older adults' may be able to adapt to age-related threats (while maximizing their potential for developmental success) by engaging in adaptive health behaviors (e.g., physical activity or adaptive sleep patterns). Finally, research on self-protective coping provides preliminary support for the assumption that psychological distress can play a mediation role in the associations between age-related challenges and health related-outcomes (i.e. cortisol dysregulation

and physical problems, Fig. 1, supporting an effect of self-protective coping on the association *a*-*b*-*d*).

Limitations of Past Research

The reviewed studies provide evidence that adaptive self-regulation can contribute to older adults' emotional, biological, and physical health. However, the extant research also incorporates important limitations and provides empirical evidence only for bits and pieces of the proposed model. This implies that more longitudinal research is needed to document the complete process linking age-related challenges and adaptive self-regulation with distress, biological processes, and physical health.

First, many of the reported studies relied on cross-sectional data, which prevents drawing any causal conclusion regarding the direction of the observed effects. Given the limited amount of longitudinal studies, it still remains uncertain whether ameliorations in older adults' emotional, biological, and physical health are, in fact, driven by self-protective cognitions and specific health behaviors.

Second, studies are lacking that examine whether adaptive behavioral and cognitive self-regulation can attenuate the psychological consequences of age-related challenges on biological dysregulation, and whether observed improvements in psychological distress stemming from the engagement in such self-regulatory strategies can mediate physical health outcomes (Fig. 1, insufficient evidence of association *a*-*b*-*d*). In addition, research has not yet clearly demonstrated whether biological processes can act as a mediator in the relationship between psychological and health-related variables. (Fig. 1, insufficient evidence of association *b*-*c*-*d*).

Third, the engagement in self-protective coping or specific health behaviors (i.e., physical activity and restorative sleep) does not always explain large portions of variance in older adults' psychological, biological, and physical health (e.g., Guszkowska, 2005; Mead et al., 2007; Wrosch et al., 2002, Wrosch, Bauer, & Scheier, 2005). This observed variability suggests that there could be other variables that may account for additional variance in older adults' health-related trajectories. In this regard, research is required that further illuminates the particular circumstances, including important risk factors for health, in which the use of self-protective coping or specific health behaviors may be more or less effective to protect an older adult's resources.

Fourth, past research has primarily focused on individual differences with regards to older adults' engagement in self-regulation, while neglecting the issue of intra-individual changes in these processes over time. However, individuals' engagement in self-regulation (e.g., adaptive sleep patterns) is likely to fluctuate over time, and inter-individual differences in intra-individual changes in the maintenance of such self-regulation strategies (e.g., increases versus declines in sleep duration) may further affect a person's health status.

Finally, while research has shown that biological, emotional, and behavioral processes can interact with each other in predicting physical health problems (e.g., Wrosch et al., 2008), this research was limited to investigating the associations between cortisol, negative affect, and sleep. However, it is likely that other psychological and biological factors contribute to changes in physical health as well. Therefore, it is important to investigate the interplay between additional psychological processes (e.g., feelings of loneliness) and biological processes (e.g., biological markers of systemic

inflammation) to deepen our understanding of how older adults can successfully cope with age-related challenges and maintain their quality of life.

Present Research

To address some of the reported limitations of previous research, this dissertation is based on a set of three interrelated longitudinal studies. The data for these studies was drawn from the longitudinal *Montreal Aging and Health Study*, a project initiated by Wrosch and colleagues in 2004, which involved a heterogeneous sample of 215 older adults from the Montreal, Quebec area. This study tracked psychological, biological and physical health variables over time.

The present research was conducted to expand an emerging and fruitful line of research on the roles of adaptive behavioral and cognitive self-regulation for healthy aging. The overarching objective of this dissertation was to shed light on the complete process linking adaptive self-regulation with age-related challenges, psychological distress, biological dysregulation, and physical illness over time. Based on ideas from the Motivational Theory of Life-Span Development (Heckhausen et al., 2010), the different studies examined the adaptive roles of specific self-regulation strategies, including behavioral engagement, such as physical activity (Study 1) and restorative sleep (Study 2), as well as cognitive disengagement, such as self-protective attributions (Study 3), in predicting older adults' emotional, biological, and physical health trajectories. It was expected that the use of behavioral and cognitive processes that are functionally adapted to a person's opportunities and constraints could have the potential to buffer normative declines in psychological, biological and/or physical health in older adulthood. In this regard, the health-related benefits of adaptive self-regulation were expected to be

particularly pronounced among "at-risk" older adults who are vulnerable to developing a disease, if they counteract some of the underlying risk factors or pathogenic mechanisms.

Study 1 (N = 157) examined whether behavioral strategies (i.e., physical activity) can exert benefits on psychological distress over two years and on physical health over four years among older adults with varying levels of a psychological challenge (i.e., high versus low baseline perceptions of stress). In addition, it was investigated whether the observed benefits of behavioral strategies (i.e., physical activity) on long-term physical health outcomes can be mediated by a short-term reduction of psychological distress (investigation of the effect of behavioral self-regulation on the association between *a-b-d*, see Fig. 1). The specific hypotheses were:

> ***Hypothesis 1.1.*** *Baseline levels of physical activity will prevent 4-year declines in physical health problems particularly among participants who perceive high baseline levels of stress, but to a lesser extent among participants who perceive low baseline levels of stress (i.e., interaction effect between baseline perceived stress and physical activity on 4-year changes in physical health).*
>
> ***Hypothesis 1.2.*** *Baseline levels of physical activity will contribute to 2-year declines in perceived stress particularly among participants who perceive high baseline levels of stress, but to a lesser extent among participants who perceive low baseline levels of stress (i.e., interaction effect between baseline perceived stress and physical activity on 2-year changes in perceived stress).*
>
> ***Hypothesis 1.3.*** *The hypothesized interaction effect between baseline levels of perceived stress and physical activity in predicting 4-year changes in physical*

health problems will be statistically mediated by 2-year changes in perceived stress.

The objective of Study 2 ($N = 157$) was to examine whether behavioral strategies (i.e., restorative sleep patterns) can ameliorate age-related deteriorations in cortisol secretion among older adults over time (investigation of the effect of behavioral self-regulation on *a-c*, see Fig. 1). The specific hypothesis was:

Hypothesis 2.1. *Across waves, longer (as compared to shorter) sleep will protect older adults from experiencing 2-year increases in diurnal cortisol secretion over time.*

Study 3 ($N = 122$) elucidates the effects of cognitive strategies (i.e., self-protective attributions) on psychological distress and biological dysregulations among older adults who experienced varying levels of a psychological challenge (i.e., high versus low loneliness). In addition, this study examined the mediating role of psychological and biological processes (i.e., psychological distress and cortisol secretion) in the association between a psychological challenge (i.e., loneliness) and a biological marker of systemic inflammation (i.e., C-reactive protein), which is likely to influence the development of disease (i.e., investigation of the effect of cognitive self-regulation on the association between *a-b-c-[d]*, see Fig. 1). The specific hypotheses were:

Hypothesis 3.1. *Baseline levels of self-protective control strategies for managing health threats (e.g., seeing the silver lining or avoiding self-blame for a problem) will prevent 2-year increases in psychological stress and diurnal cortisol secretion, as well as higher 6-year levels of the inflammatory biomarker C-reactive protein (CRP), but only among lonely participants, and not among non-*

lonely participants. (i.e., interaction effects between loneliness and self-protective control strategies in predicting 2-year changes in perceived stress and cortisol secretion and 6-year levels of CRP)

Hypothesis 3.2. *The hypothesized interaction effect between baseline levels of loneliness and self-protective control strategies in predicting 6-year levels of CRP will be statistically mediated by 2-year changes in perceived stress and cortisol secretion.*

The Different Roles of Perceived Stress in the Association Between Older Adults' Physical Activity and Physical Health

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Abstract

This 4-yr longitudinal study examined the different roles of perceived stress in the association between older adults' physical activities and physical health. We hypothesized that physical activities would exert beneficial effects on physical health by preventing chronically high levels of perceived stress. *Design and Measures:* We assessed baseline levels of physical activities and repeated measures of perceived stress and physical symptoms in three waves of data from a sample of 157 older adults. *Results:* Among participants with high (but not low) baseline levels of perceived stress, physical activity predicted a 2-yr reduction of perceived stress and a 4-yr prevention of physical health symptoms. Moreover, the interaction effect on 4-yr changes in physical symptoms was mediated by 2-yr changes in perceived stress. *Conclusions*: Physical health benefits of physical activity are particularly pronounced among older adults who perceive high levels of stress, and this effect is mediated by a prevention of chronically high perceptions of stress.

Keywords: physical health, perceived stress, physical activities, older adulthood.

Introduction

The Different Roles of Perceived Stress in the Association Between Older Adults' Physical Activity and Physical Health

Research suggests that perceptions of stress play an important role in the link between physical activity and physical health. First, physical activity has been shown to reduce perceptions of stress (Salmon, 2001). Second, physical activity and low stress are generally associated with better physical health (Cohen et al., 2007; Haskell et al., 2007). Third, the health benefits of activity are particularly evident among individuals who experience high, as compared to low, levels of stress (Brown & Siegel, 1988; Carmack et al., 1999). While these findings suggest that physical activity could benefit health by ameliorating chronically high levels of stress, this mechanism has not yet been demonstrated in longitudinal research. Here, we test this possibility in three waves of data from a 4-year longitudinal study of community-dwelling older adults. We expected that engaging in physical activity would ameliorate stress and thereby mediate good physical health in persons who perceive high levels of stress. By contrast, we did not expect these effects to be apparent among persons low in stress, as these individual are generally at a lower risk of developing health problems, and health benefits of physical activity may be less likely to operate through a reduction of low stress levels.

Physical Activities, Perceived Stress, and Physical Health

The maintenance of a physically active lifestyle is an important contributor to older adults' physical health (Centers for Disease Control and Prevention, 2010; Haskell et al., 2007; Pate et al., 1995). While some of the benefits of physical activity are attributable to modulation of biological processes involved in disease (e.g., increased

cardiorespiratory fitness, decreased blood pressure, more musculoskeletal strength, Dunn et al., 1997; Faulkner et al., 1994), there is also evidence that psychological mechanisms, such as the perception of stress, may explain beneficial health effects of physical activity. Perceived stress represents appraisals of person-environment interactions that can influence emotional and biological processes (Lazarus & Folkman, 1984). In addition, perceived stress is associated with a number of adverse physical health outcomes, likely in a causal fashion (Cohen, 1996; Cohen, Kamarck, & Mermelstein, 1983; Cohen, Tyrrell, & Smith, 1991).

In support of this process, research suggests that high levels of perceived stress contribute to negative affect (e.g., feelings of anxiety and depression) and biological and behavioral processes (e.g., dysregulated cortisol rhythms or non-adherence to medical regimes) that may heighten vulnerability to various physical health problems (Cohen et al., 1997, 2007; McEwen, 1998; Miller, Chen, & Cole, 2009; Miller, Chen, & Zhou, 2007). Moreover, physical activity is generally associated with decreased levels of perceived stress (King, Taylor, & Haskell, 1993; Norris, Carroll, & Cochrane, 1990; Salmon, 2001) and alters some of the behavioral and biological processes through which perceived stress can affect physical health outcomes (e.g., less smoking, better nutrition practice, less cortisol output or cardiovascular reactivity; Boutelle, Murray, Jeffery, Hennrikus, & Lando, 2000; Crews & Landers, 1987; Fleshner, Kennedy, Johnson, Day, & Greenwood, 2009; Rimmele, Seiler, Marti, Wirtz, Ehlert, & Heinrichs, 2009; Wankel & Sefton, 1994).

These findings suggest that physical activity has physical health benefits, which may operate partially through amelioration of perceived stress. However, several

longitudinal studies have shown that physical activity does not always explain large portions of variance in physical health outcomes (Guszkowska, 2005; Mead et al., 2007). Such a lack of stronger associations could be due to the possibility that physical activity is not equally adaptive among different groups of individuals. For example, physical activity may exert its adaptive effects on physical health particularly among individuals who are vulnerable to developing a disease, if it attenuates or counteracts some of the underlying risk factors or pathogenic mechanisms. By contrast, the health effects of physical activities may be less pronounced among individuals who are at a lower risk of developing physical disease. A corollary of this argument is that perceived stress itself could not only mediate, but also moderate, the link between physical activity and physical health (Brown, 1991; Brown & Siegel, 1988; Carmack et al., 1999). In this scenario, physical activity may be particularly beneficial for health among individuals who perceive high levels of stress, because it can ameliorate their maladaptive stress levels and some of its emotional, behavioral, and biological consequences.

We note that this process would be consistent with research indicating that people can be stressed over different periods of time, and that the chronic rather than the temporary perception of stress is detrimental to a person's physical health. Such consequences of chronic stress may be due to its harmful long-term effects on emotional, physiological, and behavioral responses that influence susceptibility to, and course of, disease (Kiecolt-Glaser et al., 2003; Miller et al., 2002, 2008, 2009; Schulz, Kirschbaum, Pruessner, & Hellhammer, 1998). Transient perceptions of stress, by contrast, should be less problematic for a person's physical health as they may not give rise to sustained patterns of biological dysregulation or maladaptive health behaviors.

Further support for the proposed process stems from research documenting that physical activity is associated with better physical health particularly among individuals who experienced many stressful life events, and that this association can be explained by psychological distress (Brown, 1991; Brown & Siegel, 1988; Carmack et al., 1999). In addition, longitudinal research showed that the deleterious effects of widowhood on functional health declines were more pronounced among community-dwelling older adults who engaged in low, as compared to high, levels of physical activities (Unger et al., 1997). Finally, experimental intervention research suggests that engaging obese women or cancer survivors in exercise programs can decrease their experience of stress (Cramer et al., 1991; Hughes et al., 2008; for a review, see Salmon, 2001).

These studies demonstrate that health benefits of physical activity can occur particularly among individuals who perceive high levels of stress, and that physical activity can reduce stress levels in vulnerable populations that confront problematic life circumstances. Nonetheless, the present literature did not document the complete process that links physical activity, perceived stress, and physical health outcomes over time. In particular, it has not been shown that the beneficial effects of physical activity on the prevention of chronically high levels of stress can mediate subsequent levels of physical health. From our theoretical perspective, however, such associations are likely to occur, and demonstrating this process in longitudinal research may contribute to our understanding of the adaptive health effects of physical activity.

The Present Research

This research examined whether physical activity would have differential health benefits in higher vs. lower stress individuals. We expected the health benefits to be most

apparent in the former group, in whom physical activity would reduce high stress levels over time. By contrast, we did not expect these benefits of physical activity to be apparent in the low-stress individuals. To start examining this possibility, we analyzed three waves of data from a 4-year longitudinal study of older adults. Our analysis focused on predicting physical health symptoms (e.g., chest pain, joint pain, or difficulty breathing) because such symptoms can be signs of a variety of underlying or developing physical diseases and have been associated with distress and biological markers of stress in previous research (Wrosch, et al., 2002, 2007, 2008). More specifically, we examined whether the interaction between baseline levels of physical activity and perceived stress would predict changes in physical symptoms over time. In addition, we expected that changes in perceived stress would mediate this interaction effect.

Method

Participants

This study is based on a sample of community-dwelling older adults who took part in the longitudinal *Montreal Aging and Health Study* (MAHS; Wrosch et al., 2007a). Participants were recruited through newspaper advertisement. The only inclusion criterion was that participants had to be older than 60 years because we were interested in examining a normative sample of older adults. In 2004, we conducted the first wave of the MAHS by assessing a heterogeneous sample of 215 older adults from the Montreal area. The 2-yr follow-up included 184 participants and 164 subjects participated in the 4-yr follow-up. Reasons for non-participation were being deceased (n = 13), having problems that prevented participation (n =17), refusing further participation (n = 8), and being unable to locate participants (n = 13). Seven additional participants were excluded

from the analyses because they did not participate in the 2-year follow-up. The final sample included 157 participants who were on average 72 years old (SD = 5.55) and 48% of the sample was male. Study attrition over four years was not significantly associated with baseline measures of the study variables, except for age. Older participants were more likely to discontinue their study participation, $t(213) = -2.30$, $p < .05$.

Materials

The main study variables included repeated measures of physical health symptoms and perceived stress, as well as baseline measures of physical activities. In addition, the study included a number of covariates associated with health-relevant sociodemographic characteristics (i.e., age, sex, education) and baseline levels of chronic health problems (see Tables 1 and 2 for zero-order correlations, means, standard deviations, and frequencies of main study variables).

Physical health symptoms were measured at baseline, 2-yr, and 4-yr follow-up by administering a symptom checklist that has been validated in previous research with older adults (Wrosch et al., 2002). Participants reported at bedtime on three typical days whether they had experienced each of 12 physical health symptoms during the day (e.g., chest pain, joint pain, or shortness of breath; PRIME MD: Spitzer et al., 1994). Daily measures of physical symptoms were significantly correlated with each other (rs_{T1} = .65 to .75, $ps_{T1} < .01$; rs_{T2} = .61 to .77, $ps_{T2} < .01$; rs_{T3} = .44 to .67, $ps_{T3} < .01$), and we obtained indicators of physical health symptoms for baseline, 2-yr, and 4-yr follow up by counting the total number of symptoms experienced across all three days (see Table 2). Levels of physical symptoms showed a linear increase across waves, $F(1, 156) = 7.71$, $p < .01$.

Table 1

Zero-Order Correlations Between Main Constructs

	1	2	3	4	5	6	7	8	9	10
1. Physical health symptoms (baseline)										
2. Physical health symptoms (2-yr follow up)	.58**									
3. Physical health symptoms (4-yr follow up)	.48**	.57**								
4. Perceived stress (baseline)	.34**	.28**	.26**							
5. Perceived stress (2-yr follow up)	.25**	.29**	.31**	.63**						
6. Perceived stress (4-yr follow up)	.33**	.31**	.30**	.68**	.76**					
7. Physical activities (baseline)	-.08	-.09	-.17*	-.11	-.07	-.17*				
8. Age	-.08	-.07	-.10	-.11	.03	-.01	-.12			
9. Sex [a]	.09	.03	.10	.11	.12	.16*	-.09	.06		
10. Education	-.20*	-.16*	-.07	-.21**	-.14	-.21**	.09	-.20*	-.14	
11. Chronic health problems	.24**	.31**	.21**	.08	.08	.12	-.08	-.06	-.03	-.05

Note. [a] Higher values represent female participants. $^{*}p < 0.05$. $^{**}p < 0.01$.

Table 2

Means, Standard Deviations, and Frequencies of Main Study Variables (N = 157)

Constructs	Mean (SD) or Percentage [a]
Physical health symptoms	
Baseline	2.59 (3.20)
2 Years	2.87 (3.50)
4 Years	3.42 (4.04)
Perceived stress	
Baseline	2.42 (0.65)
2 Years	2.42 (0.65)
4 Years	2.47 (0.73)
Hours of weekly physical activity (baseline)	2.10 (3.25)
Number of chronic health problems (baseline)	.80 (.81)
Arthritis (%)	29.90
Diabetes (%)	14.10
Cancer (%)	3.20
Lung or other respiratory disease (%)	10.80
Heart condition (%)	17.20
Difficulty bathing (%)	2.50
Difficulty managing finances (%)	2.50
Age	71.72 (5.55)
Male (%)	48.40%
Education (baseline)	2.09 (1.07)
None (%)	4.00
High school (%)	31.30
Trade (%)	28.70
Bachelor (%)	24.00
Masters or Doctorate (%)	12.00

Note. [a] Mean (SD) are presented for continuous variables

Perceived stress was measured at baseline, 2-yr, and 4-yr follow-up by administering the 10-item version of the perceived stress scale (Cohen et al., 1983). Respondents were asked to rate how frequently they experienced 10 different situations over the past month (e.g., "How often have you been upset because of something that happened unexpectedly?'' or "How often have you felt difficulties were piling up so high that you could not overcome them?"), by using a five-point Likert-type scale ranging from 1 (never) to 5 (very often). Indicators of perceived stress were obtained by averaging the ratings of the 10 items separately for baseline, 2-yr, and 4-yr follow up, αs > .87. Levels of perceived stress did not significantly change across waves, $F(1, 156) = 1.05$, $p > .05$.

Participants' engagement in *physical activity* was assessed at baseline by administering an open-response format questionnaire used in previous research (Miller, Cohen, & Herbert, 1999; Paffenbarger, Blair, Lee, & Hyde, 1993). Participants were asked if they engage in any regular activity (e.g., walking, jogging, bicycling, etc), long enough to work up a sweat. In addition, they reported how many days per week and for how long each time they engage in physical activities. This measure has been validated by previous research, showing that it predicts objective markers of fitness, such as oxygen uptake during pedal ergometry (Siconolfi, Lasater, Snow, & Carleton, 1985). In order to obtain a measure of participants' weekly physical activity, we multiplied the number of days participants were physically active by the hours participants usually engaged each time in physical activities. On average, participants engaged approximately two hours per week in physical activities (see Table 2). Note that based on the physical activity guidelines from the Centers for Disease Control and Prevention (2010), 69.4% of

our sample can be classified as insufficiently active (less than 150 min per week of total activity) and 30.6% as active (at least 150 minutes per week of total activity).

Covariates. To reduce the possibility of spurious associations, this study incorporated a number of covariates that were associated in previous research with activity engagement, perceived stress, or physical health problems. These variables included participants' *age, sex, education, and chronic health problems* (Denton, Prus, & Walters, 2004; Lee, Lindquist, Segal, & Covinsky, 2006). Education was measured by asking participants to report their highest educational degree completed (0 = none, 1 = high school, 2 = trade, 3 = undergraduate degree, 4 = graduate degree). A measure of chronic health problems was derived based on research identifying conditions associated with premature mortality in older adults (Lee et al., 2006). It included six conditions - diabetes, cancer, lung or other respiratory disease, heart condition, difficulty bathing, and difficulty managing finances (Lee et al., 2006). In addition, we incorporated the presence of arthritis into our measure, given that this disease is associated with both perceived stress and daily symptoms (Wrosch & Schulz, 2008; Thomason, Brantley, Jones, Dyer, & Morris, 1992).

