AN ABSTRACT OF THE DISSERTATION OF

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Title: Longitudinal Changes in Stress-Related Growth: Relations with Stress Severity and Depressive Symptoms

Abstract approved:

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Studies on the positive effects of stress, called stress-related growth (SRG), are increasing, and there are a variety of contradictory theoretical models and findings in the literature. This dissertation compared several of these models in two studies, using two waves of data from the Davis Longitudinal Study, a study of college alumni, roughly split between men and women ranging in age from 30 – 50 (cross-sectional $N = 1140$; longitudinal $N = 509$). The first study examined whether the effects of SRG on subsequent stressful episodes were positive (resource accumulation), negative (resource depletion), or were mediated by how individuals coped with the problem (contingent). Results showed that stress severity was weakly but positively related to SRG within time, but no direct cross-lagged effects emerged across time. However, when coping strategies were entered into the model, the within-time effects between stress severity and SRG were fully mediated by both positive and negative coping strategies, whereas the cross-lagged effects were partially mediated by positive coping strategies itself but not negative coping

strategies. The second manuscript investigated whether SRG was a protective factor against future depressive symptoms. As the literature is inconsistent, we hypothesized that stress severity would have a moderating effect on the relationship between SRG and depressive symptoms. Cross-sectional results indicated the effect of SRG on depressive symptoms were moderated by the severity of the stressor – it was only in the high stress group that SRG was inversely related to depressive symptoms for the within-time analyses with both the cross-sectional and longitudinal samples. However, the longitudinal analyses showed a different pattern of results. In the cross-lagged model, there was no effect of SRG on subsequent depressive symptoms, although there was a weak effect of depressive symptoms at Time 1 being positively related to SRG at Time 2.

Taken together, these results present a highly complex relationship between stress, coping, SRG, and depressive symptoms. The effect of SRG on future outcomes of subsequent stressful episodes is mediated by the type of coping strategies used and dependent upon the degree of stressfulness of the problems faced.

Longitudinal Changes in Stress-Related Growth:
Relations with Stress Severity and Depressive Symptoms

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I understand that my dissertation will become part of the permanent collection of Oregon State University libraries. My signature below authorizes release of my dissertation to any reader upon request.

Xiaoyu Bi, Author

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CONTRIBUTION OF AUTHORS

My advisor and co-author, Dr. Carolyn M. Aldwin, of the two manuscripts in this dissertation kept motivating and providing me conceptual framework, thoughtful ideas, methodology, as well as writing skills for both manuscripts.

TABLE OF CONTENTS

Page

LIST OF TABLES

LIST OF FIGURES

Figure Page

DEDICATION

This dissertation is dedicated to my husband, Wing F Liu, and my son, who is expected in February.

LONGITUDINAL CHANGES IN STRESS-RELATED GROWTH: RELATIONS WITH STRESS SEVERITY AND DEPRESSIVE SYMPTOMS

GENERAL INTRODUCTION

A remarkable number of studies have documented negative effects of stress on physical (for review, see Cohen, Janicki-Deverts, & Miller, 2007) as well as mental health (for review, see Aldwin, 2007). For example, studies have shown that stress increases vulnerability to physical illness, e.g., cardiovascular disease risk (Belkic, Landsbergis, Schnall, & Baker, 2004; Krantz & McCeney, 2002), HIV/AIDS (Leserman et al., 2002; Pereira & Penedo, 2005), cancer (Antoni et al., 2006), cold (Cohen et al., 1998), and coronary artery disease (Boscarino, 2004). Also, researchers have indicated that stress is more likely to contribute to anxiety disorders (Zatzick et al., 1997; Michael, 2000), depression (Michaels et al., 1999; Galovski, Blanchard, Malta, & Freidenberg, 2003), and schizophrenia (Morrison, Frame, & Larkin, 2003; Mueser, Rosenberg, Goodman, & Trumbetta, 2002). However, within the past three decades, researchers have begun noticing a variety of positive results from stressful experiences as well (Aldwin & Stokols, 1988; Tedeschi & Calhoun, 1995). Since then, a growing number of studies have concentrated on the positive outcomes of stress (for reviews, see Lindley & Joseph, 2004; Park & Helgeson, 2006; Schaefer & Moos, 1992).

Definition of Stress-Related Growth

These positive outcomes have been labeled as stress-related growth (SRG; Aldwin, 2007; Park, Cohen, & Murch, 1996), posttraumatic growth (PTG; Tedeschi & Calhoun, 1996, 2004), adversarial growth (Linley & Joseph, 2004), benefit finding (Affleck & Tennen, 1996), thriving (O'Leary & Ickovics, 1995), flourishing (Ryff &

Singer, 1998), personal growth (Schaefer & Moos, 1992), or positive illusion (Taylor, 1983), and so on (for reviews, see Tedeschi & Calhoun, 2004). Even though several terms are similar, there are still some obvious differences especially among positive illusion, PTG, and SRG. From Taylor's (1983; Taylor, Kemeny, Reed, Bower, & Gruenewald, 2000) perspective, these positive outcomes are not due to genuine growth but rather cognitive adaption (i.e., positive illusion and self-deception). Calhoun and Tedeschi (1999) suggest that these positive changes, characterized by five domains of new possibilities, relating to others, personal strength, spiritual change, and appreciation of life (Tedeschi & Calhoun, 1996), are "true" results when and only when subjects encounter "highly challenging" stressors (Calhoun & Tedeschi, 1999,) or "seismic" events (Tedeschi & Calhoun, 2004). However, other researchers (Aldwin & Levenson, 2004; Park, 2009) argue that people can also obtain positive outcomes from less radical and acute stressors or from positive events such as childbirth and marriage. I endorse Aldwin, Levenson, and Park's arguments and view SRG as a broader term than PTG. The term of SRG means "actual or veridical changes that people have made in relation to their experience with an identified stressful or traumatic event" (Park, 2009, p. 12). In general, the term of SRG is used throughout my proposal, but sometimes when specific studies are referred to, other terms are used as well.

Significance of Stress-Related Growth

Examining SRG is important for three reasons. First, it is well-known that stress is universal and has a variety of deleterious effects on people's physical and mental health. Strongly grounded in this perspective, most previous research narrows our concentration on exclusively negative aspects of stress. However, SRG studies

widen our focus to its positive feature as well and thus provide us a more integrative picture (Joseph & Linley, 2009).

Second, perceived SRG may have significant implications for better psychological health (for reviews, see Algoe & Stanton, 2008; Helgeson, Reynolds, & Tomich, 2006). Participants with higher levels of SRG are much more likely to have better well-being (for a review, see Helgeson et al., 2006) and lower levels of depressive symptoms (Carver & Antoni, 2004). Given the positive effects of SRG, it may provide an alternative way to improve psychological health in clinical intervention programs (Antoni et al., 2001).

Finally, SRG may be related to better physical health (for a review, see Bower, Epel, & Moskowitz, 2008; Pressman & Cohen, 2005). For example, participants with higher levels of SRG are much more likely to have lower levels of morbidity in people who have suffered heart attacks (Affleck, Tennen, Croog, & Levine, 1987) and diabetes (Moskowitz, Epel, & Acree, 2008), and to have less negative changes in biological HIV markers (Bower, Kemeny, Taylor, & Fahey, 1998; Milam, 2006). These results suggest that perceived SRG may have significant implications for better physical health and potentially can be used in clinical intervention programs (Antoni et al., 2001).

Statement of Problem

Given the importance of SRG, a number of studies have examined the predictors, mediators, and outcomes of SRG. Since SRG derives from stress, stress severity may be an important factor for SRG (Schaefer & Moos, 1992). Some researchers argue that the development of SRG is closely related to coping, which mainly plays a mediating role between stress severity and SRG (Aldwin, 2007; Park, Aldwin, Fenster, & Snyder, 2008; Schaefer & Moos, 1992). Also, the relation

between depressive symptoms and SRG is a critical issue in SRG studies, with orthogonal, negative, and positive relations found (for reviews, see Algoe & Stanton, 2008; Helgeson et al., 2006).

However, empirical research on longitudinal relations between SRG, stress severity, coping, and depressive symptoms is lacking. Therefore, little information is known about how SRG is influenced by the three variables and how SRG subsequently influences them over the lifespan of people.

Purpose of the Dissertation

This dissertation is composed of two manuscripts. Its overall purpose is to examine longitudinal relations between SRG, stress severity, coping, and depressive symptoms in large samples of young and middle-aged college alumni over five years. The first manuscript (i.e., Study 1) investigates potential reciprocal paths between stress severity and SRG as well as the longitudinal mediating effects of coping over two waves. Specifically, it examines how previous levels of stress severity predict subsequent SRG, and in turn how the SRG levels affect the stressfulness of subsequent episodes. Furthermore, it tests how positive and negative coping strategies play different mediating roles on the potential reciprocities between stress severity and SRG over time.

The second manuscript (i.e., Study 2) focuses on potential moderating effects of stress severity on the relations between SRG and depressive symptoms. Namely, do the relations between SRG and depressive symptoms depend on different levels of stress severity? Specifically, we examine when the level of stress severity is low, are SRG and depressive symptoms are unrelated to one another? When the level of stress severity is high, are they negatively related to one another?

Organization of the Dissertation

Chapter 1 mainly discusses overall theoretical and empirical SRG literature for both Study 1 and Study 2 because the two manuscripts share a common thread of examining the longitudinal relations between SRG and other variables in this dissertation. First, we introduce and make links between SRG and three theoretical models throughout this dissertation, including generalized resistance resources theory (GRRs, Antonovsky, 1979, 1987), conservation of resources theory (COR, Hobfoll, 1988), and deviation amplification model (DAM, Aldwin & Stokols, 1988; Aldwin, Sutton, & Lachman, 1996). Then, we briefly review several overarching issues in SRG, particularly focusing on longitudinal empirical studies in SRG changes and two measurement issues.

Chapter 2 presents Study 1 entitled *Does coping mediate the relations between stress severity and stress-related growth? Findings from the Davis Longitudinal Study*. First, we review both theoretical and empirical literature in stress severity, coping, and SRG, primarily focusing on the mediating effects of coping on stress severity and SRG. Next, research questions and hypotheses are proposed, and then study design and data analyses are described in detail.

Chapter 3 present Study 2 entitled *The Effects of Stress Severity on Reciprocal Relations between Stress-Related Growth and Depressive Symptoms: Findings from the Davis Longitudinal Study*. Following the same format, we review and discuss both theoretical and empirical literature in SRG and depressive symptoms, and then describe our research questions, hypotheses, methodological design, and statistical analyses.

Chapter 4 is about overall discussion of this dissertation. We briefly summarize the results of both Study 1 and Study 2, discuss the limitations, and address the potential clinical implications in practice.

Overall Literature Review

Because both Study 1 and Study 2 examine longitudinal changes of SRG over two waves, this section reviews the literature related to this question. First, we describe transactional model of stress and coping and then review the development of SRG theories. Based on this literature, we discuss how a developmental perspective shapes and contributes to our understanding about SRG, i.e., how different life stressors are connected with each other over time. Second, we briefly introduce three overarching theories of GRRs, COR, and DAM and describe their propositions about SRG changes over time. The three theories also provide specific conceptual explanations for Study 1 related to stress severity, coping, and SRG and for Study 2 related to depressive symptoms and SRG, which will be reviewed in Chapter 2 and Chapter 3, respectively. Third, we review empirical literature in the longitudinal variations of SRG, and provide plausible explanations for divergent findings. Finally, two measurement issues including SRG dimensions and SRG measures items are discussed.

Transactional Model of Stress and Coping

A transactional model of stress and coping has been proposed by Lazarus and his colleagues (1966; Lazarus & Folkman, 1984). From their perspective, "psychological stress is a particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well-being" (Lazarus & Folkman, 1984, pp. 19). Unlike previous stress theories (Holmes & Rahe, 1967; Dohrenwend, Krasnoff,

Askenasy, & Dohrenwend, 1978) that define stress as an external event, Lazarus and Folkman (1984) argue that stress arises from the transaction between person and environment in which cognitive appraisal is the key factor. There are two categories of appraisal, primary and secondary. Primary appraisal refers to the evaluation of stressfulness of events/stimulus as a harm/loss, a threat, or a challenge. Secondary appraisal evaluates the types of resources at one's disposal which impacts upon the perceived stressfulness of the problem. The appraisal of stress varies from one individual to another, so it is not surprising that an external stressor such as midterm examinations may have remarkably different meaning to students (Folkman & Lazarus, 1985).

Once people have appraised their stressful events, they use a series of coping strategies to deal with stressors. From the perspective of the transactional approach, coping is simultaneously influenced by personal and environmental factors, which in turn are impacted by coping results. In other words, person, environment, and coping are all reciprocally influencing each other over time. In addition, Lazarus and Folkman (1984) point out that coping is not a static but a dynamic process over a person's lifespan, indicating that coping is specific to certain episodes and tends to differ from one stressor to another. Thus, coping strategies are less consistent across situations. Based on the process-oriented perspective, Lazarus and Folkman (1984) define coping as "constantly changing cognitive and behavioral efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person" (pp. 141*).*

In summary, by taking into account both personal and environmental factors into their transactional model, Lazarus and Folkman (1984) switch the focus from only external parts of stressors to people's perception of stressors and greatly advance

the development of stress studies. Although controversial (Dohrenwend, Dohrenwend, Dodson, & Shrout, 1984; Hobfoll, 1989), this emphasis on the centrality of perception has allowed for innovative approaches to stress research including a new focus on the positive outcomes of stress (i.e., SRG).

Historical Overview of Stress-Related Growth

Even though the term of SRG has only recently been proposed (Aldwin et al., 1996; Park et al., 1996), the general construct can trace back to nearly 50 years ago. Frankl (1963) discussed the positive meaning that he gained when he was in concentration camps. For example, he described that "we can discover this meaning in life …… by the attitude we take toward unavoidable suffering". Caplan (1964) found that even though life crises have negative effects on people, they might also potentially provide an impetus for psychological growth. Haan (1982) also found that "stress benefits people, making them more tender, humble, and hardy" (p. 255).

Others have emphasized that SRG is a process which unfolds over time. Antonovsky (1979) has proposed a salutogenic model to describe positive responses to stress. That is, if people are able to successfully utilize generalized resistance resources (GRRs), they are much more likely to achieve positive outcomes, which in turn lead to an increase in GRRs. Moos and Schaefer (1986) find that life crises can "enrich" people's lives and improve personal relationships, such as becoming closer to family members. Based on Lazarus and Folkman's (1984) transactional framework, Schaefer and Moos (1992) develop a conceptual model to explain how personal and environmental factors shape positive outcomes from life crises and transitions, and in turn how personal growth influences the person and the environment.

Hobfoll's conservation of resource theory (COR; 1988) argues that in dealing with stress, despite resource loss being common and powerful, people are also capable of gaining resources such as development of mastery and positive outlooks. By examining combat exposure among veterans, Aldwin, Levenson, and Spiro (1994) found that even under remarkably traumatic situations, most veterans reported positive consequences (e.g., increased coping skills and self-esteem). Tedeschi and Calhoun (1996; 2004) especially emphasize how people gained growth in struggle with highly stressful events. Based on Schaefer and Moos's (1992) conceptual model, Park et al. (1996) significantly promoted the development of SRG research by designing the first quantitative SRG measure and hypothesizing that SRG occurs by making meaning (Park, 2009).

In recent years, SRG has received considerable attention and been examined among a variety of stressors. For example, it has been widely explored among patients who suffer from cancer especially breast cancer (Manne, Ostroff, Winkel, Goldstein, Fox, & Grana, 2003; Schwarzer, Luszczynska, Boehmer, Taubert, & Knoll, 2006; Sear, Stanton, & Danoff-Burg, 2003; Stanton, Bower, & Low, 2006; Tomich & Helgeson, 2004), HIV-AIDS (Milam, 2002, 2006; Siegel, Schrimshaw, & Pretter, 2005), and heart attacks (Affleck et al., 1987). Also, researchers have examined SRG from specific stressful events such as bereavement (Davis, Nolen-Hoeksema, & Larson, 1998), disasters (McMillen, Smith, & Fisher, 1997), combat (Aldwin et al., 1994), terrorist attacks (Butler et al., 2005; Park et al., 2008), sexual assault (Frazier, Conlon, & Glaser, 2001) and so on.

Even though these studies show that SRG can happen in a variety of contexts, most of them merely examine SRG from a single stressor and do not take a developmental approach (Aldwin, 2007; Aldwin et al., 1994; Aldwin et al., 1996;

Schaefer & Moos, 1992; Park et al., 1996). Does SRG from one stressor predict SRG from another stressor? Does previous SRG play a beneficial role on subsequent SRG attainment across stressors?

There is a controversy over the developmental course of SRG, and relatively few studies examine this phenomenon. The next section will describe relevant theories that explore this topic from a developmental approach and will overview existing empirical literature.

A Developmental Perspective on SRG

Several researchers have called for a developmental perspective in SRG studies (Aldwin et al., 1994; Aldwin et al., 1996; Schaefer & Moos, 1992; Park et al., 1996), but only a few have empirically examined the developmental effects of previous positive outcomes on people. For example, Elder (1974) documented how middle-class children gained positive effects from economic deprivation, and how these positive outcomes influenced their future academic success and marital status. Aldwin et al. (1996) found that a majority of respondents said their prior stressful experiences were useful for them to cope with their current stressful episodes. Also, few theories have provided convincing evidence to explain how SRG changes over time. Given this limitation, this section describes three distinct theoretical perspectives which serve as overarching theories for understanding the development of SRG. Further, how SRG is related to stress severity, coping, and depressive symptoms over time is addressed in Study 1 and Study 2.

Generalized Resistance Resources (GRRs)

The term of generalized resistance resources (GRRs; Antonovsky, 1979) describes how people facilitate stress and how they gain positive outcomes from stress. Generally, GRRs refer to anything that can successfully overcome or resolve

stressors, including physical and biochemical GRRs (e.g., nervous system and immunopotentiating mechanisms), artifactual-material GRRs (e.g., money and power), cognitive and emotional GRRs (e.g., wisdom), valuative-attitudinal GRRs (e.g., coping strategies), interpersonal-relational GRRs (e.g., social supports/ties), and macrosociocultural GRRs (e.g., religion). From his points of view, health is not a dichotomous variable simply categorized as health versus disease, but is a continuum variable moving between salutogenic outcomes of "health ease" and pathogenic outcomes of "health dis-ease". Furthermore, people's health outcomes are significantly influenced by GRRs. Namely, the fewer GRRs yield the more negative outcomes of health dis-ease (e.g., depressive symptoms); conversely, the more GRRs yield the more positive outcomes of health ease, and these positive outcomes of health ease *per se* are GRRs for future events as well (Antonovsky, 1979).

Antonovsky's (1979) GRRs theory has obvious conceptual overlaps with SRG. For example, the mobilized resources of social ties, wisdom, and coping strategy (Antonovsky, 1979) are similar to the SRG dimensions of improved relation to others (Tedeschi & Calhoun, 1996), wisdom (Aldwin & Levenson, 2004), and improved coping skills (Schaefer & Moos, 1992), respectively. Given these connections, GRRs model provides a theoretical framework to examine how SRG (i.e., positive outcomes of stress) is related to stress severity and depressive symptoms (i.e., negative outcomes of stress).

Conservation of Resources Theory (COR)

COR argues that psychological stress is generated when resources that people make an effort to maintain, protect, and build are threatened or lost. These resources include objects (e.g., home, car), conditions (e.g., marriage, employee), personal characteristics (e.g., mastery, self-esteem), and energy (e.g., money, knowledge).

Given the fact that (a) resource gains are less powerful than equivalent resource loss, (b) people always invest resources to cope with stress and to gain future resources, and (c) stress arises from resource loss, it is reasonable to assume that people under stress are at high risk of depleting their resources and developing negative loss spirals (e.g., psychological distress) over time (Hobfoll, 1988; Hobfoll & Lilly, 1993).

In contrast to GRRs, COR (Hobfoll, 1988) provides an alternative theoretical framework to explain stress and health since it mainly focuses on the negative aspects of stress – resource loss and depressive symptoms. But, still some positive outcomes such as resources gains can occur. In essence, the gained resources such as improved self-efficacy and social relations (Hobfoll, 1988; Hobfoll & Lilly, 1993) are similar to SRG dimensions of personal strength and relating to others (Tedeschi & Calhoun, 1996). However, relative to resource loss, resource gains or SRG are much smaller, less influential, and less likely to be replenished over time (Hobfoll, 1988; Hobfoll & Lilly, 1993).

Deviation Amplification Model (DAM)

The Deviation amplification model (DAM) of stress and coping, proposed by Aldwin and Stokols (1988; Aldwin et al., 1996), includes a deviation countering process and a deviation amplification process. In response to stress, the former refers to decrease stress levels to maintain homeostasis whereas the latter refers to magnify the effects of stress, either negatively or positively, mediated by coping strategies. Specifically, if stress is mainly dealt with by positive coping strategies, positive spirals (e.g., high SRG) are much more likely to be generated. Conversely, if stress is mainly dealt with by negative coping strategies, negative spirals (e.g., high depressive symptoms) are much more likely to be produced.

Therefore, from the perspective of DAM (Aldwin et al., 1996), without considering the mediating effects of coping strategies, it is too arbitrary to conclude that stress generates the positive spiral of high SRG (Antonovsky, 1979) or the negative spiral of low SRG (Hobfoll, 1988). In this sense, neither the GRRs nor the COR is complete, and the DAM provides a more comprehensive picture about the relations between stress, SRG, and depressive symptoms.

Summary of GRRs, COR, and DAM

Longitudinal changes of SRG – Theoretical arguments. GRRs, COR, and DAM formulate different developmental patterns for SRG, typically represented by increasing, decreasing, or contingent trajectories, respectively. GRRs (Antonovsky, 1979) argue that SRG is a common outcome of stress, and initial SRG facilitates subsequent SRG attainment. COR (Hobfoll, 1988) proposes that SRG is less likely to occur over time because resource loss always far exceeds resource gains/SRG. DAM (Aldwin et al., 1996), however, posits that SRG is primarily mediated by coping. That is, if positive coping strategies are used, SRG increases over time; if negative coping strategies are used, SRG decreases over time. In addition, along with the discussion of the longitudinal changes of SRG, the three theories describe the relations between SRG, stress severity, coping, and depressive symptoms, which will be discussed in more detail in Study 1 and Study 2.

GRRs view health as a continuous variable, suggesting that positive outcomes and negative outcomes of stress can not occur simultaneously. Thus, when positive outcomes are high, negative outcomes are inevitably low, and vice versa. In other words, positive outcomes and negative outcomes are inversely related.

SRG and stressful events – Theoretical arguments. The three models all emphasize the unfolding process of SRG from multiple stressful events instead of

from a single stressor. These theories can reasonably explain the longitudinal changes of SRG because the stressful events which naturally occur over people's lives tend to be different from one time point to another (Aldwin, 2007; Aldwin et al., 1996).

Overview of Longitudinal Studies of Stress-Related Growth

Even though most studies argue that SRG is an incremental development process (for review, see O'Leary, Alday, & Ickovics, 1998), the cross-sectional nature of the research designs does not adequately assess whether SRG is stable or changes over time. Given this limitation, longitudinal studies may be more conducive to depicting the variations in SRG.

Longitudinal Changes of SRG – Empirical Findings

Mixed results. To date, empirical studies on longitudinal changes of SRG are still lacking. Affleck et al. (1987) and McMillen et al. (1997) have conducted longitudinal studies to assess SRG but have not examined the changes of SRG over time. Davis et al. (1998) measured the changes of SRG among bereaved people by merely asking whether they gained SRG or not, simply categorizing them into four groups, i.e., always reported SRG, lost early SRG, reported SRG later, and never reported SRG.

Researchers have found mixed results when comparing mean levels of SRG over time. For example, Schwarzer and his colleagues (2006) showed that in general SRG increased from one month to 12 months after cancer surgery, whereas Butler et al. (2005) found that among the five SRG domains proposed by Tedeschi and Calhoun (1996), all except spiritual change significantly decreased over six and half months. Some researchers (Urcuyo, Boyers, Carver, & Antoni, 2005) failed to find any changes of SRG at three, six, and 12 months after cancer surgery. Frazier et al. (2001) found a variable pattern of SRG even though it was not significant. Namely, the SRG

was significantly increased from two weeks (Time 1) to two months (Time 2), but decreased from two months to six months (Time 3), and then increased again at one year (Time 4). Ickovics and her colleagues (2006) found that the changes of SRG were varied by types of stressors. Specifically, for urban adolescent girls experiencing pregnancy and motherhood, SRG tended to increase over time, whereas for those experiencing death of a love one, physical threats, or interpersonal problems, SRG tended to decrease over time. Some researchers have found that the longitudinal changes of SRG were varied by groups, with a significant increase in the intervention group and no changes in the comparison group (Antoni et al., 2001; McGregor, Antoni, Boyers, Alferi, Blomberg, & Carver, 2004).

However, limited information is known about whether previous SRG can predict subsequent SRG, and only a few studies have begun to examine this question (Butler et al., 2005; Hart, Vella, & Mohr, 2008; Manne et al., 2003). Butler et al. (2005) found that initial SRG levels significantly predicted lower SRG levels at six and half months. Similarly, Hart et al. (2008) found that baseline SRG positively predicted subsequent SRG levels. However, Manne et al. (2003) failed to find significant changes for both breast cancer patients and their partners over three waves.

Limitations of existent literature. In summary, the studies in the longitudinal changes of SRG are still inconclusive. In general, studies by comparing mean levels of SRG have showed that SRG is typically represented by patterns of increasing (Schwarzer et al., 2006), decreasing (Butler et al., 2005), variable (Frazier et al., 2001), or no changes (Manne et al., 2003; Urcuyo et al., 2005) over time. Also, previous SRG may have negative (Butler et al., 2005), positive (Hart et al., 2008), or no effects on subsequent SRG (Manne et al., 2003). Closer inspections show that

these discrepancies in the unfolding process of SRG may be due to the following reasons.

First, age, sex, and sample sizes may account for the differences in SRG. Most studies examine SRG among middle- or early-aged adults (range of mean age from 44.7 to 62.7 years) with the exception of Frazier et al's study in sexual assault survivors (mean age of 27). This may indicate that SRG in younger people is less stable than in older people. In addition, most studies examine the changes of SRG among females and thus little is known about the changes among males over time. Since women generally report higher levels of SRG than men (Park et al., 1996; Tedeschi & Calhoun, 1996), there may have gender differences in longitudinal changes of SRG as well. Moreover, sample size is a big concern in some studies. For example, using latent growth curve modeling, Manne et al. (2003) failed to find the changes of SRG over time, which was potentially due to too small sample size (N = 162) to detect significant changes.

Second, these studies do not investigate whether SRG from prior stressors can predict SRG from subsequent stressors which naturally occur over people's lives. Generally, most of them merely examine the unfolding process of SRG from a single life stressor. Even though Ickovics et al. (2006) compared SRG across four stressors and found that pregnancy and motherhood or death of a love one led to higher levels of PTG than interpersonal problems in urban adolescent girls, they did not look at whether SRG from pregnancy and motherhood predicted SRG from other stressors such as interpersonal problems.

Third, these studies do not control the potential effect of time interval (i.e., the duration between two assessed times) on the changes of SRG over time. Generally, the time interval of aforementioned studies are relatively short, i.e., within several

weeks, months, or one year. On the one hand, these time interval may not be long enough for the development of SRG (Calhoun & Tedeschi, 1998; Schaefer & Moos, 1998). On the other hand, they may be too long so that initial obtained SRG is lost after a year (Davis et al., 1998; Frazier et al., 2001).

Fourth, the divergent results on changes of SRG may be confounded by stress severity. For example, Schwarzer et al (2006) examined cancer patients ranging from stage Ⅰ to stage Ⅳ, whereas Urcuyo et al. (2005) examined cancer patients from stage 0 to stage Ⅱ. Because people with more severe stressors were much more likely to report higher levels of SRG than those experiencing less severe stressors (Stanton et al., 2006), it is reasonable to assume that the different results of Schwarzer et al.'s increasing pattern of SRG (2006) and Urcuyo et al.'s stable pattern of SRG (2005) may be due to severity of cancer disease.

Fifth, the coping strategies that people use to deal with stress may influence the relations between stress severity and SRG over time (Aldwin et al., 1996; Schaefer & Moos, 1992). Even though researchers (Butler et al., 2005; Urcuyo et al., 2005) explored the effects of coping on SRG over time, they simply examined the prediction of coping on SRG without considering the effects of stress severity on coping. Therefore, the potential mediating effects of coping on stress severity and SRG were absent from the aforementioned studies.

Covariates of SRG. Age, sex, and time interval will be controlled as covariates in both Study 1 and Study 2 given the possible relations with SRG, stress severity, coping strategies, and depressive symptoms. In the overview section, we discuss their relations with the common variables of SRG in both Study 1 and Study 2. In regards to their relations with stress severity and coping strategies and with depressive symptoms, we will separately discuss them in Study 1 and Study 2.

Age and SRG. The relations between age and SRG are still mixed (for review, see Stanton e t al., 2006). Some studies have found non-significant relations between them (Aldwin et al., 1996; Collins, Taylor, & Skokan, 1990; Cordova, Cunningham, Carlson, & Andrykowski., 2001; Kinsinger et al., 2006; Jennings, Aldwin, Levenson, Spiro, & Mroczek, 2006; Littlewood, Vanable, Carey, & Blair, 2008; Siegel, Schrimshaw, & Pretter, 2005; Tomich & Helgeson, 2004). However, negative (Aldwin et al., 1996; Lechner et al., 2003; Manne et al., 2003; Polatinsky & Esprey, 2000; Powell, Rosner, Butollo, Tedeschi, & Calhoun, 2003) and positive relations (McMillen, Zuravin, & Rideout, 1995) between age and SRG are also found.

Sex and SRG. In general, women are more likely to report higher levels of SRG than men (Carboon, Anderson, Pollard, Szer, & Seymour, 2005; Littlewood, Vanable, Carey, & Blair, 2008; Park et al., 1996; Tedeschi & Calhoun, 1996; Weiss, 2002). For example, Tedeschi and Calhoun (1996) found that the SRG reported by traumatized women was twice greater than that of traumatized men did. In contrast, some studies have not found sex differences in SRG (Andrykowski, Brady, & Hunt, 1993; Butler et al., 2005; Ho, Chan, & Ho, 2004; Cheng, Wong, & Tsang, 2006; Fromm, Andrykowski, & Hunt, 1996; Lechner et al., 2003; McCausland & Pakenham, 2003; Pollard & Kennedy, 2007), which is possibly due to the unbalanced data in sex (i.e., more women than men) and insufficient power to detect significant sex differences in SRG.

Time interval and SRG. Time is a critical factor in the development of SRG (Tedeschi & Calhoun, 2004; Tomich & Helgeson, 2004). Most existing studies have ignored this variable when measuring SRG in multiple waves. Time interval in this dissertation refers to the duration between stressful events at two waves, calculated by the ending time at Time 2 minus the ending time at Time 1. Previous studies have

shown that people who report SRG initially may lose it later or people who do not report SRG initially may report it later (Davis et al., 1998; Frazier et al., 2001). This implies a high likelihood of no predictable effects of earlier SRG on subsequent SRG. In other words, if the earlier reported SRG disappears later, it will be very difficult to examine the prediction of SRG over time.

With regard to my study, although Time 1 data was collected at 1996 and Time 2 data was collected at 2001, the reported time interval may be different given varying event ending times at both waves. For example, the time interval for one person is six years (stressor ending years at Time 1 is 1994 and at Time 2 is 2000), and for another person it is two years (stressor ending years at Time 1 is 1996 and at Time 2 is 1998). If SRG at Time 1 has weaker effects on SRG at Time 2 for the first person whereas it has stronger effects on SRG at Time 2 for the second person, it is entirely possibly that the four years difference in time interval partially accounts for these divergent results. Therefore, time interval will be controlled in Study 1 and Study 2.

Measurement Issues of Stress-Related Growth

SRG measures are significantly related to the validity of reported SRG (Park & Lechner, 2006; Tomich & Helgeson, 2004). Although several measures have been developed and used to assess people's growth from stress (Aldwin et al., 1996; Park et al., 1996; Tedeschi & Calhoun, 1996), there still are some debates about these measures, primarily represented by two measurement issues. Namely, is SRG a unidimensional or multidimensional construct? Should SRG surveys include both positively and negatively worded items?

Dimensions of SRG. In general, most qualitative and quantitative studies find SRG is a multidimensional concept. Using open-ended questions, several qualitative studies (Frazier et al., 2001; Fromm et al., 1996; Kennedy, Tellegen, Kennedy, &

Havernick, 1976; McMillen, Smith, & Fisher, 1997) have found SRG is composed of multiple domains, such as improved positive attitude, tolerance and appreciation, religiosity, and decreased perception of money (Kennedy et al., 1976). By reviewing previous literature on life crises and personal growth, Schaefer and Moos (1992) theoretically suggest that SRG primarily includes three domains, i.e., enhanced social resources, enhanced personal resources, and acquisition of coping skills.

Most quantitative research has supported the multidimensional concept of SRG. Park et al. (1996) developed the *Stress-Related Growth Scale* (SRGS) to test the three domains (Schaefer and Moos, 1992), but eventually found that SRG was a unidimensional construct. Other measures, however, established SRG as a multidimensional concept. For example, the *Posttraumatic Growth Inventory* (PTGI) designed by Tedeschi and Calhoun (1996) found SRG included greater appreciation of life and changed sense of priorities, warmer/more intimate relationships with others, greater sense of personal strength, recognition of new possibilities or path for one's life, and spiritual development. Kelley (2006) and Aldwin et al. (2008) found five factors from *Learn from the Low Point* and *Advantage* (Aldwin et al., 1996), including values, resources, confidence, vulnerabilities, and advantages.

Closer examination suggests that Park et al. (1996) and Tedeschi and Calhoun (1996) may not have accurately detected the dimensions of SRG because the principal component analysis (PAC) they use does not match with their research questions and hypotheses. In specific, PAC assumes that there is no correlation across different components (Ramsey & Schafer, 2002; Widaman, 2007). Because Park et al. (1996) argued that the three factors of SRG were interrelated, it is inappropriate to perform PAC. Similarly, Tedeschi and Calhoun (1996) found five components were moderately (r =.35) or highly (r =.63) correlated, which also violate the underlying

assumption of PAC. In this case, Aldwin et al. (2008) and Kelley's (2006) results are more convincing. Based on theoretical and empirical SRG findings, they proposed a relation between SRG indicators and their underlying latent construct of SRG, and then used confirmatory factor analysis to verify this relation.

Items of SRG measures. It is also necessary to underscore item wording effects on SRG measures (Park & Lechner, 2006; Tomich & Helgeson, 2004). Most quantitative measures such as the SRGS (Park et al., 1996) and PTGI (Tedeschi & Calhoun, 1996) are much more likely to create positive bias responses (Park & Lechner, 2006; Tomich & Helgeson, 2004). This may be because they only assess participants' SRG in a positive direction rather than also focusing on a negative direction such as perceived weakness. *Learn from Low Points and Advantages* (Aldwin et al, 1994, 1996) may avoid this problem because it uses both positively and negatively worded items to assess participants' SRG. Given the two measurement issues, *Learn from Low Points and Advantages* is used in both Study 1 and Study 2.

Overview of the Present Studies

In general, the primary purpose of this proposal is to contrast the three theoretical models of GRRs, COR, and DAM by examining longitudinal relations between stress severity, coping, SRG, and depressive symptoms in a large sample of Davis Longitudinal Study (DLS) across two times (Time 1 = 1996, Time 2 = 2001). The main purpose of Study 1 is to test the longitudinal relations between stress severity and SRG. That is, does stress severity affect SRG and does SRG affect stress severity? Do reciprocal relations exist between stress severity and SRG? The other goal is to examine potential longitudinal mediating effects of coping strategies (both positive coping and negative coping) on stress severity and SRG. Presumably, the

effect of stress severity on SRG will be significantly mediated by positive instead of negative coping strategies.

Study 2 examines the effects of stress severity on the relations between two outcomes of stress – depressive symptoms and SRG. Specifically, it is hypothesized that both cross-sectional relations and longitudinal reciprocal relations are stratified by levels of stress severity. That is, when the level of stress severity is low, SRG and depressive symptoms are unrelated, whereas when the level of stress severity is high, they are negatively related.

Does Coping Mediate the Relations between Stress Severity and Stress-Related Growth? Findings from the Davis Longitudinal Study

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Abstract

The present study examined the relationship between stress severity and stress-related growth (SRG), especially whether it is mediated by coping strategies. Longitudinal analyses among young and middle-aged adults (N = 509) assessed twice over five years by mail survey showed that stress severity had weakly positive prediction on SRG, but no cross-lagged effects between them. However, when taking account of the mediating effect of both positive and negative coping strategies, stress severity had no direct but only indirect effects on SRG. Furthermore, there were only significant cross-lagged effects between SRG and positive coping strategies rather than negative coping strategies, indicating that previous SRG did increase subsequent positive coping strategies, and generally if people use more positive coping, they were more likely to report higher levels of SRG later.

Key Words: Stress Severity, Coping Strategies, Stress-Related Growth, Mediating Effects, Cross-Lagged Effects

Even though stress especially traumatic events are much more likely to motivate the development of SRG (Tedeschi & Calhoun, 2004), not all subjects report SRG (Aldwin et al., 1994). For example, even though around 90% of veterans reported positive outcomes from the tremendously traumatic event of combat exposure, still there were 10% of veterans who did not report positive effects (Aldwin et al., 1994). Similarly, among respondents who lost their family members, 73% reported that they gained some positive outcomes (Davis et al., 1998). Despite the high prevalence of SRG among people, the psychological mechanisms between stress and SRG have not been well-identified. One possible mediator is coping strategies that people use to handle their stressors (for a review, see Aldwin, 2007). Given the potential link between stress severity, coping, and SRG, the major purpose of Study 1 is to examine the mediating effects of coping strategies on stress severity and SRG, especially different pathways between positive action coping (PAC) and negative action coping (NAC). In the following sections, we will first discuss the significance of this study and then review three theoretical perspectives and existent empirical literature in stress severity, coping strategies, and SRG.

Significance of This Study

As mentioned in Chapter 1, promoting SRG is particularly important for better physical and psychological health. By examining the mediating effects of different coping strategies on stress severity and SRG, this study may provide better understanding and offer evidence or guidelines for clinicians or other researchers to improve people's SRG in practice. For example, if only PAC strategies have significant mediating effects on stress severity and SRG, counselors may focus on promoting people's PAC to improve their SRG levels.

In addition, the facilitating process of coping on SRG may work in a reciprocal way (Aldwin et al., 1996; Lechner & Weaver, 2009; Schaefer & Moos, 1992). That is, when people report SRG especially a high level of SRG, they may also build certain coping strategies and confidence, and thus further promote their SRG over time. However, most existing empirical literature has examined the predicting or mediating effects of coping strategies on SRG, whereas few studies have addressed the facilitating effects of SRG on coping strategies (Bower et al., 2009). If these relations are identified, it may provide a dynamic and developmental perspective to understand the improvement of both coping and SRG over time.

Literature Review

Stress Severity and SRG

Stress severity, referring to "how troubling or disturbing stressful events are to persons" (Aldwin et al., 1996), may have significant determinant effects on the positive outcomes of stress – SRG (Schaefer & Moos, 1992). However, both theoretical models and empirical studies are mixed in this question.

Theoretical models. Antonovsky (1979, 1987) argued that people suffering from highly severe stress are much more likely to report high levels of SRG, which will decrease subsequent stress severity levels. In contrast, Hobfoll (1988) argued that these people are less likely to report SRG, which will increase subsequent stress severity levels and thus make them more vulnerable to future stressors.

Summary of theoretical models. The two theories describe divergent reciprocal relations between stress severity and SRG, which are presented in two aspects. On the one hand, will stress severity facilitate (Antonovsky, 1979) or undermine (Hobfoll, 1988) SRG development? On the other hand, will SRG decrease (Antonovsky, 1979) or increase (Hobfoll, 1988) subsequent stress severity?

Empirical findings. Empirical studies find inconclusive results as well. Some studies have shown that stress severity and SRG are positively and linearly related, i.e., the more stressful events, the higher SRG levels reported (for a review, see Stanton et al., 2006). For example, Aldwin et al. (1994) found that combat veterans who suffered from greater combat exposure tended to report higher levels of SRG. Tedeschi and Calhoun (2004) argued that highly stressful events were much more likely to challenge individuals' existing world views and thus to promote potential positive outcomes. However, a curvilinear relation between stress severity and SRG has also been found (Fontana & Rosenheck, 1998; Lechner et al., 2003). For example, Lechner et al. (2003) found that cancer patients at Stage Ⅱ had significant higher SRG than those at Stage IV.

Summary of empirical studies. Overall, these empirical studies are limited by their cross-sectional data (Aldwin et al., 1994; Lechner et al., 2003; Tedeschi & Calhoun, 2004). Therefore, little information is known about longitudinal effects of SRG on subsequent stress severity or vice versa. Given this fact, in Study 1 we use a two-wave cross-lagged model to test (a) the predicting effects of stress severity on SRG; (b) the predicting effects of SRG on stress severity; and (c) their potential reciprocal relations within five years.

Coping and SRG

Empirical findings. Certain kinds of coping strategies may have significant adaptational effects (Lazarus & Folkman, 1984) such as promoting SRG (Aldwin, 2007; Holahan, Moos, & Schaefer, 1996; Shaefer & Moos, 1992; Tedeschi & Calhoun, 1995). For example, positive coping strategies, such as problem-focused coping (Koenig, Pargament, & Nielsen, 1998; McCausland & Pakenham, 2003; Park & Fenster, 2004), active coping (Armeli, Gunthert, & Cohen, 2001; Luszczynska,

Mohamed, & Schwarzer, 2005; Urcuyo et al., 2005), positive reinterpretation (Lechner, Carver, Antoni, Weaver, & Phillips, 2006; Park et al., 1996), acceptance (Butler et al., 2005; Urcuyo et al., 2005), and emotional social support (Frazier, Conlon, Steger, Tashiro, & Glaser, 2006; Thornton & Perez, 2006), are strong predictors of SRG. PAC, consisting of both problem-focused and emotion regulation strategies, has also been found to predict SRG (Aldwin et al., 1996; Kelly, 2006).

The relations between negative coping strategies and SRG are mixed. Studies have found positive (Park et al., 2008) or no relations (Park et al. 1996; Urcuyo et al., 2005) between them. However, some studies have found the relations are varied by the types of negative coping strategies, resulting in negative (Butler et al. 2005), positive (Butler et al., 2005; Pargament, Koenig, & Perez, 2000; Widows, Jacobsen, Booth-Jones, & Fields, 2005), or no relations (Butler, 2005; Pargament et al., 2000; Widows et al., 2005). For example, Pargament et al (2000) found passive religious deferral and punishing god reappraisal had no relations with SRG, but other negative religious coping strategies were positively related to SRG. Similarly, Butler et al. (2005) found self distraction and denial were positively related to baseline SRG, whereas behavioral disengagement and emotional venting were negatively related to it.

Summary of empirical studies. These inconsistent results may be accounted for by different types of negative coping strategies. For example, in Pargament et al.'s (2000) study, the negative coping merely referred to negative religious coping. However, in Butler et al.'s (2005) study, the negative coping was much broader including self distraction, denial, behavioral disengagement, emotional venting, and self-blame.

In addition, time interval between baseline and follow-up SRG may account for the inconsistencies. For example, even though denial, behavioral disengagement, and emotional venting were associated with baseline SRG at nine weeks, they were unrelated to follow-up SRG at six and half months (Butler et al., 2005).

Mediating Effects of Coping on Stress Severity and SRG

Theoretical arguments. COR, GRRs, and DAM address the potential mediating effect of coping strategies on stress severity and SRG in different ways. Antonovsky (1979) argued that coping – one of the most effective GRRs – can impede or overcome stressors. The higher the stress severity is experienced, the more coping strategies and GRRs are utilized, and thus the more positive outcomes/SRG are reported. Since the SRG is a GRR per se, it strengthens subsequent coping strategies and thus successfully decreases following stress severity.

However, Hobfoll (1988) argued that coping is a process that consumes a lot of resources. The higher the stress severity is experienced, the more coping strategies are employed, and thus the more resources are depleted. Consequently, fewer resources/SRG are available to counteract future stress, and thus a negative spiral is more likely to occur over time.

The DAM (Aldwin et al., 1996) is a more comprehensive model because its amplification model synthesizes both GRRs and COR. Aldwin et al. argued that stress severity can result in both high and low levels of SRG over time, primarily mediated by types of coping strategies. In other words, if people use more positive coping strategies to deal with stress, they will have higher levels of SRG and lower levels of stress severity subsequently; if people use more negative coping strategies to deal with stress, they will have lower levels of SRG and higher levels of stress severity subsequently.

Empirical discrepancies. Even though a number of empirical studies have investigated the relations between coping and SRG, only a few (Aldwin et al., 1996; Park et al., 2008) have examined the possible mediating effects of coping on stress severity and SRG. Aldwin et al. (1996) found that stress severity was negatively associated with positive coping strategies, which in turn were positively related to SRG. Conversely, stress severity was positively associated with negative coping strategies, which in turn were negatively related to SRG. Park et al. (2008) provided slightly different mediating results. They found that stress severity significantly and positively predicted both positive and negative coping strategies, but positive rather than negative coping had stronger effects on SRG. However, due to cross-sectional designs, these studies were unable to control the effect of previous stress severity on subsequent coping as well as the effect of previous coping on subsequent SRG. Therefore, limited information is provided by these cross-sectional studies, indicating the necessity to further examine the mediating effects using longitudinal data.

Covariates of Age, Sex, and Time Interval

Age, sex, and time interval are controlled in Study 1. Since the relations of age, sex, and time interval with SRG have been addressed in previous chapter, here only the relations with stress severity and coping strategies are explained. In general, younger adults are more likely to appraise events as more stressful (Aldwin, Sutton, Chiara, & Spiro, 1996; Boeninger, Shiraishi, Aldwin, & Spiro, 2009) and to use more negative coping strategies and fewer positive coping strategies (Aldwin et al., 2009). Women instead of men are more likely to report higher levels of stress severity (Hogan, Carlson, & Dua, 2002; Ptacek, Smith, & Zanas, 1992; Tamres, Janicki, & Helgeson, 2002) and to use more emotion-focused and problem-focused coping (Tamres et al., 2002). The time interval between stressors may have significant

impact on stress severity since concurrent multiple-stressors have more negative outcomes than isolated life stressors (Evans, 2004). Because coping strategies tend to be different across stressors (for reviews, see Aldwin 2007) and because the present study examines coping strategies under particular stressful contexts, it is plausible to assume that in the present study previous coping strategies are unrelated or only moderately related to subsequent coping strategies no matter the time interval between stressors.

Research Questions and Hypotheses

The major purpose of Study 1 is to use longitudinal mediation models to examine the longitudinal relations between stress severity, SRG, and coping after controlling for the covariates of age, sex, and time interval. One of the advantages of the longitudinal mediation models is to test within-time mediating effects at each wave as well as cross-time mediating effects simultaneously (MacKinnon, 2008).

The first primary aim of Study 1 is to examine whether there are longitudinal relations between stress severity and SRG over two waves, which is illustrated in Figure 1.1. This two-wave model mainly includes three parts, which are autoregressions, within-time direct effects, and cross-lagged effects. The autoregressive effects include stress severity at Time 1 to stress severity at Time 2 and SRG at Time 1 to SRG at Time 2. The within-time direct effects include stress severity at Time 1 to SRG at Time 1 and stress severity at Time 2 to SRG at Time 2. The cross-lagged effects include stress severity at Time 1 on SRG at Time 2 and SRG at Time 1 on stress severity at Time 2, controlling for the covariates of age, sex, and time interval. Based on the literature reviewed above, we make the following hypotheses:

Hypothesis 1a: If Antonovsky (1979) is correct, the autoregression of stress severity should be negative (i.e. β_1) while that of SRG (i.e. β_2) should be positive; stress severity should positively predict SRG (i.e., β_3, β_4, and β_6 are positive); SRG at Time 1 should negatively predict stress severity at Time 2 (i.e., β_5 is negative).

Hypothesis 1b: If Hobfoll (1988) is correct, the autocorrelation of stress severity (i.e. β_1) should be positive while that of SRG (i.e. β_2) should be negative; stress severity should negatively predict SRG (i.e., β_3, β_4, and β_6 are negative); SRG at Time 1 should positively predict stress severity at Time 2 (i.e., β_5 is positive).

Hypothesis 1c: If Aldwin et al. (1996) are correct, the direct effects of stress severity on SRG should be weak because most of the effects are mediated by coping strategies.

The second aim is to test possible longitudinal mediating effects of PAC on stress severity and SRG, which is illustrated in Figure 1.2. Figure 1.2 includes three autoregressions, two within-time mediating effects, and five cross-lagged mediating effects. Specifically, the three autoregressions are stress severity, PAC, and SRG over two waves. Besides the autoregression, two additional aspects are illustrated in Figure 1.2. One aspect is the within-time mediating effect of PAC on stress severity and SRG, and the other includes the lagged mediating effects of stress severity at Time 1 on SRG Time 2 via PAC at Time 1, and the lagged mediating effects of SRG at Time 1 on SRG at Time 2 via PAC at Time 2.

Hypothesis 2: We hypothesize that if Aldwin et al. (1996) are correct, most effects of stress severity on SRG should be positively mediated by PAC both within and across time.

Hypothesis 2a: Autoregressions of stress severity at Time 1 to stress severity at Time 2, SRG at Time 1 to SRG at Time 2, and PAC at Time 1 to PAC at Time 2

should be positive. Because coping strategies are different from situation to situation (Aldwin, 2007; Lazarus & Folkman, 1984), we further hypothesize that the PAC should be moderately correlated over time.

Hypothesis 2b: PAC should significantly and positively mediate the effect of stress severity on SRG within Time 1 and Time 2.

Hypothesis 2c: PAC at Time 1 should significantly and positively mediate the effect of stress severity at Time 1 on SRG at Time 2 as well as the effect of SRG at Time 1 on SRG at Time 2.

The third aim is to test longitudinal mediating effects of NAC on stress severity and SRG (see Figure 1.3). Similar to Figure 1.2, three autoregressive effects including stress severity, NAC, and SRG, and two within-time mediating effects of NAC on stress severity and SRG at each wave are examined.

Hypothesis 3: In addition to the positive autoregressive effect of stress severity and SRG, the autocorrelation of NAC is also positive and moderate. As earlier studies using this dataset showed no significant effect from NAC to (Bi, Taylor, & Aldwin, under review), we hypothesize that NAC has no effect on SRG and thus the relations between stress severity and SRG are not mediated by NAC both within time and across time.

Hypothesis 4: In regard to the effect of age, sex, and time interval, we hypothesize that younger people report higher levels of stress severity and SRG, and use fewer PAC and more NAC. Similarly, women report higher levels of stress severity and SRG, and use more PAC and NAC. The time interval between two events is negatively related to stress severity and SRG at time 2 and unrelated to PAC and NAC at time 2.