Results

Data Analyses

The hypotheses were tested by conducting two sets of analyses. In the first set, we examined whether baseline levels of perceived stress would moderate the effect of baseline levels of physical activities on changes in older adults' physical health symptoms over time. To this end, we conducted two separate regression analyses predicting 2-yr and 4-yr levels in physical health symptoms by baseline levels of physical symptoms, physical activities, and perceived stress (step 1), and the interaction between

physical activities and perceived stress (step 2). All analyses controlled for a number of covariates (i.e., age, sex, education, and chronic health problems), and predictor variables were standardized prior to conducting the analyses.

In the second set, we investigated whether the interaction between baseline levels of physical activities and perceived stress in predicting changes in physical health symptoms would be mediated by a reduction of perceived stress. To test this hypothesis, we conducted bootstrap analyses (Preacher & Hayes, 2008), which examined whether 2-yr changes in perceived stress exert an indirect effect on the interaction effect between physical activity and perceived stress in predicting changes in physical health symptoms. The analysis was based on 5000 bootstraps and the indirect effect was evaluated as significant if the bias-corrected 95% confidence interval did not cross zero (see Preacher & Hayes, 2008).

Perceived Stress as a Moderator

The results of the first set of analyses showed that baseline levels of physical symptoms were significantly associated with 2-yr levels, $F(1, 149) = 48.32$, $B = .50$, $SE = .07$, $R^2 = .20$, $p < .01$, and 4-yr levels of physical symptoms, $F(1, 149) = 29.16$, $B = .42$, $SE = .08$, $R^2 = .14$, $p < .01$. Moreover, the first step of the analyses indicated that of the covariates, only chronic health symptoms significantly predicted 2-yr increases in physical symptoms, $F(1, 149) = 7.19$, $B = .18$, $SE = .07$, $R^2 = .03$, $p < .01$. The other covariates and the main effects of physical activity and perceived stress were unrelated to 2-yr or 4-yr changes in physical symptoms, Fs$(1, 149) < 3.00$, $ps > .05$. However, the second step of the analyses confirmed a significant interaction between physical activities and perceived stress in predicting 4-yr changes in physical symptoms, $F(1, 148) = 4.20$, B

= -.16, *SE* = .08, R^2 = .02, $p < .05$. We note that the interaction between physical activity and perceived stress remained significant if we controlled in additional analyses for 2-yr or 4-yr changes in physical activity, and that this interaction was not found in the analysis of 2-yr changes in physical symptoms, $F(1, 148) = .94$, $p > .05$. Further, neither the quadratic main effect for physical activity nor the quadratic interaction effect between physical activity and perceived stress predicted 4-yr changes in physical symptoms.

Figure 2 illustrates the significant interaction effect. We plotted the association between hours of weekly physical activity (0 to 5 hours, where 5.35 hours represented one standard deviation above the sample mean) and 4-yr changes in physical health symptoms for participants who perceived high (+1 SD) and low (-1 SD) baseline levels of stress (Aiken & West, 1991). The pattern of results indicated that, above and beyond baseline levels of physical health symptoms, the highest symptom levels after 4 years were found among participants who reported high baseline levels of perceived stress and did not engage in physical activities. A calculation of the simple slopes confirmed this interpretation by demonstrating that physical activity was significantly associated with fewer increases in physical symptoms over time among participants who perceived high baseline levels of stress, *B* = -.34, *SE* = .16, $p < .01$, but not among their counterparts who perceived low levels of stress, *B* = -.04, *SE* = .10, $p > .05$.

Perceived Stress as a Mediator

To examine whether 2-yr changes in perceived stress would mediate the observed interaction effect on 4-yr changes in physical symptoms, we conducted bootstrap analyses. To this end, we used the "indirect SPSS macro" (Preacher & Hayes, 2008), and repeated the above-reported analysis for predicting 4-yr changes in physical health

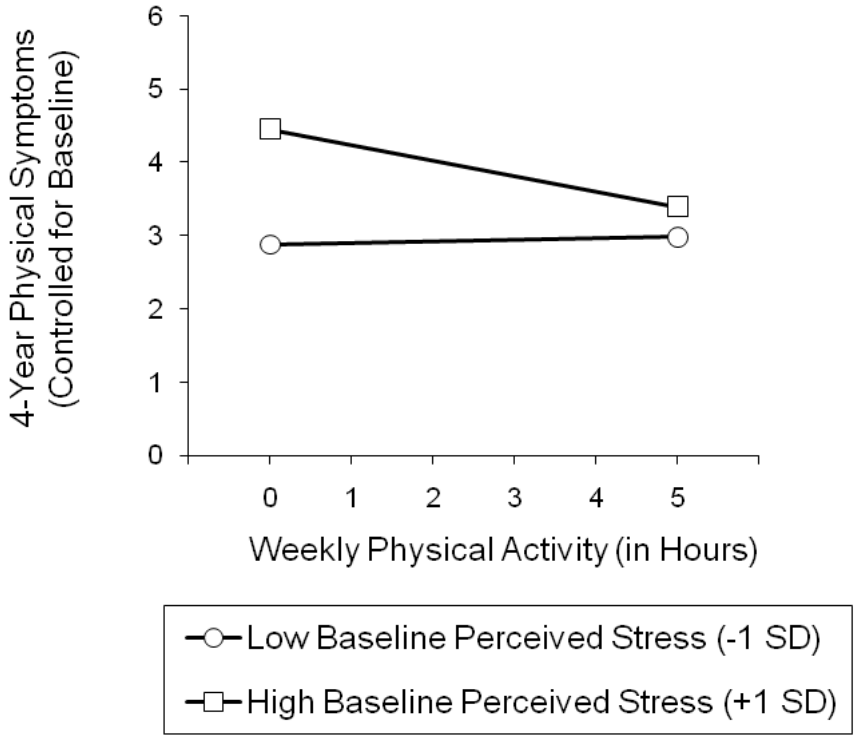

Figure 2 Associations between baseline levels of physical activity and 4-yr changes in physical health symptoms, separately for participants who experienced high and low baseline levels of perceived stress.

symptoms, and additionally incorporated 2-yr levels of perceived stress as a potential mediator. Note that this analysis controlled for baseline perceived stress, which implies that the mediating variable was independent of its baseline levels and represents 2-yr changes in perceived stress.

Figure 3 illustrates the mediation model tested. The results of the analysis demonstrated that the interaction between baseline levels of physical activity and perceived stress significantly predicted changes in perceived stress over 2 years, $F(1, 148) = 10.10$, $B = -.22$, $SE = .07$, $R^2 = .04$, $p < .01$ (see path A in Figure 2). The obtained pattern for this interaction effect closely resembled the results found for predicting 4-yr changes in physical symptoms. Physical activity was significantly associated with a reduction of perceived stress over 2 years. This association was apparent among participants with high, but not low, baseline perceived stress (+1 SD: $B = -.29$, $SE = .02$, $p < .01$ vs. -1 SD: $B = .13$, $SE = .02$, $p > .05$).

The findings further showed that increased levels of perceived stress over 2 years were independently associated with increases in physical symptoms over 4 years, $F(1, 147) = 4.67$, $B = .20$, $SE = .09$, $R^2 = .02$, $p < .05$ (see path B in Figure 3). Moreover, the analyses demonstrated that the interaction effect between baseline levels of physical activities and perceived stress in predicting 4-yr changes in physical symptoms was rendered non-significant, $F(1, 147) = 2.13$, $B = -.12$, $SE = .08$, $R^2 = .01$, $p = .15$, if 2-yr changes in perceived stress were included as a potential mediator into the model (see path C in Figure 3). In fact, the bootstrap analyses confirmed that 2-yr changes in perceived stress exerted a significant indirect effect on the interaction between physical activities and perceived stress in predicting 4-yr changes in participants' health symptoms (95%

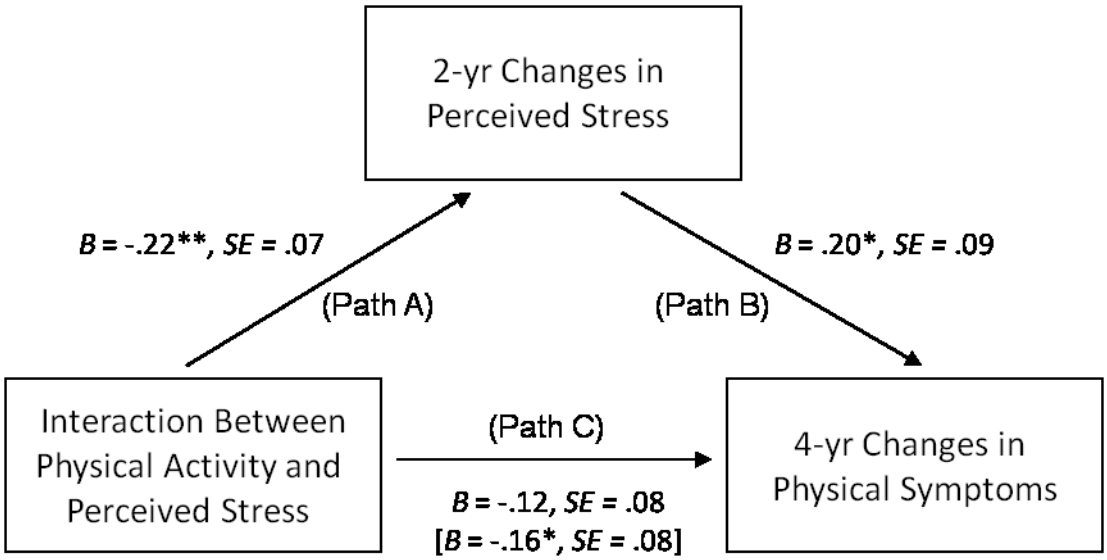

Figure 3 Mediation model examining the effect of 2-yr changes in perceived stress on the association between baseline levels of physical activity and perceived stress on 4 yr-changes in physical health symptoms.

BCI [-.196, -.002]). This pattern of results suggests that the reduction of perceived stress mediated the beneficial effect of physical activity on fewer physical symptoms among participants who perceived high baseline levels of stress.

Discussion

This longitudinal study examined the roles of perceived stress in the association between physical activities and older adults' physical health. We hypothesized that engaging in physical activities would have health benefits among individuals who perceived high (but not low) levels of stress. The study's results supported our hypotheses by demonstrating significant interaction effects between baseline levels of physical activity and perceived stress on 2-yr changes in perceived stress and 4-yr changes in physical symptoms. These results showed that among participants with high baseline levels of perceived stress, those who frequently engaged in physical activities experienced a reduction of perceived stress over 2 years, and fewer increases in physical health symptoms over 4 years. No effects of physical activity on changes in perceived stress or physical symptoms were obtained among participants who perceived only low stress levels at baseline.

In addition, 2-yr reductions in perceived stress exerted a significant indirect effect on the interaction between baseline levels of physical activities and perceived stress in predicting 4-yr changes in physical health problems. These findings demonstrate that physical activity has the potential to ameliorate chronically high perceptions of stress and thereby produce long-term benefits on physical health.

The study's findings are important for different reasons. First, they substantiate previous research suggesting that associations between physical activity and physical

health can be more pronounced among individuals with high, as compared to low, levels of stress (Brown, 1991; Brown & Siegel, 1988; Carmack et al., 1999; Unger et al., 1997). The present study extends this line of work by demonstrating that such effects also appear among community-dwelling older adults. This implies that physical activity may be especially beneficial for physical health among vulnerable older adults who perceive stress. By contrast, older adults who are better adjusted psychologically may have more favorable health trajectories, and as such the benefits of physical activity in them are less pronounced. However, it is important to note that the majority of our sample consisted of "young-old" individuals and many of the more severe health stressors occur in later phases of older adulthood (Smith & Baltes, 1997). Accordingly, it is possible that physical activity could become more adaptive among participants with low baseline levels of stress, when new age-related stressors emerge in their future. A corollary of this argument is that the observed interaction effect between physical activity and perceived stress may become smaller in later stages of the life course when individuals increase their likelihood of encountering more physical stressors based on age-normative biological declines (Baltes, 1997). In such circumstances, we would expect to observe strong health effects of physical activity - independent of participants' baseline stress levels - given that physical activity can ameliorate the psychological impact of emerging stressors and provide direct biological benefits (e.g., musculoskeletal strength, Faulkner et al., 1994) to counteract age-normative health declines (for research showing general effects of physical activity on physical health, see also Simonsick et al., 1993).

Second, the findings document an important mechanism that can explain health-related variability in old age. This mechanism is associated with the prevention of

chronically high levels of perceived stress. In particular, it shows that physical activity can not only reduce elevated perceptions of stress over time, but such reductions in stress levels can further protect older adults' physical health. This pattern of findings provides an explanation for the above-discussed stronger health effects of physical activity among individuals who perceive high (as compared to low) levels of stress. Physical activity may prevent stressed individuals from entering into a downward spiral, characterized by chronically high perceptions of stress and subsequent increases in physical health problems.

Finally, we think that our findings have implications for models of psychosocial determinants of illness, as they point to complex and dynamic interactions between health-promoting behaviors (e.g., physical activity) and psychological risk factors (e.g., perceived stress). Our study suggests that the presence of a psychological risk factor can generally enhance a person's likelihood of developing physical symptoms and thus increases the importance and adaptive value of engaging in health-promoting behaviors. In turn, the engagement in health-promoting behaviors can contribute to long-term physical health outcomes if the behavior has the potential to attenuate the psychological risk factor. We therefore suggest that theory and research should extend the present analysis in long-term longitudinal studies and examine additional psychological risk factors (e.g., depression or anxiety) as well as other health-promoting behaviors (e.g., adaptive sleeping or eating patterns) to identify the health behaviors that are particularly well suited to prevent chronic levels of different psychological problems (for effects of physical activity and stressors on different psychological problems, see Carmack et al., 1999). Research along these lines may not only contribute to improved psychological

theories of physical health, but may also provide important information that can be used to promote the physical health of individuals who experience psychologically problematic situations.

There are limitations to this research that need to be addressed in future studies. First, the predictor and outcome variables were assessed with self-report measures. In this regard, we note that our study did not assess participants' memory capacity, which could influence the validity of self-report measures. However, given that the reliability of our perceived stress measure remained high across time (alphas > .87), and that our sample of older adults was on average relatively healthy at baseline, we think that it is unlikely that our findings are due to memory impairments. In addition, self-reports could be influenced by other dispositional constructs, such as negative affectivity or neuroticism (Portella, Harmer, Flint, Cowen, & Goodwin, 2005; Watson & Pennebaker, 1989). While such general biases are less likely to occur in longitudinal analyses because change scores should be less affected by disposition-based individual biases, we note that our study included measures of neuroticism and negative affectivity (Costa & McCrae, 1992; Watson, Clark, & Tellegon, 1988), and follow-up analyses revealed that these variables did not affect the reported interaction effects. Nonetheless, we suggest that future studies should use objective measures of memory capacity, physical health, and activities (e.g., Mora, DiBonaventura, Idler, Leventhal, & Leventhal, 2008; Nasreddine et al., 2005; Parker, Strath, & Swartz, 2008) to substantiate the conclusions drawn from our study.

Second, our findings suggest that perceived stress and physical activity can be relatively independent from each other, which allowed us to demonstrate significant interaction effects. However, future research is needed to identify the variables that

determine individual differences in the association between perceived stress and activity engagement. In this regard, it would be interesting to identify psychological factors that help maintain high levels of physical activity among older adults who encounter stressful life circumstances. Such factors could be related to social support or perceptions of control and should be included in future studies.

Third, our analyses did not identify the mechanisms through which physical activities attenuated perceived stress, and through which the latter predicted subsequent health problems. In this regard, the stress-reducing effects derived from physical activity may be associated with changes in attentional focus or improved coping patterns. Physical activity typically turns people's attention away from stressful circumstances (Bahrke & Morgan, 1978) and therefore could provide a temporary respite from life stress, or physical activity may serve a beneficial restorative function that allows people to deal with stressful circumstances more effectively. In addition, exercise could counteract some of the endocrine and immune dysregulation that is often associated with high stress levels (see Cohen et al., 2007).

Finally, while our measure of physical symptoms may represent a reliable non-specific proxy for developing disease and mortality among the elderly (Sha, Callahan, Counsell, Westmoreland, Stump, & Kroenke, 2005), the analyses did not examine the development of more severe health problems (e.g., cancer, functional limitations, or mortality) or the health benefits deriving directly from physical activity (e.g., cardiorespiratory fitness or musculoskeletal strength, Dunn et al., 1997; Faulkner et al., 1994). We argue that our approach has some important advantages because it has the potential to detect physical health changes across different diseases. However, we note

that supplemental analyses showed that our predictor variables were unrelated to changes in chronic health problems, functional limitations, or mortality. Nonetheless, future research should conduct long-term follow-ups of individuals' chronic health problems, given that our sample was just approaching a life phase during which the likelihood of experiencing chronic health problems rapidly increases (Smith & Baltes, 1997). In addition, such an approach should examine a wider range of psychosocial, cognitive, and health variables (e.g., cardiorespiratory fitness) to provide a more comprehensive picture of the pathways to healthy aging.

Sleep Duration Buffers Diurnal Cortisol Increases in Older Adulthood

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Abstract

This study examined the long-term associations between reports of sleep duration and diurnal cortisol secretion in older adulthood. It was hypothesized that longer sleep would protect older adults against increases in diurnal cortisol secretion over time. We tested this hypothesis using three waves of data from a 4-year longitudinal study involving 157 older adults. Results from growth curve and cross-lagged panel analyses demonstrated that levels and increases in sleep duration buffered long-term elevations of diurnal cortisol secretion. Reversed analyses indicated that diurnal cortisol secretion did not predict changes in sleep duration over time. These results were independent from sociodemographic characteristics (i.e., age, sex, partnership status, and education) and health-related variables (i.e., chronic illness, medication usage, body mass index, and smoking). They suggest that long sleep exerts restorative functions and protects older adults from exhibiting increases in diurnal cortisol secretion over time.

Keywords: cortisol secretion, sleep duration, older adulthood

Introduction

Sleep Duration Buffers Diurnal Cortisol Increases in Older Adulthood

Cortisol is a hormone that is secreted by the hypothalamic-pituitary-adrenocortical (HPA) axis. Across the day, cortisol secretion follows a circadian rhythm, reaching the highest levels shortly after awakening and progressively declining until bedtime (Van Cauter & Turek, 1994). Cortisol has widespread regulatory influences in the body which include the mobilization of glucose, the regulation of fluid volume, the modulation of immune function, and various central nervous system effects on behaviors related to feeding, sleeping, and learning (Sapolsky et al., 2000). However, physical or psychological stressors can disrupt the HPA axis homeostasis (Heim et al., 2000; Miller et al., 2007; Schaeffer & Baum, 1984; Weiner, 1992). When patterns of cortisol release become dysregulated, either through increased or decreased output, there are likely to be adverse physiological implications, which may alter vulnerability to various mental and physical illnesses (Bjorntorp & Rosmond, 1999; Lupien et al., 2009; Raison & Miller, 2003).

Age-related elevations in cortisol have been found both in natural settings and in response to laboratory-induced challenge (Almeida et al., 2009; McEwen & Stellar, 1993, Kern et al., 1996; Otte et al., 2005; Van Cauter et al., 1996, 2000; Wrosch et al., 2008 McEwen & Stellar, 1993). These processes may occur because common age-related challenges (e.g., health threats or loss of resources, Baltes et al., 1979; Heckhausen et al., 2010) can trigger emotional distress and increase the release of cortisol into the circulation (Wrosch et al., 2004). In addition, age effects on cortisol secretion may reflect an impairment of the HPA axis feedback control and could underlie a constellation of dysregulation in other bodily systems (Boscaro et al., 1998; McEwen & Sapolsky, 1995;

Seeman & Robbins, 1994; Wilkinson, Peskind, & Raskind, 1997). However, despite the importance of understanding the age-related trajectories in HPA axis functioning, a consistent pattern of associations between cortisol level and age has not been established. Several studies observed considerable variability in the age-related changes of cortisol secretion (Lupien et al., 1998, 2005; Seeman et al., 1997), and other research did not find an association between diurnal cortisol output and age (e.g., Greenspan, Rowe, Maitland, McAloon-Dyke, & Elahi, 1993).

To provide an explanation for this inconsistent pattern of findings, we suggest that diurnal cortisol may not increase among all older adults, and that some of them are protected from exhibiting cortisol increases. In addition, such individual differences could be reliably associated with behavioral factors that are likely to modulate cortisol release. To this end, we suggest that sleep duration could be one such factor that has the potential to influence trajectories of older adults' diurnal cortisol secretion (Balbo et al., 2010). Preliminary support for this argument stems from cross-sectional research demonstrating that shorter, as compared to longer, sleep duration was associated with higher cortisol levels across day in a cohort of adults (Kumari et al., 2009). Moreover, chronic short sleepers have been found to exhibit higher nocturnal cortisol levels than chronic long sleepers (Späth-Schwalbe, Scholler, Kern, Fehm, & Born, 1992). Further, extant laboratory studies have indicated that experimental sleep restriction can predict increases in cortisol levels both during the night of acute sleep loss (Weibel, Follenius, Spiegel, Ehrhart, & Brandenberger, 1995; Weitzman, Zimmerman, Czeisler, & Ronda, 1983) and, if wakefulness was prolonged, during the following day (Leproult, Copinschi, Buxton, & Van Cauter, 1997; Spiegel, Leproult, Van Cauter, 1999; Van Cauter et al.,

2000). Finally, there is evidence suggesting that adaptive sleep patterns can attenuate the adverse effects of elevated cortisol levels on older adults' physical health. These health benefits have been explained by the possibility that the restorative function of sleep can down-regulate high levels of cortisol over time (Wrosch et al., 2008).

We note that the link between cortisol and sleep is likely bi-directional. Indeed, there is evidence that cortisol can be associated with changes in sleep patterns, although such effects have not been found consistently across studies (for a review, see Steiger, 2003). These mixed findings suggest that cortisol may play an indirect role in causing sleeping problems, in which it needs to interact with other biological processes to have effects (for a review, see Buckley and Schatzberg, 2005; Shaver, Martire, Beach, & Scheier, 2002).

Together, the reported research suggests that long sleep could buffer age-related increases in diurnal cortisol secretion, whereas shorter sleep may lead to increases in older adults' cortisol output. Further, such restorative functions of long sleep may be especially important in older adulthood when age-related challenges contribute to psychological and biological disturbances (Wrosch et al., 2007a, 2008), and older adults could benefit from adaptive behavioral patterns that support homeostasis. Finally, because cortisol could also influence changes in sleep patterns, conclusions about the direction of effects cannot be drawn from cross-sectional evidence, and the existing short-term longitudinal studies leave unexamined whether sleep duration contributes to sustained increases of cortisol secretion. Thus, longitudinal research is needed that tracks changes in individuals' sleep duration and cortisol output over a prolonged period of time to examine the long-term associations between older adults' sleep duration and diurnal

cortisol secretion.

To address this gap in the literature, the present research examined the long-term associations between reports of sleep duration and diurnal cortisol secretion in older adulthood. We expected that cortisol levels would generally increase over time, and that this effect could be moderated by older adults' sleep duration. More specifically, we hypothesized that shorter sleep (and declines in sleep duration) would amplify age-related increases in subsequent cortisol secretion, whereas longer sleep (and increases in sleep duration) would protect against them. In addition, we considered the possibility that both levels of and changes in cortisol could also contribute to changes in sleep duration over time.

Method

Participants

The study is based on a 4-year longitudinal sample of community-dwelling older adults who took part in the *Montreal Aging and Health Study* (MAHS; Wrosch et al., 2007a). Participants were recruited through newspaper advertisement. The only inclusion criterion was that participants had to be older than 60 years because we were interested in examining a normative sample of older adults.

In 2004, we conducted the first wave of the MAHS by assessing a heterogeneous sample of 215 older adults from the Montreal area. The 2-yr follow-up included 184 participants and 164 subjects participated in the 4-yr follow-up. Reasons for non-participation were being deceased (n = 13), having problems that prevented participation (n =17), refusing further participation (n = 8), and being unable to locate participants (n = 13). Seven additional participants were excluded from the analyses because they did not

participate in the 2-yr follow-up. Thus, the final sample included 157 participants. Study attrition over four years was not significantly associated with baseline measures of the study variables, except for participants' age. Older participants were more likely to discontinue their study participation, $t(213) = -2.30$, $p < .05$.

Materials

The analyses incorporated repeated measures of participants' diurnal cortisol rhythms and sleep duration. In addition, we included a number of sociodemographic (i.e., age, sex, partnership status, and education) and health-related covariates (i.e., chronic illness, medication usage, body mass index, and smoking; see Table 3 for means, standard deviations, and frequencies of main study variables).

Diurnal cortisol rhythms were assessed across waves on three non-consecutive typical days. We asked the participants to collect saliva samples as they engaged in their normal daily activities. On each of the three days, the participants collected five saliva samples (by using salivettes) at specific times of the day: awakening, 30 minutes after awakening, 2 p.m., 4 p.m., and before bedtime. Participants were asked not to eat or brush their teeth immediately prior to saliva collection to prevent contamination with food or blood. The actual time of day was recorded by the participants for all of the collected saliva samples. They were provided with a timer that they had to set at 30 min at the time they collected their first saliva sample after awakening. Compliance with the 30 minutes measure was generally good, as indicated by small deviations from 30 minutes after wakening at T1 ($M = 3.51$ minutes), T2 ($M = 4.65$ minutes), and T3 ($M = 2.45$ minutes). To ensure compliance concerning the collection of the afternoon and evening samples, participants

Table 3

Means, Standard Deviations, and Frequencies of Main Study Variables (N = 157).

Constructs	Mean (SD) or Percentage [a]
Cortisol (AUC) (log nmol/L h)	
4 Years	13.45 (2.66)
2 Years	13.22 (2.47)
Baseline	12.71 (2.59)
Sleep duration (T1) (minutes)	
4 Years	416.80 (88.33)
2 Years	410.03 (79.18)
Baseline	401.62 (82.66)
Age (yrs)	71.72 (5.55)
Male (%)	48.40
Number of chronic health problems (baseline)	2.26 (1.63)
No chronic health problems (%)	10.80
One chronic health problem (%)	24.80
Two or three chronic health problems (%)	43.90
Four or more chronic health problems (%)	20.50
Cortisol-related medication	2.21 (1.82)
BMI (baseline)	25.69 (3.73)
Underweight (BMI ≤ 18.4) (%)	2.50
Normal weight (18.5 ≤ BMI ≤ 24.9) (%)	38.90
Overweight (25 ≤ BMI ≤ 29.9) (%)	49.70
Obese (BMI ≥ 30) (%)	8.90
Current smoker (%)	10.20
Education (baseline)	2.09 (1.07)

Note. [a] Mean and standard deviation (SD) are presented for continuous variables. AUC = area under the curve; BMI = body mass index. Education was indexed as 0 = no education, 1 = high school, 2 = trade, 3 = masters or doctorate.

were called at 2 p.m. and 4 p.m. They were further instructed to collect the last sample of the day by themselves at the time they went to bed. The saliva samples were stored in participants' home refrigerators until they were returned to the lab 2–3 days after collection was completed (for stability of cortisol concentrations in these conditions, see Clements and Parker, 1998). After the saliva containers were returned to the lab, they were frozen until the completion of the study. Cortisol analysis was performed at the University of Trier, in duplicate, using a time-resolved fluorescence immunoassay with a cortisol-biotin conjugate as a tracer (Dressendörfer, Kirschbaum, Rhode, Stahl, & Strasburger, 1992). The intra-assay coefficient of variation was less than 5%; the inter-assay variability from cortisol analyses performed at the University of Trier has been found to be routinely below 10%.

All raw cortisol values were log-transformed to obtain normally distributed cortisol data. Total diurnal cortisol secretion was indexed by calculating the area under the curve (AUC) for each collection day using trapezoidal estimation (based on hours after awakening). The AUC was chosen as the outcome for this report because, of the various daytime cortisol metrics, it is the best proxy for overall tissue exposure to the hormone, and thus most likely to relate to sleep patterns and other distal health outcomes (Rodenbeck, Huether, Ruther, & Hajak, 2002). Given that some saliva samples may have been contaminated with blood or food, we excluded samples across the three waves of data collection because they deviated more than 3 SDs from the mean cortisol secretion for the time of day (1.2%). In cases in which a single saliva sample was missing, we replaced the missing value with the sample mean before calculating AUC. Within each

wave, AUC estimates were averaged across collection days to obtain a stable indicator of individual differences in diurnal cortisol secretion.

Sleep duration was measured using items from the Brief Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman, & Kupfer,1989). In all three assessments, we asked the participants to report for the majority of recent days and nights during the past month (a) the time they usually laid down to go to sleep, (b) the time they usually got out of bed in the morning, (c) how long it took them to fall asleep after they had laid down to go to sleep, (d) how many minutes of sleep they had lost because they woke up in the middle of the night, and (e) how many minutes of sleep they had lost because they woke up earlier than their usual time to get up. Global indicators of sleep duration for each of the three assessments were calculated by subtracting the minutes individuals spent in bed during the night without sleeping from the total minutes individuals spent in bed during the night.

Covariates. To minimize spurious associations, we controlled for variables that have been shown in previous research to be associated with cortisol secretion or sleep duration (Gangwisch et al., 2006; Patel, Malhotra, White, Gottlieb, & Hu, 2006; Wrosch et al., 2009). The covariates included baseline levels of participants' age, sex, partnership status, education, chronic illness, cortisol-related medication usage, body mass index, and smoking. Partnership status was coded as 1 (being married or cohabitating) and 2 (being separated, divorced, or widowed). Education was measured by asking participants to report their highest educational degree completed (0 = none, 1 = high school, 2 = trade, 3 = undergraduate degree, 4 = graduate degree). Usage of medications that could be associated with cortisol secretion was assessed by counting the number of medications

that either contained glucocorticoids and/or can influence the HPA-axis activity (e.g., antidepressants, β-blockers, or anti-inflammatory drugs). Body mass index was calculated in kg/m^2. Smoking was indexed as whether or not participants used cigarettes daily. Levels of chronic illness were measured by asking participants to report whether they were affected by 17 different health problems (e.g., coronary heart disease, cancer, high blood pressure, or arthritis). A count variable was computed to obtain an indicator of how many different chronic illnesses each participant experienced.

Data Analyses

To test the study's hypotheses, we performed two sets of analyses. The first set applied cross-lagged panel analyses and examined with four regression models whether previous levels of sleep duration would predict subsequent 2-yr changes in diurnal cortisol secretion (and whether levels of cortisol would predict 2-yr changes in sleep duration). The analyses were controlled for the previously described covariates.

The second set applied growth-curve analysis (utilizing HLM 6.0, Raudenbush, Bryk, Cheong, & Congdon, 2004) to examine whether cortisol levels would increase over 4-yrs of study, and whether 2-yr increases in sleep duration would ameliorate this effect. We used growth curve analysis because these models allowed us to examine within-person changes across time and to identify between-person predictors of individual differences in within-person changes (Bryk & Raudenbush, 1987). More specifically, we estimated in the Level-1 model the within-person variability in participants' cortisol secretion over four years (using data from T1, T2, and T3) as a function of years since study entry and a residual term. In the subsequent Level-2 model, we examined whether 2-yr changes in sleep duration (and baseline levels of sleep duration and the covariates)

would predict individuals differences in longitudinal trajectories obtained in the Level-1 model. A measure of 2-yr changes in sleep duration was obtained in a regression analysis, which predicted sleep duration assessed at T2 from the baseline indicator of sleep duration, and saved the standardized residuals. In order to shed further light on the temporal associations between sleep duration and cortisol secretion, we finally reversed the analyses to examine whether 2-yr increases in cortisol levels (using residualized change scores) would predict 4-yr changes in sleep duration.

Results

Sample Description and Preliminary Analyses

Table 3 provides a description of the sample. Participants were on average about 72 years old and approximately half of the sample was female. They reported an average of 2-3 chronic health problems, and their mean BMI was at the intersection of normal weight to overweight. Eighty percent of the participants reported that they used one or more medications that either contained glucocorticoids and/or can influence the HPA-axis activity (e.g., antidepressants, β-blockers, or anti-inflammatory drugs). Thirty-six percent obtained an undergraduate degree or a higher education, and the minority of the sample smoked. The socio-demographic characteristics and health status obtained in the sample were within the normative range for older adults residing at home (Aging NACO, 2006).

Participants slept at night on average between 402 minutes (baseline) and 417 minutes (4-yr follow-up) and their sleep ranged across waves from 212 to 592 minutes. Measures of sleep duration were significantly correlated with each other across waves ($rs = .55$ to $.56$, $ps < .01$). In addition, Figure 4 illustrates the raw cortisol values. The sample as a whole

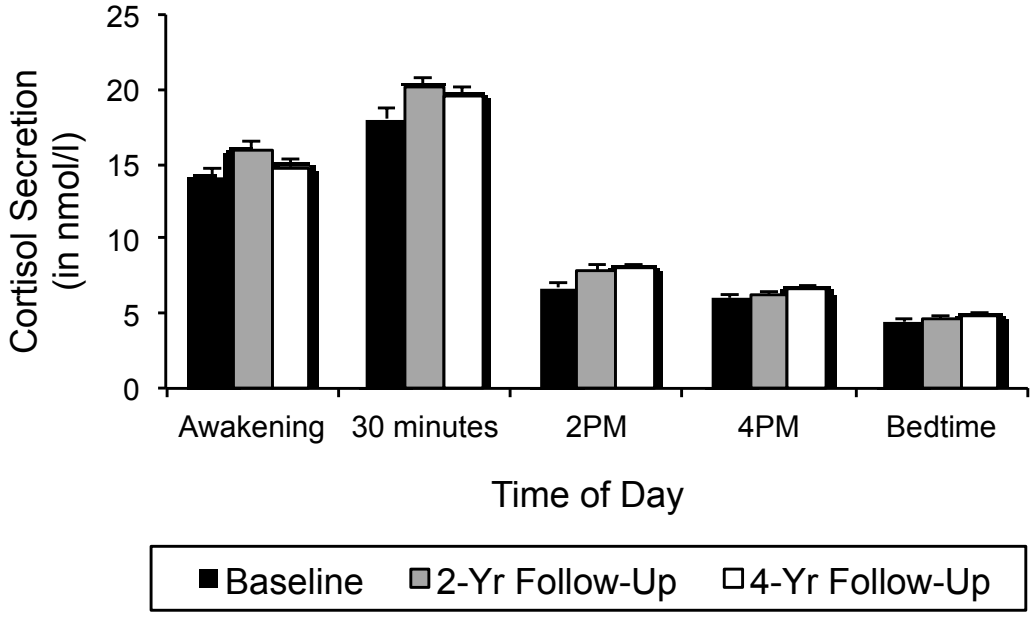

Figure 4 Means and standard errors of salivary cortisol secretion across three assessment days at baseline, 2-yr, and 4-yr follow-up.

exhibited typical patterns of cortisol secretion in all three study waves, demonstrating high cortisol levels at awakening, peaking 30 minutes after awakening, and continuously decreasing over the later part of the day until bedtime. The calculated day-to-day AUC values were considerably correlated within each wave of data-collection (rs = .46 to .67, $ps < .01$), and averaged AUC estimates were significantly correlated across waves (rs = .30 to .45, $ps < .01$).

2-Year Associations Between Sleep Duration and Cortisol Secretion

We conducted cross-lagged panel analyses with four separate regression models to examine whether baseline and 2-yr levels of sleep duration would predict subsequent 2-yr changes in cortisol secretion or vice versa. The results of the analyses are illustrated in Figure 5 and showed that 2-yr, and 4-yr levels of the outcome variables were significantly associated with previous levels of the outcomes. While these findings indicate considerable stability in cortisol and sleep duration over time, they also demonstrate that higher baseline levels of sleep duration were associated with smaller 2-yr increases in cortisol secretion, $F(1, 147) = 5.95$, $R^2 = .03$, $p < .05$, and higher 2-yr levels of sleep duration predicted smaller increases in cortisol secretion over the subsequent 2-yr period, $F(1, 147) = 6.98$, $R^2 = .04$, $p < .01$. However, neither baseline levels nor 2-yr levels of cortisol secretion were significantly associated with subsequent changes in sleep duration, $Fs(1, 147) < 3.44$, $R^2s < .01$, $ps > .05$.[1]

[1] We note that the pattern of results obtained in our analyses remained identical if individual differences in compliance with the 30-minutes measure cortisol were taken into account. In addition, subsequent analyses showed that neither depressive symptomatology (CES-D), nor a dichotomous distinction between taking versus not taking any cortisol-related medicine, or any of the 17 assessed chronic health problems (if analyzed separately) explained the reported effects.

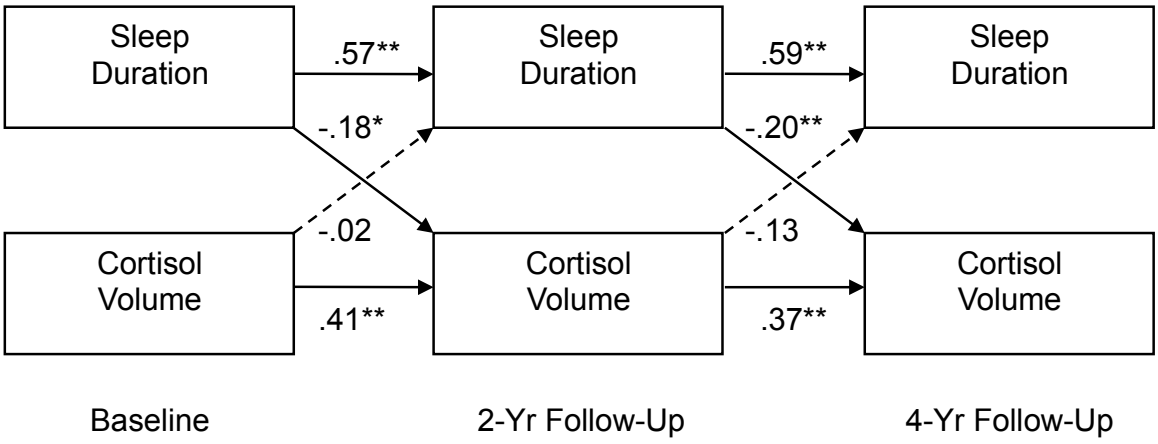

Figure 5 Cross-lagged panel analyses examining the longitudinal associations between baseline, 2-yr, and 4-yr levels of sleep duration and cortisol secretion. Values represent standardized regression coefficients.

Predicting 4-Year Trajectories of Cortisol Secretion.

We conducted growth-curve models to examine whether cortisol would increase over 4 years of study and whether these increases could be moderated by 2-yr changes in sleep duration. The results of these analyses are summarized in Table 4 and show that baseline levels of cortisol secretion (i.e., Level-1 intercept) were significantly different from zero. Moreover, the Level-1 slope was significantly positive, which implies that levels of cortisol secretion exhibited a linear increase over time. Finally, there was significant variability around the averaged Level-1 intercept and slope, χ^2s(156) > 223, *ps* < .01, indicating the presence of reliable individual differences in baseline levels of, and changes in, cortisol secretion.

In the Level 2-model, we attempted to explain this variability and predicted individual differences in Level-1 intercept and slope by the centered baseline scores of sleep duration, 2-yr changes in sleep duration, covariates, and a random residual term. The results from the Level-2 model revealed that baseline levels of sleep duration were negatively associated with baseline levels of cortisol secretion (see effects on intercept in Table 4), indicating that participants who reported shorter sleep duration at baseline concurrently exhibited higher diurnal cortisol secretion than participants who slept longer. Of the sociodemographic and health-related covariates, age, sex, partnership status, and BMI significantly predicted baseline levels of diurnal cortisol volume (i.e., Level-1 intercept). As shown in Table 4, older individuals, men, participants without a partner, and participants with a higher BMI secreted significantly higher diurnal cortisol levels at baseline than younger individuals, women, participants with a partner, and participants with a lower BMI. Moreover, except for partnership status, none of the

Table 4

Results of Growth-Curve Analyses predicting 4-Yr Changes in Diurnal Cortisol Volume (AUC) by Sociodemographic Factors, Baseline Levels of Health-Related Variables, and Baseline Levels and 2-Yr Changes of Sleep Duration.

	Diurnal Cortisol Volume (AUC)			
	Intercept [a]		Slope	
	β (*SE*)	T-ratio	β (*SE*)	T-ratio
Level-1	12.73 (0.17)	73.89**	0.20 (0.06)	3.30**
Level-2 Predictors				
Age	0.09 (0.03)	3.35**	-0.01 (0.01)	-0.98
Sex [b]	-1.43 (0.38)	-3.80**	0.19 (0.14)	1.40
Partnership status [c]	1.31 (0.35)	3.74**	-0.40 (0.13)	-2.96**
Education	0.29 (0.15)	1.89	-0.02 (0.06)	-0.34
Chronic illness	0.13 (0.12)	1.10	0.05 (0.04)	-1.21
Medication	0.08 (0.05)	1.41	0.01 (0.02)	0.60
BMI	0.09 (0.04)	1.94*	-0.02 (0.02)	-1.03
Smoking	1.01 (0.69)	1.46	-0.08 (0.20)	-0.40
T1 Sleep duration	-0.01 (0.00)	-3.34**	0.00 (0.00)	1.22
Δ T1-T2 Sleep duration	-0.03 (0.16)	-0.21	-0.18 (0.05)	-3.30**

Note. Level-1 model had 156 *dfs*. Level-2 model had 146 *dfs*. [a] The intercept represents participants' levels of cortisol secretion at study entry, and the slope represents the within-person associations between years since study entry and cortisol secretion. [b] Higher values represent females. [c] Higher values represent participants without a partner. * $p < .05$. ** $p < .01$.

covariates significantly predicted 4-yr changes in cortisol secretion (i.e., Level-1 slope). Participants who were married or cohabitating (β = .58, *SE* = .15, *T-ratio* = 3.85, $p < .01$) exhibited larger increases in their initially lower levels of cortisol secretion over 4 years as compared to their counterparts who did not have a partner (β = -.18, *SE* = .14, *T-ratio* = -1.22, $p > .10$). Finally, the analysis demonstrated that 2-yr changes in sleep duration (but not baseline levels of sleep duration) significantly predicted 4-yr changes in cortisol secretion (see effects on slope in Table 4).

Figure 6 illustrates the effect of 2-yr changes in sleep duration on 4-yr changes in cortisol secretion by plotting the trajectories of cortisol secretion across measurements, separately for participants who exhibited increases (+1 *SD*) and decreases (-1 *SD*) in levels of sleep duration over the first two years of study. The observed pattern of results demonstrates that levels of cortisol secretion did not change significantly among participants who experienced increases in their sleep duration over the first two years of study (β = 0.02, *SE* = 0.08, *T-ratio* = 0.24, $p > .10$). By contrast, participants who reported decreases in sleep duration over the first two years of the study displayed steep increases in cortisol secretion over the four-years study period (β = 0.38, *SE* = 0.08, *T-ratio* = 4.68, $p < .01$).

Predicting 4-Year Trajectories of Sleep Duration.