Method

Sample and Procedure

All participants in this study are from the Davis Longitudinal Study (DLS), an ongoing study of alumni of the University of California, Davis (Aldwin et al., 1996; Yancura & Aldwin, 2009). The study began in 1968-1970, with new cohorts added every ten years. The initial survey was mailed to every alumnus who graduated in 1967, l968, and 1969. For subsequent cohort surveys, a random sample of recent graduates was asked to participate. The present study uses two-wave data collected in 1996 (Time 1) and 2001 (Time 2) from young and middle-aged alumni of the DLS (Aldwin et al., 1996). Because a new cohort was added (1999 alumni) in 2001 who did not participate in the 1996 study (n = 228), these individuals are not included in any analyses.

Table 1.1 displays the procedure of sample size selection. Participants were asked to indicate their most recent low points (i.e., serious stressors), ranging from problems with children to death of a family member, and then to rate the stress severity of their most recent low point, ranging from 1 (*not at all stressful)* to 7 (*most stressful thing ever experienced)*. If participants did not report stress severity, they were excluded from the present study. In this case, for 1996 data 30 respondents were excluded and 898 were included, and the excluded respondents had higher levels of family income than those included, $t(900) = 1.69$, $p < .05$. For 2001 data, 36 participants were excluded and 1,140 were included (among them, 220 was a new cohort), and the excluded respondents were younger than those kept, $t(1,167) = 1.99$, $p < .05$.

Then, the two dataset without missing stress severity were merged and only those answering both Time 1 and Time 2 were kept, resulting in the sample size of

592. There were few statistically significant demographic differences between the participants excluded and those included in the longitudinal analysis. For 1996 data, those excluded respondents were 3.56 years younger than those included, $t(895) = -.6.71$, $p < .001$. Minority groups were much more likely to be excluded than European Americans (11.55% *vs*. 6.98%), $X^2(1, n = 890) = 5.34$, $p < .05$. Respondents with lower income were much more likely to be excluded, $t(881) = -3.14$, $p < .001$, and married respondents were much more likely to be included than respondents with other marital status (23.60% *vs.* 31.58%), $X^2(1, n = 893) = 6.58$, $p = .01$.

The time interval was calculated by subtracting the ending year at Time 2 from the ending year at Time 1. Given missing values in the ending year at both times, other related variables were used to compute it. If a stressor was ongoing and its ending year was missing, the ending year was recoded as the year of survey. In this case, 149 and 55 observations were replaced for 1996 and 2001 ending years, respectively. If a stressor's ending year was missing, but its starting year was not missing and the aftermath of the stressor was not ongoing, the low ending year was calculated based on event duration. If an event lasted less than a year (i.e., 1-6 on 10 Likert-scale), the ending year was coded as the starting year. In this way, 201 and 81 observations were replaced for 1996 and 2001 data, respectively. If an event lasted more than a year (i.e., 7-10 on 10 Likert-scale), the low ending year was computed by the starting year plus one year for those event durations that were about a year (i.e., 7 on 10 Likert-scale) or more than a year (i.e., 8 on 10 Likert-scale), 2 years to 4 years for those event durations from 2 to 4 years (i.e., 9 on 10 Likert-scale), or 5 years for those event duration five+ years (i.e., 10 on 10-Likert-scale), with the maximum year

for the low ending year being 1996 and 2001. Finally, 64 and 6 observations were replaced for 1996 and 2001 data, respectively.

If respondents reported the same type of stressors and starting years in 1996 and 2001 and the stressors were over in 1996, these respondents were excluded (n = 33). If the ending year in 2001 data occurred prior to the ending year in 1996 data, these respondents were also excluded (n = 19). If the ending year in 1996 data occurred prior to 1991, those respondents were also excluded (n = 31). Thus, the final sample size for the present study was 509 whose time interval ranged from 0 to 11 years.

As Table 1.2 shows that among the 509 respondents, nearly half of them (44.01%) were male, and their age ranged from 28 to 74 (M_{age} = 44.24, SD = 7.38). The majority (93.47%) were European Americans. As these were alumni, all of them had at least a bachelor's degree and nearly half (52.45%) had advanced degrees. Approximately two-thirds (76.28%) of the participants were married and ten percent (10.67%) were single or had never been married. The majority worked full time (79.72%) and the modal income was between $75,000 and $99,999.

Measures

Demographics or covariates. The questionnaire asked participants to indicate their age, sex (0 = *Male*, 1 = *Female)*, and beginning year and ending year of their recent stressors. As described above, the time interval between two stressors were calculated by the ending year at Time 2 minus the ending year at Time 1. Since the ending year ranges from 1991 to 1996 at Time 1 and from 1996 to 2001 at Time 2, the scope of time interval is from 0 to 11 years.

Stress severity. Participants were asked to rate how troubling or disturbing their most recent stressors to them on a 7-point Likert scale (1 = *Not at all stressful,* to 7 = *Most stressful thing ever experienced*).

California Coping Inventory (CCI). Coping was assessed by the *California Coping Inventory* (CCI; Aldwin, 1994; Aldwin et al., 1996; Aldwin, Shiraishi, Cupertino, 2001). The 50 items were rated on a 4-point Likert scale (0 = *Not at all*, 3 = *Used a lot*). Participants were asked to identify their most recent stressors and the frequency with which they used each strategy. Previous factor analyses showed that the CCI includes five coping subscales: *Positive action coping* (20 items)*, negative action coping* (12 items)*, prayer* (3 items)*, withdrawal* (11 items)*, and substance use* (3 items) (Aldwin et al., 2001). In our study, we focused on two subscales of *Positive Action Coping* (PAC) and *Negative Action Coping* (NAC), which contained both problem-focused and emotion-focused coping, e.g., "focused on managing the problem", "told myself to calm down", "imagined ways of retaliating", and "expressed hostility to the other person(s)".

Stress-Related Growth. SRG is a latent variable including factors of confidence, resources, values, advantages, and vulnerabilities derived from the *Learn from the Low Point and Advantages* checklists (Aldwin et al, 1994). The SRG measure consists of two sections. First, respondents were asked if they could turn any part of their low point to their advantage and checked as many as five response options including "no", "yes, emotional well-being", "yes, tangible advantage/gain", "yes, developed a new philosophy/attitude toward life", and "yes, other". All the four "yes" items were separately coded as 1, which were then summed to obtain a total advantages score ranging from 0 to 4. Second, respondents indicated how much they learned from the experience of their low point on a scale of 0-3, where 0 = *Not at all*

to 3 = *A lot*. Items were divided into four domains: confidence (3 items), resources (3 items), values (4 items), and vulnerabilities (4 items) (Aldwin et al., 2009; Kelly, 2006). Sample items include "I could stand on my own two feet" (confidence), "I had positive social resources (e.g., good friends, neighbors, family)" (resources), "Religion/spirituality is very important to me" (values), and "There are some situations that I cannot do anything about" (vulnerabilities).

In this study, a modified version of Kelly's SRG construct (2006) was used. First, due to missing values in the 14 items, the four factors derived from them were created by computing the mean (Acock, 2006) rather than total scores of the answered items in each factor. For example, if one of three resources items does not answered, the resources factor will contain the mean of the two answered items; if all items are answered, the resources factor will contain the mean of all three items; if no items are answered, the resources factor is set to missing. Second, Bi et al. (under review) examined the stability of the factor structure of SRG by randomly splitting the 2001 DLS data into two equal halves. Both factor structures were identical, but there was one residual correlation between SRG indicators of values and resources in both subsamples. The residual correlation is positive, which is probably due to the common underlying factor of social support, as both the values and the resources indicators are related to friends and family. Longitudinal factorial invariance for SRG was also examined (Bi et al., under review), which showed that weak factorial invariance, i.e., equal factor loadings, was the best fit to the data.

Statistical Analyses

SEM was used to examine three hypothesized cross-lagged models (Figure 1.1, Figure 1.2, & Figure 1.3) controlling for age, sex, and time interval at Time 1. If the hypothesized models did not have good fits to the data or if some hypothesized

paths were not significant, the models would be re-estimated by dropping non-significant path one by one until all remained paths were significant with good model fits. For example, if the two cross-lagged effects in the first model (Figure 1.1) were not significant, the model would be re-run by dropping the cross-lagged path with the larger *p* value. If another path was still insignificant, the same step would be performed. The final model would be the one with a good model fit and all statistically significant paths.

Analyses were conducted using Mplus 5.21 (Muthén & Muthén, 2009). Maximum likelihood estimation and missing at random were used to handle parameter estimation and missing data. Four model fit criteria including chi-square statistic, comparative fit index (CFI), root mean square of error of approximation (RMSEA), and standardized root mean squared residuals (SMSR) were used to evaluate the three hypothesized models. Since chi-square statistic is greatly influenced by large sample size ($N = 509$ in present study), a significant score of chi-square statistic is expected and may be unreliable as a model fit index (Kline, 2004). Given this fact, this study mainly relied on other three model fit criteria to assess whether the hypothesized SEM model fit the data or not. The CFI greater than .90 (Kline, 2004), the RMSEA from .05 to .08 (Browne & Cudeck, 1993), and the SMSR less than .08 indicate acceptable model fit (Hu & Bentler, 1999).

Results

Demographic Descriptions

A wide variety of types of problems were reported at both time points (See Table 1.3). The most frequently reported problems at Time 1 were career or work problem (16.90%), marital/relationship problems (9.82%), parent's health (8.25%), physical health problems (self) (7.47%), and parent's death (6.88%). At Time 2,

however, the pattern of stressors had changed as the respondents moved into mid-life. Even though career and work problems were still the most stressful problem at Time 2, the percentage was decreased a lot. The number of respondents who lost their parents increased nearly two-fold, indicating that the DLS participants were shifting their focus from work and career to parents' deaths. Specifically, the most frequently reported problems were career/work problems (11.79%), parent's deaths (11.20%), marital/relationship problem (10.81%), physical health problems (self) (8.45%), and parent's health (8.25%). Despite this change in types of problems faced, there were no differences in the mean levels of stress severity across the two time points (5.27 and 5.31, respectively; See Table 1.4), which suggested that most participants reported moderately high life problems on the 7-point stressfulness scale.

As mentioned above, there were four SRG factors (i.e., confidence, vulnerabilities, resources, and values) derived from 14 items of learn from low points. Given missing values in these items, the four SRG factors were calculated by mean instead of total scores of the answered items. The mean values of all four factors ranged from 0 to 3 (see Table 1.4 for *M* and *SD*). "I could stand on my own two feet" was the most frequently checked SRG confidence item (85.34% at Time 1 and 87.88% at Time 2). Two SRG resources items of "I had positive psychological resources (e.g., ability to cope)" and "I had positive social resources (e.g., good friends, neighbors, family)" were frequently checked (93.79% & 92.02% at Time 1; 94.74% & 92.26% at Time 2). Two SRG values items of "family is very important to me" and "taking care of myself is very important to me" were most frequently reported (95.69% & 91.15% at Time 1; 95.29% & 93.95% at Time 2). The least frequently checked item of SRG vulnerabilities was "my health prevented me from doing as much as I would have liked" (16.60% at Time 1 and 20.79% at Time 2).

With regard to the four binary items of SRG advantage checklist, the summed total score was computed. The most frequently checked item was "developed new philosophy/attitude towards life" (45.95% at Time 1 and 41.85% at Time 2).

Correlations among Study Variables

Table 1.5 presents the correlations among all of the study variables for both Time 1 and Time 2. The discussion here will primarily focus on correlations within time.

Age had only a modest correlation with the study variables. The hypothesis that younger people rated their problems as more stressful was not supported. At Time 1, older adults were slightly more likely to rate their stressors as more stressful (r = .09, p < .05), but the correlation was not significant at Time 2 (r = -.04). The only consistent cross-time correlation was the PAC: older people were more likely to use PAC at both time points (rs = .11 & .12, ps< .05 & .01). Consistent with the hypotheses, females reported problems as slightly more stressful (rs = .11 & .10, ps < .05). They also reported higher levels of SRG indicators at both time points (rs ranging from .10 to .21) and more likely to use PAC within both time points (rs = .19 & .08, ps< .05 & .001). Stress severity was significantly related to both PAC and NAC (rs ranging from .13 to .23) as well as most SRG indicators (rs ranging from .08 to .27). PAC was positively associated with all SRG indicators at both time points (rs ranging from .29 to .60). In contrast, NAC was only consistently correlated with SRG confidence (rs = .15 & .12, ps< .001 & .01) as well as with SRG vulnerabilities (rs = .28 & .27, ps < .001). All SRG indicators were positively correlated within time (rs ranging from .12 to .68).

The time interval was unrelated to most variables in present study, except the negative relations with stress severity and SRG advantages at Time 2, indicating that

the longer time interval, the lower the reported stress (r = -.15, p < .001), and the fewer the perceived advantages, (r = -.09, p < .05). Given the time interval was unrelated to most variables in the present study, it was not included in all SEM models.

Exploratory Analyses of Type of Stressors and SRG

Exploratory analyses using ANOVA in 2001 DLS dataset were conducted to test whether mean levels of SRG were varied by type of stressors. 32-item Low Points Scale (Aldwin, et. al, 1996) was used to examine people recent low points in their lives. Boeninger, Shiraishi, & Aldwin (2003) categorized the 32 items of recent low points into seven types of stressors, including family, work, health, angst, parents' health, bereavement, and others. We followed their classification, and compared the mean level of SRG among the seven types of stressors. In general, the ANOVA results showed that there were no significant differences in the type of stressors and SRG indicators, at least for SRG resources and SRG confidence, and few differences were found between types of stressors and the other three SRG indicators. Further effect size results showed that the variance in SRG indicators were explained by types of stressors were very small, which were 1.10%, 1.40%, 1.90%, 2.00%, and 2.80% for SRG confidence, resources, values, advantages, and vulnerabilities, respectively.

The mean of SRG values were varied by type of stressors, F(6, 1,119) = 3.71, p < .01. Specifically, respondents experiencing bereavement stressor (M = 2.25) reported higher mean level of SRG values than those experiencing family-related stressor (M = 2.02, p < .05), work-related stressor (M = 1.97, p < .001), and other type of stressor (M = 1.97, p < .05). There also had significant differences in the mean level of SRG vulnerabilities among types of stressors, F(6, 1,112) = 3.80, p < .001, with respondents experiencing health stressor (M = 1.19) reporting higher mean

vulnerabilities score than those experiencing stressors related to parents' health (M = .97, p < .05), and bereavement (M = 1.00, p < .05). The mean level of SRG advantages were also significant different, F(6, 1,129) = 5.38, p < .001. Namely, respondents experiencing work-related stressor (M = 1.23) reported higher score than those experiencing family-related stressor (M = .98, p < .05), parents' health (M = .86, p < .01), and other stressors (M = .71, p < .001), and respondents experiencing angst (M = 1.17) reported higher score than those with other stressors (M = .71, p < .05).

Longitudinal Analyses of Stress Severity and SRG

The hypothesized SEM model of stress severity and SRG was constructed based upon the theoretical model presented in Figure 1.1. The overall fit statistics suggested that the hypothesized model had a good fit to the data: X^2(64, N = 509) = 174.951, p < .001, CFI = .936, RMSEA = .058, and SRMR = .050. However, age was not significantly related to SRG at Time 1, and the cross-lagged paths between stress severity and SRG were also not significant. Therefore, the model was re-run, dropping those three paths.

The re-estimated model results showed that the overall fit was still good: X^2(67, N = 509) = 179.903, p < .001, CFI = .934, RMSEA = .058, and SRMR = .053, accounting for 8.1% and 33.3% of the variation in SRG at Time 1 and Time 2, respectively. As can be seen in Figure 1.4, both age and sex had positive direct effects on stress severity at Time 1 (βs = .107 & .123, p < .05). These results indicated that older people and women reported higher levels of stress severity than younger people and men, as hypothesized.

The autoregressive effects were significant for both stress severity (β = .310, p < .001) and for SRG (β = .567, p < .001), indicating that both were moderately to strongly influenced by the prior assessments. Further, while the within-time

associations between stress severity and SRG were significant (βs = .100 & .084, ps < .05), the cross-lagged paths were not significant.

This complex pattern of results provides partial support for both Hobfoll (1988) and Antonovsky (1979). One the one hand, the positive autocorrelation for stress severity supports Hobfoll's accumulation of stress model, whereas the positive autocorrelation for SRG, as well as the positive within-time effects between stress severity and SRG, support Antonovsky's proposition about accumulation of SRG over time. However, the lack of cross-lagged correlations contradicts both models, suggesting that the two models may be incomplete. Thus, we examined Aldwin et al.'s (1996) hypothesis of the mediating effect of coping strategies on stress severity and SRG.

Coping as a Mediator between Stress Severity and SRG

The second model estimated the mediating effects of PAC on stress severity and SRG both within time and across times, along with the autoregressive effects of stress severity, PAC, and SRG. The hypothesized model yielded a good model fit: $X^2(85, N = 509) = 215.096$, $p < .001$, CFI = .944, RMSEA = .055, and SRMR = .045. However, there were three non-significant paths, including the effect of age on SRG at Time 1, stress severity at Time 1 to SRG at Time 2, and PAC at Time 1 to stress severity at Time 2.

After dropping the three paths, the effect from SRG at Time 1 to stress severity at Time 2 became non-significant, and this path was then deleted in the final model (see Figure 1.5). The final model still fit well to the data: $X^2(89, N = 509) = 220.647$, $p < .001$, CFI = .944, RMSEA = .054, and SRMR = .047, accounting for 59.6% and 58.1% of the variation in SRG at Time 1 and Time 2, respectively.

Although stress severity had weak direct effects on SRG within time in the previous analysis (see Figure 1.4), after PAC was added to the model, stress severity no longer had direct effects on SRG within time (see Figure 1.5). However, stress severity had positive direct effects on PAC at both times (β = .189, p < .001, & β = .086, p < .05). Further, PAC had moderate to strong direct effects on SRG at both times (βs = .732 & .532, ps < .001).

As in the previous analyses, the autocorrelation of SRG over time (β = .553, p < .001) was still higher than that of stress severity over time (β = .310, p < .001). Consistent with our hypothesis, there was a moderate autocorrelation of PAC (β = .341, p < .001).

SRG at Time 1 did play a beneficial role on subsequent PAC, i.e., the higher SRG at Time 1, the more PAC at Time 2 (β = .203, p < .01), supporting Antonovosky's model. However, contrary to our hypothesis, the PAC at Time 1 had a negative direct effect on SRG at Time 2 (β = -.223, p < .01), supporting Hobfoll's model. The indirect effects among stress severity, PAC, and SRG were examined as well (see Table 1.6). As indicated above, stress severity had no direct effects on SRG within time when the mediating effect of PAC was taken into account. However, the indirect effects via PAC were significant at Time 1(β = .138, p < .001) as well as at Time 2 (β = .046, p < .01). These mediational effects within time support Aldwin and her colleagues' arguments (1996) that the effects of stress severity on SRG are mainly mediated by PAC.

Besides the direct effect from SRG at Time 1 to SRG at Time 2, its indirect effect was also significant, mediated by the PAC at Time 2 (β = .108, p < .01). Thus, the total effect from SRG at Time 1 to SRG at Time 2 was .641(p < .001).

Also, the PAC had a longitudinal mediating effect between stress severity at Time 1 and SRG at Time 2, which was largely positive ($\beta = .095$, $p < .001$). Although one of the indirect pathways was negative, the majority was positive (see Table 1.6).

In general, this pattern of results supports Aldwin et al.'s (1996) DAM. Namely, if people use the PAC to deal with their stressors, they are more likely to have a higher level of SRG, which in turn increases their positive coping strategies in future, leading to a higher level of subsequent SRG. The only contrary finding was the negative pathway from PAC at Time 1 to SRG at Time 2. However, as can be seen in Table 1.6, the positive indirect pathways ($\beta = .650$) compensated for the negative direct pathway ($\beta = -.223$), leading to a positive total effect ($\beta = .427$, $p < .001$).

The third model examined the mediating effects of NAC on stress severity and SRG. The theoretical model yielded a good fit: $X^2(88, N = 509) = 250.023$, $p < .001$, CFI = .913, RMSEA = .060, and SRMR = .061. However, there were several non-significant paths, including age to NAC and SRG at Time 1, sex to NAC at Time 1, stress severity at Time 1 to SRG at Time 2, NAC at Time 1 to stress severity at Time 2, and SRG at Time 1 to stress severity at Time 2. The modification indices indicated that the within-time effect from NAC to SRG were significant, which made sense based on Park et al.'s (2008) findings. After trimming the six non-significant paths and adding the two significant paths, the model was re-estimated and also produced a good fit (Figure 1.6): $X^2(93, N = 509) = 247.002$, $p < .001$, CFI = .918, RMSEA = .057, and SRMR = .060. Although the model was a good fit, the effect sizes of NAC were much weaker than of PAC, accounting for 9.8% and 33.5% of variation in SRG at Time 1 and Time 2. In contrast, the effect sizes of PAC were 59.6% and 58.1%, as reported earlier, suggesting that positive coping strategies rather

than negative coping strategies are more important for SRG development. As shows in Figure 1.6, the autocorrelations of stress severity and SRG were .310 and .563 (*p*s < .001). Similar to PAC in Figure 1.5, NAC also has a moderate cross-time correlation (β = .314, p < .001), which supports the hypothesis of a moderate association between coping processes over time.

In contrast to PAC, age and sex had no direct effects on NAC. However, the effect from age to stress severity, and sex to stress severity as well as to SRG, were still significant (βs = .107, .123, & .258, *p*s < .05, .01, & .001, respectively). Even though the NAC had direct effects on SRG at both times (β = .171, p < .001, & β = .107, p < .05), the effects were very weak. As expected, both the NAC and SRG had no significant cross-lagged effects on one another.

The indirect effects for this model were also examined (see Table 1.7). Contrary to our hypothesis, NAC had positive mediating effects at both Time 1 (β = .034, p < .01) and Time 2 (β = .028, p < .05). However, the magnitudes of these effects were much smaller than those of PAC. The total indirect effect of NAC on SRG at Time 2 was positive (β = .13, p < .001).

Further, there was no significant meditational effect between stress severity at Time 1 and SRG at Time 2 for NAC itself. However, the indirect effects via more paths were significant. These paths included NAC at Time 1 and SRG at Time 1 (β = .018, p < .05), NAC at Time 1 and Time 2 (β = .008, p < .05), and stress severity and NAC at Time 2 (β = .008, p < .05), which yielded a total indirect effect was .034 (p = .001).

Neuroticism, Stress Severity, Coping, and SRG

Given the moderate to high levels of autocorrelation of stress severity, coping, and SRG, we conducted an exploratory analysis to determine whether personality –

specifically, neuroticism – was a potential confounding factor for the three autocorrelations. Partial correlations were used to examine the impact of neuroticism assessed by the EPI-Q (Floderus, 1974) on the autocorrelations of stress severity, coping, and SRG. In all instances, the autocorrelations remained significant, and most did not change more than one point.

Discussion

Summary of Results

The major purpose of this study was to test potential reciprocal paths between stress severity and SRG and further to analyze how these relations were mediated by coping strategies over two time points. Specifically, we examined how previous levels of stress severity predicted subsequent SRG levels, and, in turn, how SRG levels affected the stress severity of subsequent episodes. Furthermore, we investigated whether and how positive and negative coping strategies played different mediating roles on the potential reciprocities between stress severity and SRG over time.

As reviewed earlier, the literature on stress severity and SRG is mixed, both theoretically and empirically. Anotonovosky (1979) predicted that stress severity mobilizes resources and that there is an accumulation of resources over time. Thus, stress severity should be positively related to SRG both cross-sectionally and longitudinally. That is, SRG at Time 1 is positively related to SRG at Time 2, and negatively related to stress severity at Time 2.

In contrast, Hobfoll (1988) suggested that coping with stress depletes resources and creates further vulnerabilities. Thus, stress severity should be negatively related to SRG both cross-sectionally and longitudinally. That is, SRG at

Time 1 is negatively related to SRG at Time 2 and positively related to stress severity at Time 2.

Finally, Aldwin et al. (1996) argued that the long-term effects of a stressor are mediated by coping. They proposed a contingent model – the relations between SRG and future stressors should be mediated through the type of coping strategies. Thus, positive or negative associations can be found over time, varying by coping strategies.

The first hypothesis addressed the within-time and cross-lagged relations between stress severity and SRG. Stress severity had weakly positive effects on SRG within time, supporting Antonovsky's (1979) positive associations between stress severity and SRG. However, there were no cross-lagged effects from either stress severity at Time 1 to SRG at Time 2 or from SRG at Time 1 to stress severity at Time 2. Therefore, neither Antonovsky nor Hobfoll's (1988) theories were supported.

However, when coping strategies were entered into the model, stress severity now had significant indirect effects on SRG, mediated by PAC and NAC. Further, there were significant cross-lagged effects between coping and SRG – at least for PAC, but not NAC.

The results supported Aldwin et al.'s (1996) DAM. That is, stress severity leads to more positive coping, which in turn leads to higher SRG levels both within and across time. Reciprocally, SRG at Time 1 leads to higher positive coping at Time 2, reinforcing the probability of experiencing SRG at Time 2, supporting Hypothesis 2.

Hypothesis 3 was only partially supported. Unexpectedly, we found that NAC was positively associated with SRG at both time points. This is similar to Park et al.'s (2008) study, which found weak associations between negative coping and PTG. Further, none of the cross-lagged effects with NAC were significant. Thus, even

though the NAC had significant mediating effects between stress severity and SRG within time, it did not have direct effects across time. In other words, NAC at Time 1 by itself did not directly mediate the effects from stress severity at Time 1 to SRG at Time 2, although the indirect path via SRG was significant. Also, the SRG at Time 1 did not predict subsequent NAC.

Another interesting result from this study was there were negative instead of positive direct and indirect effects from PAC at Time 1 to SRG at Time 2. This finding partially supported Hobfoll's (1988) argument that in the process of dealing with stressors, coping strategies do consume resources, which leads to lower levels of SRG afterwards. However, the indirect effects from stress severity at Time 1 to SRG at Time 2 via other paths were positive, which yielded the total indirect effect was significantly positive. Therefore, in general, Aldwin et al.'s (1996) DAM was still supported; namely, if people use PAC to cope with their stressors, they are more likely to report higher levels of SRG later.

The present study only examined one part of the DAM; namely that, the mediating effects of the PAC and NAC on stress severity and SRG. However, the possible mediating effects between stress severity and the negative outcomes of stress (e.g., depressive symptoms) via coping strategies were not tested. Therefore, future studies need to test another aspect of this model.

Age Effects

The relation between age and stress severity was also examined. However, contrary to our hypothesis, age was positively associated with perceived stress severity, i.e., older adults reported higher levels of stress severity than younger adults. This result was not consistent with prior studies. For example, Boeninger et al. (2009) found older people were less likely to report higher levels of stress severity

than younger people. This was possibly due to two reasons. First, there were demographic differences between our study and Boeninger et al.'s study. Specifically, the respondents in the present study (M_{age} = 44.23) were 20 years younger than those respondents in Boeninger et al.'s study (M_{age} = 65.02). Also, in the present study, nearly half of the respondents (44.01%) were male, whereas Boeninger et al.'s study exclusively included males. Second, differing stressor characteristics may also explain the divergent results between the two studies. Boeninger et al.'s study was looking at everyday stressors; in contrast, the present study was looking at major life stressors. Thus, the contrast between type of stressors, age and sex differences in stress ratings (see Tamres et al., 2002) may account for the differences between these studies.

Further, there were no direct effects of age on SRG at either time point. However, there were significant indirect paths through both stress severity and coping. Age was positively related to both stress severity and coping, which in turn increased SRG. Therefore, older adults are more likely to use positive coping strategies to increase their chances of more positive outcomes from their stressful events. Given that the positive coping strategy assessed here included both problem-focused coping and self-regulation strategies, these results are consistent with existing literature, which shows possible increases in self-regulation with age (e.g., Aldwin, Yancura, & Boeninger, in press; Berg & Upchurch, 2007).

The relations between age and coping strategies, including both PAC and NAC, were mixed in previous studies. We found that middle-aged adults used more PAC than younger people, supporting that these older adults in some ways cope with stressful events much better than younger adults (Coates & Blanchard-Fields, 2008). However, no age differences were found in the utilization of NAC, contradicting

earlier studies (Aldwin, 1991; McCrae, 1989); namely that, older people were less likely to use negative coping (e.g., escapist and hostile strategies) than younger people. In addition, others (e.g., Folkman, Lazarus, Pimley, & Novacek, 1987) found that older people were more likely to use escapist coping strategies. The divergence between these studies may be due to the difference in the age structure of the samples. The present study primarily compared middle-aged with younger adults and did not have the older range of ages found in the other studies.

Sex Effects

Consistent with our hypotheses and much of the literature (for a review, see Tamres et al. [2002]), sex was associated with stress severity, with women reporting higher levels of stress than men. However, the findings regarding sex and coping were inconsistent with our hypothesis. In this study, we found that women were more likely to use PAC than men, whereas no sex difference was found in the utilization of NAC. This result is conflicting with previous findings that men were more likely to use problem-focused coping than women, but were less likely to use emotion-focused coping (Pearlin & Schooler, 1978; Stone & Neale, 1984). However, in Tamres et al.'s meta-analysis study, they found that compared with men, women were more likely to use both problem- and emotion-focused coping, whereas no sex differences were found in some specific coping behaviors, such as venting and self-blame. Since the PAC in this study includes both problem-focused and emotion-regulation coping strategies, and the NAC includes blame (e.g., "blamed others"), venting (e.g., "yelled or cursed" and "threw or punched things"), the results of the present study are consistent with the findings of Tamres et al. (2002).

Coping Strategies across Time

This study examined coping with specific situations instead of general coping styles. Therefore, the coping strategies over time should be moderately correlated (Fondacaro & Moos, 1987; McCrae, 1989). However, the autocorrelation of PAC in present study was relatively high, $r = .50$. This was possibly because more than 10% participants whose stressors at Time 1 (but extended time) were similar to their stressors at Time 2 were included in the present study given that this study was interested in how previous stressors influenced subsequent (and continuing) stressors (Note that respondents had to deal with a continuing stressor – that is, cases in which the respondents reported the same ending date of a stressor in 1996 and 2001 were omitted). Thus, it is not surprising that the cross-time correlations might be higher than expected.

Limitations and Future Studies

This study is the first to examine both within-time and cross-time mediating effects of PAC and NAC on stress severity and SRG. But, notably, there were several limitations.

Demographic characteristics. First, given the sample's homogenous sociodemographic characteristics, we must be careful in generalizing the results of present study to a broader population. Specifically, most participants were adults in (or approaching) mid-life, mostly ranging in age from 30's to 50's, and few older adults were included in this study. Thus, the lack of a wider range in age limits the generalization of the findings. Also, most respondents in the present study were highly educated European Americans, and their average family income was high. Therefore, the results of this study need to be replicated among people with different socioeconomic status (SES), such as other ethnic groups, less educated and lower income people, because individuals with lower SES might reported higher or lower

levels of SRG than those with higher SES (for a review, see Lechner & Weaver, 2009).

SRG across different time points. The autocorrelation of SRG was surprisingly high. In part, this may reflect the same stressors with the coping stability mentioned earlier. That is, individuals whose stressors were ongoing from Time 1 to Time 2 were included so that we could examine the development of SRG. It is also possibly due to confounding factors of personality (Evers et al., 2001; Garnefski, Kraaij, Schroevers, & Somsen, 2008). Thus, we conducted exploratory analyses to examine the partial autocorrelations for SRG indicators after neuroticism was taken into account. The results showed that neuroticism had only minimal effects on these autocorrelations. However, the present study did not test whether other relevant personality factors (e.g., extraversion & conscientiousness) (Garnefski et al., 2008) make contributions to SRG continuity over time.

Methodological issues. The results of this study are limited by its two waves of data. Researchers (MacKinnon, 2008; Singer & Willett, 2003) have argued that three or more waves of data tend to provide more precise results. Thus, future studies should use more than two waves of data to further explore the longitudinal mediating effects of coping on stress severity and SRG. Nonetheless, this is one of the first studies to examine how SRG in one episode influences SRG in future episodes.

It should also be noted that SRG in the present study was assessed retrospectively. Some researchers (Park & Lechner, 2006; Tennen & Affleck, 2009) have argued that the retrospective assessment of SRG might be unable to correctly capture the self-reported SRG. One possible reason is people may denigrate their past selves and exaggerate their current selves to show their increased SRG levels. The other possible reason is that people may overestimate the severity of their stressors

and correspondingly may report higher levels of SRG. However, our study suggested that stress severity levels were lower for more temporally-distant episodes, and thus we may have underestimated the levels of SRG. Given the limitation of the retrospective method, future studies should employ other SRG measurements, such as reports of others and comparison groups (for a review, see Park & Lechner, 2006).

In spite of these limitations, this study makes several meaningful contributions to the existing SRG literature. The study showed that the effects of stress severity on SRG were primarily mediated by the PAC both within time and across time. This finding provides strong evidence to the DAM (Aldwin et al., 1996) for the importance of coping strategies especially the PAC on the development of SRG from stressors. As mentioned earlier, though, only the positive spiral of the DAM model was examined, and future research should also test for negative spirals.

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Table 1.1

Sample Selection Procedures

Selection	DLS 1996	DLS 2001
Total sample	928	1,176/229 (new cohort)
No stress severity	30	36
Then, sample size is	898	1,140/220 (new cohort)
Combined	592	
Report the same stressors, and is over in 1996	33	
Ending points at Time 2 are prior to Time 1	19	
Sample size	540	
Ending points at Time 1 were prior to 1991	31	
Final sample size	509	

Table 1.2

Demographic Characteristics (N = 509)

Demographic	n	%
Sex		
Male	224	44.01
Female	285	55.99
Ethnicity		
Anglo	472	93.47
African-American	2	.40
Hispanic	9	1.78
Pilipino	2	.40
Asian	25	4.95
American Indian	3	.59
Other	6	1.19
Education		
BA/BS	139	27.31
Credential/licensure	80	15.72
Working towards higher degree	23	4.52
MA/MS	163	32.02
PhD	37	7.27
DVM	14	2.75
MD	25	4.91
JD	28	5.50
Marital Status		
Single/never married	54	10.67
Cohabitating	22	4.35
Married	386	76.28
Separated	3	.59
Divorced	37	7.31
Widowed	4	.79
Employment Status		
Employed full-time	405	79.72
Employed part-time	60	11.81
Unemployed	13	2.56
Homemaker	26	5.12
Retired	4	.79

Table 1.3

Recent Low Points at Two Times (N = 509)

Types of Stressors	Time 1 – 1996 (%)	Time 2 – 2001 (%)
Career/work problems	16.9	11.79
Marital/relationship problems	9.82	10.81
Parent's health	8.25	8.25
Physical health problems (self)	7.47	8.45
Parent's death	6.88	11.20

Table 1.4

Means and Standard Deviations of Variables (N = 509)

Variable	Time 1 – 1996		Time 2 – 2001	
	M	*SD*	*M*	*SD*
Age	44.23	7.38		
Sex (*1 = female*)	.56	.50		
Time Interval	4.67	2.12		
Stress Severity	5.27	1.27	5.31	1.31
Positive Action Coping	24.74	10.35	29.96	10.36
Negative Action Coping	6.99	5.64	7.76	6.45
SRG Confidence	1.55	.80	1.63	.78
SRG Vulnerabilities	1.07	.55	1.05	.55
SRG Resources	2.09	.77	2.14	.76
SRG Values	2.07	.65	2.12	.65
SRG Advantages	1.12	.91	1.01	.87

Table 1.5

Correlations of Study Variables (N = 509)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. Age	--																		
2. Sex (*1 = female*)	-.12**	--																	
3. Stress Severity 1	.09*	.11*	--																
4. PAC 1	.11*	.19***	.22***	--															
5. NAC 1	.04	.06	.20***	.35***	--														
6. SRG Confidence 1	.04	.21***	.09*	.60***	.15***	--													
7. SRG Vulnerabilities 1	.10*	.18***	.27***	.34***	.28***	.36***	--												
8. SRG Resources 1	.07	.17***	.01	.47***	.07	.49***	.14**	--											
9. SRG Values 1	.03	.21***	.02	.44***	.08	.47***	.21***	.68***	--										
10. SRG Advantages 1	-.02	.13**	.18**	.41***	.07	.43***	.17***	.21***	.28***	--									
11. Stress Severity 2	-.04	.10*	.31***	.07	.02	.10*	.09*	-.01	.09*	.05	--								
12. PAC 2	.12**	.08*	.08	.50***	.09*	.37***	.17***	.32***	.27***	.31***	.13**	--							
13. NAC 2	-.04	.03	.18**	.10*	.32***	.08	.16***	-.02	.02	.02	.23***	.24***	--						
14. SRG Confidence 2	.02	.14**	.07	.34***	.04	.42***	.20***	.27***	.28***	.24***	.11**	.54***	.12**	--					
15. SRG Vulnerabilities 2	.04	.13**	.16***	.20***	.12**	.19***	.36***	.10*	.09	.14**	.16***	.29***	.27***	.35***	--				
16. SRG Resources 2	.11*	.18***	.04	.30***	.07	.28***	.08	.43***	.33***	.11*	-.01	.42***	.01	.54***	.12**	--			
17. SRG Values 2	.05	.16***	.04	.30***	.08*	.31***	.16***	.34***	.48***	.21***	.08*	.40***	.04	.48***	.17***	.58***	--		
18. SRG Advantages 2	-.02	.10*	.16***	.25***	-.02	.25***	.09*	.12**	.17***	.35***	.16***	.40***	.14**	.41***	.16***	.24***	.30***	--	--
19. Time Interval	.03	-.06	-.03	-.04	-.04	-.03	-.04	-.01	-.03	.05	-.15***	-.05	-.03	-.03	-.01	-.00	-.07	-.09*	--

Note. Sample size varies slightly across correlations. PAC = Positive Action Coping; NAC = Negative Action Coping; SRG = Stress-Related Growth.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 1.6

Direct, Indirect, and Total Effects of Stress Severity, PAC, and SRG (N = 509)

Predictors	Outcomes	Direct Effect	Mediator	Indirect Effect	Total Indirect Effect	Total Effect
Sex	Pos96	.182***	Stress96	.023*	.023*	.205***
Stress96	SRG96		Pos96	.138***	.138***	.138***
Age	SRG96		Pos96	.081*	.095**	.095**
			Stress96 & Pos96	.015*		
Sex	SRG96	.145***	Pos96	.133***	.150***	.294***
			Stress96 & Pos96	.017*		
Age	Stress01		Stress96	.033*	.033*	.033*
Sex	Stress01		Stress96	.038**	.038**	.038**
Stress96	Pos01		Stress01	.027*	.119***	.119***
			Pos96	.064**		
			SRG96 & Pos96	.028*		
Pos96	Pos01	.341***	SRG96	.149**	.149**	.489***
Age	Pos01		Pos96	.038*	.067**	.067**
			Stress96 & Stress01	.003		
			Stress96 & Pos96	.007[a]		
			Pos96 & SRG96	.016		
			Stress96 & Pos96 & SRG96	.003		
Sex	Pos01		Pos96	.062**	.133***	.133***
			SRG96	.029*		
			Stress96 & Stress01	.003		
			Stress96 & Pos96	.008*		
			Pos96 & SRG96	.027*		
			Stress96 & Pos96 & SRG96	.003		
Stress01	SRG01		Pos01	.046**	.046**	.046**
Pos96	SRG01	-.223**	Pos01	.181***	.650***	.427***
			SRG96	.390***		
			SRG96 & Pos01	.079**		
SRG96	SRG01	.533***	Pos01	.108**	.108**	.641**
Age	SRG01		Pos96	-.025	.057**	.057**
			Stress96 & Pos96	-.005		
			Pos96 & Pos01	.020*		
			Pos96 & SRG96	.043*		
			Stress96 & Stress01 & Pos01	.002		
			Stress96 & Pos96 & Pos01	.004		
			Pos96 & SRG96 & Pos01	.009		
			Stress96 & Pos96 & SRG96	.008		
			Stress96 & Pos96 & SRG96 & Pos01	.002		
Sex	SRG01		Pos96	-.041*	.182***	.182***
			SRG96	.077**		
			Stress96 & Pos96	-.005		
			Pos96 & Pos01	.033**		
			SRG96 & Pos01	.016*		
			Pos96 & SRG96	.071***		

Table 1.6.
continued

Predictors	Outcomes	Direct Effect	Mediator	Indirect Effect	Total Indirect Effect	Total Effect
			Stress96 & Stress01 & Pos01	.002		
			Stress96 & Pos96 & Pos01	.004*		
			Pos96 & SRG96 & Pos01	.014*		
			Stress96 & Pos96 & SRG96	.009*		
			Stress96 & Pos96 & SRG96 & Pos01	.002		
Stress96	SRG01		Pos96	-.042*	.095***	.095***
			Stress01 & Pos01	.014*		
			Pos96 & Pos01	.034**		
			Pos96 & SRG96	.074***		
			Pos96 & SRG96 & Pos01	.015*		

Note. *Sex*: 0 = *men*, 1 = *women*. Sample size varies slightly across correlations. PAC = Positive Action Coping; NAC = Negative Action Coping; SRG = Stress-Related Growth.
* $p < .05$. ** $p < .01$. *** $p < .001$. [a]$p = .051$.

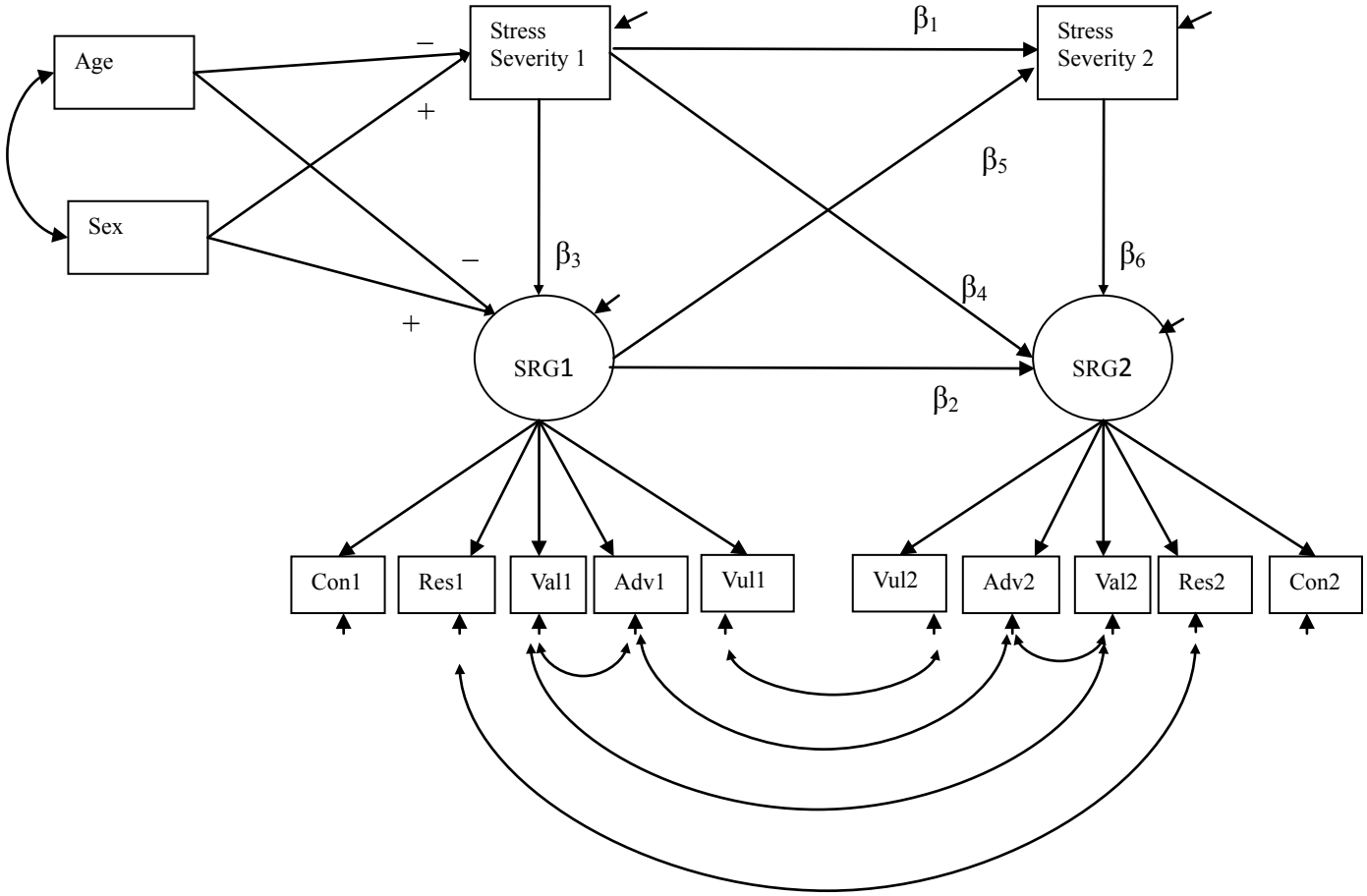
Table 1.7

Direct, Indirect, and Total Effects of Stress Severity, NAC, and SRG (N = 509)

Predictors	Outcomes	Direct Effect	Mediator	Indirect Effect	Total Indirect Effect	Total Effect
Age	NAC96		Stress96	.022*	.022*	.022*
Sex	NAC96		Stress96	.025*	.025*	.025*
Stress96	SRG96		NAC96	.035**	.035**	.035**
Sex	SRG96	.258***	Stress96 & NAC96	.004*	.004*	.262***
Age	Stress01		Stress96	.033*	.033*	.033*
Sex	Stress01		Stress96	.038*	.038**	.038**
Stress96	NAC01		Stress01	.068***	.132***	.132***
			NAC96	.064***		
Age	NAC01		Stress96 & Stress01	.007*	.014*	.014*
			Stress96 & NAC01	.007*		
Sex	NAC01		Stress96 & NAC96	.008*	.016*	.016*
			Stress96 & Stress01	.008*		
Stress01	SRG01		NAC01	.023*	.023*	.023*
Stress96	SRG01		NAC96 & SRG96	.020**	.034***	.034***
			NAC96 & NAC01	.007*		
			Stress01 & NAC01	.007*		
NAC96	SRG01		NAC01	.033**	.130***	.130***
			SRG96	.096**		
Sex	SRG01		SRG96	.145***	.149***	.149***
			Stress96 & Stress01 & NAC01	.001		
			Stress96 & NAC96 & NAC01	.001		
			Stress96 & NAC96 & SRG96	.002*		

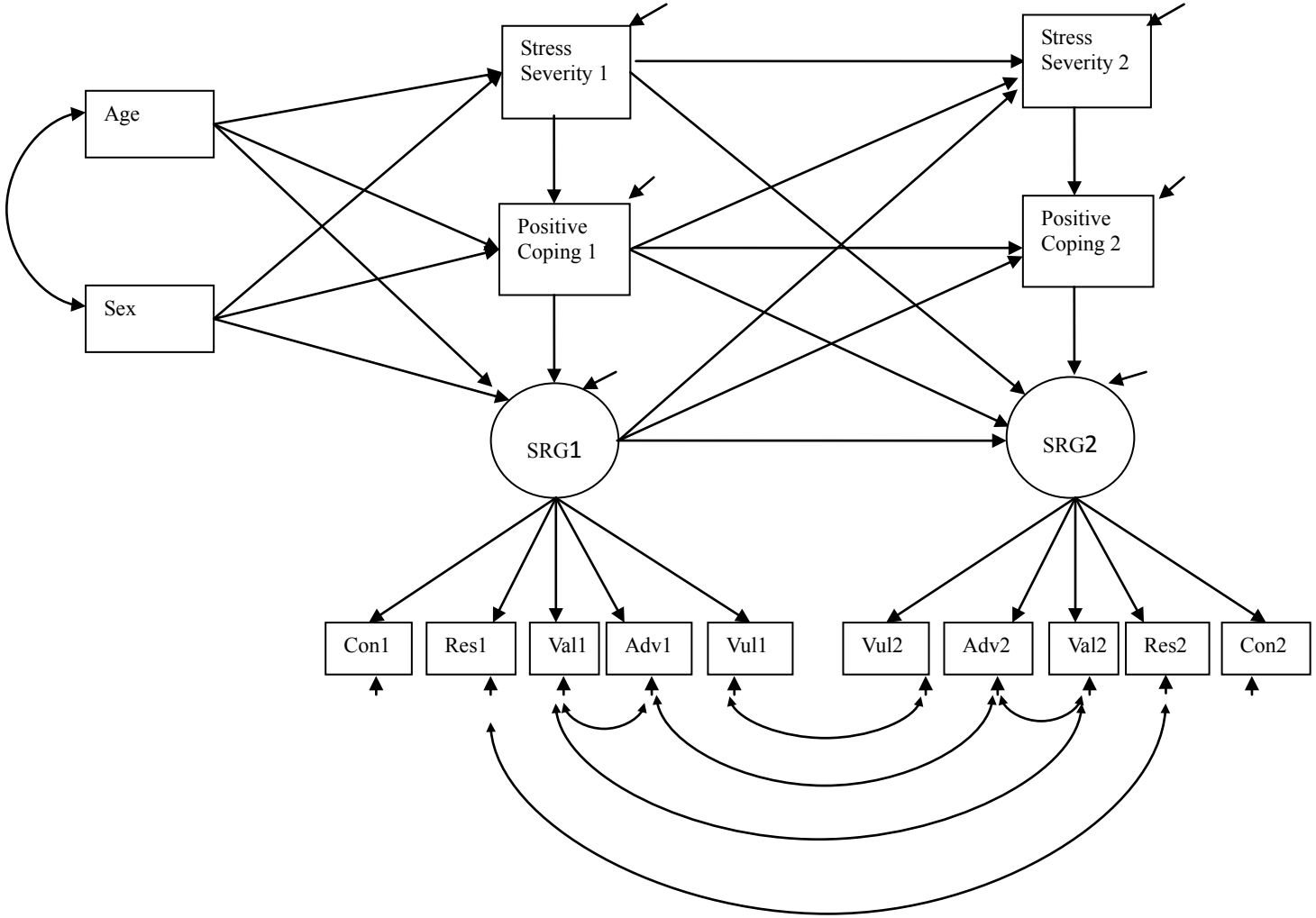
Note. Sex: 0 = *men*, 1 = *women*. Sample size varies slightly across correlations. PAC = Positive Action Coping; NAC = Negative Action Coping; SRG = Stress-Related Growth. * $p < .05$. ** $p < .01$. *** $p < .001$.
The total indirect effect from stress96 to SRG01 is .034, $p = .001$.