We next reversed the previously reported growth curve analysis, and predicted in the Level-1 model the within-person variability in sleep duration (using data from T1, T2, and T3) as a function of years since study entry and a residual term. In the Level 2 model, we estimated between-person variation in the within-person intercept and slope of sleep duration as a function of the centered baseline scores of cortisol levels, 2-yr changes in

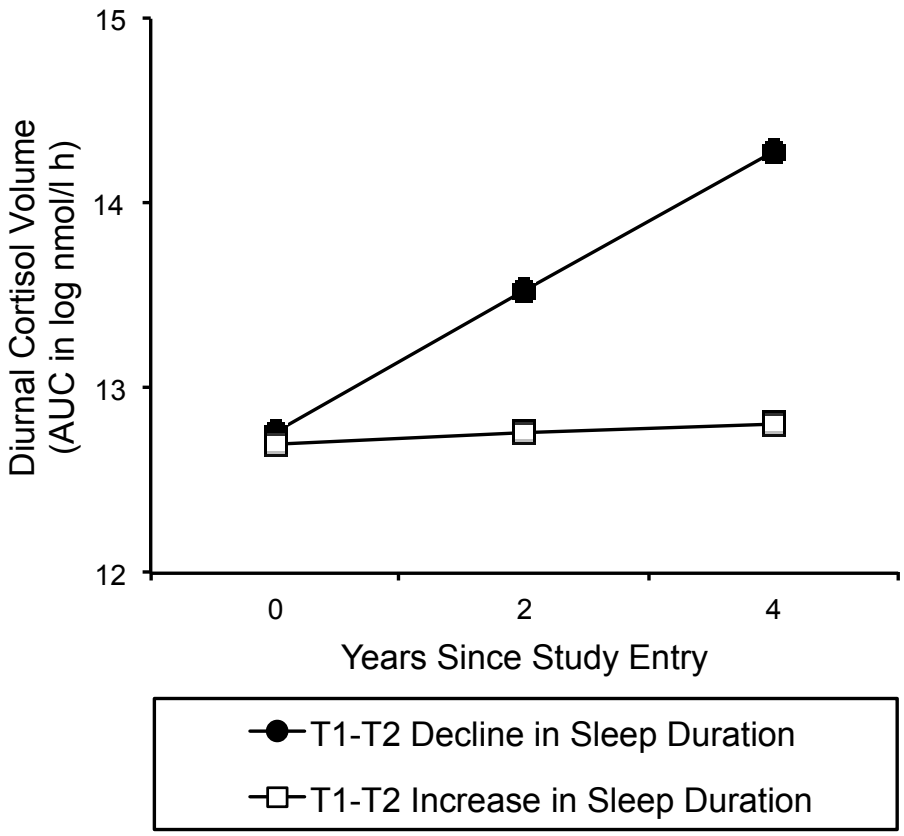

Figure 6 Changes in cortisol secretion across four years, separately for participants who reported increases (+1 SD) and declines (-1 SD) in levels of sleep duration over the first two years of study.

cortisol levels, covariates, and a random residual term. The results of the Level-1 model showed that baseline levels of sleep duration were significantly different from zero, β = 402.11, *SE* = 5.89, *T-ratio* = 68.24, *df* = 156, $p < .01$, and increased significantly over 4 years of study, β = 4.08, *SE* = 1.68, *T-ratio* = 2.43, *df* = 156, $p < .05$. In addition, there was significant variability around the averaged Level-1 intercept and slope, χ^2s(156) > 189, *ps* < .01. Explaining this variability, the Level-2 model showed that baseline levels of cortisol secretion, β = -6.39, *SE* = 2.70, *T-ratio* = -2.36, *df* = 146, $p < .05$, as well as 2-yr changes in cortisol secretion, β = -5.40, *SE* = 2.20, *T-ratio* = -2.45, *df* = 146, $p < .05$, were negatively associated with baseline levels of sleep duration. The latter finding mirrors the results from the cross-lagged panel analyses by demonstrating that those participants who exhibited declines in cortisol secretion over two years reported higher baseline levels of sleep duration. Of the covariates, only sex, β = -28.81, *SE* = 13.07, *T-ratio* = -2.20, *df* = 146, $p < .05$, significantly predicted baseline levels of sleep duration (i.e., Level-1 intercept). Men reported longer sleep at baseline as compared to women. In addition, partnership status significantly predicted 4-yr changes in sleep duration (i.e., Level-2 slope), β = -8.39, *SE* = 3.70, *T-ratio* = -2.26, *df* = 146, $p < .05$, revealing that participants with a partner reported larger increases in sleep duration over four years than their counterparts who did not have a partner. Finally, the Level-2 model also showed that neither baseline levels of cortisol secretion nor 2-yr changes in cortisol secretion significantly predicted 4-yr changes in participants' sleep duration.

Discussion

The study's findings demonstrate that older adults experienced increases in diurnal cortisol secretion over time, and that this effect was forecasted by individual

differences in sleep duration. More specifically, results from cross-lagged panel analyses showed that lower, as compared to higher, baseline and 2-yr levels of sleep duration forecasted increases in cortisol secretion over the subsequent two years. Further, growth-curve models substantiated these results by demonstrating that cortisol secretion generally increased over four years of study, and that 2-yr increases in sleep duration ameliorated 4-yr increases in cortisol secretion. In fact, our analyses showed a steep increase in 4-yr cortisol secretion among older adults who experienced declines in their sleep duration over the first two years of study. By contrast, no significant changes in cortisol output were found among their counterparts who increased their sleep duration. Finally, reversed analyses showed that levels of and 2-yr changes in cortisol secretion were not associated with subsequent changes in participants' sleep duration. Together, this pattern of findings supports the hypothesis that sleep patterns can have a directional, and possibly restorative, effect on older adults' diurnal cortisol secretion, and that long sleep can prevent age-normative increases in cortisol output.

It is important to note that although the cross-lagged panel analyses showed that higher baseline and 2-yr levels of sleep duration ameliorated subsequent 2-yr increases in cortisol secretion, the results from the growth curve model showed that baseline levels of sleep duration did not forecast 4-yr changes in cortisol secretion. To this end, our analyses suggest that this pattern of findings may have emerged because some individuals exhibited changes in their sleep duration over the first two years of study, which were reliable predictors of 4-yr changes in cortisol secretion. Thus, levels of sleep duration can be a significant predictor of older adults' cortisol changes over two years, and subsequent changes in cortisol secretion are more likely to emerge as a function of alterations in

sleep duration during these two years.

These data are the first to provide direct longitudinal evidence for a mechanism that links longer sleep duration with the prevention of long-term increases in older adults' diurnal cortisol secretion. The identification of this mechanism is important because older adulthood is often associated with increasing levels of age-related challenges (e.g., health threats and loss of resources, Baltes et al., 1979; Heckhausen et al., 2010), which can elicit psychological distress and dysregulate the HPA axis (Wrosch et al., 2004; Wrosch et al., 2007a). Thus, long sleep has the potential to ameliorate the adverse consequences of age-related challenges and emotional distress on increases in older adults' diurnal cortisol output, which could in turn reduce their risk of experiencing subsequent physical health declines. By contrast, short sleep could give rise to increased levels of perceived stress and negative emotions (Hamilton et al., 2007), which may dysregulate a person's HPA-axis. In addition, cortisol disturbances among older adults experiencing poor sleep could derive from their engagement in maladaptive behaviors (e.g., coffee consumption or sedentary behaviors), which they may adopt to cope with increased fatigue (e.g., Digdon & Rhodes, 2009). These conclusions are consistent with previous work theorizing that good sleep can down-regulate levels of cortisol secretion, and through this mechanism explain the observed buffering effect that restorative sleep patterns can have on the association between cortisol and subsequent physical health problems (Wrosch et al., 2008).

In addition, our research indicated that older adults experienced a fairly small, but significant, increase in their sleep duration over time (approximately 4 minutes each year). This finding is consistent with results from meta-analysis documenting that sleep

duration typically decreases until age 60 among healthy adults, but plateaus thereafter (Ohayon, Carskadon, Guillrminault, & Vitiello, 2004). However, some studies have shown that sleep duration can also decline in older adulthood, and mounting evidence suggests that such effects are due to older adults' medical comorbidities rather than age per se (Foley et al., 2001; Ohayon & Vechierrini, 2002; Vitiello, Moe, & Prinz, 2002). The latter possibility further implies that sleep could become reduced over time when older adults experience an onset of severe physical health problems. Subsequently conducted analyses showed that such quadratic effects were not observed in our study, which we attribute to the relatively good health of the study participants. Nonetheless, we would expect such declines in sleep duration to emerge in later waves of our study, and suggest that this process would be likely to set in motion a downward spiral, characterized by the onset of severe health problems, reduced sleep length, increases in cortisol secretion, and subsequent physical disease.

Finally, the reported results may have some implications for clinical treatment. If restorative sleep can down-regulate increases in cortisol levels, clinical interventions should target older adults' sleep patterns (e.g., Morin et al., 1994). Such interventions could prevent the adverse consequences of age-normative challenges on the dysregulation of biological processes. Moreover, given that dysregulated cortisol patterns can forecast a variety of disease-related processes (Dekker et al, 2008; Smith, Ben, Beswick, Yarnell, Lightman, & Elwood, 2005), interventions along these lines could further contribute to the maintenance of older adults' physical health.

There are limitations to this study that need to be addressed in future research. First, we used a self-report measure to assess sleep duration. Although such self-reports

of sleep duration have been validated with objective sleep assessments (Lockley, Skene, & Arendt, 1999), they could be biased by other psychological factors. In this regard, it is important to note that we analyzed changes in sleep duration over time as predictor and outcome variable, and such change measures are likely to partial out some of the potential biases found in self-reports. In addition, demonstrating that changes in sleep duration were associated with subsequent changes in cortisol output lessens the possibility that the reported findings are driven by sleep pathology. In fact, clinically significant sleeping problems could underlie individual differences in sleep duration and were not assessed in our study. However, we would expect that such sleeping problems are more likely to be associated with levels, as compared to changes, of sleep duration. In addition, we note that our sleep data were within the normative range of healthy older adults' sleep duration in the general population (Ohayon, 2004). Nonetheless, future research should include objective sleep measures and assess sleep pathology to replicate the obtained findings.

Second, our analysis focused on sleep duration and did not examine sleep efficiency (i.e., the time that individuals sleep relative to the time they spent in bed), which can also exert restorative functions (e.g., Cacioppo et al., 2002a; Dew et al., 2003). In this regard, it would have been possible to incorporate a measure of sleep efficiency into our analyses. We did not pursue this possibility because the indices used for computing these variables largely overlap and sleep duration and sleep efficiency were strongly correlated in our study (rs > .75, ps < .01). However, we note here that repeating the analyses with sleep efficiency (instead of sleep duration) did not produce the same effects, and levels of, or changes in, sleep efficiency were not significantly associated

with subsequent changes in cortisol output. However, given that previous work has shown that sleep efficiency can also predict health-related outcomes above and beyond sleep duration (Cohen et al., 2009), future research is needed to examine the conditions under which sleep duration and sleep efficiency predict different outcomes.

Third, we focused in our analysis on the overall volume of cortisol secretion because we reasoned that sleep duration may be particularly likely to associate with cumulative indices of cortisol output across the day. However, other research has shown that sleep duration may also be associated with cortisol slope and awakening levels of cortisol (Kumari et al., 2009). Follow-up analyses of our data showed that this was not the case for our sample, as levels of or changes in sleep duration were unrelated to changes in these alternative cortisol indices. This may imply that short sleep can influence long-term increases in cortisol secretion across the entire day, and such effects are less likely to be detected if only portions of the diurnal rhythm or changes in cortisol slope are analyzed.

Fourth, although our results were independent from a number of sociodemographic and health-related variables, some of the covariates were associated with participants' cortisol secretion. In this regard, the findings that lower baseline cortisol levels were associated with a younger age, being female, having a partner, and a lower BMI support the validity of our measures as they reflect associations commonly found in other research (e.g., Almeida et al., 2009; Fraser, Ingram, Anderson, Morrison, Davies, & Connell, 1999; Kudielka et al., 1998). However, it is interesting to note that, over time, participants who had a partner exhibited larger cortisol increases, but also longer sleep, than their single counterparts. This suggests that having a partner in older

adulthood can be both, a protective factor and a risk factor. In fact, such processes could derive from the possibility that although older adults often benefit from close relationships (Löckenhoff & Carstensen, 2004) and co-sleeping has been associated with reports of better sleep (Troxel, 2010), having a partner could also increase stress in the context of severe disability or becoming a caregiver (e.g., Brodaty & Hadzi-Pavlovic, 1990). While our study could not shed more light on these possibilities, we suggest that future research should conduct couple studies and track older adults' physical health, sleep, and cortisol output over time.

Finally, our analyses did not examine the complete process that could be associated with cortisol increases in old age. In this regard, our theoretical model would assume that specific age-related challenges could trigger emotional distress and dysregulate older adults' cortisol secretion (Wrosch et al., 2004, 2007), which may increase their likelihood of developing subsequent physical disease. In this cascade of events, adaptive sleep patterns and other protective factors (e.g., coping, Wrosch & Schulz, 2008) could prevent chronically high levels of psychological distress and cortisol secretion and contribute to good physical health. Thus, future studies should include a wider range of contextual, psychological, and physical health variables to illuminate how older adults can maintain adaptive biological functioning and good physical health.

Health-Related Self-Protection Predicts Psychological and Biological Benefits Among Lonely Older Adults

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Abstract

Objectives: This longitudinal study ($N = 122$) examined whether health-related self-protection (e.g., using positive reappraisals or avoiding self-blame) prevents lonely older adults from exhibiting 2-year increases in stress-related psychological and biological problems (i.e. perceived stress and diurnal cortisol) as well as higher levels of systemic inflammation (CRP) after 6 years. **Methods:** Levels of diurnal cortisol (AUC) and perceived stress were measured at baseline and 2-year follow-up, and levels of systemic inflammation were measured at 6-year follow-up. The main predictors included baseline levels of loneliness and health related self-protection. **Results:** Among lonely participants, baseline self-protection was associated with an amelioration of 2-year increases in psychological stress and diurnal cortisol volume, and predicted lower CRP after 6 years. These associations were not found among non-lonely participants. In addition, mediation analyses demonstrated that the buffering effect of self-protection on 6-year levels of CRP among lonely older adults was statistically mediated by 2-year changes in cortisol volume. **Conclusions:** These findings suggest that lonely older adults may ameliorate stress-related psychological and biological disturbances if they engage in self-protection to cope with their health threats.

Key Words: diurnal cortisol; C-reactive protein; loneliness; self-protection, aging.

Abbreviations: AUC = area under the curve; CRP = C-reactive protein; BMI = body-mass-index; SES = socioeconomic status.

Introduction

Feelings of loneliness are increasingly recognized to compromise quality of life (Cacioppo et al., 2002b; Heinrich & Gullone, 2006). Such adverse consequences of loneliness are likely to be especially pronounced in older adulthood (Hawkley & Cacioppo, 2007) when individuals experience a normative loss of resources and increasing health threats (Baltes, Cornelius, & Nesselroade, 1979; Heckhausen et al., 2010). Indeed, lonely older adults may find managing health-related threats particularly challenging, and the stress that ensues may contribute to patterns of biological dysregulation (e.g., cortisol disturbance or heightened systemic inflammation). However, theory and research also suggest that older adults can cope with health threats and prevent such problems if they engage in self-protective control strategies (Heckhausen et al., 2010; Wrosch et al., 2009). Some examples of self-protective control strategies are positive reappraisals, where a person focuses on positive aspects in the context of a problem, and the avoidance of self-blame for the occurrence of a health problem itself. In the current research we tested these ideas using four waves of data from a longitudinal study of older adults. We expected that the adoption of self-protective strategies would prevent lonely older adults, over time, from exhibiting increasing levels of psychological stress, as well as alterations in cortisol output and heightened systemic inflammation.

Mounting evidence suggests that feelings of loneliness increase people's vulnerability to a variety of physical health problems (Cacioppo et al., 2002b; Kiecolt-Glaser et al., 1984; Russell et al., 1997; Seeman, 2000; Sugisawa, Liang, & Liu, 1994). Moreover, the processes that underlie the loneliness-health link could be related to individuals' increased vulnerability to psychological stress and its biological sequelae

(Hawkley & Cacioppo, 2010). In this regard, lonely individuals may accumulate health-related problems because they construe their world as threatening (Cacioppo, Hawkley, Rickett, & Masi, 2005), and these views trigger stress-related disturbances in endocrine functioning as well as elevated levels of the inflammatory biomarker C-reactive protein (Cacioppo et al., 2000; Kiecolt-Glaser et al., 1984; Steptoe, Owen, Kunz-Ebrecht, & Brydon, 2004).

In support of such a process, research has demonstrated that lonely individuals perceive more stress than non-lonely individuals (Cacioppo et al., 2000; Cacioppo, Hawkley, & Bernston, 2003; Cacioppo, Hughes, Waite, Hawkley, & Thisted, 2006). Increased levels of perceived stress, in turn, can contribute to dysregulation of immune function via behavioral (e.g., smoking or not exercising), autonomic, or neuroendocrine pathways (Cohen & Kessler, 1997; McEwen, 1998; Miller et al., 2009). Indeed, studies have documented that feelings of loneliness are associated with poor health behaviors, greater diurnal cortisol secretion (Cacioppo et al., 2000; Kiecolt-Glaser et al., 1984), and higher levels of the inflammatory biomarker CRP (McDade, Hawkley, & Cacioppo, 2006). A recent study that used functional genomic methodology found evidence suggesting that loneliness was associated with an underlying resistance to glucocorticoid signaling (Cole, Hawkley, Arevalo, Sung, Rose, & Cacioppo, 2007). This tendency could explain why lonely individuals simultaneously have both high levels of cortisol and inflammatory biomarkers.

Empirical evidence further suggests that associations between feelings of loneliness and patterns of morbidity and mortality can be particularly strong in older adulthood (Hawkley & Cacioppo, 2007). This effect may be related to the above-

discussed biological consequence of loneliness, given that CRP can play a key role in the development of a wide range of physical health problems associated with aging (e.g., cardiovascular disease, functional disability, or mortality; Jenny et al., 2007; Kuo et al., 2006; Ridker et al., 2002; Ridker & Silvertown, 2008; Strandberg, 2000). Further, the stress-related psychological and biological disturbances associated with loneliness could accrue from the experience of increasing health threats in older adulthood (e.g., physical symptoms, functional declines, or chronic illness; Wrosch et al., 2004). Such adverse effects of common age-related challenges may be aggravated among lonely older adults because they lack emotionally satisfying social networks that are useful for regulating the distress derived from the occurrence of normative health threats. By contrast, older adults who perceive themselves as socially embedded should be less likely to accumulate stress-related problems because their social ties may facilitate the management of health-related challenges. This argument is consistent with research documenting that the adaptive value of satisfying personal relationships and emotional closeness becomes increasingly important for older adults' emotion regulation and the resulting benefits for mental and physical health (Carstensen, 1992; Carstensen et al., 1999; Löckenhoff & Carstensen, 2004; Watkins, 1997).

To forestall the mental and physical health declines associated with age-related challenges, the *Motivational Theory of Life-Span Development* postulates that older adults should engage in self-protective control strategies (Heckhausen et al., 2010). These control strategies involve making positive reappraisals or external attributions for health threats (e.g., seeing the silver lining or avoiding self-blame for problems) and have been shown to benefit older adults' mental and physical health (Wrosch et al., 2000, 2006,

2009). Such adaptive effects are likely to be observed if self-protective strategies foster acceptance of, and disengagement from, problems that cannot be overcome through a person's active coping behaviors (Heckhausen et al., 2010; Heckhausen & Schulz, 1995).

The documented benefits of self-protective strategies in older adulthood imply that their use could become particularly important for ameliorating stress-related psychological and biological disturbances among older adults who are lonely and experience health threats. Given that lonely older adults often lack social networks that could provide emotional and/or tangible support, internal self-protection processes could facilitate the management of health threats, and thereby prevent downward spirals in mental and physical health. By contrast, the use of health-related self-protection may generally be less influential among older adults who do not feel lonely, as they can rely on emotionally satisfying networks that help them to adjust to age-related challenges. In sum, we propose that health-related self-protection can become paramount among lonely older adults because the emotional benefits of these strategies are likely to counteract some of the psychological and biological processes through which feelings of loneliness contribute to physical health problems.

To test this hypothesis, we analyzed data from a 6-year longitudinal study of older adults and compared psychological and biological outcomes among relatively lonely versus non-lonely participants. We expected that lonely participants would experience increases in psychological stress and diurnal cortisol output over a 2-year follow-up period, as well as heightened levels of the inflammatory biomarker CRP after 6 years of study. In addition, we predicted that higher baseline levels of health-related self-protection would prevent these psychological and biological problems from occurring

among lonely participants. Finally, we tested whether the inflammatory benefits associated with lonely participants' self-protection would occur via prospective reductions in psychological stress and cortisol output.

Method

Participants

The data stem from a longitudinal study of community-dwelling older adults known as the *Montreal Aging and Health Study* (Wrosch, Schulz, Miller, Lupien, Dunne, 2007b). Participants were recruited through newspaper advertisements. The only inclusion criterion was that participants had to be older than 60 years because we were interested in examining a normative sample of older adults. After contacting the laboratory, participants were invited for an initial appointment. Participants who were unable to visit the lab were assessed in their homes.

In 2004, we enrolled the first wave of a heterogeneous sample of 215 older adults from the Montreal area. The 2-year and 4-year follow-ups included 184 and 164 participants, respectively. 137 subjects participated in the 6-year follow-up. Study attrition from baseline to 6-year follow-up was attributable to being deceased ($n = 23$), refusing to participate further ($n = 9$), being unable to locate participants ($n = 19$), or having other personal problems that precluded participation ($n = 27$). Study attrition was not significantly associated with baseline measures of the study variables, except for participants' age. Older, as compared to younger, participants were more likely to discontinue their study participation over time, $t(128.91) = -2.46$, $p < .01$. The analyses for testing our hypotheses are based on 122 participants because we excluded fifteen participants who did not provide data on CRP, cortisol, or perceived stress.

Materials

The main study variables incorporated measures of participants' health-related self-protection, loneliness, perceived stress, diurnal cortisol rhythms, and CRP. In addition, the study included a number of sociodemographic (i.e., age, sex, and socioeconomic status) and health-related (i.e., chronic illness, smoking, and body-mass-index) covariates.

Health-related self-protection was measured at baseline by administering three items from a previously validated self-report questionnaire (Wrosch et al., 2009). These items were developed on the basis of the "Motivational Theory of Life-Span Development" (Heckhausen et al., 2010) and represent core aspects of self-protective secondary control (i.e., external attributions and positive reappraisals; Wrosch et al., 2000; Wrosch & Heckhausen, 2002). The specific items were "Even if my health is in very difficult condition, I can find something positive in life ", "When I am faced with a bad health problem, I try to look at the bright side of things", or "When I find it impossible to overcome a health problem, I try not to blame myself." The items were answered with 5-point Likert-type scales (0 = *almost never true* to 4 = *almost always true*), and an indicator of participants' health-related self-protection was obtained by computing a mean score of the three items (α = .73).

Loneliness was measured at baseline by asking participants at the end of three non-consecutive typical days to what extent they felt "lonely" or "isolated" during the day. These two items have been used for assessing loneliness in previous research (Pressman, Cohen, Miller, Barkin, Rabin, & Treanor, 2005). Participants responded to the items by using 5-point Likert-type scales, ranging from 0 (*very slightly or not at all*) to 4

(*extremely*). For each day, the two items were correlated, *r*s = .36 to .59, *p*s < .01, and we computed sum scores of the two items (M_{D1} = .40, SD_{D1} = 1.00; M_{D2} = .36, SD_{D2} = 1.04; M_{D3} = .39, SD_{D3} = .94). Daily measures of loneliness were significantly correlated with each other across days (*r*s = .34 to .61, *p*s < .01), and we obtained a global indicator of loneliness by computing a sum score of the three daily ratings (α = .82).

Perceived stress was measured at baseline and 2-year follow-up by administering the 10-item version of the perceived stress scale (Cohen et al., 1983). Participants were asked to rate how frequently they experienced 10 different situations over the past month (e.g., "How often have you been upset because of something that happened unexpectedly?'' or "How often have you felt difficulties were piling up so high that you could not overcome them?"), by using 5-point Likert-type scales, ranging from 1 (*never*) to 5 (*very often*). Global indicators of perceived stress were obtained by averaging the ratings of the 10 items separately for baseline and 2-yr follow up (αs > .87). We computed a measure of change in perceived stress over two years by predicting in a regression analysis the 2-yr follow-up levels from the baseline levels of perceived stress and saving the standardized residuals for further analysis. Levels of perceived stress were correlated with each other across waves (see Table 6), and did not significantly change over time, $t(121) = 1.61$, $p > .10$.

Diurnal cortisol rhythms were measured across 3 non-consecutive typical days, at both baseline and 2-year follow-up. We asked the participants to collect saliva samples as they engaged in their normal daily activities. On each of the 3 days, participants collected five saliva samples (by using salivettes) at specific times of the day: awakening, 30 min after awakening, 2 PM, 4 PM, and bedtime. Participants were asked not to eat or

brush their teeth immediately prior to saliva collection to prevent contamination with food or blood. They were provided with a timer that they had to set at 30 min at the time they collected their first saliva sample after awakening. To ensure compliance concerning the collection of the afternoon and evening samples, participants were called at 2 PM and 4 PM. They were further instructed to collect the last sample of the day by themselves at the time they went to bed. The actual time of day was recorded by the participants for all of the collected saliva samples. The saliva samples were stored in participants' home refrigerators until they were returned to the lab 2–3 days after collection was completed (Clements & Parker, 1998). After the saliva containers were returned to the lab, they were frozen until the completion of the study. Cortisol analysis was performed at the University of Trier, in duplicate, using a time-resolved fluorescence immunoassay with a cortisol-biotin conjugate as a tracer (Dressendörfer et al., 1992). The intra-assay coefficient of variation was less than 5%; the interassay variability has been found to be routinely 10%.

Across both assessments, we obtained typical patterns of cortisol secretion over the three days, demonstrating high levels at awakening (*M*s = 12.45 to 13.91, *SD*s = 6.33 to 8.58), peaking 30 minutes after awakening (*M*s = 16.76 to 19.90, *SD*s = 9.96 to 12.27), and continuously decreasing over the later part of the day (2 PM: *M*s = 5.38 to 6.68, *SD*s = 2.99 to 4.06; 4 PM: *M*s = 4.88 to 5.39, *SD*s = 3.06 to 4.11; and bedtime: *M*s = 3.18 to 3.70, *SD*s = 3.06 to 4.77). All raw cortisol values were log-transformed to stabilize variance, and total diurnal cortisol secretion was indexed by calculating the area under the curve (AUC) for each collection day using trapezoidal estimation (based on hours after awakening). AUC was chosen because it captures individual differences in

cumulative tissue exposure to cortisol, which should in turn affect the immune system's capacity to regulate systemic inflammation. AUC was calculated for days on which participants provided five saliva samples (on average 5.52 of the 6 days). For each wave, AUC was averaged across collection days to obtain a stable indicator of individual differences in diurnal cortisol secretion. We computed a measure of changes in cortisol secretion over time by predicting 2-yr follow-up levels from baseline levels of cortisol (AUC) and saving the standardized residuals. Participants' diurnal cortisol levels were significantly correlated with each other across waves (see Table 6), and did not significantly increase from baseline to 2-yr follow-up, $t(121) = .59$, $p > .10$.

C-reactive protein was measured as an indicator of systemic inflammation at 6-year follow-up. CRP was not measured during earlier waves of the study. We collected capillary whole blood from participants on filter paper by using a finger-prick. A disposable, single-use lancet was used to deliver a controlled, uniform puncture to the finger, and up to five drops of blood were collected on filter paper designed for this purpose (Whatman 903, GE Healthcare, Piscataway, NJ). Samples were allowed to dry and subsequently stored in a freezer with desiccant in resealable plastic bags until completion of the study. CRP was analyzed in the Laboratory for Human Biology Research at Northwestern University using a high-sensitivity enzyme immunoassay protocol (McDade, Burhop, & Dohnal, 2004; Williams & McDade, 2009). Prior validation of the blood spot CRP method has shown good sensitivity and reliability, as well as high correlations between CRP levels obtained from matched plasma and blood spot samples (McDade et al., 2004). The median C-RP value at 6-yr follow-up was 0.98 mg/L (25^{th} percentile: 0.51 and 75^{th} percentile: 2.13).

Covariates. To minimize the likelihood of spurious associations, we included a number of covariates into our analyses that could influence distress, cortisol, or CRP. These variables included baseline levels of participants' age, sex, socioeconomic status (SES), chronic illness, smoking, and body-mass-index (BMI). SES was assessed by averaging the standardized scores of participants' highest level of education, yearly family income, and perceived socioeconomic status (α = .69; Adler, Epel, Castellazzo, & Ickovics, 2000). Baseline levels of chronic illness were measured by counting the presence of 17 different health problems (e.g., coronary heart disease, cancer, osteoarthritis, or diabetes). Smoking was indexed as whether or not cigarettes were used daily. Finally, BMI was calculated in kg/m^2.

Data Analyses

We tested our hypotheses in three sets of regression analyses. The first analysis examined whether health-related self-protection would be associated with lower 6-year levels of C-reactive protein (CRP) in lonely (but not in non-lonely) participants. The second set tested whether health-related self-protection would also predict 2-year reductions of perceived stress and diurnal cortisol secretion in lonely (but not in non-lonely) participants. The hypotheses from the first two sets were evaluated by testing the interaction effects between health-related self-protection and loneliness on the respective outcomes for significance. The third set of analyses investigated whether 2-year reductions in perceived stress and/or diurnal cortisol secretion would statistically mediate lower 6-year levels of CRP. Mediation was examined by conducting bootstrap analyses (Preacher & Hayes, 2008), which examined whether 2-year changes in perceived stress and cortisol secretion would exert an indirect effect on the interaction between health-

related self-protection and loneliness in predicting 6-year levels of CRP. The mediation analysis was based on 5000 bootstraps and the indirect effect was evaluated as significant if the bias-corrected 95% confidence interval of the indirect effect did not cross zero (Preacher & Hayes, 2008). All analyses controlled for sociodemographic (i.e., age, sex, and socioeconomic status) and health-relevant (i.e., chronic illness, smoking, and body-mass-index) covariates, and predictor variables were standardized before conducting the analyses.

Results

Sample

Table 5 provides a description of the sample, and Table 6 reports the zero-order correlations between the main constructs. Participants used in the analyses were on average 72 years old and approximately half of the sample was female. They reported an average of 2-3 chronic health problems, and their mean BMI was between normal and overweight. Thirty-four percent obtained an undergraduate degree or a higher education, and the minority of the sample smoked. The participants' socio-demographic characteristics and health status were within the normative range for older adults residing at home (Aging NACO, 2006).

Main Analyses

We examined whether health-related self-protection was associated with CRP among lonely participants by conducting a hierarchical regression analysis. The analysis predicted 6-year levels of CRP by baseline levels of loneliness and self-protection and the covariates (Step 1), followed by the interaction between loneliness and self-protection (Step 2). The results of the analyses are reported in Table 7. None of the incorporated

Table 5

Means, Standard Deviations, and Frequencies of Main Study Variables (N = 122).

Constructs	Mean (SD) or Percentage [a]
C-reactive protein (mg/L)	
6 Years	1.57 (1.68)
Perceived stress	
2 Years	2.44 (0.64)
Baseline	2.37 (0.62)
Diurnal cortisol (AUC) (log nmol/L h)	
2 Years	13.19 (2.51)
Baseline	12.87 (2.69)
Self-protection (baseline)	3.06 (0.78)
Loneliness (baseline)	1.13 (2.59)
Number of chronic health problems (baseline)	2.24 (1.61)
Age	71.61 (4.50)
Male (%)	49.2
Socioeconomic status	3.30 (1.07)
Education (baseline) [b]	2.09 (1.09)
Yearly family income (baseline) [c]	1.57 (1.23)
Perceived socioeconomic status (baseline)	6.25 (1.71)
Smoking (baseline) (%)	8.2
Body-mass-index (baseline)	25.54 (3.69)

Note. [a] Mean and standard deviation (SD) are presented for continuous variables. [b] Education was indexed as 0 = no education, 1 = high school, 2 = trade, 3 = masters or doctorate. [c] Income was indexed as 0 = less than $17,000, 1 = up to $34,000, 2 = up to $51,000, 3 = up to $68,000, 4 = up to $85,000, 5 = more than $85,000

Table 6

Zero-Order Correlations Between Main Constructs.

	1	2	3	4	5	6
1. C-reactive protein (6-year)						
2. Cortisol volume (2-year)	.29**					
3. Cortisol volume (baseline)	.23**	.31**				
4. Perceived stress (2-year)	.12	.00	.00			
5. Perceived stress (baseline)	-.05	-.05	-.09	.66**		
6. Loneliness (baseline)	.04	-.02	-.06	.21*	.29**	
7. Self-protection (baseline)	-.21*	-.11	-.07	-.27**	-.28**	-.13

Note. $^{*}p \leq .05$. $^{**}p \leq .01$.

Table 7

Hierarchical Regression Analyses Predicting Levels of C-Reactive Protein After 6 Years by Baseline Levels of Loneliness and Health-Related Self-Protection.

	6-Year levels of C-reactive protein	
Baseline predictors	R^2	β
Main effects		
Loneliness	.00	.01
Self-protection	.02	-.13
Interaction		
Loneliness X self-protection	.04*	-.21*

* $p \leq .05$. ** $p \leq .01$. Results were controlled for age, sex, socioeconomic status, chronic health problems, smoking, and body-mass-index. R^2 values represent the unique proportion of variance explained in each step of analyses. β represents standardized regression coefficient in each step of analyses.

covariates or main effects were significantly associated with 6-year levels of CRP, *F*s(1, 113) < 2.14, R^2*s* < .01, *ps* > .10, except for smoking, $F(1, 113) = 10.62$, $R^2 = .08$, $p < .01$. Participants who smoked at baseline exhibited higher 6-year levels of CRP than their non-smoking counterparts, $\beta = .30$, $p < .01$. Of importance, the second step of the analysis demonstrated a significant two-way interaction effect between loneliness and self-protection on 6-year levels of CRP, $F(1, 112) = 5.54$, $p = .02$.

To illustrate the significant interaction effect, we plotted the association between health-related self-protection and 6-year levels of CRP for participants who experienced high (+1 SD) and low (-1 SD) baseline levels of loneliness (46). As depicted in Figure 7, baseline levels of self-protection were significantly associated with lower levels of CRP after 6 years among lonely participants, $\beta = -.42$, $p < .01$, but not among their non-lonely counterparts, $\beta = .13$, $p = .38$. Consistent with our hypotheses, these findings suggest that to the extent participants experienced higher baseline levels of loneliness, health-related self-protection was increasingly associated with lower 6-year levels of CRP.

Next, we examined whether health-related self-protection would also be associated with 2-year reductions of perceived stress and diurnal cortisol secretion among lonely (but not non-lonely) participants. To this end, we repeated the previously reported regression analysis; this time predicting 2-year changes in perceived stress and diurnal cortisol levels as the outcome variables.

The results of the analyses are reported in Table 8. The main effects of loneliness and self-protection as well as the covariates were unrelated to the outcome variables, *F*s(1, 113) < 1.82, R^2s < .01, *p*s > .05, except for SES, $F(1, 113) = 7.90$, $R^2 = .06$, $p < .01$, and BMI, $F(1, 113) = 3.97$, $R^2 = .03$, $p < .05$. Participants who reported a higher SES, $\beta =$

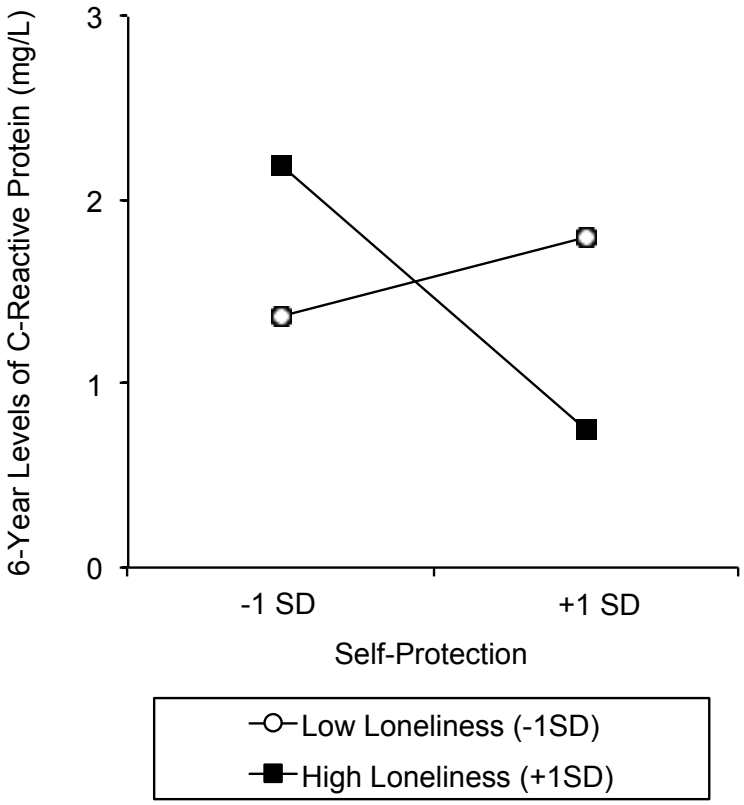


Figure 7 Associations between baseline levels of health-related self-protection and 6-year levels of C-reactive protein, separately for participants who did and did not feel lonely at baseline. Results were plotted one standard deviation above and below the sample means of self-protection (*SD* = .78) and loneliness (*SD* = 2.59).

Table 8

Hierarchical Regression Analysis Predicting 2-Year Changes in Perceived Stress and Cortisol Volume by Baseline Levels of Loneliness and Health-Related Self-Protection.

	2-Year changes in perceived stress		2-Year changes in cortisol volume	
Baseline predictors	R^2	β	R^2	β
Main effects				
Loneliness	.00	-.03	.00	.07
Self-protection	.01	-.13	.01	-.08
Interaction				
Loneliness X self-protection	.03*	-.19*	.03*	-.18*

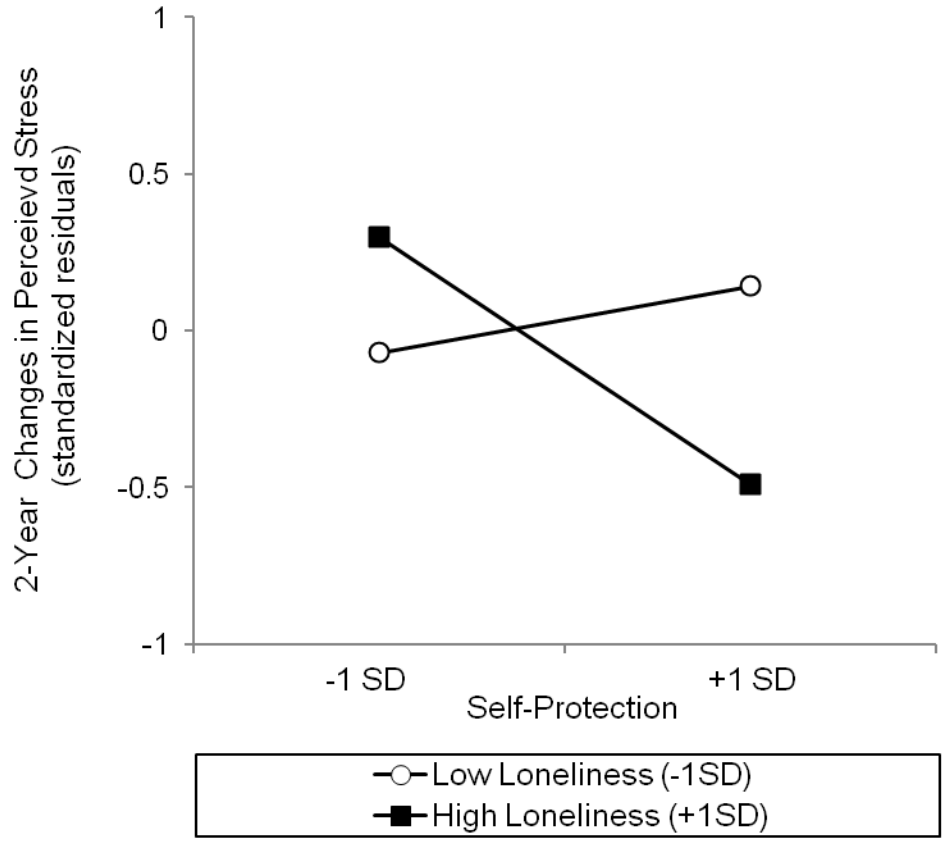
* $p \leq .05$. ** $p \leq .01$. Results were controlled for age, sex, socioeconomic status, chronic health problems, smoking, and body-mass-index. R^2 values represent the unique proportion of variance explained in each step of analyses. β represents standardized regression coefficient in each step of analyses.

.28, $p < .01$, or a higher BMI, $\beta = .19$, $p < .05$, exhibited larger 2-year increases in diurnal cortisol secretion. Moreover, similar to the previous analysis, the second step of the analyses revealed significant two-way interactions between loneliness and self-protections in predicting 2-year changes in perceived stress, $F(1, 112) = 3.90$, $p = .05$, and 2-year changes in diurnal cortisol secretion, $F(1, 112) = 4.03$, $p < .05$.

Figure 8 illustrates the significant two-way interactions by plotting the associations between health-related self-protection and 2-year changes in perceived stress (upper panel), and 2-year changes in diurnal cortisol secretion (lower panel) for participants who experienced high (+1 SD) and low (-1 SD) levels of loneliness. The obtained pattern of results indicated that self-protection significantly predicted 2-year decreases in both perceived stress, $\beta = -.39$, $p = .02$, and cortisol secretion, $\beta = -.34$, $p = .03$, but only among lonely participants. By contrast, no significant associations between self-protection and changes in perceived stress or diurnal cortisol secretion were observed among participants who did not feel lonely, βs $< .15$, $ps > .33$. These findings demonstrate that to the extent participants felt lonely, health-related self-protection was increasingly associated with smaller increases over time in perceived stress and diurnal cortisol secretion.

Finally, we examined whether self-protection buffered against loneliness-related elevations in CRP at 6-years through preventing earlier increases in perceived stress and cortisol output. To this end, we repeated the previously reported analyses for predicting 6-year levels of CRP by additionally incorporating 2-year changes in perceived stress and cortisol secretion as potential multiple mediators (using the "indirect SPSS macro") (44).