Figure 1.1. Cross-Lagged Theoretical Model of Stress Severity and Stress-Related Growth



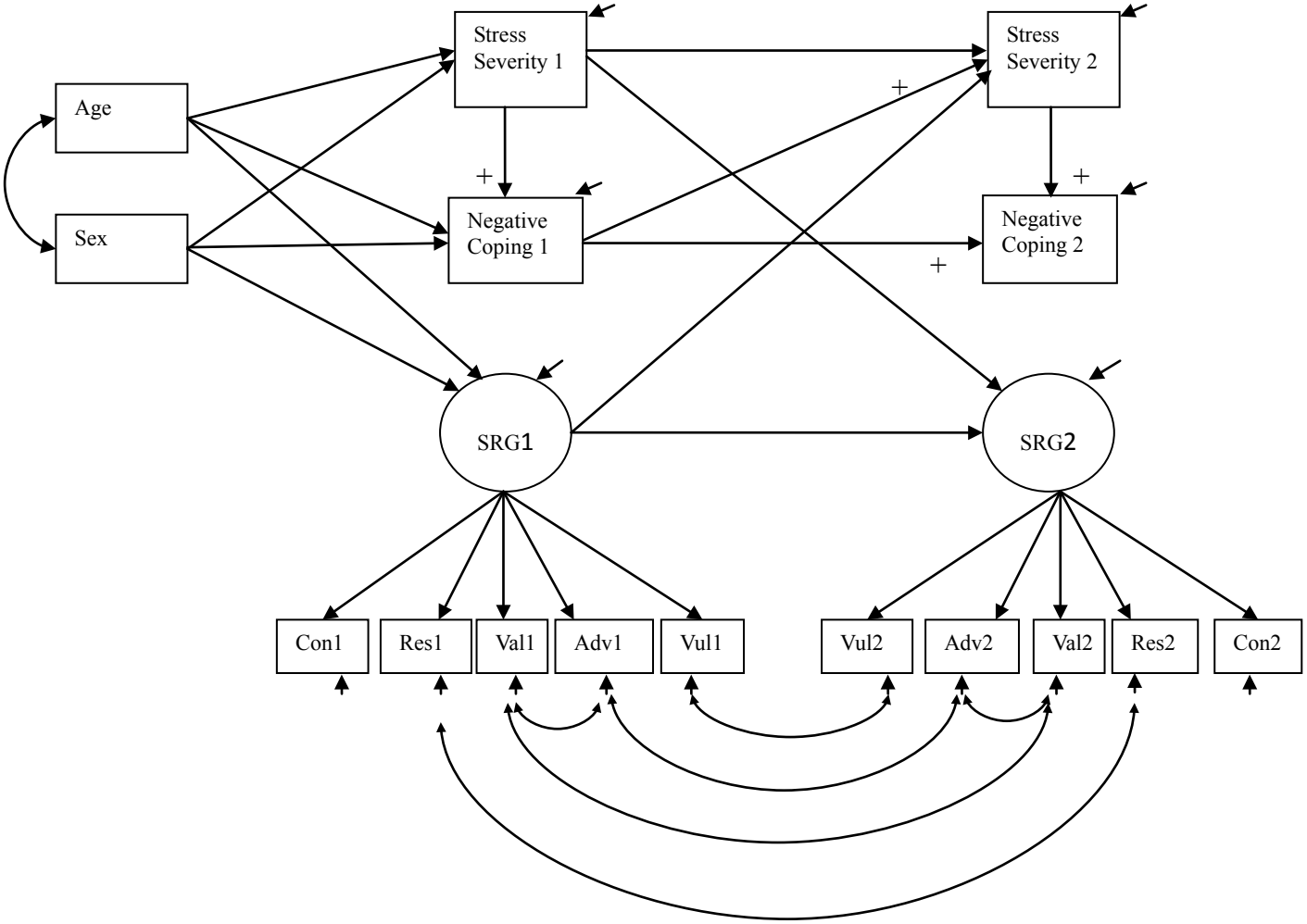
Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities.

Figure 1.2. Longitudinal Theoretical Mediating Model of Positive Action Coping



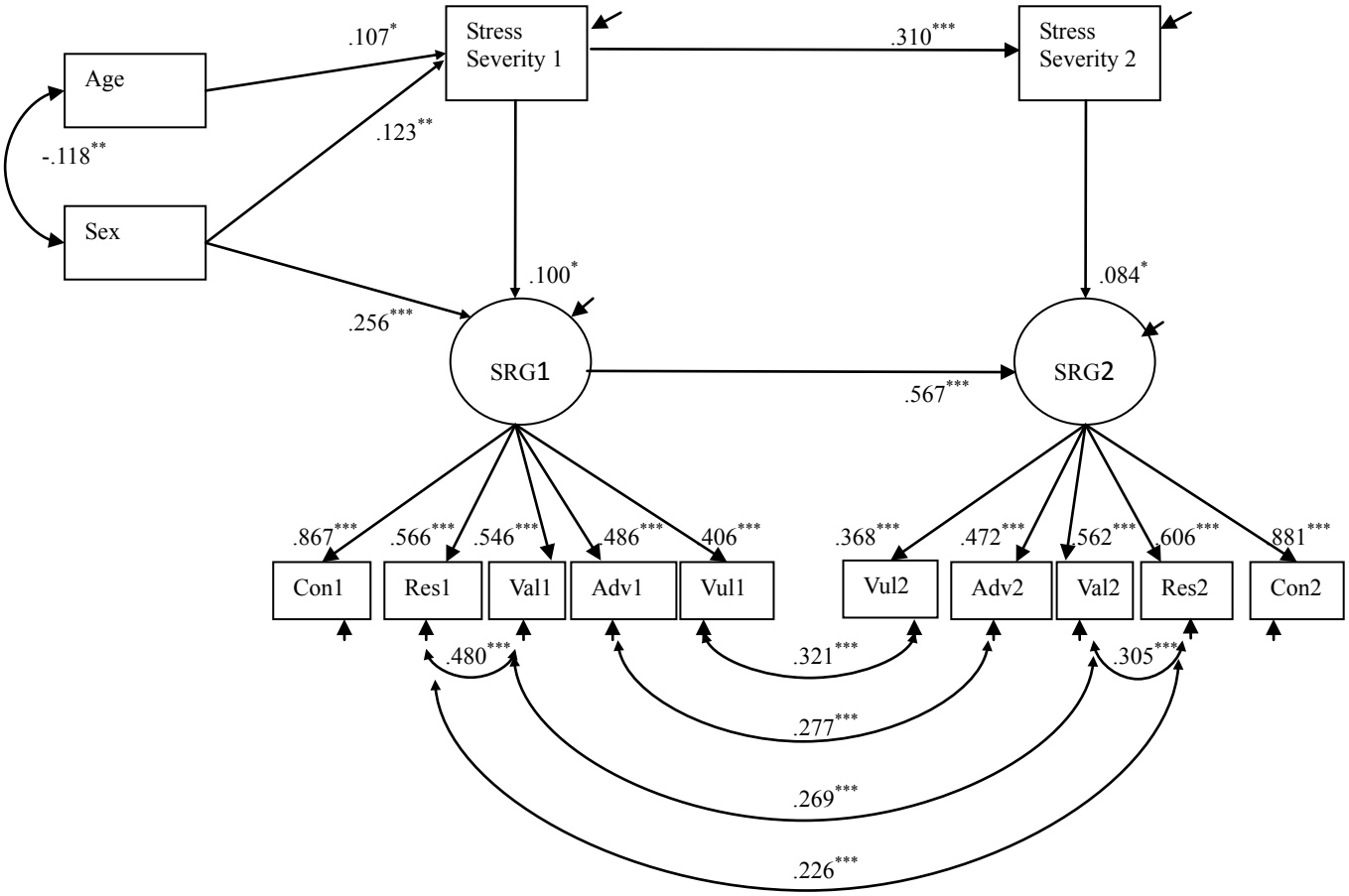
Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities.

Figure 1.3. Longitudinal Theoretical Mediating Model of Negative Action Coping



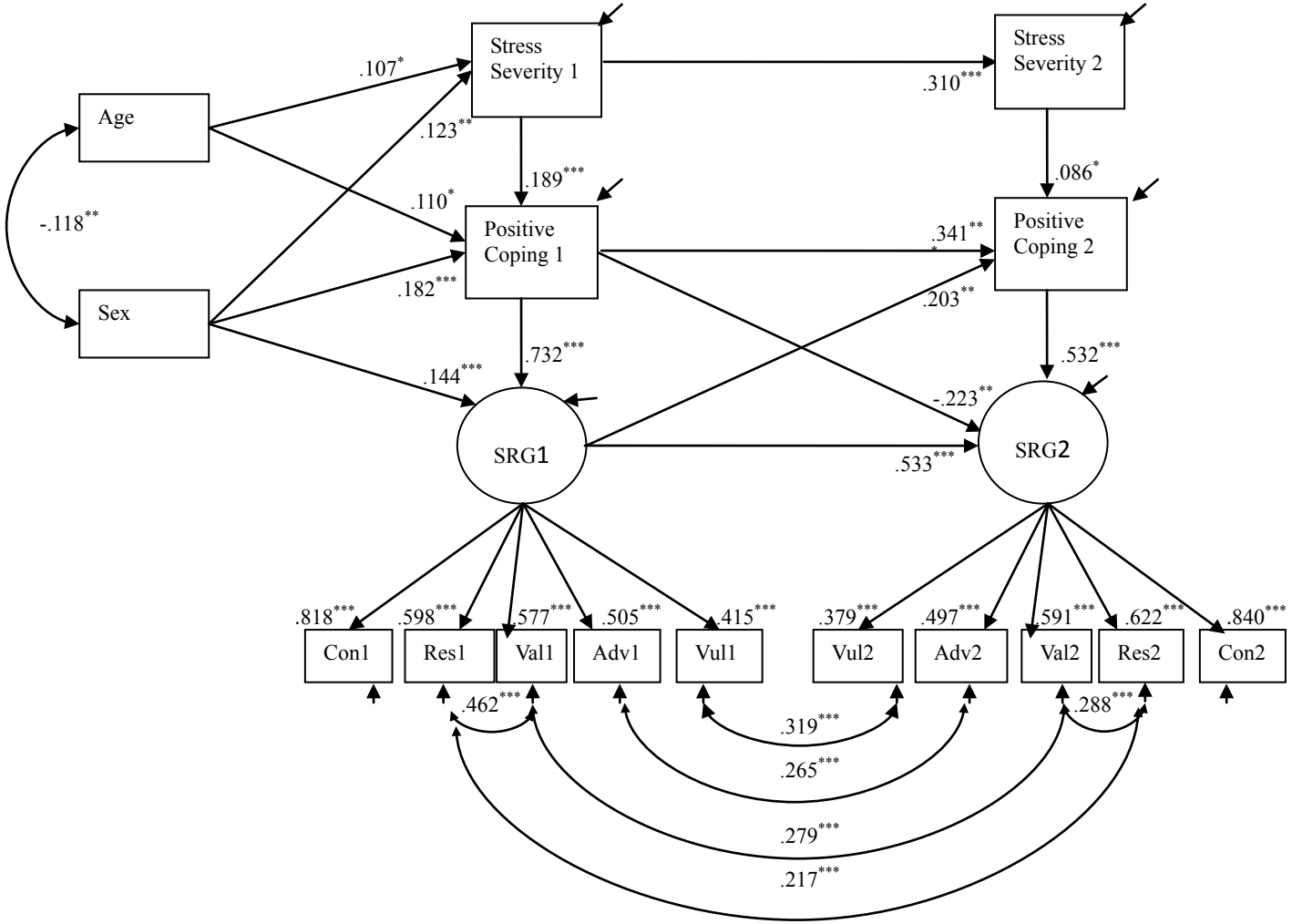
Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities.

Figure 1.4. Final Cross-Lagged Model of Stress Severity and Stress-Related Growth



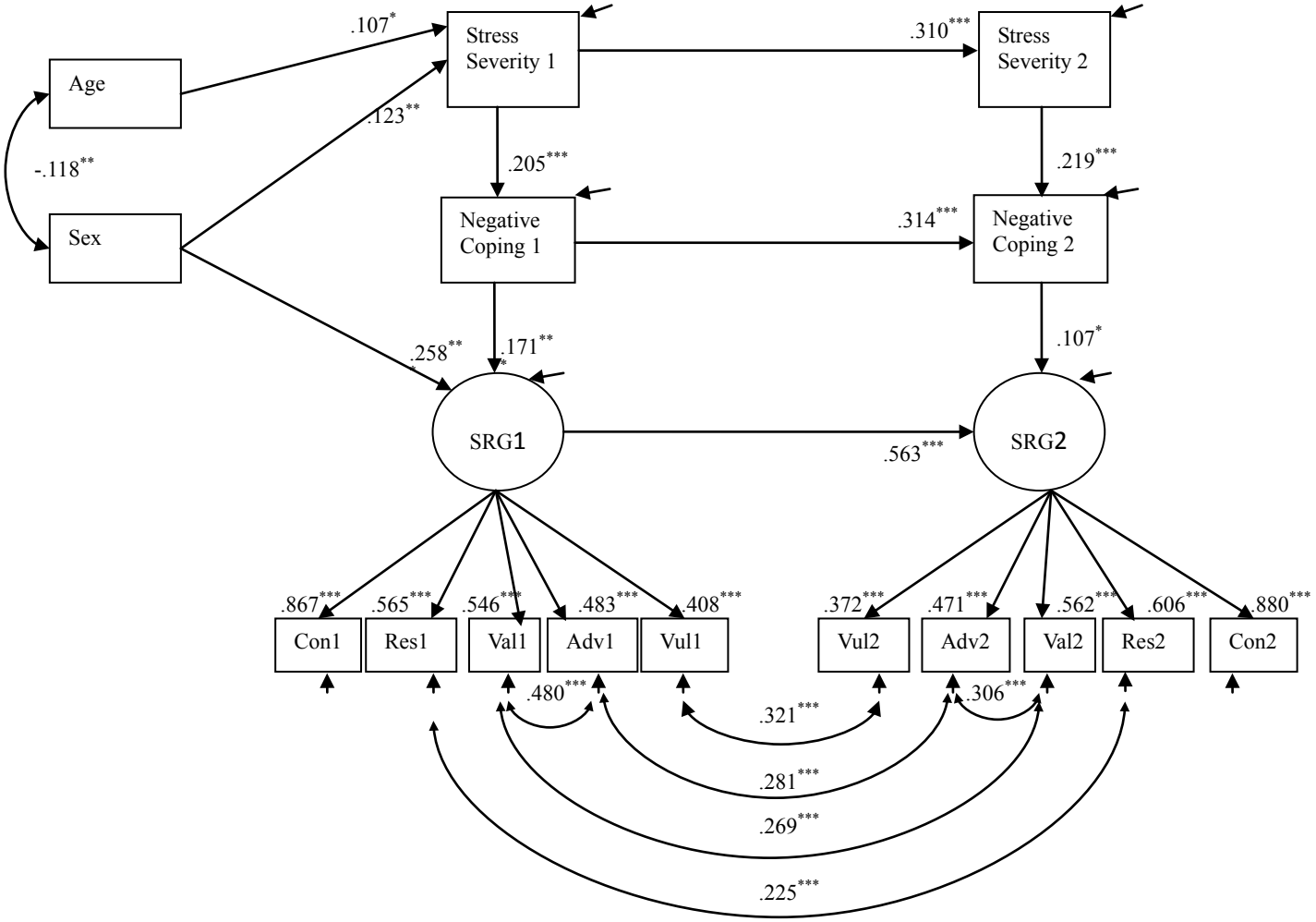
Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities.

Figure 1.5. Final Longitudinal Mediating Model of Positive Action Coping



Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities

Figure 1.6. Final Longitudinal Mediating Model of Negative Action Coping



Note. Con = Confidence; Vul = Vulnerabilities; Res = Resources; Val = Values; Adv = Advantages

The Effects of Stress Severity on Reciprocal Relations between Stress-Related Growth and Depressive Symptoms: Findings from the Davis Longitudinal Study

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Abstract

The moderating effects of stress severity on the relationships between stress-related growth (SRG) and depressive symptoms were examined among young and middle-aged adults surveyed at two points in time over five years. Cross-sectional analyses (N = 1,140) found that in the low stress severity group, SRG and depressive symptoms were unrelated to each other, while in the high stress severity group, SRG was related to reduced depressive symptoms. Longitudinal analyses (N = 509) showed that under low stress, SRG and depressive symptoms were orthogonal. However, under high stress, SRG predicted lower depressive symptoms within time, while previous depressive symptoms predicted higher subsequent SRG. However, further analyses showed that there were no statistically significant differences in the longitudinal models across stressor groups, suggesting that the effect is fairly weak.

Key words: stress-related growth, depressive symptoms, stress severity, moderating, cross-lagged effects

Study 1 examined the reciprocal relations between stress severity and SRG, as mediated by coping strategies over time. However, there is an additional intriguing question in the literature on the relations between negative outcomes of depressive symptoms and positive outcomes of SRG from stressful events (Helgeson et al., 2006; Tedeschi & Calhoun, 2004). That is, does SRG greatly reduce depressive symptoms? Or do depressive symptoms motivate or impede the development of SRG? A majority of studies examining the relations between SRG and depressive symptoms are cross-sectional. Therefore, there is limited knowledge about longitudinal relations between SRG and depressive symptoms. The present study will use longitudinal meditional analyses (MacKinnon, 2008) to explore both within-time and cross-lagged effects and to determine how SRG and depressive symptoms are related to one another when stress severity is taken into account.

Significance of This Study

It is well known that depressive symptoms are one of the common negative outcomes of stress and are highly prevalent among persons. For example, every year around 17 million adult Americans experience depression (http://www.apa.org/topics/depress/recover.aspx#). Depression can evoke serious problems with families (Whisman, 2006), interfere with physical functioning (Glassman & Shapiro, 1998; Rantanen, Penninx, Masaki, Lintunen, Foley, & Guralnick, 2000), reduce work productivity (Lerner et al., 2004), and even lead to suicide (for a review, see Berman, 2009). Some preventive interventions, such as cognitive behavioral interventions (Hollon & Beck, 2004), have been used to decrease depressive levels. Given the potential beneficial effects of SRG, promoting SRG levels to decrease depressive symptoms might be a plausible approach in clinical intervention programs (Antoni et al., 2001; Anotni, Carver, & Lechner, 2009).

Identifying the relations between SRG and depressive symptoms is especially important because different relations have different implications for clinical intervention

programs. If the higher levels of SRG predict lower levels of depressive symptoms (for reviews, see Algoe & Stanton, 2008; Linley & Joseph, 2004), SRG will provide an alternative treatment to reduce the levels of depressive symptoms (Zoellner & Maercker, 2006). Otherwise, it may be misleading for clinical interventions in that higher levels of SRG are related to worse psychological outcomes (Tomich & Helgeson, 2004) or no significant positive effects at all (Cordova, Cunningham, Carlson, & Andrykowski, 2001).

Second, it may be worthwhile to maintain depressive symptoms to stimulate SRG after occurrence of stress (Calhoun & Tedeschi, 1999), if the higher levels and enduring depressive symptoms motivate the development of SRG (Tedeschi & Calhoun, 2004). Otherwise, people have to meaninglessly suffer from depressive symptoms for a long period of time and this may result in seriously detrimental outcomes.

Third, in this study we argue that the relations may be stratified by stress severity, with no relations in a low stress severity group and negative relations in a high stress severity group. If these relations are supported, it will provide important guidelines for clinical intervention programs. That is, SRG only has adaptive effects on depressive symptoms in a high stress severity group.

Literature Review

The major purpose of Study 2 is to examine whether the relations between SRG and depressive symptoms are stratified by level of stress severity. Besides generalized resistance resources (GRRs; Antonovsky, 1979), conservation of resources (COR; Hobfoll, 1988), and deviation amplification model (DAM; Aldwin et al., 1996), two more models are incorporated in Study 2, i.e., functional-descriptive model (Tedeschi & Calhoun, 1996, 2004) and dynamic model of affect (DMA, Zautra, 2003), to depict conceptual arguments about SRG and depressive symptoms. Because the five theories describe complicated relations between SRG and depressive symptoms, to make it much easier to understand, we draw eight

figures (see Figure 2.1) to summarize their ideas. Then, we compare the five theories and discuss what are suggested by them.

Theoretical Models of SRG and Depressive Symptoms

Generalized resistance resources (GRRs). Antonovsky (1979) argued that health is a continuous variable moving from one end to another on the health ease (e.g., GRR/SRG) and dis-ease continuum (e.g., depressive symptoms). In stressful contexts, which end is produced is primarily determined by the extent of available GRRs/SRG. That is, the more adequate GRRs available, the higher level of GRRs/SRG and the lower level of depressive symptoms, which in turn further reinforces subsequent SRG and decreases subsequent depressive symptoms. Conversely, the fewer GRRs available, the higher level of depressive symptoms and the lower level of GRRs/SRG, which in turn further reinforces subsequent depressive symptoms and decreases subsequent SRG. Therefore, SRG and depressive symptoms are inversely related and counteracting each other over time (see Figure 2.1).

Conservation of resources (COR). Hobfoll's (1988; Hobfoll & Lilly, 1993) studies imply that the relations between SRG and depressive symptoms are varied by stress severity levels. That is, if the level of stress severity is low, SRG and depressive symptoms are typically unrelated or independent of each other (Hobfoll, 1988; Hobfoll & Lilly, 1993), whereas if the level of stress severity is high, they tend to be negatively related (Hobfoll & Lilly, 1993). Furthermore, Hobfoll argued that since resource loss is more influential than resource gain, SRG does not predict or only marginally predicts depressive symptoms, while depressive symptoms do have significantly negative effects on SRG. In other words, SRG does not serve as a protective factor for depressive symptoms but depressive symptoms serve as a risk factor for SRG (see Figure 2.1).

Functional-descriptive model. Tedeschi and Calhoun (2004) specifically focus on the effects of depressive symptoms on SRG from highly traumatic events. They argued that

depressive symptoms are positively related to SRG because certain extents of the depressive symptoms are necessary to motivate the development of SRG. Namely, the higher level of depressive symptoms, the higher level of SRG produced (see Figure 2.1). Therefore, from their points of view, depressive symptoms coexist with SRG.

Deviation amplification model (DAM). Aldwin et al. (1996) argued that stressors can result in either a deviation countering or a deviation amplification process. When the level of stress severity is low, neither depressive symptoms nor SRG tends to occur (i.e., a deviation countering process). Therefore, SRG and depressive symptoms seem to be unrelated. When the level of stress severity is high, a deviation amplification process is likely to happen, represented by either positive spirals (e.g., high levels of SRG) or negative spirals (e.g., high levels of depressive symptoms). Therefore, the deviation amplification process suggests inverse relations between SRG and depressive symptoms (see Figure 2.1).

Dynamic model of affect (DMA). Zautra (2003) explicitly differentiated the relations between positive outcomes and negative outcomes by the level of stress severity. Namely, if stress severity level is low, positive outcomes and negative outcomes are relatively independent; in contrast, if stress severity level is high, they are inversely related. Moreover, Zautra (2003) argued that positive outcomes have few beneficial effects or reducing functions on depressive symptoms, whereas depressive symptoms have detrimental effects or decreasing functions on positive outcomes of stress, i.e., SRG in our study (see Figure 2.1). In part, this is because pursuing positive outcomes is intrinsically motivated by human life instead of by decreasing negative outcomes (Zautra, 2003).

Discussions of the five theoretical models. In summary, the five theories proposed different relations between SRG and depressive symptoms. Closer inspection shows that these divergences may be because of the level of stress severity. Specifically, Tedeschi and Calhoun (2004) argued that the positive outcomes only result from traumatic events, whereas

other researchers (Aldwin et al., 1996; Antonovsky, 1979; Hobfoll, 1988; Zautra, 2003) argued that besides traumatic events, SRG can be derived from high stress events as well. Researchers (Aldwin et al., 1996; Hobfoll, 1988; Zautra, 2003) also have mentioned that low stress events may not be sufficient enough to evoke SRG.

If we define no relations between SRG and depressive symptoms as an orthogonal model, negative relations as a unidimensional model, and positive relations as a covariation model, the five theories along with the levels of stress severity can be categorized as follows. In a low stress severity group, an orthogonal model (Aldwin et al., 1996; Hobfoll, 1988; Zautra, 2003) may be supported because low level of stress may not be sufficient enough for the development of SRG and depressive symptoms. In a high stress severity group, a unidimensional model (Aldwin et al., 1996; Antonovsky, 1979; Hobfoll, 1988; Zautra, 2003) may be supported because both high levels of SRG and depressive symptoms can be reduced by one another. In a traumatic stress group, a positive covariation model (Tedeschi & Calhoun, 2004) may be supported because SRG and depressive symptoms from trauma are robust enough and thus neither one is easily offset by the other.

In addition, there are controversies among the theories about whether the relations between SRG and depressive symptoms are unidimensional or reciprocal. Aldwin et al. (1996) and Antonovsky (1979) have argued that they are reciprocally related over time. That is, SRG has effects on depressive symptoms and so depressive symptoms on SRG. Hobfoll (1988) and Zautra (2003), however, have argued that they are unidirectionally related; namely that only depressive symptoms have effects on SRG. Given this controversy, this question is examined in the present study as well.

Empirical Studies of SRG and Depressive Symptoms

Empirical studies are also mixed in the relations between SRG and depressive symptoms, primarily revolving around three questions. Does SRG suppress depressive

symptoms or vice versa? Does SRG coexist with or is it independent from depressive symptoms? Is their causality unidirectional or reciprocal?

An orthogonal model of SRG and depressive symptoms. A majority of cross-sectional studies have found that SRG is independent of or unrelated to depressive symptoms when they are examined in relatively low levels of stress severity, e.g., early-stage breast cancer (Antoni et al., 2001; Cordova et al., 2001; Hobfoll & Lilly, 1993; Sears et al., 2003) or undergraduates' mixed stressors (Park et al., 1996, study 2 & 3; Park & Fenster, 2004). Other studies have found that they are negatively (Hobfoll & Lilly, 1993; Siegel et al., 2005) or positively associated (Edmonds & Hooker, 1992; Hobfoll, Canetti-Nisim, & Johnson, 2006). Because of their cross-sectional designs, however, these studies are unable to address causal directions between SRG and depressive symptoms over time.

A unidimensional model of SRG and depressive symptoms. The unidimensional model can be examined from two directions. On the one hand, previous SRG has been shown to have beneficial effects on subsequent depressive symptoms; namely that, higher level of initial SRG predicts lower levels of depressive symptoms afterwards (for reviews, see Algoe & Stanton, 2008; Linley & Joseph, 2004). On the other hand, previous depressive symptoms have been found to have resistant effects on subsequent SRG; namely that, higher initial level of depressive symptoms predicts lower levels of SRG afterwards (Hart et al., 2008).

A positive covariation model of SRG and depressive symptoms. A covariation model includes two aspects as well. Unlike the unidimensional model, in the covariation model initial SRG predicts an increased level of subsequent depressive symptoms (Tomich & Helgeson, 2004), indicating that perceived SRG can have detrimental effects on depressive symptoms. Also, the predictive effect of previous depressive symptoms on subsequent SRG has been examined, represented by a positive (Pollard & Kennedy, 2007; Wrosch & Miller,

2009) rather than a negative relation in the unidimensional model (Hart et al., 2008). For example, depressive symptoms act as an impetus to promote SRG levels later (Pollard & Kennedy, 2007) or to abandon unattainable goals (Wrosch & Miller, 2009).

Stratified effects of stress severity on depressive symptoms and SRG. It is difficult to directly compare stress severity across studies because each uses different criteria to assess stress severity (e.g., stages of disease or different points of Likert-scale). However, it is still possible to make rough comparisons, and results suggest that the level of stress severity may account for the inconsistent relations between SRG and depressive symptoms across studies.

The potential stratified effects of stress severity can be illustrated by three cross-sectional studies of Hobfoll. Hobfoll & Lilly (1993) found that SRG was unrelated to psychological distress among both a community sample and a student sample. However, when stress was taken into account, SRG and psychological distress were negatively related to one another, especially among community samples, who had higher levels of stress severity than the student samples. When SRG and depressive symptoms were examined in the context of terrorism in Israel, Hobfoll et al. (2006) found that they were positively related with one another. These results provide evidence for us to hypothesize that the cross-sectional relations between SRG and depressive symptoms may be stratified by the level of stress severity.

The longitudinal effects of SRG on depressive symptoms seem to vary by stress severity as well. Specifically, without considering stress severity, SRG alone had no long-term predictive effects on depressive symptoms (Tomich & Helgeson, 2004). When stress severity levels were high, SRG had beneficial effects on depressive symptoms, i.e., a unidimensional model (Carver & Antoni, 2004; Davis et al., 1998; Frazier et al., 2001; Ickovics et al., 2006; McMillen et al., 1997; Schwarzer et al., 2006). However, when the

levels of stress severity were traumatic, SRG had detrimental effects on subsequent depressive symptoms, i.e., a positive covariation model (Tomich & Helgeson, 2004).

In a similar vein, the longitudinal effects of depressive symptoms on subsequent SRG also vary from one level of stress severity to another. That is, when the level of stress severity was low, depressive symptoms were independent from or had no effects on SRG (Abraido-Lanza, Guier, & Colon, 1998). When the level of stress severity was high, depressive symptoms negatively predicted SRG (Hart et al., 2008). When the level of stress severity was traumatic, depressive symptoms motivated or were positively related to the development of SRG (Pollard & Kennedy, 2007; Tedeschi & Calhoun, 1996).

Reciprocal relations between SRG and depression. Researchers have specifically underscored the mutual relations between SRG and depressive symptoms (Aldwin et al., 1996; Antonovsky, 1979; Hobfoll, 1988). However, existing empirical studies have rarely examined the reciprocal relations (Hart et al., 2008). Instead, two unidirectional effects have been widely investigated. Some studies have examined if previous SRG has adaptational effects or disruptive effects on subsequent depressive symptoms. Other studies, however, have attempted to test if previous depressive symptoms impede or motivate SRG development afterwards. To my knowledge, the reciprocal relations between SRG and depressive symptoms have rarely been simultaneously examined. Although Hart et al. (2008) examined the reciprocal relations, they did not test the relations at varying levels of stress severity. Specifically, Hart et al. found that initial decreased depressive symptoms led to subsequent increases in SRG, and in turn the improved SRG further reduced subsequent depressive symptoms.

Covariates of age, sex, and time interval. Because the relations between age, sex, and time interval have been addressed in the overall literature review section, we only focus on their relations with depressive symptoms here. Generally, women (Nolen-Hoeksema &

Hilt, 2009) and younger adults (Penninx, 2006) are more likely to experience depressive symptoms, although it also has been found that depressive symptoms increased with age (Kennedy, 1996).

The time interval may affect depressive symptoms levels given the duration and prediction of depressive symptoms. Specifically, even though most patients recover from depression within one year (Keller, Shapiro, Lavori, & Wolfe, 1982), some patients still suffered from depression at 16 months (Rounsaville, Prusoff, & Padian, 1980), two years, five years (Keller et al., 1982), 10 years (Mueller et al., 1996), or even longer (Keller & Boland, 1998). In addition, previous depressive symptoms predict subsequent depressive symptoms (Rudolph, Flynn, Abaied, Groot, & Thompson, 2009; Tram & Cole, 2006). With regard to our study, since the time interval between two stressors varies from a short period of time to five years or longer, it is entirely possible that depressive symptoms at time 2 are influenced by depressive the time interval.

Research Questions and Hypotheses

The purpose of this study is to evaluate how the relations between SRG and depressive symptoms are stratified by stress severity both in cross-sectional models (Figure 2.2 & Figure 2.3) and two-wave cross-lagged models (Figure 2.4 & Figure 2.5). Because the present study mainly examines respondents' major life stressors, it will only test the relations between SRG and depressive symptoms in low and high stress severity groups. Specifically, the following hypotheses are examined:

Hypothesis 1: We hypothesize that the cross-sectional relations between SRG and depressive symptoms in 2001 will be stratified by the levels of stress severity.

Hypothesis 1.a: In the low stress severity group, SRG and depressive symptoms will not be correlated with one another (Figure 2.2).

Hypothesis 1.b: In the high stress severity group, SRG and depressive symptoms will be negatively correlated with one another (Figure 2.3).

Hypothesis 2: We hypothesize that both the within-time effects and the cross-lagged effects between SRG and depressive symptoms will be stratified by the levels of stress severity.

Hypothesis 2.a: In the low stress severity group, the within-time effects from SRG to depressive symptoms will be non-significant (Figure 2.4), whereas in the high stress severity group, the within-time effects will be significantly negative (Figure 2.5).

Hypothesis 2.b: In the low stress severity group, the cross-lagged effects between SRG and depressive symptoms will be non-significant (Figure 2.4), whereas in the high stress severity group, the cross-lagged effects will be significantly negative (Figure 2.5).

Hypothesis 2.c: In both the low and the high stress severity groups, the autoregressive effects of SRG and depressive symptoms will be positive (Figure 2.4 & 2.5).

Hypothesis 3: Younger participants will report higher levels of SRG and lower levels of depressive symptoms than older participants. Women will report both higher levels of SRG and depressive symptoms than men. Time interval is negatively related to both SRG and depressive symptoms at Time 2.

Methods

Sample and Procedure

This study used both cross-sectional and longitudinal Davis Longitudinal Study (DLS) data to examine the relations between SRG and depressive symptoms. Given the bigger sample size of the 2001 DLS, the cross-sectional study was examined by 2001 DLS instead of 1996 DLS data. After excluding 36 respondents who did not report their stress severity levels, the final sample size in the cross-sectional study was 1,140. The excluded

respondents were younger than the included respondents, $t(1167) = 1.99$, $p < .05$, and no more demographic differences were found between them.

Among the 1,140 respondents included, around half of them (45.62%) were male, a majority (83.86%) of them were European Americans, approximate two-thirds (65.72%) of the participants were married and one-fifth (20.41%) were single or never married, and ranged in age from 23 to 79 ($M = 44.45$, $SD = 11.06$). Because they were university alumni, all of them had at least a bachelor's degree and nearly half (48.94%) had advanced degrees including MA/MS, PhD, DVM, MD, or JD. More than three-quarters worked full time (78.10%) and the modal income was between $75,000 and $99,999.

We used the median level of stress severity to divide the sample into two groups. Given the median of 5 on a 7-point scale of stress severity, respondents who rated their stress severity from 1-5 were classified as the low stress severity group and those who rated their stress severity either 6 or 7 were classified as the high stress severity group. As Table 2.1 shows that 51.84% ($n = 591$) and 48.16% ($n = 549$) of participants reported low and high levels of stress severity, respectively. In the low stress severity group, there were more males ($n = 305$) than females ($n = 279$), whereas in the high stress severity group, there were fewer males ($n = 210$) than females ($n = 335$), $t(1,127) = -.466$, $p < .001$. No other significant differences were found in age, income, ethnic, education, and marital status between the two stress severity groups (see Table 2.1).

Among the 509 respondents in the longitudinal study, nearly half of them (44.01%) were male, the majority (93.47%) were European Americans, and participants ranged in age from 28 to 74 ($M = 44.24$, $SD = 7.38$). All respondents had at least a bachelor's degree and half of them (52.45%) had advanced degrees. Around three-quarters (76.28%) of the participants were married, and ten percent (10.67%) were single or had never been married.

The majority worked full time (79.72%) and their modal income was between $75,000 and $99,999.

For the longitudinal data, the level of stress severity was divided into two groups by the median score of 5 in 1996, which yielded the same low and high stress severity groupings as the cross-sectional study. Statistical analyses showed that there were no significant differences in demographic characteristics between the two groups (see Table 2.2).

Measures

Demographics or covariates. Respondents were asked to indicate their age, sex, beginning and ending years of the most recent stressors. Time interval was calculated by the ending year at Time 2 (ranging from 1996 to 2001) subtracting the ending year at Time 1 (ranging from 1991 to 1996), which yielded the range of the time interval from 0 to 11 years.

Stress severity. The stressfulness of the most recent stressors was rated on a 7-Likert scale (1 = *Not at all stressful,* to 7 = *Most stressful thing ever experienced*).

Stress-related growth. *Learn from the Low Point and Advantages* (Aldwin et al., 1994) was used to assess SRG. In this study, SRG is a latent variable, including factors of advantages, confidence, resources, values, and vulnerabilities. First, respondents were asked to check as many as five response options to indicate whether they could find advantages from their most recent stressors. The five response options included "no" (coded as 0), "yes, emotional well-being" (coded as 1), "yes, tangible advantage/gain" (coded as 1), "yes, developed a new philosophy/attitude toward life" (coded as 1), and "yes, other" (coded as 1). Then, the total score of the checked items was summed, ranging from 0 to 4. Second, 14 items were used to assess how much respondents learned from the most recent stressors on a 3-point scale (0 = *Not at all*, to 3 = *A lot*). The 14 items included four SRG factors: confidence (3 items), resources (3 items), values (4 items), and vulnerabilities (4 items) (Aldwin et al., 2009; Kelly, 2006). Sample items include "I could stand on my own two feet"

(confidence), "I had positive social resources (e.g., good friends, neighbors, family)" (resources), "Religion/spirituality is very important to me" (values), and "There are some situations that I cannot do anything about" (vulnerabilities).

Depressive symptoms. We used the 11-item version (Kohout, Berkman, Evans, & Cornoni-Huntley, 1993) of the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) to assess participants' depressive symptoms within the past week. The CES-D is a self-report measure, with items scaled from 0 to 2 (0 = *Hardly ever or never*, 1 = *Some of the time*, 2 = *Much or most of the time*). It includes four factors, i.e., somatic complaints (4 items), depressed affect (3 items), positive affect (2 items), and interpersonal problems (2 items) (Kohout et al., 1993). Based on the results of Kohout et al. (1993), we generated four factors by separately summing the items for each factor. The internal consistency reliabilities are high and Cronbach's coefficient α are .76 and .80 in 1996 and 2001, respectively.

Statistical Analyses

Hypothesis 1 examined whether the relations between stress severity and depressive symptoms varied by stress severity groups in cross-sectional data. Before testing this hypothesis, multiple-group confirmatory factor analyses (CFA) were used to determine whether the factor structures for SRG and depressive symptoms were the same in each group. First, the CFA tested whether the factor structures of the CES-D and SRG measures were invariant across the two stress severity groups, and second, whether the relations between these two latent variables (and the covariates) were different across groups, following the procedure indicated by Thompson and Green (2006).

In Stage 1, the same equal form (i.e., whether the indicators load on the same factors) was examined within each of the stress severity group. If both equal forms had good fits to the data, then the comparison of factor invariance across groups was performed in Stage 2. These multiple-group CFA testing factor invariance in this stage included four nested steps,

corresponding to the increased constraints of measurement invariance (e.g., equal form, equal factor loadings, equal indicators intercept, and equal indicator error variance). The difference in fit (i.e., ΔX^2) was examined at each step. If the fit did not significantly worsen, the next step, utilizing more constraints, was performed. These analyses evaluated the same factor invariance simultaneously for both SRG and depressive symptoms within each group and then across the groups. Note that the relations between the two latent variables (and the covariates) were freely estimated both within and across groups.

Having established the nature of the simultaneous measurement models, we then conducted the analyses testing Hypothesis 1 in Stage 3. In this stage, we constrained the relations between SRG and depressive symptoms to be equal across groups, and tested the ΔX^2 between the constrained model in this stage and the freely estimated model in Stage 2 to determine if that decreased the fit.

Similar procedures were applied to test Hypothesis 2, which examined both within-time and cross-lagged relations between SRG and depressive symptoms in the two stress severity groups. We repeated the measurement model analyses estimating SRG and depressive symptoms simultaneously at both time points. The within-time relations (including covariates), autocorrelations, and cross-lagged relations were freely estimated in these models. To test the hypotheses, we then constrained the within-time and cross-lagged relations to be equal across groups and examined whether this affected the fit (i.e., ΔX^2).

Analyses were conducted using structural equation modeling (SEM; Kline, 2004) in Mplus 5.21 (Muthén & Muthén, 2009). Maximum likelihood estimation and missing at random were used to handle parameter estimation and missing data, respectively. To assess model fit, we examined the Chi-square value, a CFI, an RMSEA, and a SRMR. The CFI greater than .90 (Kline, 2004), the RMSEA from .05 to .08 (Browne & Cudeck, 1993), and the SRMR less than .08 (Hu & Bentler, 1999) indicate a reasonable model fit.

Results

Demographic Descriptions

Demographic descriptions for cross-sectional study. Reported low points in 2001 ranged from problems with children to death of a family member. The three most frequently reported problems were career or work problems (12.05%), parent's death (10.55%), and marital/relationship problems (9.94%). Table 2.3 shows the mean and standard deviation for the cross-sectional study variables. "Learning that they could stand on their own two feet" was the most endorsed confidence item (88.02%). "Learning that family was important" was the most frequently checked item for values, with 95.20% of the individuals endorsing this item. "Having positive psychological resources" was the most endorsed resources item (94.13%). The most frequently checked item for vulnerabilities was "There are some situations that I can't do anything about," with 85.98% of the respondents endorsing it. Of the four dichotomous items assessing whether the individuals could turn anything about the situation to their advantage, the most frequently endorsed item was "Developing a new philosophy/attitude towards life" (42.98%). Only about half of the individuals, however, were able to find even one advantage.

The indicators of depressive symptoms were also examined (see Table 2.3 for mean and standard deviation). A quarter of respondents (25.20%) did not experience any somatic complaints, 70.79% of respondents scored from 1 to 4, whereas only 4.01% people scored 5 to 8, which led to a mean of 1.62 in somatic complaints. Nearly half (44.35%) of the respondents did not report any depressed affect, 50.09% reported total depressed affect from 1 to 3, and only 5.56% reported total depressed affect from 4 to 6, yielding a mean score of 1.20. Because more than half of the respondents (55.03%) reported a total positive affect score of 4 and only 4.90% reported a total score in positive affect from 0 to 2, this yielded a high mean score of 3.17. Almost three quarters (74.95%) of respondents did not experience

interpersonal problems, 24.31% reported a total score from 1 to 2, and only 0.71% reported a total score from 3 to 4. Thus, the mean level for interpersonal problems (M = .36) was remarkably low.

Demographic descriptions for the longitudinal sample. A wide variety of types of problems were reported at both time points. The most frequently reported problems at Time 1 were career or work problem (16.90%), marital/relationship problems (9.82%), parent's health (8.25%), physical health problems (self) (7.47%), and parent's death (6.88%). At Time 2, however, the pattern of stressors had changed as the respondents moved into mid-life. Even though career and work problems were still the most frequently reported stressful problems at Time 2, the percentages decreased. The number of people who lost their parents almost doubled at Time 2, indicating that the DLS participants were shifting their focus from work and career to parents' death. Specifically, the most frequently reported problems were career/work problems (11.79%), parent's death (11.20%), marital/relationship problem (10.81%), physical health problems (self) (8.45%), and parent's health (8.25%). Despite this change in types of problems, there were no significant differences in the mean levels of stress severity across the two time points (5.27 and 5.31, respectively), which suggested that most participants reported moderately high problems on the 7-point stressfulness scale.

Table 2.5 shows the mean and standard deviation for the variables in the longitudinal study. "I could stand on my own two feet," was the most frequently checked SRG confidence item (85.34% at Time 1 & 87.88% at Time 2). The least frequently checked SRG vulnerabilities item was "my health prevented me from doing as much as I would have liked" (16.60% at Time 1, 20.79% at Time 2). Two SRG resources items of "I had positive psychological resources (e.g., ability to cope)" and "I had positive social resources (e.g., good friends, neighbors, family)" were frequently checked (93.79% & 92.02% at Time 1; 94.74% & 92.26% at Time 2). Two SRG values items of "family is very important to me" and

"taking care of myself is very important to me" were most frequently reported, with 95.69% and 91.15% participants reporting them at Time 1, and 95.29% and 93.95% at Time 2. The summed total score was computed for the four binary items of the SRG advantage checklist. The most frequently checked item was "developed new philosophy/attitude towards life" (45.95% at Time 1 & 41.85% at Time 2).

With regard to the depressive symptoms in 1996, around one quarter (26.95%) of respondents did not experience any somatic complaints. Most respondents (65.07%) had a low score ranging from 1 to 3, and only 7.98% respondents had a total score from 4 to 8, which yielded the mean score in somatic complaints as 1.46. Of the respondents, 37.57% did not report any depressed affect, 58.45% reported total depressed affect from 1 to 3, and only 3.98% reported total depressed affect from 4 to 6, yielding the mean score of 1.26. Because more than half of the respondents (52.78%) did not report positive affect feelings and only 3.97% reported a total positive affect score either 3 or 4, the mean score was low (M = .82). A high percent of respondents (78.40%) did not experience interpersonal problems, 21.20% had a total score from 1 to 2, whereas only 0.40% reported a total score of 3 and no one reported a total score of 4. Thus, the mean level (M = .29) for the interpersonal problems was remarkably low.

Correlations between Cross-Sectional Study Variables

Table 2.3 shows the correlation matrix of variables examined among the cross-sectional study variables in the overall sample. The hypothesis that women would report higher SRG and depressive symptoms was partially supported. Sex was positively related to all SRG indicators (rs ranging from .11 to .20, ps < .001), but was only positively related to one indicator of depressive symptoms, i.e., depressed affect (r = .10, p < .01).

The hypothesis that younger respondents reported higher SRG and less depressive symptoms was not supported. Age was unrelated to most SRG indicators, except the

contradictory correlations of SRG values (r = .06, p < .05) and SRG advantages (r = -.09, p < .01), indicating that age might have no direct effect on the latent variable of SRG. Also, age was negatively related to three indicators of depressive symptoms (rs ranging from -.14 to -.23, ps < .01 & .001). These correlations showed that it was older people rather than younger people who reported lower levels of depressive symptoms.

The relations between the indicators of SRG and depressive symptoms were varied across indicators. The indicator of SRG resources was negatively related to most indicators of depressive symptoms (rs ranging from -.08 to -.14, ps < .01 & .001), whereas the indicator of SRG vulnerabilities was positively related to most indicators of depressive symptoms (rs ranging from .23 to .28, ps < .001). The other three SRG indicators were only positively related to positive affect of depressive symptoms (rs = .13, .19, & .11, respectively, ps < .001).

Table 2.4 shows the correlations between the indicators of SRG and depressive symptoms in the cross-sectional study in both the low and the high stress severity groups. Because our hypotheses focused on the differing relations between SRG and depressive symptoms in two stress severity groups, the descriptions here primarily concentrated on the correlations between them. Even though the correlation matrix showed very similar patterns in the two groups, in general the high stress severity group had slightly higher negative as well as positive relations among the indicators of SRG and depressive symptoms. Specifically, in the low stress severity group, the negative correlations ranged from -.09 to -.12 (ps < .05 & .01) and the positive correlations ranged from .10 to .26 (ps < .05, .01, & .001). In the high stress severity group, the negative correlations ranged from -.09 to -.18 (ps < .05, .01, & .001), and the positive correlations ranged from .09 to .29 (ps < .05 & .001).

Correlations between the Longitudinal Study Variables

The correlations between the longitudinal study variables were also examined (see Table 2.5). In general, the within-time correlations between SRG and depressive symptoms indicators were very similar across Time 1 and Time 2. The correlations were primarily negative between SRG resources and depressive symptoms indicators, and were primarily positive between SRG vulnerabilities and depressive symptoms indicators.

Table 2.5 also shows the cross-time correlations between SRG and depressive symptoms indicators. Generally, SRG vulnerabilities and resources had salient correlations with the indicators of depressive symptoms, whereas SRG confidence, advantages, and values were unrelated or only related to one indicator of depressive symptoms. The SRG vulnerabilities indicator was significantly related to all of the depressive symptoms indicators, with the exception of SRG vulnerabilities at Time 2 with interpersonal problems at Time 1. These significant within-time and cross-time correlations between SRG vulnerabilities and depressive symptoms indicators indicated that SRG vulnerabilities might be one of the depressive symptoms indicators in the present study given the close relations between vulnerability and depressive symptoms established in previous studies (Abramson, Metalsky, & Alloy, 1989; Beck, Rush, Shaw, & Emery, 1979; Metalsky & Joiner, 1992). The SRG resources indicator was also significantly related to most of the depressive symptoms indicators. Unlike SRG vulnerabilities, however, the correlations between SRG resources and depressive symptoms were not stable over time. For example, SRG resources indicator at Time 1 was unrelated to depressed affect at Time 2, whereas SRG resources indicator at Time 2 was negatively related to depressed affect at Time 1.

The correlation of sex, age, and time interval with SRG and depressive symptoms indicators were also indicated in Table 2.5. Sex was positively related to all SRG indicators at both Time 1 and Time 2, whereas age was unrelated to most SRG indicators. In contrast, sex was only related to two indicators of depressive symptoms over the two time points,

whereas age was related to half of the depressive symptoms indicators, and primarily negatively related. These results indicated that women reported higher levels of SRG than men, which was consistent with our third hypothesis; and perhaps older individuals reported lower levels of depressive symptoms than younger individuals, which was contrary to our third hypothesis.

The last row in Table 2.5 shows the correlation between time interval and the other variables in the longitudinal study. The time interval was unrelated to most of the variables, except depressed affect and somatic complaints at Time 1 and SRG advantages at Time 2 (*r*s = -.14, -.16, & -.09, respectively). The results were inconsistent with our hypotheses about the negative relations of the time interval with SRG and depressive symptoms at Time 2. Given this finding, the time interval was not included in all of the following cross-lagged models.

Because the research questions mainly focused on the potential differing relations between SRG and depressive symptoms across stress severity groups, Table 2.6 only displays the within-time and cross-time correlations between SRG and depressive symptoms indicators in the low and high stress severity groups. With regard to the within-time correlations at Time 1, in the low stress severity group, there were four significantly weak negative correlations (*r*s ranging from -.13 to -.19, *p*s < .05 & .01) and three positive correlations (*r*s ranging from .20 to .27, *p*s < .001), whereas in the high stress severity group, there was one significantly weak negative correlation (*r* = -.13, *p* < .05) and six moderate positive correlations (*r*s ranging from .23 to .34, *p*s < .001).

There were some salient differences across groups regarding the cross-time correlation between SRG and depressive symptoms indicators. Specifically, the negative relations between SRG indicators at Time 1 and depressive symptoms indicators at Time 2 were very similar in the low (*r* = -.13, *p* < .05) and high stress severity groups (*r* = -.14,

$p < .05$), whereas there were three positive correlations in the low stress severity group (rs ranging from .18 to .21, ps < .01 & .001) and five positive correlations in the high stress severity group (rs ranging from .14 to .23, ps < .05). Among the indicators of depressive symptoms at Time 1 and SRG at Time 2, there was one more negative correlation in the low stress severity group (rs = -.14 & -.21, ps < .05 & .001) than in the high stress severity group (r = -.15, p < .05). There were two more positive correlations in the high stress severity group (rs ranging from .18 to .20, ps < .01 & .001) than in the low stress severity group (rs ranging from .15 to .28, ps < .05 & .001). These results indicated that the relations between SRG and depressive symptoms might be different across groups in the cross-lagged model, which was examined in the following sections.