The results of the mediation analyses are illustrated in Figure 9. Consistent with


1
0.5
0
-0.5
-1
2-Year Changes in Perceievd Stress
(standardized residuals)
-1 SD
+1 SD
Self-Protection
Low Loneliness (-1SD)
High Loneliness (+1SD)

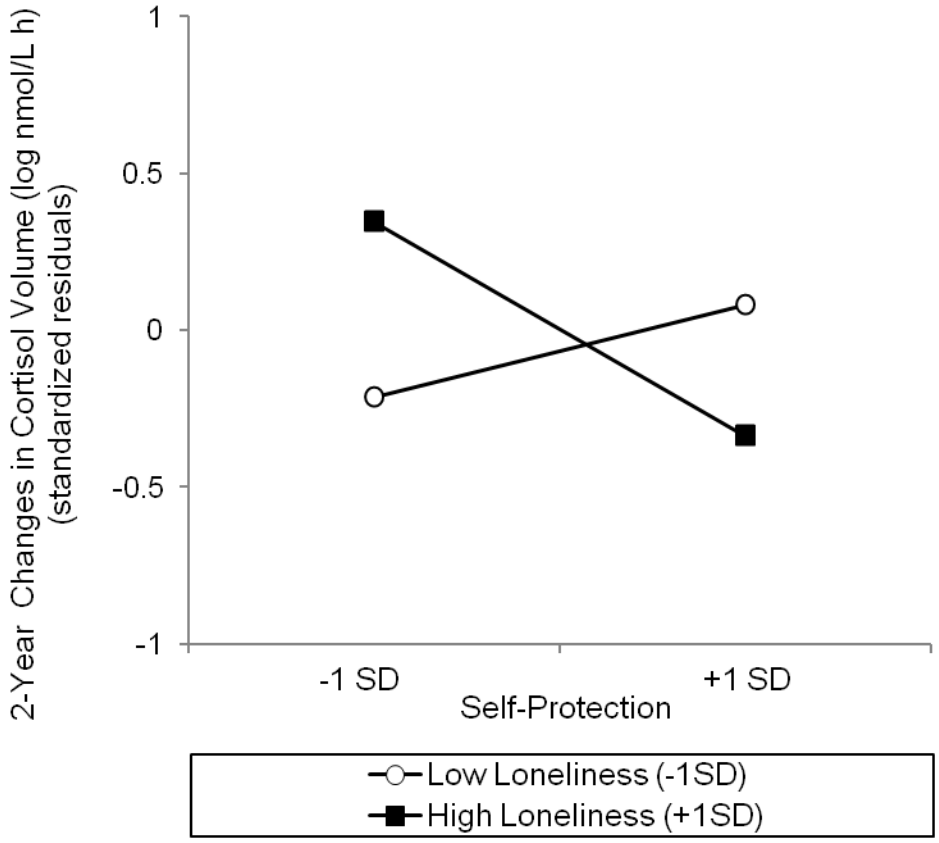
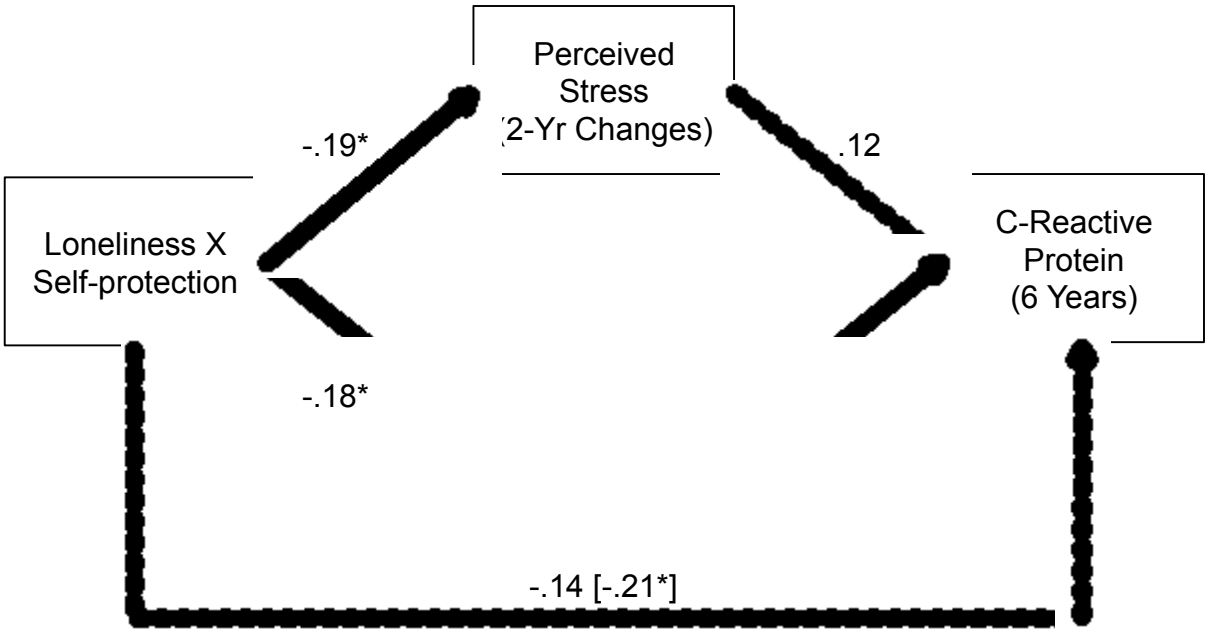


Figure 8 Associations between baseline levels of health-related self-protection and 2-year changes in perceived stress and cortisol volume, separately for participants who did and did not feel lonely at baseline. Results were plotted one standard deviation above and below the sample means of self-protection (*SD* = .78) and loneliness (*SD* = 2.59).



Note. Values represent standardized regression coefficients. Solid lines indicate significant paths in the mediation analysis. Bootstrap analyses showed that 2-yr changes in diurnal cortisol volume exerted a significant indirect effect on the association between the interaction of loneliness and health-related self-protection with 6-yr levels of CRP.

Figure 9 Multiple mediation model examining whether 2-year changes in perceived stress and cortisol volume mediate the interaction effect between health-related self-protection and loneliness on 6-year levels of C-reactive protein.

above reported results, the interaction between health-related self-protection and loneliness significantly contributed to 2-year changes in perceived stress and diurnal cortisol secretion. In addition, 2-year increases in diurnal cortisol secretion, $F(1, 110) = 8.04$, $\beta = .25$, $R^2 = .05$, $p < .01$, but not in perceived stress, $F(1, 110) = 1.92$, $\beta = .12$, $R^2 = .01$, $p = .17$, were associated with higher levels of CRP after 6 years. Moreover, the significant interaction effect between self-protection and loneliness on 6-year levels of CRP was rendered non-significant, $F(1, 110) = 2.52$, $\beta = -.14$, $R^2 = .02$, $p > .10$, if both mediators were included in the analysis. Finally, the bootstrap analysis clarified which of the included variables showed a significant indirect (i.e., mediation) effect. In this regard, the results were consistent with a scenario in which 2-year changes in cortisol secretion underlied the interaction between self-protection and loneliness in predicting lower 6-year levels of CRP (95% BCI [-.161, -.001]). By contrast, the results of the analyses showed that changes in perceived stress were not responsible for the observed interaction's capacity to predict 6-year levels of CRP (95% BCI [-.116, .005]). Together, this pattern of results lends support to a scenario in which health-related self-protection contributed to a decline of lonely participants' diurnal cortisol section over 2 years, which in turn predicted lower levels of CRP after 6 years of study.

Discussion

The results from this study suggest that self-protective strategies may ameliorate some of the psychobiological consequences of loneliness in older adulthood. More specifically, the findings showed that, among lonely older adults, the use of self-protection buffered against 2-year increases in perceived stress and cortisol secretion, and was associated with lower 6-year levels of C-reactive protein (CRP). In addition,

mediation analyses showed that the obtained differences in CRP were partly attributable to self-protection's influence on cortisol output during the initial two years of the study. No effects of self-protection were observed among older adults who did not feel lonely.

This pattern of findings suggests that among lonely older adults, the capacity to cope internally with common health threats is a contributor to later psychobiological outcomes. Those lonely older adults who reframe problematic health circumstances positively and do not blame themselves for health-related threats can prevent increases in perceived stress and diurnal cortisol secretion. Further, by reducing cortisol output, self-protective strategies seem to be able to forecast lower CRP four years later. By contrast, lonely older adults who do not use self-protective strategies to manage their health threats are at risk of feeling greater stress over time, and showing higher levels of biomarkers associated with disease and disability. Finally, the results imply that using health-related self-protection is less influential among older adults who do not feel lonely, presumably because they can rely on adaptive socio-emotional processes that facilitate the management of age-normative health threats. These conclusions are consistent with life-span developmental theories of socio-emotional and motivational functioning, which postulate that adaptive control striving (Heckhausen et al., 2010) and emotionally gratifying social relationships (Carstensen et al., 1999) can foster pathways to successful aging.

Moreover, our findings contribute to knowledge about how psychosocial factors modulate crosstalk between glucocorticoids and inflammation (Chrousos & Gold, 1992; Cole et al., 2007; Miller et al., 2008). We found that self-protective strategies forecasted a reduction in cortisol secretion two years later. This reduced cortisol output was, in turn,

associated with lower CRP four years later. At first blush, these findings may seem difficult to reconcile, as cortisol generally has anti-inflammatory properties. However, sustained exposure to high levels of cortisol may render innate immune cells partially resistant to glucocorticoid inhibition, allowing inflammation to escape normal regulatory controls (Miller et al., 2008; Raison & Miller, 2003). Indeed, evidence of glucocorticoid resistance has emerged in functional genomic studies of lonely individuals (Cole et al., 2007).

Overall, the study's findings have important implications for understanding pathways to successful aging. Given that they can accentuate the impact of age-related challenges, feelings of loneliness are likely to increase older adults' levels of stress and its attendant biological sequelae (Hawkley & Cacioppo, 2007). In such circumstances, the emotional benefits derived from the use of self-protective strategies can protect emotional resources and free up time and energy for the pursuit of other important (health-related) activities (Heckhausen et al., 2010). Through this mechanism, self-protective processes are likely to buffer against the psychobiological consequences of loneliness. Given inflammation's role in the pathophysiology of several major diseases (Hotamisligil, 2006; Scrivo, Vasile, Bartosiewicz, & Valesini, 2011), this mechanism may have widespread benefits for older adults' long-term health.

In addition, the findings contribute to the literature on socio-emotional functioning. Although there is evidence that loneliness can affect mental and physical health, and the biological processes that underlie them, such effects have not been found consistently across studies (Cacioppo et al., 2002b; Doane & Adam, 2010; Hawkley et al., 2006; Miller, Kemeny, Taylor, Cole, & Visscher, 1997). Our research provides an

explanation for these inconsistent findings by suggesting that variability in the effects of loneliness can be associated with individual differences in self-protective coping. This implies that socio-emotional problems can interact with self-regulation processes in predicting health-related outcomes, and adaptive self-regulation becomes particularly important for promoting successful aging among individuals who feel lonely.

Finally, the present research may have some implications for clinical treatment. Given that the use of adaptive control strategies can be improved in therapy (Nathan, 2007), interventions should aim at teaching lonely older adults how to engage in self-protective control strategies. The implementation of such programs could reduce psychological stress among lonely older adults, and perhaps as a result dampen their cortisol output and systemic inflammation.

There are limitations to this study. First, we measured levels of CRP only at 6-year follow-up and did not include earlier assessments of CRP. This implies that the results reporting effects on 6-year levels of CRP need to be interpreted cautiously as they are based on longitudinal, but not prospective, analyses. As a consequence, it is possible that individual differences in CRP levels were already present at baseline and could have contributed to participants' coping responses and feelings of loneliness. Moreover, it prevents us from definitively concluding that psychological factors were associated with CRP because they triggered changes in cortisol level. However, there are several reasons to believe that this limitation does not seriously compromise the overall interpretation of findings. First, baseline measures of psychological variables predicted reliable 2-yr changes in diurnal cortisol secretion, which were associated with 6-yr levels of CRP. Second, it seems unlikely that CRP has the potential to influence coping responses, but

only does so among lonely individuals. Third, our empirical results are consistent with general models of health suggesting that the association between psychological risk factors and immune function can be conferred through stress hormones, such as cortisol (Kiecolt-Glaser et al., 2002; Miller et al., 2009). Nonetheless, we recommend that future studies should assess psychological, endocrine, and immune variables over multiple time points to substantiate our findings.

Second, we focused in our analysis on the overall volume of cortisol secretion because we reasoned that such cumulative concentrations of cortisol output across day are particularly likely to exert reliable associations with psychological and immune variables. However, other research has demonstrated that loneliness is also associated with a flattened diurnal cortisol slope and an increased cortisol awakening response (Kiecolt-Glaser et al., 2002). Follow-up analyses of our data showed that this was not the case for our sample, as changes in these alternative cortisol indices were neither associated with the interaction between loneliness and self-protection nor with 6-year levels of systematic inflammation. These findings may imply that associations between cortisol and psychological or immune variables can be underestimated if research analyzes only a portion, or the shape, of the diurnal cortisol rhythm.

Third, although our analysis included a number of covariates, there may be underlying personality constructs that could have influenced older adults' feelings of loneliness and coping responses, and through this process caused the observed effects on participants' biological disturbances. A trait that could produce such effects is neuroticism (Gunthert, Cohen, & Armeli, 1999; Portella et al., 2005). We note that our study included a baseline measure of neuroticism (Costa & McCrae, 1992), and

subsequently conducted follow-up analyses revealed that all reported effects remained significant if individual differences in neuroticism were taken into account.

Finally, our research did not examine the complete process that could be associated with the adaptive management of age-related challenges. In fact, our theoretical model (Wrosch et al., 2004, 2006) would suggest that the observed effects of psychological predictors on inflammation could contribute to a higher likelihood of developing subsequent physical disease (Miller et al., 2009). Although, we could not examine the physical health changes derived from excessive systemic inflammation in the present study, analyses of subsequent waves of our study may shed light on this possibility. We therefore feel that future research on the psychobiological pathways to successful aging is warranted and has the potential to contribute to maintaining older adults' quality of life.

GENERAL DISCUSSION

Summary of Research Findings

The overall objective of this dissertation was to shed light on the longitudinal processes linking adaptive self-regulation with age-related challenges, psychological, distress, biological dysregulation, and physical illness over time. To this end, we conducted three different studies to examine the adaptive roles of specific self-regulation strategies, including behavioral engagement (e.g., physical activity in Study 1 and restorative sleep in Study 2) and cognitive disengagement (e.g., self-protective attributions in Study 3), in predicting older adults' emotional, biological, and physical health trajectories. On a more general level, we expected that the use of behavioral and cognitive processes that are functionally adjusted to a person's opportunities and constraints could have the potential to buffer normative declines in older adults' psychological, biological and physical health. Further, the health-related benefits derived from adaptive self-regulation were expected to be particularly pronounced among "at-risk" older adults who are vulnerable to developing a disease.

The studies' results strongly supported these hypotheses. Study 1 identified the engagement in physical activity as an effective behavioral mechanism that can counteract chronic perceptions of stress. In particular, the study's findings demonstrated that older adults' engagement in physical activity forecasted a reduction of stress perceptions over two years and fewer physical health problems over four years among participants who perceived high levels of stress at baseline, but to a lesser extent among their counterparts who perceived low levels of stress at baseline. Importantly, mediation analyses indicated that the long-term physical health benefits among highly stressed individuals were

statistically explained by a short-term reduction of stress perceptions. These findings suggest that physical activity can exert benefits on older adults' physical health by preventing chronically high perceptions of stress.

Study 2 attempted to uncover behavioral self-regulation strategies that can also be maintained in the event of serious age-related declines that may undermine the possibility of engaging in more active health behaviors. In particular, Study 2 was designed to investigate the long-term associations between reports of sleep duration and diurnal cortisol secretion in older adulthood. Results indicated that older adults experienced increases in diurnal cortisol secretion over time, and that this effect was forecasted by individual differences in sleep duration. More specifically, higher levels of and increases in sleep duration buffered long-term elevations of participants' diurnal cortisol secretion. By contrast, lower levels of and declines in sleep duration predicted a steep increase in cortisol secretion over time. In addition, reversed analyses showed that levels of or changes in diurnal cortisol secretion did not predict changes in sleep duration over time. These findings suggest that longer sleep represents a useful behavioral strategy to counteract age-associated increases in diurnal cortisol secretion over time.

While the former two studies focused on the adaptive value of specific types of behavioral self-regulation in adjusting to age-related challenges, Study 3 demonstrated that cognitive self-regulation aimed at disengaging from problems can be beneficial in the face of certain challenges that can hardly be addressed by a person's behavioral coping efforts. In this regard, we showed that feelings of loneliness may be one such problem that may best be dealt with through a person's cognitive efforts to internally adjust personal standards and perceptions to match current life circumstances. In particular,

Study 3 found that, among lonely older adults, internal self-protection predicted a reduction of psychological stress and diurnal cortisol volume over two years, and lower levels of CRP after 6 years. These effects were not obtained among non-lonely older adults. In addition, mediation analyses revealed that the observed short-term reduction of cortisol secretion statistically explained the effects of self-protective control on lonely participants' lower long-term levels of systematic inflammation. These findings suggest that older adults'stress-related psychological and biological disturbances deriving from feelings of loneliness can be prevented by the use of internal self-protection to cope with emerging health threats.

While the findings from the three reported studies are consistent with the current body of literature on the adaptive value of self-regulation for managing adverse challenges in older adulthood (e.g., Baltes & Baltes, 1990; Carstensen et al., 1999; Hall et al., 2010; Hamilton et al., 2007; Heckhausen & Schulz, 1995), they also extend previous research lines and contribute some meaningful and new insights to this area of research. First, in contrast to prior work, the reported studies are all based on longitudinal data, which allowed us to obtain some information about the direction of effects. In this regard, our results indicate that ameliorations in older adults' emotional, biological, and physical health can, in fact, be driven by specific health behaviors and self-protective cognitions. Second, the present research explains some of the observed inconsistencies in the literature with regards to the effects of behavioral (i.e., physical activity and restorative sleep) and cognitive (e.g., self-protective coping) self-regulation on older adults' psychological, biological, and physical health. In this respect, our findings suggest that variability in the effects of adaptive self-regulation may in part be due to the presence of

additional risk factors for health. In particular, we showed that behavioral and cognitive processes can interact with health-related risk factors in predicting positive outcomes, and that adaptive self-regulation becomes particularly important for promoting successful aging among "at risk" individuals. Third, our results expand previous research by demonstrating that adaptive self-regulation can attenuate the negative effects of adverse challenges on psychological distress and biological dysregulation, and that the resulting improvements in a person's psychological and biological processes stemming from the engagement in adaptive self-regulation can confer subsequent health benefits. In this respect, our research lends empirical support to the assumption that changes in cortisol patterns derived from psychological stressors can act as a reliable mediator in the relationship between psychological and health-related variables (Fig. 1, preliminary evidence for the association a-c-[d]). Fourth, the present research adds a novel dimension to the knowledge of adaptive behavioral and cognitive self-regulation by examining not only between-person changes in the engagement of self-regulation over time, but also individual differences in within-person changes in the engagement of self-regulation over time. In this regard, our findings indicate that the engagement in behavioral self-regulation (i.e., adaptive sleep patterns) is likely to fluctuate over time, and that inter-individual differences in intra-individual changes in the maintenance of such self-regulation strategies (e.g., increases versus declines in sleep duration) can significantly affect a person's health status. Fifth, while previous research was predominantly concerned with the effects of behavioral tendencies and cognitions related to goal adjustment (e.g., Heckhausen et al., 2001; Wrosch & Heckhausen, 1999, 2002; Wrosch et al., 2000, 2002), the present research has refined this approach by examining the adaptive

value of concrete behaviors (as compared to behavioral tendencies) and self-protective cognitions across different situations. Given that behavioral tendencies may not always translate into matching behaviors, we think that assessing the frequency of specific behaviors lends additional validity to the interpretation of findings. Finally, the incorporation of additional psychological (e.g., perceived stress or feelings of loneliness), biological (e.g., cortisol secretion or biological markers of systematic inflammation) and physical (e.g., daily physical symptoms) variables into the different studies allowed us to improve our knowledge of the complex interplay between health-related processes and adaptive self-regulation in predicting successful aging.

Hence, the various strengths inherent to our empirical approach enabled us to comprehensively assess the different adaptive roles played by behavioral and cognitive self-regulation in preventing the adverse effects of age-related challenges on older adults' psychological, biological, and physical health outcomes. By unraveling the particular circumstances in which the use of specific health behaviors or self-protective coping can be more or less effective to protect an older adult's resources, this dissertation makes a unique contribution to the growing research on self-regulation and elaborates extant theories of lifespan development. The various advantages associated with the present research (e.g., longitudinal assessments, examination of inter- and intra-individual change, and inclusion of actual behaviors) are likely to guide future studies that attempt to elucidate the effectiveness of adaptive self-regulation across the life-span.

Theoretical Contributions

The results from the present research contribute to life-span theories of successful

aging (e.g., Baltes & Baltes, 1990; Carstensen et al., 1999; Heckhausen & Schulz, 1995) by confirming some of their predictions and extending these theories in different ways. In this regard, the studies' findings illustrate that increasing age-related challenges can set in motion a downward spiral characterized by psychological distress and biological dysregulations, which in turn may instigate further deteriorations in a person's long-term health outcomes. Of importance, they also demonstrate that such an adverse cascade of psychological and health-related problems may not take place per se, but only if older adults are unable to adequately address some of the pathogenic processes associated with emerging age-related challenges. These observations are consistent with a previously proposed model on the importance of adaptive self-regulation for managing age-related health declines (Wrosch et al., 2004, 2006).

In addition, our findings reveal the particular circumstances in which the use of specific cognitive and behavioral self-regulation strategies can be especially effective for counteracting age-related losses. In particular, we demonstrate that the engagement in proactive health behaviors can become paramount for managing psychological and biological losses when opportunities for actively controlling a problem are present (e.g., perceived stress and cortisol dysregulation). However, if a person encounters reduced opportunities for actively controlling an adverse challenge (e.g., socio-emotional problems), a self-protective cognitive coping modus that fosters acceptance of, and disengagement from, the specific problem may be the optimal strategy. This implies that in order for older adults to successfully address a wide range of different threats, they are required to continuously adjust and re-adjust to their shifting circumstances by switching between self-regulation strategies that promote adaptive changes in external conditions

versus internal perceptions. This interpretation is consistent with control theories which posit that a balance between persistent goal pursuit and flexible goal adjustment is crucial in adapting to the obstacles and crises of personal development (e.g., Brandtstaedter & Renner, 1990; Carver & Scheier, 1990; Heckhausen & Schulz, 1995; Heckhausen, Wrosch, & Schulz, 2010; Rothbaum, Weisz, & Snyder, 1982). Moreover, our findings also lend additional support to prior studies documenting that the adaptive value of behavioral engagement and cognitive disengagement strategies depends on the availability of opportunities for actively controlling a problem (e.g., Heckhausen & Brim, 1997; Heckhausen & Schulz, 1995; Wrosch et al., 2000, 2006).

The finding that older adults draw on personal resources to adjust to a wide range of adverse challenges, including psychological (i.e., loneliness and stress) and biological problems (i.e., cortisol dysregulations), illustrates the remarkable adaptation that takes place in day-to-day interactions between older individuals and their environment. While the accumulation of adverse challenges and losses is an inevitable part of the aging process, it appears that older adults can preserve considerably high levels of emotional, biological, and physical health if they selectively invest behavioral and cognitive efforts to deal with age-related threats. This conclusion is consistent with research showing that there is much variability in the aging process, and that a substantial amount of older adults can maintain well-being and functional autonomy, despite the accumulation of irreversible problems and developmental constraints (Charles, 2010; Heckhausen et al., 1989; Heckhausen & Schulz, 1995; Rowe & Kahn, 1998).

Moreover, the current findings make clear that behavioral changes in older adulthood do not simply derive from efforts to cope with loss, but rather reflect active

adaptation to particular circumstances, social niches, and environments, which are inevitably shaped by the perception of time (Carstensen et al., 1999). According to Socioemotional Selectivity Theory, the perception of time, especially the recognition of approaching endings, is thought to exert a considerable influence on the selection and pursuit of goals (Carstensen, 1991). More specifically, in older adulthood, when future time is constrained, individuals become increasingly motivated to shift their priorities toward emotionally gratifying goals (e.g., maintaining a close relationship), in an attempt to successfully regulate the emotional turmoil associated with age-appropriate developmental concerns (Baltes & Carstensen, 1998; Carstensen, 1995, 2006). In the present research, the proposed benefits of selecting emotionally meaningful goals were manifest in so far as older adults who perceived themselves as socially embedded were less likely to experience a cascade of stress-related problems, possibly because their social ties facilitated the emotion regulation of emerging age-related challenges. In contrast, among lonely older adults who were unable to draw on socio-emotional resources to regulate the distress deriving from age-related challenges, the health-compromising consequences of common threats were found to be particularly strong. These findings are consistent with prior research demonstrating that the adaptive value of emotional closeness with significant others becomes increasingly important in older adulthood (Carstensen, 1992; Carstensen, Issacowitz, & Charles, 1999). In fact, the endorsement of socio-emotional goals has been found to promote older adults' emotional regulation and the resulting benefits on psychological well-being and physical health (Löckenhoff & Carstensen, 2004; for a review see Watkins, 1997).

In addition, these findings are the fist to demonstrate that internal self-regulation becomes particularly important for promoting successful aging if socio-emotional functioning breaks down. In this respect, we show that lonely older adults who reframe problematic health circumstances positively and do not blame themselves for health-related threats can prevent increases in stress-related psychological and biological problems. By contrast, lonely older adults who do not use such self-protective strategies to manage their health threats are at increased risk of experiencing greater stress and higher levels of biomarkers associated with disease and disability. This implies that socio-emotional problems can interact with internal self-regulation in predicting health-related outcomes. In this regard, our research makes a valuable contribution to the literature, as it empirically combines central ideas from life-span developmental theories of socio-emotional and motivational functioning, which state that adaptive control striving (Heckhausen et al., 2010) and emotionally gratifying social relationships (Carstensen et al., 1999) can foster pathways to successful aging.

Finally, the results also contribute novel insights to general models of stress and disease (e.g., Cohen et al., 1997; Kiecolt-Glaser et al., 2002; Miller, Chen, & Cole, 2009), as they uncover the specific pathways through which psychological risk factors "get under the skin" to influence long-term health outcomes. In general, these models converge upon the idea that cortisol represents a primary mechanism through which chronic stressors get inside the body to influence disease. In addition, some of them also specify that stress can give rise to disease by increasing levels of cortisol secretion, thereby exposing bodily tissues to elevated concentrations of the hormone. If prolonged, this process is believed to lead to tissue damage and subsequent disturbances in various

biological systems, such as cytokine-mediated inflammation, which in turn may contribute to a wide spectrum of health problems and disease (Miller et al., 2002; Segerstom & Miller, 2004). Our findings provide empirical evidence for these theoretical assumptions by demonstrating that stressors predispose a person to a broad array of psychological, biological, and physical health problems, and that dysregulation of cortisol secretion serves as a major mechanism through which stressors exert a considerable impact on immune function.

However, it has to be recognized that the extant research on the associations between psychological stressors, cortisol output, immune functioning, and physical health has yielded mixed results. While some research suggests that psychological stress does not always lead to dysregulation of a person's cortisol output (Burke, Davis, Otte, & Mohr, 2005; McEwen, 2000; Miller et al., 2002), other studies have struggled with demonstrating reliable effects of cortisol secretion on physical health problems (Wrosch et al., 2008) or immune functioning (Miller, Cohen, Pressman, Barkin, Rabin, & Treanor, 2004). In this regard, our findings suggest that some of the observed inconsistencies in the literature may be due to differences in individuals' self-regulatory capacities. In particular, the studies' findings point out that individuals' engagement in adaptive self-regulation can alleviate the psychological and biological problems deriving from adverse challenges (i.e., feelings of loneliness and perceived stress), and that the resulting amelioration of chronic stress and cortisol output can bring about better long-term health outcomes (i.e., improved immune functioning and physical health). The identification of this self-regulatory pathway implies that the linkage between stress and health is more complex than previously thought, and that theoretical models would benefit from taking

into account the potential influence of self-regulation on the interplay between psychological, biological, and physical factors.

Clinical Implications

The finding that adaptive self-regulation plays a key role in adjusting to age-related losses in general, and especially among certain high-risk groups of older adults has far-reaching implications for clinical practice.

If specific cognitive and behavioral self-regulation strategies can ameliorate declines in older adults' emotional, biological, and physical health, interventions should teach older adults' how to use specific behavioral and cognitive strategies. Since these strategies are most effective if they are functionally-adjusted to a person's particular circumstances, individuals would benefit from learning how to recognize whether behavioral engagement or cognitive disengagement may be the optimal strategy for counteracting a specific problem. In addition to identifying and selecting functionally-adapted self-regulation strategies, older adults also need to acquire the necessary skills to translate the selected strategies into practice. In circumstances in which opportunities for actively overcoming an adverse challenge are present, it would be important to assist older adults in adaptively engaging in and maintaining proactive behaviors, including physical activity and adaptive sleep (e.g., for non-pharmacological sleep interventions, see Morin, Culber, & Schwartz; for physical activity interventions, see King, Rejeski, Buchner, 1998). However, in the face of challenges that can hardly be addressed by a person's active coping efforts (e.g., chronic health conditions or functional decline), emphasis should be put on promoting the adaptive use of cognitive coping strategies that foster goal disengagement, such as positive re-appraisal and external attributions of

failure experiences (e.g., for interventions on secondary coping**,** see Heckhausen & Dweck, 1998). Teaching individuals how to identify problems, think through solutions, and finally act on them may help them recognize and select the optimal strategy, and additionally provide the required skills to take actions (e.g., Nathan & Gorman, 1998). In the light of specific psychological challenges, such as socio-emotional problems or psychological distress, it could also be beneficial to include stress management techniques and emotion-enhancing strategies that ameliorate confidence, reduce perceived stress, or tackle increasing loneliness in the face of dwindling social networks with advancing age. Thus, for interventions to be effective they need to integrate a larger set of cognitive, behavioral, and educational tools (e.g., goal-setting, self-monitoring, behavioral skill training, feedback, support, and relapse-prevention training) that is tailored to the individuals' particular needs.

Given that certain psychological (i.e., feelings of loneliness or perceived stress) and biological (i.e., increased cortisol secretion or inflammatory markers) threats may be important precursors to a person's long-term physical health problems, it could also be useful to screen older adults for specific psychological and biological risk factors as part of an early identification program. Identifying high-risk groups of older adults before they develop sever diseases would allow practitioners to determine follow-up procedures in a way that ameliorates the course of disease or, in some cases, even prevents the disease process from developing. In addition, early screenings of health-related risk factors coupled with customized risk education may be effective in increasing individuals' motivation to adopt preventative health-related strategies. Indeed, it has been shown that individuals' risk perception can predict the engagement in actual health

promoting behaviors (e.g., Brewer, Chapman, Gibbons, Gerrard, McCaul, & Weinstein, 2007).

Although our findings suggest that "at risk" older adults may be in particular need of adaptive self-regulation for maintaining adequate levels of functioning, it has to be noted that – independent of health-related risk factors - the benefits of adaptive self-regulation may increase with advancing age when unavoidable losses of resources are likely to impose an increasing burden on the elderly at large. Based on these considerations, it is important that programs aimed at improving older adults' self-regulatory capacities do not only target high-risk groups of older adults but also take into consideration the needs of the elderly community in general. Given the large and increasing percentage of older adults in the general population, such a community-based approach could be an important and cost-effective alternative to the limited health services available to the elderly. Offering community-based interventions that include psycho-educational and cognitive-behavioral tools to target changes in health-related cognitions and behaviors may equip older adults with the necessary skills to adequately address a variety of different challenges.

Hence, by empowering older adults to draw on their personal resources to adequately manage emerging age-related challenges, they may become capable of successfully avoiding a downward spiral characterized by maladaptive control strategies, psychological distress, biological dysregulations, and physical health problems. As such, the implementation of a comprehensive community-based approach may not only be promising for encouraging older adults to play an active role in their own health care, but also for significantly reducing the costs for public health services.

Limitations and Future Research

While the present studies answered important questions with regard to the self-regulation of age-related challenges in older adulthood, they are also associated with limitations that need to be addressed in future research.

The present research suggests that cognitive efforts that foster goal disengagement, such as self-protective attributions of failure experiences, may be well-suited to protect emotional, biological, and physical health in the light of uncontrollable problems (e.g., feelings of loneliness). By contrast, engaging in specific proactive strategies may be particularly adaptive for a person's overall health if a problem appears to be controllable through a person's behavioral coping efforts (e.g., perceived stress). This implies that in order for older adults to select the appropriate coping strategy, they need to determine whether a specific problem is controllable or uncontrollable through active coping efforts. However, while in some cases it may be quiet obvious that a problem cannot be actively overcome (e.g., widowhood), there are a number of other adverse challenges for which the level of controllability may be less clear (e.g., psychological problems). For instance, some problems may be "somewhat controllable" and "somewhat uncontrollable", and others may shift from being controllable towards being uncontrollable as individuals advance in age (e.g., slowly progressing functional problems). This implies that it could be possible that some problems may be manageable through behavioral engagement and cognitive disengagement. While we recognize that perceptions of stress and feelings of loneliness could be such problems that involve uncontrollable and controllable parts, follow-up analyses of the reported studies demonstrated that behavioral engagement (i.e., physical activity and sleep) did not buffer

the health-compromising effects derived from loneliness on immune function, and cognitive disengagement (i.e., positive re-appraisal) did not alleviate the negative consequences of perceived stress. Nonetheless, because we think that it is conceptually possible that some problems may respond to both behavioral and cognitive adjustment strategies, we suggest that future research should incorporate measures of perceived and objective controllability of problems and track individuals across time and different situations to shed further light on this issue.

Further, while the present research demonstrates that goal disengagement through self-protective attributions can lead to better psychological and biological functioning among lonely individuals, it may also be possible that disengaging from unattainable goals may not always result in positive outcomes (Wrosch et al., 2003). If individuals let go from goals that are no longer attainable but have difficulty with refocusing their resources on the pursuit of other meaningful and more realistic goals, they could also develop negative emotions, such as feelings of aimlessness or emptiness (Carver & Scheier, 1999; Wrosch et al., 2003). Given these considerations, we raise the possibility that lonely older adults who disengaged from uncontrollable health problems via self-protective cognitions may have experienced health-related benefits because this process enabled them to redirect their energy and commitment towards valuable alternative goals. While this explanation would be consistent with prior research with older adults demonstrating that withdrawing energy and commitment from an unattainable goal may only be adaptive if individuals have the opportunity to engage with valuable alternative goals (Wrosch et al., 2003), such associations have not been found consistently (Wrosch, Amir, & Miller, 2011; Dunne, Wrosch, & Miller, 2011). Because the present research

was not designed to address these questions, we suggest that future research should thoroughly study the role played by goal re-engagement in self-regulatory processes.

In addition, the present studies demonstrate that reliable individual differences in both baseline levels (i.e., adaptive sleep patterns) and intra-individual changes in behavioral processes over time (i.e., increases versus declines in sleep duration) can be independently associated with health-related outcomes. While we did not find significant intra-individual changes in physical activities and self-protective cognitions, we suggest that future research on understanding the functions of self-regulation should carefully take into account the potential influences deriving from intra-individual changes over time because if such variations take place within a person they are likely to exert a considerable impact on a persons' health. In this regard, it may also be useful to carefully uncover the key variables that can explain some of the inter- and intra-individual variability in the capacity to adequately regulate adverse challenges over time. Identifying the specific psychological variables (e.g., social support or perceptions of control) that foster the adoption and maintenance of adaptive behavioral (i.e., physical activity) and cognitive strategies (e.g., self-protective attributions) among the elderly in general, and particularly among "at risk" older adults could be helpful in this respect. Hence, by studying the issue of intra-individual changes in self-regulation capacities at large, as well as the processes that underlie not only baseline individual differences in self-regulation but also intra-individual changes in these differences over time, researchers may obtain important information that can be used to promote the adaptive use of these strategies in the elderly population.

While our research has contributed to the literature by uncovering the specific circumstances in which adaptive behavioral and cognitive self-regulation can be particularly well-suited to maintain emotional, biological and physical health, there are several issues that would benefit from further research. For example, future research endeavors should study whether the observed detrimental effects of increased cortisol secretion on subsequent levels of systemic inflammation can forecast the development of physical diseases in the long-run (Fig. 1, association a-c-d). In addition, it may also be meaningful to investigate whether such effects on physical health can trigger the emergence of additional age-related challenges (Fig. 1, feedback loops). In this context, it would also be important to address the development of more severe health problems (e.g., cancer, functional limitations, or mortality), and to include additional biological variables that are likely to play a major function in the association between cortisol secretion and physical health (e.g., biological markers of functioning of the immune, metabolic, or skeletal systems).

Given that comprehensively addressing these issues was beyond the scope of this dissertation, we suggest that future research should conduct fine-grained longitudinal studies to track older adults over a prolonged period of time and include additional self-regulation strategies (e.g., self-correction, or planning), as well as a wider range of self-report and objective measures of contextual, psychological, and physical health variables. We are confident that research along these lines has the potential to further illuminate the psychological, biological, physical biological pathways through which adaptive self-regulation processes contribute to healthy aging.

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Appendix A:
Consent Form

CONSENT FORM TO PARTICIPATE IN RESEARCH

This is to state that I agree to participate in a program of research being conducted by Dr. Carsten Wrosch of the Psychology Department of Concordia University.

A. PURPOSE

I have been informed that the purpose of the research is to study older adults' goal management, well-being, and health.

B. PROCEDURES

This research will involve a questionnaire and 15 salivary cortisol samples collected over the course of three typical days. A research assistant will go to the participant's home to administer part of a questionnaire on goal management, well-being and health, as well as to explain the saliva collection procedure. The rest of the questionnaire will be filled in by the participant while alone and should take approximately one hour to complete. The saliva collection will involve chewing a provided cotton swab for one minute before replacing it in its salivette. The saliva collection will be performed five times a day at specific times. The participant will receive phone calls from the research assistant to remind him/her to take a salivary cortisol sample. The participant will receive $50 for participating in the study. There should be no risks or discomfort involved in answering the questions or collecting the salivary cortisol samples. The participant's name will not be attached to the questionnaire, although the signatures and names on the consent forms will be collected and stored separately by the supervising professor. The participant is
free to refuse to answer any question that makes him or her uncomfortable answering.

B. CONDITIONS OF PARTICIPATION

- I understand that I am free to withdraw my consent and discontinue my participation at anytime without negative consequences. Even if I discontinue my participation, I will receive $50.
- I understand that my participation in this study is CONFIDENTIAL (i.e., the researcher will know, but will not disclose my identity)
- I understand that the data from this study might be published.

I HAVE CAREFULLY STUDIED THE ABOVE AND UNDERSTAND THIS AGREEMENT. I FREELY
CONSENT AND VOLUNTARILY AGREE TO PARTICIPATE IN THIS STUDY.

NAME (please print) _____

SIGNATURE _____

WITNESS SIGNATURE _____

DATE _____

Appendix B:
Daily Questionnaire (Wave 1)

#: _____

DAY 1

Date: _____

Please record the exact time when you took your saliva sample.

1st Saliva Sample: ***(Label: 1-1)***   I woke up at _____ h _____ min
2nd Saliva Sample: ***(Label: 1-2)***   Exact time : _____ h _____ min
3rd Saliva Sample: ***(Label: 1-3)***   Exact time : _____ h _____ min
4th Saliva Sample: ***(Label: 1-4)***   Exact time : _____ h _____ min
5th Saliva Sample: ***(Label: 1-5)***   Exact time : _____ h _____ min

After the last saliva sample of the day, please respond to the questions on the back of this page.

DAY 1 (cont'd)

Today, have you been bothered by…….

	NO	YES
1. Stomach pain		
2. Back pain		
3. Pain in your arms, legs or joints (knees hips, etc.)		
4. Pain or problems during sexual intercourse		
5. Headaches		
6. Chest pain		
7. Dizziness		
8. Fainting Spells		
9. Feeling your heart pound or race		
10. Shortness of breath		
11. Constipation, loose bowels, or diarrhea		
12. Nausea, gas or indigestion		

To what extent did you experience each of the following emotions today? Check the appropriate box next to the emotion.

	Very slightly or not at all	A little	Moderately	Quite a bit	Extremely
1. Lonely					
2. Stressed					
3. Sad					
4. Upset					
5. Hostile					
6. Isolated					
7. Overwhelmed					
8. Unhappy					
9. Angry					

#: _____

DAY 2

Date: _____

Please record the exact time when you took your saliva sample.

1st Saliva Sample: ***(Label: 2-1)***   I woke up at _____h_____min
2nd Saliva Sample: ***(Label: 2-2)***   Exact time : _____h_____min
3rd Saliva Sample: ***(Label: 2-3)***   Exact time : _____h_____min
4th Saliva Sample: ***(Label: 2-4)***   Exact time : _____h_____min
5th Saliva Sample: ***(Label: 2-5)***   Exact time : _____h_____min

After the last saliva sample of the day, please respond to the questions on the back of this page.

DAY 2 (cont'd)

Today, have you been bothered by…….

	NO	YES
1. Stomach pain		
2. Back pain		
3. Pain in your arms, legs or joints (knees hips, etc.)		
4. Pain or problems during sexual intercourse		
5. Headaches		
6. Chest pain		
7. Dizziness		
8. Fainting Spells		
9. Feeling your heart pound or race		
10. Shortness of breath		
11. Constipation, loose bowels, or diarrhea		
12. Nausea, gas or indigestion		

To what extent did you experience each of the following emotions today? Check the appropriate box next to the emotion.

	Very slightly or not at all	A little	Moderately	Quite a bit	Extremely
1. Lonely					
2. Stressed					
3. Sad					
4. Upset					
5. Hostile					
6. Isolated					
7. Overwhelmed					
8. Unhappy					
9. Angry					

DAY 3

Date: _____

Please record the exact time when you took your saliva sample.

1st Saliva Sample: ***(Label: 3-1)***   I woke up at _____h_____min
2nd Saliva Sample: ***(Label: 3-2)***   Exact time : _____h_____min
3rd Saliva Sample: ***(Label: 3-3)***   Exact time : _____h_____min
4th Saliva Sample: ***(Label: 3-4)***   Exact time : _____h_____min
5th Saliva Sample: ***(Label: 3-5)***   Exact time : _____h_____min

After the last saliva sample of the day, please respond to the questions on the back of this page.

DAY 3 (cont'd)

Today, have you been bothered by…….

	NO	YES
1. Stomach pain		
2. Back pain		
3. Pain in your arms, legs or joints (knees hips, etc.)		
4. Pain or problems during sexual intercourse		
5. Headaches		
6. Chest pain		
7. Dizziness		
8. Fainting Spells		
9. Feeling your heart pound or race		
10. Shortness of breath		
11. Constipation, loose bowels, or diarrhea		
12. Nausea, gas or indigestion		

To what extent did you experience each of the following emotions today? Check the appropriate box next to the emotion.

	Very slightly or not at all	A little	Moderately	Quite a bit	Extremely
1. Lonely					
2. Stressed					
3. Sad					
4. Upset					
5. Hostile					
6. Isolated					
7. Overwhelmed					
8. Unhappy					
9. Angry					

Appendix C:
Aging and Health Questionnaire (Wave 1)

Concordia
UNIVERSITY

Aging & Health

Date: _____

Dear Participant,

We want to thank you for participating in this study. Without your answers, we could not do research. Please keep in mind that all the information you provide is absolutely confidential and will only be used for research purposes. If you have any questions or need further clarification, please do not hesitate to contact us.

Dr. Carsten Wrosch
Principal Researcher

Dominique Dussault
Project Coordinator
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Email: cwlab@alcor.concordia.ca

Medication Use

1. Have you used any medications in the past three months? Please include both prescription and over-the-counter medications.

   ☐ YES ☐ NO ➔ Go to the next page

2. If you answered yes to the previous question, please list all medications you have used during the **past three months**:

MEDICATION 1:	
Name _____	Dosage _____
Frequency _____	Reason taken _____
Start date _____	Stop date _____
MEDICATION 2:	
Name _____	Dosage _____
Frequency _____	Reason taken _____
Start date _____	Stop date _____
MEDICATION 3:	
Name _____	Dosage _____
Frequency _____	Reason taken _____
Start date _____	Stop date _____
MEDICATION 4:	
Name _____	Dosage _____
Frequency _____	Reason taken _____
Start date _____	Stop date _____
MEDICATION 5:	
Name _____	Dosage _____
Frequency _____	Reason taken _____
Start date _____	Stop date _____
MEDICATION 6:	
Name _____	Dosage _____
Frequency _____	Reason taken _____
Start date _____	Stop date _____

Activities of Daily Living

Please answer the following questions regarding your daily chores. Place a check under "No" if you do not experience any difficulty with the specific chore. If you do experience some difficulty with that chore, we would like you to first evaluate the amount of: 1) difficulty completing the chore; 2) physical strain involved **and** 3) emotional strain experienced with this chore, using the scale below. Please write the corresponding number under each of the "yes" columns.