Factor Model Fit of Depressive Symptoms

CFA was used to verify the model fit on the four factors described by Kohout and his colleagues (1993). The model had an excellent fit using the 2001cross-sectional data: $X^2(2, N = 1{,}140) = 3.361$, $p = .186$, CFI = .998, RMSEA = .025, SRMR = .010. The same model was replicated using the longitudinal data, which had excellent fit to the data as well. Specifically, the fit statistics for the 1996 data were $X^2(2, N = 509) = 10.357$, $p < .01$, CFI = .967, SRMR = .029, and for the 2001 data were $X^2(2, N = 509) = 2.278$, $p = .320$, CFI = .997, RMSEA = .017, SRMR = .017. These results indicated that the four factors model of CES-D had a good fit, and thus was used in our SEM analyses.

Evaluating Between-Group Differences in the Relations between SRG and Depressive Symptoms in the Cross-Sectional Sample

The cross-sectional relation between SRG and depressive symptoms was estimated using multiple-group analyses. Specifically, we started with a one-factor CFA model in both groups.

One-group CFA model. In Stage 1, we first conducted a one-group CFA model in the low stress severity group and then in the high stress severity group. The hypothesized models between SRG and depressive symptoms (Figure 2.2 & 2.3) did not fit well to the data. In the low stress severity group, the fit statistics were $X^2(39, N = 591) = 219.217$, $p < .001$, CFI = .874, RMSEA = .088, SRMR = .076, and in the high stress severity group, they were $X^2(39, N = 549) = 220.249$, $p < .001$, CFI = .853, RMSEA = .092, SRMR = .080. The results of the hypothesized model showed that the effects from age to SRG and from sex to depressive symptoms were not significant in either model. The modification indices suggested that SRG vulnerabilities might be one indicator of depressive symptoms, which makes sense given the close relations between vulnerabilities and depressive symptoms (Abramson et al., 1989; Beck et al., 1979; Metalsky & Joiner, 1992). Therefore, the two models were re-estimated dropping the two non-significant paths and adding one factor loading from depressive symptoms to SRG vulnerabilities.

As shown in Table 2.7, in both stress severity groups, the re-estimated models had good fits to the data. Specifically, in the low stress severity group, the fit statistics were $X^2(40, n = 591) = 157.952$, $p < .001$, CFI = .918, RMSEA = .071, SRMR = .060, and in the high stress severity group, they were $X^2(40, n = 549) = 136.027$, $p < .001$, CFI = .922, RMSEA = .066, SRMR = .049. The fit for the two re-estimated models was significantly better than the two previous ones (i.e., hypothesized models). The fit statistics for the low stress severity group were $\Delta X^2(1, n = 591) = 61.265$, $p < .001$, and for the high stress severity group were $\Delta X^2(1, n = 549) = 84.222$, $p < .001$. The re-estimated models showed that both models had good fits to the data, indicating that it was possible to proceed with multiple-group analysis of equal form (Brown, 2006; Thompson & Green, 2006).

Multiple-group CFA of equal form. In the first step of Stage 2, we assumed that the latent variables of SRG and depressive symptoms load on the same indicators across groups.

Also, in this step, the covariance between SRG and depressive symptoms and the factor loadings between latent variables and their indicators were all freely estimated across groups. As shown in Table 2.7, this multiple-group CFA provided a good fit to the data: $X^2(80, N = 1140) = 293.979$, $p < .001$, CFI = .920, RMSEA = .069, SRMR = .055. Because the equal form model had an overall good fit, a model with equal factor loadings was evaluated next.

Multiple-group CFA of equal factor loadings. In the second step of Stage 2, all factor loadings were constrained to be equal across groups, while the covariance between SRG and depressive symptoms and the intercepts of all SRG and depressive symptoms indicators were freely estimated. Table 2.7 showed that this model had good fit statistics: $X^2(88, N = 1140) = 324.666$, $p < .001$, CFI = .911, RMSEA = .069, SRMR = .060. The difference in X^2 (i.e., ΔX^2) between this model and the previous model was significant: $\Delta X^2(8, N = 1140) = 30.686$, $p < .001$, indicating that the model with equal factor loadings was significantly worse than the previous model. Therefore, the multiple-group CFA of equal form was used to analyze the hypothesized model (Figure 2.2 & 2.3) to compare the potential covariance difference between SRG and depressive symptoms across stress severity groups.

Assessment of covariance difference between latent variables. As indicated above, the multiple-group CFA of equal form with the covariance between SRG and depressive symptoms freely estimated (i.e., the first step of Stage 2) had a good fit to the data. Figure 2.6 and 2.7 show the cross-sectional covariance between SRG and depressive symptoms in the low ($b = .000$, $p = .947$) and high ($b = -.039$, $p < .001$) stress severity groups, respectively. The results seemed to support our first hypothesis that SRG and depressive symptoms were unrelated in the low stress severity group and were negatively related in the high stress severity group.

However, in order to test whether the differences in the covariance across groups were statistically significant or not, we calculated a chi-square difference in the models between

the freely estimated covariance and equally constrained covariance. Multiple-group CFA was used to constrain the covariance (Figure 2.2 & 2.3) to be equal, which yielded the model fit was $X^2(81, N = 1140) = 305.011$, $p < .001$, and a covariance was -.006. The chi-square difference (i.e., ΔX^2) between the model with this constraint and the model without the constraint was $\Delta X^2(81 - 80 = 1) = 305.011 - 293.980 = 11.032$, $p < .001$. These results indicated that there were significant differences in the covariance between SRG and depressive symptoms across the two stress severity groups. Therefore, our first hypothesis that there were stratified relations between SRG and depressive symptoms by stress severity was strongly supported.

Evaluating Between-Group Differences in the Relations between SRG and Depressive Symptoms in the Longitudinal Sample

Both the within-time and cross-lagged effects between SRG and depressive symptoms were examined in the low (Figure 2.4) and the high (Figure 2.5) stress severity groups. Applying the same procedure for estimating the cross-sectional difference across groups, we examined whether there were significant group differences in the within-time and cross-lagged effects.

One-group CFA model. In Stage 1, the one-group CFA model was examined in the low and high stress severity groups, independently. The two hypothesized models (Figure 2.4 & 2.5) results indicated that SRG vulnerabilities indicator was one of indicators of depressive symptoms, and the residuals for vulnerabilities, SRG resources, SRG values, SRG advantages, CES-D positive affect, and CES-D somatic complaints were autocorrelated. Therefore, the hypothesized models were re-estimated by adding the factor loading from depressive symptoms to SRG vulnerabilities as well as the autocorrelated residuals. The re-estimated models yielded good fits to the data (see Table 2.8) both in the low stress severity group: $X^2(151, n = 283) = 235.367$, $p < .001$, CFI = .940, RMSEA = .044, SRMR = .062, and

in the high stress severity group: $X^2(151, n = 226) = 271.691$, $p < .001$, CFI = .909, RMSEA = .059, SRMR = .079. Because the fit statistics for both models were acceptable, multiple-group analysis of equal form was examined in the next stage.

Multiple-group CFA of equal form. In the first step of Stage 2, the form of the latent variable and their indicators were assumed to be equal, while all other variances and paths were freely estimated across groups. Table 2.8 shows that in this step, the multiple-group CFA fit well to the data: $X^2(302, N = 509) = 507.059$, $p < .001$, CFI = .925, RMSEA = .052, SRMR = .070. Thus, in the next step, the multiple-group CFA using equal factor loadings was assessed.

Multiple-group CFA of equal factor loadings. In this step, all factor loadings across groups were set to be equal, whereas all other variances, covariance, and paths were freely estimated. The multiple-group CFA results showed that this measurement invariance had a good fit to the data (see Table 2.8): $X^2(318, N = 509) = 531.334$, $p < .001$, CFI = .922, RMSEA = .051, SRMR = .073. The chi-square difference (i.e., ΔX^2) between this model and the multiple group CFA of equal form was not significant: $\Delta X^2(16, N = 509) = 24.275$, $p = .084$. The results indicated that there were no significant differences in the fit between this model and the previous model. Therefore, the multiple-group CFA of equal factor loadings and intercepts was tested in the next step.

Multiple-group CFA of equal factor loadings and intercepts. In the third step of Stage 2, we constrained both factor loading and intercepts (i.e., means) of the indicators were equal, while left the errors, covariance, and other effects freely estimated. Table 2.8 shows that the model fit was good: $X^2(332, N = 509) = 558.342$, $p < .001$, CFI = .918, RMSEA = .052, SRMR = .073. However, further tests of the difference in the chi-square difference between this model and the previous model showed that the current model was a much worse

fit to the data, $\Delta X^2(\Delta df = 14) = 27.008$, $p < .05$. Thus, the multiple-group CFA with equal factor loadings was used in the following analyses.

Assessing cross-group difference in the relations between SRG and depressive symptoms. Figure 2.8 and 2.9 show the cross-lagged model results with equal factor loadings in the low and high stress severity group (To make the illustrated figures easier to read, we did not include the autocorrelated residuals even though they were included in the statistical analyses). The results showed that our second hypothesis seemed to be partially supported and that the within-time and cross-lagged effects between SRG and depressive symptoms were different across groups. Specifically, in the low stress severity group, the within-time effect from SRG to depressive symptoms at Time 1 was not significant (b = -.111, p = .113), but it was significant at Time 2 (b = -.163, $p < .05$). The cross-lagged effects between SRG and depressive symptoms were not significant (b = .093 & .001). These results suggested that in the low stress severity group, mostly there were no significant relations between SRG and depressive symptoms either within time or across time (Hobfoll, 1988; Aldwin et al., 1996; Zautra, 2003).

In the high stress severity group, the within-time effect from SRG to depressive symptoms was statistically significant (bs = -.174 & -.168, ps < .05). The cross-lagged effect from depressive symptoms at Time 1 to SRG at Time 2 was also statistically significant (b = .174, $p < .05$), whereas it was not significant from SRG at Time 1to depressive symptoms at Time 2 (b = .136, p =.076). These within-time results indicated that SRG and depressive symptoms were unidimensionally related; namely that when one was high, the other was low (Antonovsky, 1979; Aldwin et al., 1996; Zautra, 2003). The positive cross-lagged effect from depressive symptoms at Time 1 to SRG at Time 2 indicated that previous depressive symptoms motivated the development of subsequent SRG (Tedeschi & Calhoun, 2004). The non-significant cross-lagged effect from SRG at Time 1 to depressive symptoms

at Time 2 indicated that when the level of stress severity was high, SRG still had no effects on depressive symptoms over time (Zautra, 2003).

The analyses above, however, did not determine whether the group difference in the relations between SRG and depressive symptoms was substantively significant or not. To test this difference, the four paths between SRG and depressive symptoms were simultaneously constrained to be equal across groups, and then the chi-square difference between this model and the model with freely estimated the four paths was calculated. The chi-square in the constrained model was $X^2(322, N = 509) = 534.246$, $p < .001$. Thus, the chi-square difference was $\Delta X^2 = 534.246 – 531.334 = 2.912$, $\Delta df = 322 – 318$, $p = .573$, indicating that there was no substantively significant differences in the relations between SRG and depressive symptoms across the two stress severity groups.

The autoregressive effects of SRG and depressive symptoms were positive in both groups, which was consistent with our second hypothesis. Specifically, in the low stress severity group, the autoregressive effects of SRG and depressive symptoms were *b*s = .557 and .486 (*p*s < .001), and in the high stress severity group, they were *b*s = .477 and .636 (*p*s < .001). These results indicated that previous SRG and depressive symptoms positively predicted subsequent changes.

Neuroticism, SRG, and Depressive Symptoms

Because the autocorrelation of SRG and depressive symptoms in both stress severity groups were high, an exploratory analysis was conducted to see whether personality – specifically, neuroticism – acted as a potential confounding factor. We used partial correlations to examine the impact of neuroticism assessed by the EPI-Q (Floderus, 1974). Results showed that all partial autocorrelations still remained significant, and most did not change more than five points, except somatic complaints and depressed affects of depressive symptoms.

Discussion

Summary of Results

Within the existing literature on the relations between SRG and depressive symptoms, findings were mixed both cross-sectionally and longitudinally. The major purpose of the present study was to examine whether the within-time and cross-lagged effects between SRG and depressive symptoms were stratified by stress severity. Specifically, in the low stress severity group, are SRG and depressive symptoms unrelated to one another? In the high stress severity group, are SRG and depressive symptoms negatively related to one another?

Earlier researchers proposed and found inconsistent relations between SRG and depressive symptoms. Antonovsky (1979) argued that SRG and depressive symptoms are negatively related to one another; namely that, previous SRG is inversely related to subsequent depressive symptoms, and vice verse. Tedeschi and Calhoun (1996; 2004), however, argued that their relations are unidirectional; namely that, depressive symptoms motivate the development of SRG, while SRG does not have effect on subsequent depressive symptoms.

The relations between SRG and depressive symptoms may be stratified by stress severity (Aldwin et al., 1996; Hobfoll, 1988; Hobfoll & Lilly, 1993; Zautra, 2003), but still there are some differences among these researchers. Hobfoll and Zautra argued when the level of stress severity is low, they are independent of one another, while when the level of stress severity is high, depressive symptoms have significant negative effects on the development of SRG over time but SRG does not have significant negative effects on depressive symptoms. Aldwin and her colleagues' arguments are partially consistent with Hobfoll and Zautra's arguments. Similar to them, Aldwin et al. proposed that SRG and depressive symptoms are unrelated when the level of stress severity is low. But, different

from them, when the levels of stress severity is high, not only depressive symptoms are related to reduced SRG over time but also SRG is related to decreased depressive symptoms.

The stratified relations between SRG and depressive symptoms were fully supported by cross-sectional samples. That is, in the low stress severity group, SRG and depressive symptoms were unrelated to one another, whereas in the high stress severity group, they were negatively related to one another. Thus, these results are consistent with the arguments of COR (Hobfoll, 1988; Hobfoll & Lilly, 1993), DAM (Aldwin et al., 1996), and DMA (Zautra, 2003).

The stratified relations between SRG and depressive symptoms were partially supported by longitudinal samples. In the low stress severity group, there were no cross-lagged effects between SRG and depressive symptoms as well as no significant within-time effects from SRG to depressive symptoms at Time 1. In the high stress severity group, SRG had significantly negative within-time effects but no significant cross-lagged effect on depressive symptoms, and the cross-lagged effect of depressive symptoms on SRG was significantly positive instead of negative.

These findings indicated that the longitudinal relations between SRG and depressive symptoms were very complicated and only partially supported the five theories described in the literature review section. Specifically, in the low stress severity group, Hobfoll (1988), Aldwin et al. (1996), and Zautra's (2003) arguments about non-significant relations between SRG and depressive symptoms were mostly supported, with the exception of the significantly negative within-time effect at Time 2, although this finding may have been due to chance. In the high stress severity group, the negative within-time relations described by Antonovsky (1979), Aldwin et al. (1996), and Zautra (2003) were supported, indicating that the higher levels of SRG reported by respondents, the lower levels of depressive symptoms they experienced. With regard to the cross-lagged effect, the positive effect from depressive

symptoms to SRG seemed to support Tedeschi and Calhoun's (2004) argument, whereas the non-significant positive cross-lagged effect from SRG to depressive symptoms2 did not provide substantial support for Hobfoll's (2006) findings but did support Zautra's argument (2003). This indicated that over a long period of time, depressive symptoms did initiate the development of SRG (Tedeschi & Calhoun, 2004), whereas the development of SRG had no effects on subsequent depressive symptoms (Zautra, 2003).

However, further statistical analyses by testing the chi-square difference between the within-time and cross-lagged effects freely estimated and equally constrained showed that there were no statistically significant differences in the four effects between SRG and depressive symptoms across stress severity groups. Thus, the stratified effect of stress severity on the relations between SRG and depressive symptoms were not fully supported by longitudinal samples.

Explanations about the Results

There are at least two possible reasons to explain why the results did not support our hypotheses in the longitudinal study samples. One explanation was that the longitudinal study might not have a large enough sample size (i.e., lack of power) to detect differences across stress severity groups. Specifically, the total longitudinal sample size was 509, with 283 and 226 respondents in the low and high stress severity groups, respectively. In contrast, the total cross-sectional sample size was 1,140, with 591 and 549 respondents in the low and high stress severity groups, respectively. Therefore, the sample size in the cross-sectional study was more than double that of in the longitudinal study, both in total and by group. However, there were fewer estimated parameters in the cross-sectional study than in the longitudinal study. Given the small sample size but more estimated parameters in longitudinal study, it might be make sense why no significant differences were found in the relations between SRG and depressive symptoms across stress severity groups.

The other plausible explanation relates to stress severity. First, there were a high proportion of respondents at the median level of stress severity in the low stress severity group. Specifically, the median level of stress severity was 5 on the 7-Likert scale of stressfulness, and respondents at the median (n = 164) accounted for 57.95% of total 283 respondents in the low stress severity group of the longitudinal samples. The high proportion of respondents at the median stress severity level might obscure the significant difference in the relations between SRG and depressive symptoms across groups. Second, approximately 30% (n = 145) of the 509 respondents reported unchanging stress severity levels over time. Among the 145 respondents, slightly more than half (54.48%) reported high stressfulness levels. Because SRG and depressive symptoms were strongly impacted by stress severity, the high proportion of respondents reporting unchanging stress severity might also obscure the stratified relations between SRG and depressive symptoms.

Given the two possible reasons, it might make sense why there were no significant differences in the final stage of the chi-square analyses. Future studies using a bigger sample size and better dealing with the variable of stress severity may find significant stratified relations between SRG and depressive symptoms by stress severity.

Double Loadings of Vulnerabilities on SRG and Depressive Symptoms

It should be noted that the factor of vulnerabilities loaded both on SRG and depressive symptoms. As mentioned before, vulnerabilities was one of the important factors for SRG since one's perceived weakness in dealing with stress may indicate respondents have better thoughts about their abilities and situations. In addition, it has been pointed out that vulnerability is one of the important aspects in depression studies (Ingram & Price, 2000). Cognitive vulnerabilities (i.e., negative thoughts towards the self or environment) were strongly related to depressive symptoms (Abramson et al., 1989; Beck et al., 1979;

Metalsky & Joiner, 1992). Therefore, it is reasonable that the factor of SRG vulnerabilities also loaded on depressive symptoms.

Limitations and Future Studies

The primary advantage of the present study was that it examined both within-time and cross-lagged effects between SRG and depressive symptoms simultaneously. Even though this study made a great contribution to the literature about the relations between SRG and depressive symptoms, there were several limitations that should be noted.

Demographic characteristics. First, given the highly homogeneous demographic characteristic of the samples, the current results may not be applicable to other populations. Specifically, the present study mainly examined middle-aged European Americans ranging in age from 30 to 50 years-old, who had at least a bachelor's degree and high levels of family income. Therefore, future studies need to replicate this study in a broader population to see how stress severity stratifies the relations between SRG and depressive symptoms. Because previous studies have shown that in comparison to those with higher socioeconomic status (SES), people with lower SES reported higher levels of SRG (for a review, see Lechner & Weaver, 2009) as well as depressive symptoms (for a review, see Lorant et al., 2003), and thus different results might be generated among lower SES individuals.

Measurement issues. Two measurement issues need to be addressed. First, two indicators of depressive symptoms, i.e., positive affect and interpersonal problems, were examined by two items in the CES-D, respectively. The too few items may not correctly capture the construct of latent variables (Hertzog, Van Alstine, Usala, Hultsch, & Dixon, 1990; Marjorie, Lackey, & Sullivan, 2003).

Second, both SRG and depressive symptoms were assessed by respondents' retrospective reports. Researchers examining both SRG (Park & Lechner, 2006; Tennen & Affleck, 2009) and depressive symptoms (for a review, see Schraedley, Turner, & Gotlib,

2002) have argued that retrospective reports may distort respondents' perceptions of their past negative events, produce response bias, and thus inaccurately capture SRG and depressive symptoms. Given these measurement limitations, future studies may use more items to examine positive affect and interpersonal problems, and combine with other assessments, such as interviews or reports by others, to estimate SRG and depressive symptoms.

SRG and depressive symptoms. Also, future studies need to further examine the relations between SRG and depressive symptoms from the following four aspects. First, given the relatively small sample size in the longitudinal study, future studies need to examine whether the relations between SRG and depressive symptoms are stratified or moderated by stress severity among larger longitudinal samples.

Second, this study did not investigate the relations between SRG and depressive symptoms under traumatic conditions even though trauma is highly prevalent and has tragic impacts on people. The present study mainly examined respondents' major life stressors and then divided the rating of stress severity into two groups (i.e., low and high stress severity groups). Therefore, how SRG and depressive symptoms are related in a traumatic stress group is unknown. Are they positively related to one another as Tedeschi and Calhoun (2004) and Hobfoll et al. (2007) argued?

Third, the present study did not examine the mechanism of how SRG exerts it effect on depressive symptoms as well as the effect of depressive symptoms on SRG. Are the relations between SRG and depressive symptoms accounted for by different mechanisms, such as meaning making (Park, 2009) and shattered assumptions (Janoff-Bulman, 1992), under different levels of stress severity?

Fourth, not all potential confounding factors of personality (Evers et al., 2001; Garnefski et al., 2008; Alexander et al., 2009) were examined in the present study. As described earlier, the autocorrelation of SRG and depressive symptoms in both stress severity

groups were high. Taking into account the potential effect of neuroticism at Time 1, our exploratory analyses examined the partial correlations of the indicators of SRG and depressive symptoms over time and found that neuroticism only accounted for minimal effects of their autocorrelations. These results indicated that neuroticism was not a confounding factor. However, the present study did not examine the effect of other potential confounding factors of personality such as extraversion and conscientiousness (Anderson & McLean, 1997; Garnefski et al., 2008; Jylha & Isometsa, 2006). Therefore, it is very important for future studies to examine whether these personality factors have significant effects on the autocorrelations of SRG and depressive symptoms.

In summary, the results of this study supported the stratified or moderated effect of stress severity on the relations between SRG and depressive symptoms in the cross-sectional sample but not in the longitudinal sample. That is, the present study found the orthogonal or independent cross-sectional relation between SRG and depressive symptoms in the low stress severity group, and the unidimensional or negative cross-sectional relations in the high stress severity group. However, the significantly different relations across groups were not replicated in the longitudinal study. This may be because the smaller sample size and relatively restricted range of stress severity have obscured this pattern of results. Future studies should utilize larger samples and better deal with the variable of stress severity.