1 = very slightly or not at all
2 = a little
3 = moderately
4 = quite a bit
5 = extremely

Because of health or physical problems, do you have any difficulty or are you unable:	**No**	**Yes**		
		Difficulty	**Physical strain**	**Emotional strain**
…to eat, including feeding yourself?				
…to dress yourself?				
…to bathe or shower?				
…to use the toilet including getting to the toilet?				
…to walk around the home?				
…to get in and out of a bed or a chair?				
…to do heavy housework, like scrubbing floors or washing windows, or yard work, like raking leaves or moving?				
…to do light housework?				
…to do shopping for personal items?				
…to prepare meals?				
…to manage money, such as paying bills?				
…to use the phone?				

Personal information

1. **Sex** ☐ Female ☐ Male
2. **Age** _____ yrs.
3. **First Language** ☐ English ☐ French ☐ Other_____
4. **Family Status?**

   ☐ married
   ☐ live with partner but not married
   ☐ single
   ☐ divorced; please indicate since when _____
   ☐ widowed; please indicate since when _____

5. **Highest Level of Education Completed**

   ☐ None
   ☐ High School
   ☐ Collegial or Trade School
   ☐ Bachelor's Degree
   ☐ Masters or Doctorate Degree

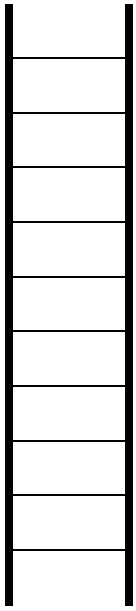
6. **Working status:** ☐ Retired ☐ Still working ☐ Never worked outside the house
7. **Profession (before retirement)** _____
8. **Current Family income (per year):**

   ☐ Less than 17 000$ ☐ 17 001$ - 34 000$ ☐ 34 001$ - 51 000$
   ☐ 51 001$ - 68 000$ ☐ 68 001$ - 85 000$ ☐ more than 85 000$

9. Height: _____
10. Body weight: _____

SES and Finances

1. **Think of this ladder as representing where people stand in our society. At the top of the ladder are the people who are the best off, those who have the most money, most education, and best jobs. At the bottom are the people who are the worst off, those who have the least money, least education, and worst jobs or no job. Please, place an X on the rung that best represents where you think you stand on the ladder?**



2. **Using a scale from 0 to 10 where 0 means " the worst possible financial situation" and 10 means " the best possible financial situation," how would you rate your financial situation these days?**

Worst										**Best**
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
0	1	2	3	4	5	6	7	8	9	10

3. **In general, would you say you (and your family living with you) have <u>more money</u> than you need, <u>just enough</u> for your needs, or <u>not enough</u> to meet your needs?**

☐ More money than you need ☐ Just enough money ☐ Not enough money

4. **How difficult is it for you (and your family) to pay your monthly bills?**

☐ Very difficult ☐ Somewhat difficult ☐ Not very difficult ☐ Not at all difficult

Health

1. If you think about your **current physical condition**, which of the following would be your goal for the next few months?

Improve my health	Maintain my current health	Minimize health declines
☐	☐	☐
1	2	3

1a. How important is it to you to attain your health goals? (check one)

Not at all ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 **Extremely**

1b. How likely do you think it is that you will attain your health goals? (check one)

Not at all ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 **Extremely**

1c. To what extent do you feel that attainment of your health goals depends mostly on what <u>you</u> do? (check one)

Not at all ☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 **A great deal**

Please check the box in the column "Yes" if you experienced the symptom <u>often in the last month</u>, **"**No" if you did not.

During the past month, have you often been bothered by…….	**YES**	**NO**
13. Stomach pain		
14. Back pain		
15. Pain in your arms, legs or joints (knees hips, etc.)		
16. Pain or problems during sexual intercourse		
17. Headaches		
18. Chest pain		
19. Dizziness		
20. Fainting Spells		
21. Feeling your heart pound or race		
22. Shortness of breath		
23. Constipation, loose bowels, or diarrhea		
24. Nausea, gas or indigestion		

Physical Health

Please answer the following questions about your physical health.

	NO	YES	NOT SURE
1. Do you currently have high blood pressure?			
2. Do you currently have problems with an irregular heart beat or chest pain?			
3. Have you ever been told that you have coronary heart disease or coronary artery disease?			
4. Have you ever had a heart attack?			
5. Have you ever been treated for congestive heart failure?			
6. Have you ever had major surgery? (IF YES:) What? _____			
7. Have you ever had a stroke?			
8. Do you currently have osteoarthritis, fibromyalgia, osteoporosis, or any other serious muscular or bone problem?			
9. Do you currently have asthma, emphysema, chronic bronchitis, chronic obstructive lung disease, or any other serious respiratory problems?			
10. Do you currently have stomach ulcers, irritable bowel syndrome, or any other serious problems with your stomach or bowels?			
11. Do you have diabetes?			
12. Do you currently have problems with your kidneys?			
13. Do you have cirrhosis or any other serious liver problems?			
14. Do you currently have cancer? (IF YES:) What type _____			
15. Do you currently have rheumatoid arthritis, lupus, acquired immune deficiency syndrome, multiple sclerosis, scleroderma, or any other autoimmune problem?			

Physical Health (cont'd)

	NO	YES	NOT SURE
16. Do you currently have problems with blood circulation in your legs, hemophilia, or any other blood-related problems?			
17. Do you have epilepsy or any other neurological problems?			
18. Do you currently have an overactive or underactive thyroid, or any other thyroid problems?			
19. Do you currently have any problems with your vision or hearing?			
20. Do you currently have asthma, bronchitis, or emphysema?			
21. Do you currently have persistent skin trouble (e.g., eczema)?			
22. Do you currently have recurring stomach trouble, indigestion, or diarrhea?			
23. Do you currently have migraine headaches?			
24. Are you constipated all or most of the time?			
25. Do you have chronic sleeping problems?			
26. Do you currently have any other health problems that I have not asked you about? (IF YES:) What? _____			

Please indicate by checking the appropriate box, to what extent you have experienced any of the following cold symptoms during the past couple of weeks.

	None	Mild	Moderately	Severe	Very severe
Nasal congestion					
Sneezing					
Runny nose					
Cough					
Feeling under the weather					
Scratchy/sore throat					
Headaches					
Fever					

Health Management

To what extent does each of the following statements apply to you? For each statement, please indicate the extent to which of the following statements *usually* applies to you.

	Almost Never True	Seldom True	Some-times True	Often True	Almost Always True
1. I invest as much time and energy as possible to improve my health.					
2. Even if my health is in very difficult condition, I can find something positive in life.					
3. If I develop a new health problem, I immediately get help from a health professional (e.g., doctor, nurse).					
4. When I decide to do something about a health problem, I am confident that I will achieve it.					
5. I do whatever is necessary to be as healthy as I possibly can be.					
6. When a treatment doesn't work for a health problem I have, I try hard to find out about other treatments.					
7. When I am faced with a bad health problem, I try to look at the bright side of things.					
8. Once I decide what I need to do to improve my health, I avoid things that could distract me from doing these things.					
9. If I have a health problem that gets worse, I put in even more effort to get better.					
10. When I first notice a health problem, I try to get as much advice as I can from people who might know something about the problem.					
11. When I find it impossible to overcome a health problem, I try not to blame myself.					
12. I often think about how important good health is to me.					

Well-Being

1. Below are five statements with which you may agree or disagree. Please indicate your agreement with each item by putting a check in the appropriate box next to the statement.

	Strongly Disagree	**Disagree**	**Slightly Disagree**	**Neither Agree nor Disagree**	**Slightly Agree**	**Agree**	**Strongly Agree**
1. In most ways my life is close to my ideal.							
2. The conditions of my life are excellent.							
3. I am satisfied with my life.							
4. So far I have gotten the important things I want in life.							
5. If I could live my life over, I would change almost nothing.							

2. Using a 0 to 10 scale, where 0 means *no control at all* and 10 means *very much control*, how would you rate the amount of control you have over your <u>life</u> overall these days?

No control **Total control**

☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐

0 1 2 3 4 5 6 7 8 9 10

Well-Being

This scale consists of a number of words that describe different feelings and emotions. Read each item and indicate to what extent you experienced the following emotions **during the past year**.

	Very slightly or not at all	**A little**	**Moderately**	**Quite a bit**	**Extremely**
10. Interested					
11. Distressed					
12. Excited					
13. Upset					
14. Strong					
15. Guilty					
16. Scared					
17. Hostile					
18. Enthusiastic					
19. Proud					
20. Irritable					
21. Alert					
22. Ashamed					
23. Inspired					
24. Nervous					
25. Determined					
26. Attentive					
27. Jittery					
28. Active					
29. Afraid					

Well-Being

The questions in this scale ask you about your feelings and thoughts **during the last month**. In each case, you will be asked to indicate how often you have felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate the response option that seems like a reasonable estimate.

In the last month, how often have you …	**Never**	**Almost Never**	**Some-times**	**Fairly Often**	**Very Often**
1. …been upset because of something that happened unexpectedly?					
2. …felt that you were unable to control the important things in your life?					
3. …felt nervous and "stressed"?					
4. …felt confident about your ability to handle your personal problems?					
5. …felt that things were going your way?					
6. …found that you could not cope with all the things that you had to do?					
7. …been able to control irritations in your life?					
8. …felt that you were on top of things?					
9. …been angered because of things that happened that were outside of your control?					
10. …felt difficulties were piling up so high that you could not overcome them?					

Well-Being

Below is a list of the ways you might have felt or behaved. Please indicate by a check how often you have felt this way **during the past week** using the following scale.

Rarely or None of the Time = Less than 1 Day
Some or a Little of the Time = 1 – 2 Days
Occasionally or a Moderate Amount of the Time =3 - 4 Days
Most or All of the Time = 5 - 7 Days

During the past week ….	**Less than 1 Day**	**1 – 2 Days**	**3 – 4 Days**	**5 – 7 Days**
1. I was bothered by things that usually don't bother me.				
2. I had trouble keeping my mind on what I was doing.				
3. I felt depressed.				
4. I felt that everything I did was an effort.				
5. I felt hopeful about the future.				
6. I felt fearful.				
7. My sleep was restless.				
8. I was happy.				
9. I felt lonely.				
10. I could not get "going."				

Goal Adjustment

During their lives people cannot always attain what they want and are sometimes forced to stop pursuing the goals they have set. We are interested in understanding how you usually react when this happens to you. Please indicate the extent to which you agree or disagree with each of the following statements, as it usually applies to you.

If I have to stop pursuing an important goal in my life…	**Strongly Disagree**	**Disagree**	**Neutral**	**Agree**	**Strongly Agree**
1. It's easy for me to reduce my effort towards the goal.					
2. I convince myself that I have other meaningful goals to pursue.					
3. I stay committed to the goal for a long time; I can't let it go.					
4. I start working on other new goals.					
5. I think about other new goals to pursue					
6. I find it difficult to stop trying to achieve the goal.					
7. I seek other meaningful goals.					
8. It's easy for me to stop thinking about the goal and let it go.					
9. I tell myself that I have a number of other new goals to draw upon.					
10. I put effort toward other meaningful goals.					

Describe Yourself

Please mark the appropriate answer for each of the statements below:

	Strongly Disagree	**Disagree**	**Agree**	**Strongly Agree**
1. I feel that I'm a person of worth, at least on an equal plane with others.				
2. I feel that I have a number of good qualities.				
3. All in all, I am inclined to feel that I am a failure.				
4. I am able to do things as well as most other people.				
5. I feel I do not have much to be proud of.				
6. I take a positive attitude toward myself.				
7. On the whole, I am satisfied with myself.				
8. I wish I could have more respect for myself.				
9. I certainly feel useless at times.				
10. At times I think I am no good at all.				

Describe Yourself

Please indicate how well each of the following statements describes you by putting a check in the appropriate box.

	Not at all true of me	Slightly true of me	True of me to some extent	Mostly true of me	Very much true of me
1. I am not a worrier.					
2. I often feel inferior to others.					
3. When I'm under a great deal of stress, sometimes I feel like I'm going to pieces.					
4. I rarely feel lonely or blue.					
5. I often feel tense and jittery.					
6. Sometimes I feel completely worthless.					
7. I rarely feel fearful or anxious.					
8. I often get angry at the way people treat me.					
9. Too often, when things go wrong, I get discouraged and feel like giving up.					
10. I am seldom sad or depressed.					
11. I often feel helpless and want someone else to solve my problems.					
12. At times I have been so ashamed I just wanted to hide.					

Health Practices

Tobacco Use

	YES	NO
1. Do you smoke cigarettes, cigars, or pipes daily?		

1a. On average how many of each do you smoke daily? _____ cigarettes

_____ cigars

_____ pipes

	YES	NO	I still smoke
2. Did you ever smoke cigarettes, cigars, or a pipe daily?			

2a. If yes, when did you quit smoking on a daily basis ? _____ Month and year

Alcohol

	YES	NO
1. Do you drink alcohol at least once a week?		

2. On average, how many times per week do you usually drink alcohol? _____

3. When you drink alcohol, on average how many drinks do you usually have? (Consider a glass of wine, a 12-ounce beer, or a shot of hard liquor equal to one drink.)

_____drinks

Activities

	YES	NO
1. Do you engage in any regular activity akin to brisk walking, jogging, bicycling, etc, long enough to work up a sweat?		
1a. If yes, how many days per week? _____ days? 1b. If yes, for how long each time? _____ minutes		
2. How many city blocks or their equivalent do you regularly walk each day? Assume 12 blocks = 1 mile. _____Blocks per day		
3. On average, how many hours per day do you watch television? _____hours		
	YES	**NO**
4. Did you participate in any other activities (e.g. bingo, cards, shopping) during the past week?		
4a. If yes, could you name these activities? 1._____ 2._____ 3._____ 4._____ 5._____		

Sleep

The following questions relate to your usual sleep habits. Your answers should indicate the most accurate reply for the majority of days and nights of the **past month**.

1. What time have you usually laid down to go to sleep? _____

2. What time have you usually gotten out of bed in the morning? _____

3. How long has it taken you to fall asleep after you have laid down to go to sleep?

   _____ minutes

4. How many minutes of sleep have you lost because you woke up in the middle of the night?

   _____ minutes

5. How many minutes of sleep have you lost because you woke up earlier than your usual time to get up?

   _____ minutes

6. How many hours of **actual** sleep do you get at night?

   _____ hours (This may be different than the number of hours you spent in bed)

7. How would you rate your sleep quality overall?

   ☐ Very good ☐ Fairly Good
   ☐ Fairly Bad ☐ Very Bad

8. How often do you have trouble staying awake while driving, eating meals, or in social activity?

   ☐ Not during the past month ☐ Less than once per week
   ☐ Once or twice per week ☐ Three or more times per week

9. Do you usually take a nap during the day?

   ☐ NO ☐ YES ➔ How many days per week? _____ days

   ➔ For how long each time? _____ minutes

Life Regrets

People make a lot of important decisions during their lives and they sometimes think that they should have done something differently than they did. For example, a person may believe that she/he would be better off today if she/he had behaved in a different way in the past. In such situations, people might regret their behaviours. In addition, they often want the negative consequences of their behaviours to be undone.

Life regrets might result from things that people have done (e.g., having pursued a fruitless goal) and from things that people have not done (e.g., not having pursued a certain goal) across a number of different life domains (e.g., work, family, spouse, health). Regrets are related to decisions in people's daily lives (e.g., not having visited a friend) and to people's long-term development (e.g., having pursued inappropriate career goals).

Please think for a moment about your life. Is there anything in your life that you regret having done or not having done? Please think about your regrets and write down your most severe life regret.

_____

_____

1. We would like to ask you some specific questions concerning the regret that you have noted.

a. Does the regret that you have noted relate to a behaviour
- ☐ that you have done
- ☐ that you have *not* done

b. When did the behaviour occur that has lead to the regret? (please try to indicate the exact number of months and years ago that the event occurred)

_____ months ago _____ years ago

c. How likely is it that the negative consequences of the event can in fact be undone?

Very Unlikely ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 **Very Likely**

d. How likely is it that the negative consequences of the event will in fact be undone?

Very Unlikely ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 **Very Likely**

e. How much effort do you invest in undoing the negative consequences of the event?

No Effort at all ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 **A Lot of Effort**

f. How strongly are you committed to undoing the negative consequences of the event?

Not at all Committed ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 **Very Much Committed**

Life Regrets (cont'd)

2. People usually experience different emotions when they think about their regrets. We would like to ask you to what extent you usually experienced the following emotions **during the past few months** when and if you thought about the regret that you noted.

	Not at all	**A little**	**Somewhat**	**Quite a bit**	**Extremely**
a. Sorrow					
b. Angry					
c. Sentimental					
d. Desperate					
e. Irritated					
f. Nostalgic					
g. Helpless					
h. Embarrassed					
i. Contemplative					

3. Below is a list of comments made by people who experienced life regrets. Please indicate how frequently these comments were true for you **during the past few months** by checking the appropriate box.

	Not at all	**Rarely**	**Sometimes**	**Often**
a. I had trouble falling asleep because I couldn't stop thinking about the regret.				
b. I woke up at night thinking about the regret.				
c. I had difficulty concentrating on my work or daily activities because thoughts about the regret kept entering my mind.				
d. Once I start thinking about the regret I find it hard to think about (focus my attention on) other things.				
e. Thoughts about the regret interfered with my ability to enjoy social or leisure activities.				

Support

The following questions ask about people in your environment who provide you with help or support. Each questionnaire has two parts. For the first part, list all the people you know. Give the person's initials and their relationship to you (see example). Do not list more than one person next to each of the numbers beneath the question. Do not list more than nine persons per question.

For the second part, circle how satisfied you are with the overall support you have using the number corresponding to the legend below. If you have no support for a question, check the words "no one", but still rate your level of satisfaction.

EXAMPLE:

Who do you know whom you can trust with information that could get you in trouble?

No one	1) T.N. (brother)	4) T.N. (father)	7)
	2) L.M. (friend)	5) L.M. (employer)	8)
	3) R.S. (friend)	6)	9)

How satisfied are you with the overall support?

Very dissatisfied	Fairly dissatisfied	A little dissatisfied	A little satisfied	Fairly satisfied	Very satisfied
1	2	3	4	5	6

1. Whom can you really count on to listen to you when you need to talk?

No one 1)	4)	7)	
2)	5)	8)	
3)	6)	9)	

How satisfied are you with the overall support?

Very dissatisfied	Fairly dissatisfied	A little dissatisfied	A little satisfied	Fairly satisfied	Very satisfied
1	2	3	4	5	6

2. Whose lives do you feel that you are an important part of?

No one 1)	4)	7)	
2)	5)	8)	
3)	6)	9)	

How satisfied are you with the overall support?

Very dissatisfied	Fairly dissatisfied	A little dissatisfied	A little satisfied	Fairly satisfied	Very satisfied
1	2	3	4	5	6

3. Whom could you really count on to help you out in a crisis situation, even though they would have to go out of their way to do so?

No one 1)		4)		7)	
2)		5)		8)	
3)		6)		9)	
How satisfied are you with the overall support?					
Very dissatisfied	**Fairly dissatisfied**	**A little dissatisfied**	**A little satisfied**	**Fairly satisfied**	**Very satisfied**
1	2	3	4	5	6

4. Whom can you talk with frankly, without having to watch what you say?

No one 1)		4)		7)	
2)		5)		8)	
3)		6)		9)	
How satisfied are you with the overall support?					
Very dissatisfied	**Fairly dissatisfied**	**A little dissatisfied**	**A little satisfied**	**Fairly satisfied**	**Very satisfied**
1	2	3	4	5	6

5. Who can you really count on to be dependable when you need help?

No one 1)		4)		7)	
2)		5)		8)	
3)		6)		9)	
How satisfied are you with the overall support?					
Very dissatisfied	**Fairly dissatisfied**	**A little dissatisfied**	**A little satisfied**	**Fairly satisfied**	**Very satisfied**
1	2	3	4	5	6

6. Whom can you really count on to give you useful suggestions that help you to avoid making mistakes?

No one 1)		4)		7)	
2)		5)		8)	
3)		6)		9)	
How satisfied are you with the overall support?					
Very dissatisfied	**Fairly dissatisfied**	**A little dissatisfied**	**A little satisfied**	**Fairly satisfied**	**Very satisfied**
1	2	3	4	5	6

7. Who will comfort you when you need it by holding you in their arms?

No one	1)	4)	7)		
	2)	5)	8)		
	3)	6)	9)		

How satisfied are you with the overall support?

Very dissatisfied	Fairly dissatisfied	A little dissatisfied	A little satisfied	Fairly satisfied	Very satisfied
1	2	3	4	5	6

8. Whom do you feel would help if a family member very close to you died?

No one	1)	4)	7)		
	2)	5)	8)		
	3)	6)	9)		

How satisfied are you with the overall support?

Very dissatisfied	Fairly dissatisfied	A little dissatisfied	A little satisfied	Fairly satisfied	Very satisfied
1	2	3	4	5	6

Personal Goals

We would like some information about the goals that you have in your life. Personal goals are objectives, plans, projects, or even "ways of being" that a person is trying to pursue in a particular span of time. These can be goals that have specific endpoints (e.g., to buy a new dining room table or get the bedroom painted) or goals that continue indefinitely (e.g., to enjoy a relationship with someone else or eat a healthy diet). Some of these goals may be short-term goals, whereas other goals may take a longer period of time to achieve.

Please think about your personal goals and write down the *most important* goals that you want to pursue. Please use a separate line for each goal. Fill as many goals as you have for a maximum of 10 goals.

LIST OF GOALS:

1. _____

2. _____

3. _____

4. _____

5. _____

6. _____

7. _____

8. _____

9. _____

10. _____