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Table 2.1

Demographic differences between stress severity groups, cross-sectional sample (N = 1,140)

Demographic characteristics	Low stress severity (*n* = 591)	High stress severity (*n* = 549)	*T* or X^2	*p*
Age (*M*)	44.63	44.28	.53	.30
Income (*M*)	4.76	4.67	1.07	.14
% Male	52.23%	38.53%	21.31	< .001
% Anglo	84.69%	82.97%	.62	.43
% Married	66.27%	65.14%	.16	.69
% Employed full-time	79.66%	76.42%	1.75	.19
% Higher education	50.60%	47.16%	1.34	.25

Table 2.2

Demographic differences between stress severity groups, longitudinal sample (N = 509)

Demographic characteristics	Low stress severity (*n* = 283)	High stress severity (*n* = 226)	*T* or X^2	*p*
Age (*M*)	43.80	44.78	-1.49	.07
Income (*M*)	4.68	4.69	-.09	.46
% Male	47.35%	39.82%	2.89	.09
% Anglo	93.59%	93.30%	.02	.90
% Married	77.58%	74.67%	.59	.44
% Employed full-time	80.92%	78.76%	.24	.62
% Higher education	45.94%	43.36%	.34	.56

Table 2.3

Correlations among the Cross-Sectional Study Variables (N = 1,140)

Variables	1	2	3	4	5	6	7	8	9	10	11
1. Sex (*1 = female*)	--										
2. Age	-.16***	--									
3. SRG Confidence	.19***	-.00	--								
4. SRG Resources	.18***	.05	.55***	--							
5. SRG Values	.20***	.06*	.50***	.56***	--						
6. SRG Advantages	.11***	-.09**	.41***	.23***	.28***	--					
7. SRG Vulnerabilities	.16***	-.04	.40***	.18***	.27***	.15***	--				
8. Somatic Complaints	.05	-.14***	.01	-.12***	.04	.03	.28***	--			
9. Depressed Affect	.10**	-.19***	-.03	-.14***	-.02	.00	.27***	.53***	--		
10. Positive Affect	.02	.08**	.13***	.24***	.19***	.11***	-.16***	-.38***	-.53***	--	
11. Interpersonal Problems	-.01	-.23***	.01	-.08**	-.06	.02	.23***	.28***	.32***	-.25***	--
M	--	44.45	1.61	2.10	2.07	1.02	1.08	1.62	1.20	3.17	.36
SD	--	11.06	.80	.76	.68	.86	.57	.15	1.37	1.05	.68

Note. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2.4

Correlations among the Indicators of SRG and Depressive Symptoms in the Cross-Sectional Study by Stress Severity Groups

Variables	SRG Confidence	SRG Resources	SRG Values	SRG Advantages	SRG Vulnerabilities
			Low stress severity group (N = 591)		
Somatic Complaints	.10*	-.09*	.12*	.04	.26***
Depressed Affect	-.00	-.15**	.02	.05	.26***
Positive Affect	.12**	.22***	.17***	.07	-.12**
Interpersonal Problems	-.01	-.14**	-.04	.03	.21***
			High stress severity group (N = 549)		
Somatic Complaints	.09*	-.16***	-.07	.01	.29***
Depressed Affect	-.10*	-.14**	-.09*	-.06	.24***
Positive Affect	.17***	.28***	.24***	.15***	-.18***
Interpersonal Problems	.02	-.03	-.08	.01	.25***

Note. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2.5

Within-Time and Across-Time Correlations among the Longitudinal Study Variables (N = 509)

Variable	SRG Con 1	SRG Res 1	SRG Val 1	SRG Adv 1	SRG Vul 1	SRG Con 2	SRG Res 2	SRG Val 2	SRG Adv 2	SRG Vul 2	Age	Sex	Time Interval	*M*	*SD*
Depressed Affect 1	-.05	-.14**	-.07	-.02	.27***	.04	-.10*	-.05	.05	.19***	-.04	.11*	-.14**	1.26	1.29
Positive Affect 1	.18***	.30***	.20***	.09	-.11*	.04	.24***	.21***	.09	-.10*	.01	.10*	.06	3.18	.99
Somatic Complaint 1	-.02	-.10*	-.03	.00	.29***	.04	-.18***	-.05	.06	.21***	-.09*	.09	-.16***	1.46	1.37
Interpersonal Problems 1	-.01	-.12**	-.10*	-.05	.09*	.03	-.03	-.06	.04	.02	-.15***	.06	.07	.29	.61
Depressed Affect 2	.00	-.08	-.05	.02	.16***	-.05	-.13**	-.04	.02	.23***	-.08	.05	.00	1.16	1.32
Positive Affect 2	.08	.17***	.19***	.06	-.09*	.13**	.25***	.22***	.11*	-.18***	.09*	.05	-.02	3.18	1.04
Somatic Complaint 2	.02	-.05	-.04	.07	.19***	-.02	-.17***	-.00	.04	.22***	-.08	.00	-.03	1.55	1.38
Interpersonal Problems 2	-.03	-.13***	-.08	.01	.10*	-.00	-.15***	-.04	.03	.20***	-.15***	.01	.01	.25	.57
Age	.04	.07	.02	-.02	.10*	.02	.11*	.05	-.02	.04	--				
Sex	.21***	.17***	.21***	.13**	.18***	.14**	.18***	.16***	.10*	.13**	-.12**	--			
Time Interval	-.03	-.01	-.03	.05	-.04	-.03	.00	-.08	-.09*	-.01	.03	-.06	--		
M	1.55	2.09	2.07	1.12	1.07	1.63	2.14	2.12	1.01	1.05	44.23	--	4.64		
SD	.80	.77	.65	.91	.55	.78	.76	.65	.87	.55	7.38	--	2.12		

Note. SRG Con = SRG Confidence; SRG Res = SRG Resources; SRG Val = SRG Values; SRG Adv = SRG Advantages; SRG Vul = SRG Vulnerabilities.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2.6

Correlations among the Longitudinal Study Variables by Stress Severity Groups

Variables	SRG Con 1	SRG Res 1	SRG Val 1	SRG Adv 1	SRG Vul 1	SRG Con 2	SRG Res 2	SRG Val 2	SRG Adv 2	SRG Vul 2
Low stress severity group (n = 283)										
Depressed Affect 1	-.03	-.16**	-.05	-.00	.22***	-.01	-.12	-.10	.00	.18**
Positive Affect 1	.09	.27***	.12	-.04	-.13*	.04	.20***	.20***	.08	-.14*
Somatic Complaint 1	-.10	-.19**	-.07	-.05	.20***	-.08	-.21***	-.12	.02	.10
Interpersonal Problems 1	.07	-.16**	-.08	-.02	.07	-.02	-.03	-.09	.04	.03
Depressed Affect 2	-.01	-.10	-.10	.03	.18**	-.05	-.13*	-.11	-.05	.20***
Positive Affect 2	.08	.18**	.21***	-.00	-.05	.16**	.26***	.24***	.14*	-.12*
Somatic Complaint 2	-.04	-.04	-.06	.07	.12	-.06	-.18**	-.05	.00	.08
Interpersonal Problems 2	-.00	-.13*	-.13	.02	.04	-.09	-.18**	-.11	-.08	.12
High stress severity group (n = 226)										
Depressed Affect 1	-.09	-.13*	-.09	-.08	.27***	.09	-.07	.02	.05	.15*
Positive Affect 1	.30***	.34***	.31***	.23***	-.09	.04	.28***	.23***	.11	-.05
Somatic Complaint 1	.04	-.03	.01	.03	.33***	.16*	-.15*	.03	.06	.27***
Interpersonal Problems 1	-.10	-.08	-.12	-.08	.13	.10	-.02	-.03	.05	.02
Depressed Affect 2	-.01	-.06	.01	-.00	.11	-.05	-.13	.04	.05	.23***
Positive Affect 2	.12	.16*	.16*	.14*	-.10	.11	.24***	.20**	.10	-.22**
Somatic Complaint 2	.06	-.07	-.01	.06	.23***	.02	-.15**	.05	.05	.33***
Interpersonal Problems 2	-.06	-.14*	-.01	-.01	.16*	.10	-.11	.04	.12	.28***

Note. SRG Con = SRG Confidence; SRG Res = SRG Resources; SRG Val = SRG Values; SRG Adv = SRG Advantages; SRG Vul = SRG Vulnerabilities.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2.7

Summary of Fit Statistics for Testing Cross-Sectional Measurement Invariance of SRG and CESD in Low and High Stress Severity Groups

	X^2	df	ΔX^2	Δdf	CFI	RMSEA	SRMR
Single Group Solutions (N =1140)							
Low stress (n = 591)	157.952***	40			.918	.071	.060
High stress (n = 549)	136.027***	40			.922	.066	.049
Measurement Invariance							
Equal form	293.979***	80			.920	.069	.055
Equal factor loadings	324.666***	88	30.686***	8	.911	.069	.060

Note. * $p < .05$. ** $p < .01$. *** $p < .001$.

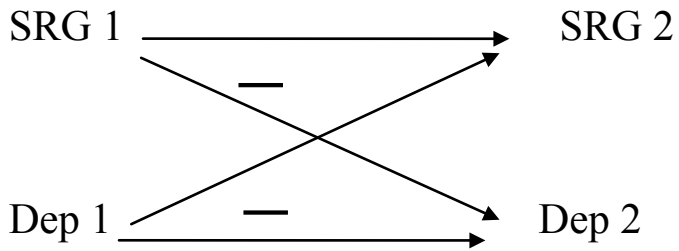
Table 2.8

Summary of Fit Statistics for Testing Longitudinal Measurement Invariance of SRG and CESD in Low and High Stress Severity Groups

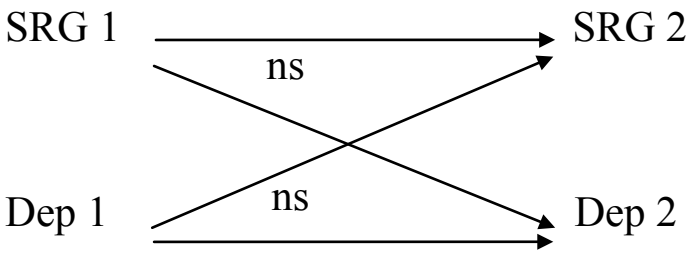
	X^2	df	ΔX^2	Δdf	CFI	RMSEA	SRMR
Single Group Solutions (N =509)							
Low stress (n = 283)	235.367***	151			.940	.044	.062
High stress (n = 226)	271.691***	151			.909	.059	.079
Measurement Invariance							
Equal form	507.059***	302			.925	.052	.070
Equal factor loadings	531.334***	318	24.275	16	.922	.051	.073
Equal indicator intercepts	558.342***	332	27.008*	14	.918	.052	.073

Note. * $p < .05$. ** $p < .01$. *** $p < .001$.

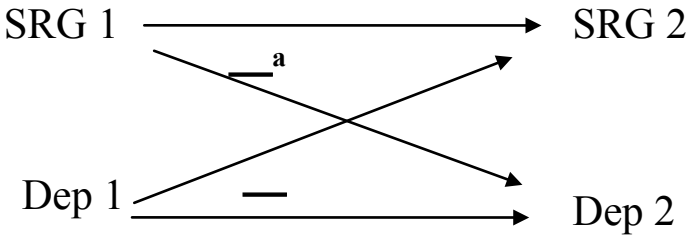
Figure 2.1. Five Theories about the Relations between Stress-Related Growth and Depressive Symptoms



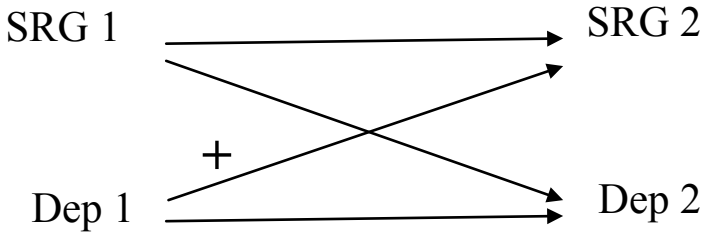
Antonovsky: When stress severity is high, SRG and depressive symptoms will be inversely related over time.



Hobfoll: When stress severity is low, SRG and depressive symptoms will be unrelated over time.

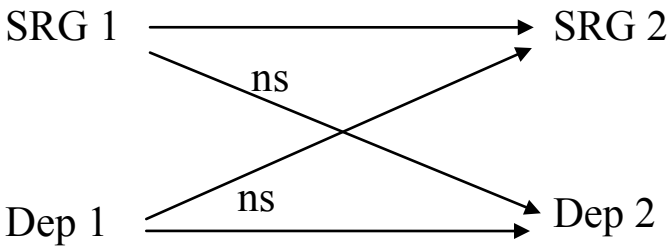


Hobfoll: When stress severity was high, SRG and depressive symptoms will be inversely related over time.
[a]marginally significant

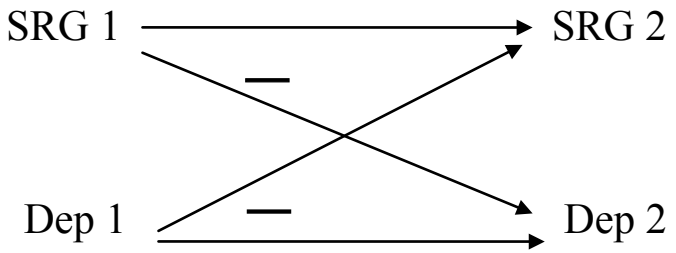


Tedeschi & Calhoun: When stress severity is traumatic, SRG and depressive symptoms will be positively related over time.

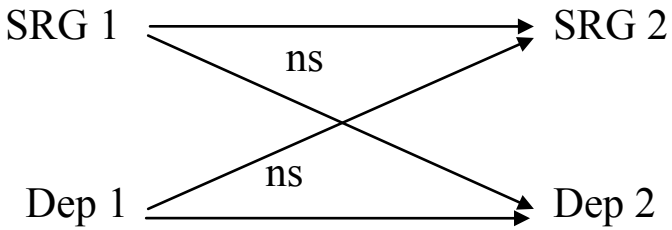
Figure 2.1. *continued*



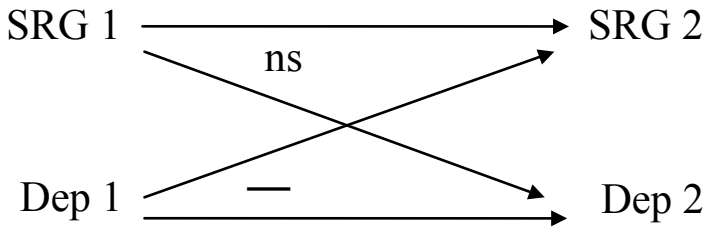
Aldwin: When stress severity is low, SRG and depressive symptoms will be unrelated over time.



Aldwin: When stress severity is high, SRG and depressive symptoms will be inversely related over time.

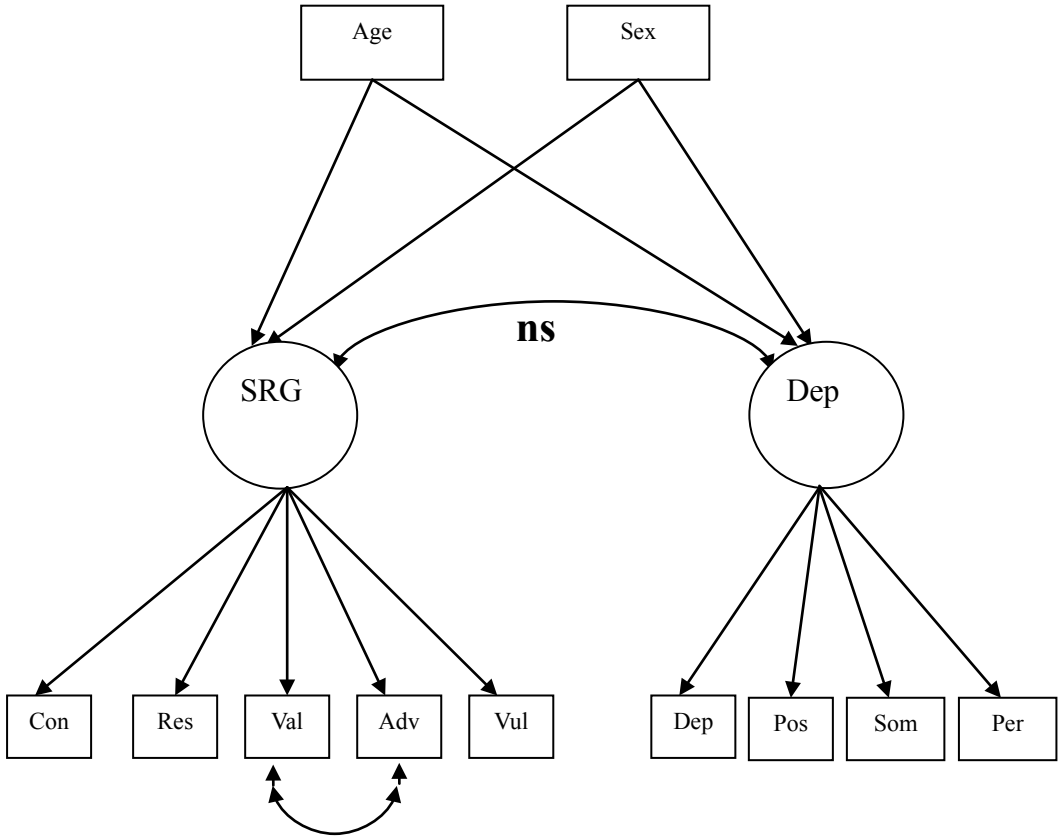


Zautra: When stress severity is low, SRG and depressive symptoms will be unrelated over time.



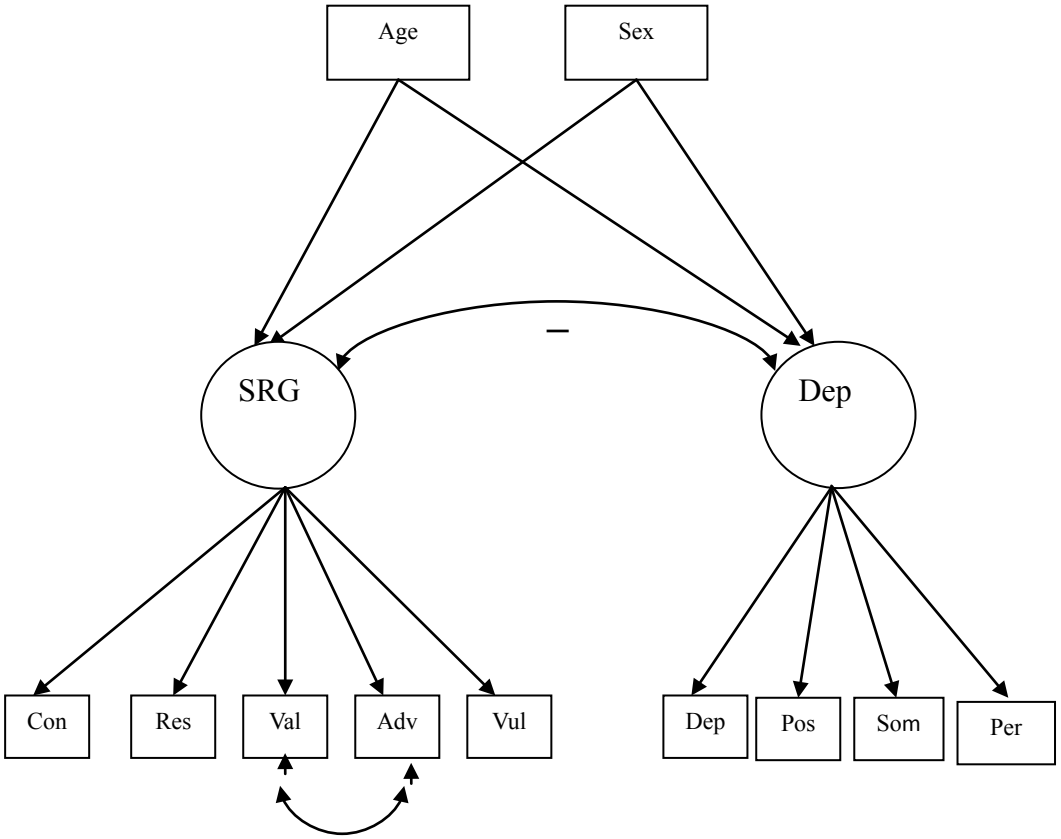
Zautra: When stress severity is high, SRG and depressive symptoms will be negatively or unrelated over time.

Figure 2.2. Theoretical Model for Cross-Sectional Relations between SRG and Depressive Symptoms in Low Stress Severity Group



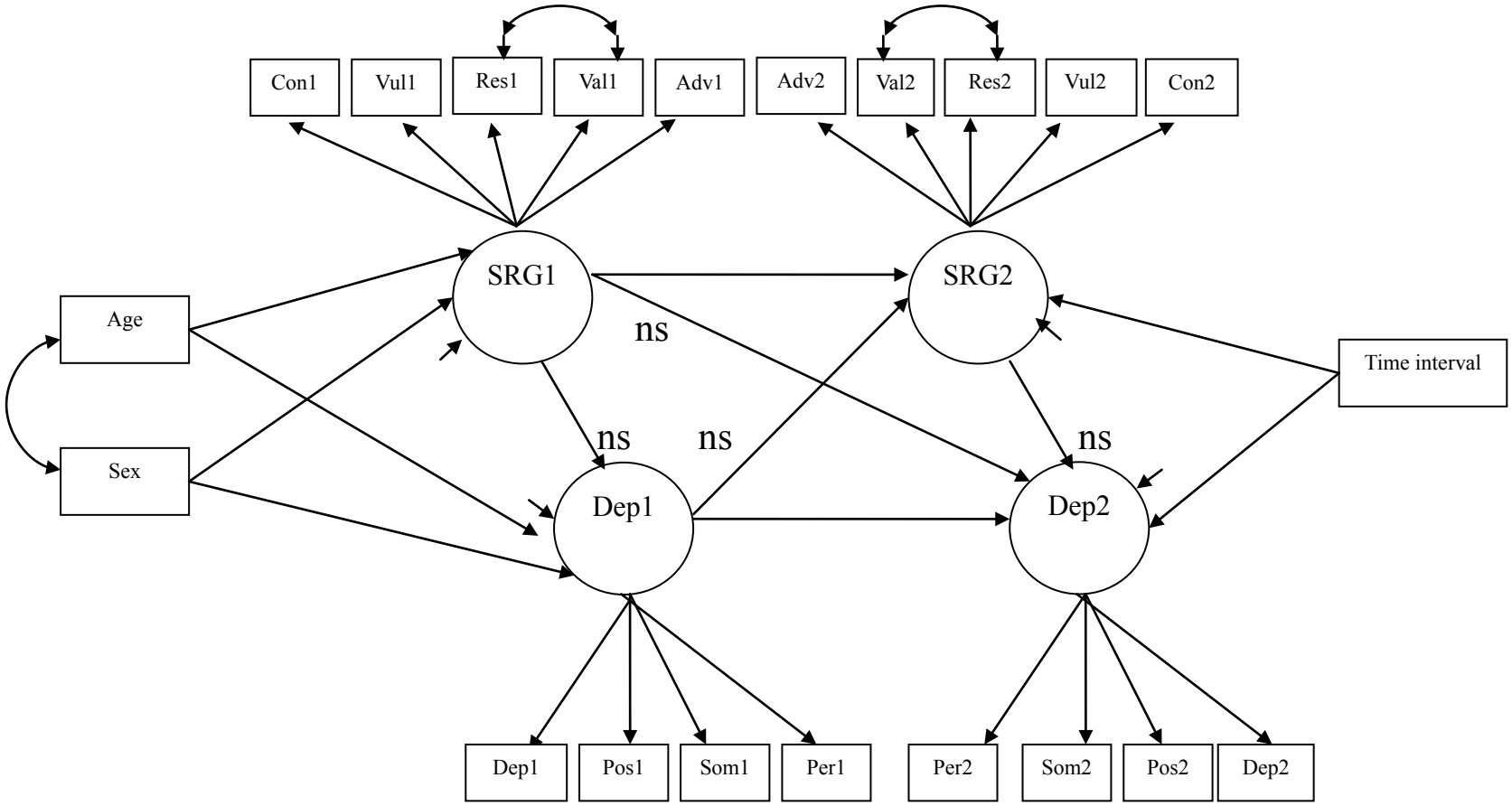
Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities; Dep = Depressed Affect; Pos = Positive Affect; Som = Somatic Complaints; Per = Interpersonal Problems.

Figure 2.3. Theoretical Model for Cross-Sectional Relations between SRG and Depressive Symptoms in High Stress Severity Group



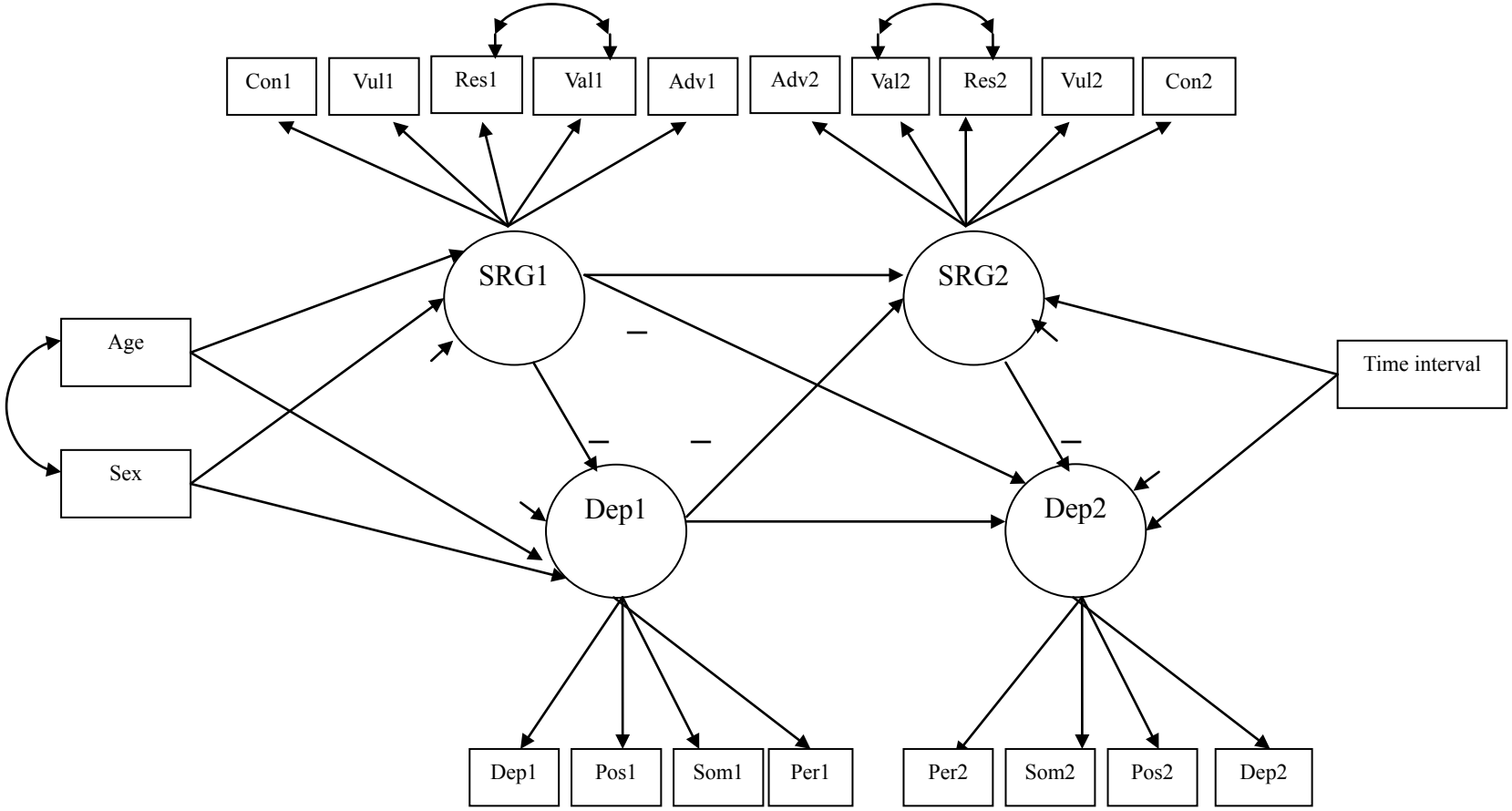
Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities; Dep = Depressed Affect; Pos = Positive Affect; Som = Somatic Complaints; Per = Interpersonal Problems.

Figure 2.4. Theoretical Model for Longitudinal Relations between SRG and Depressive Symptoms in Low Stress Severity Group



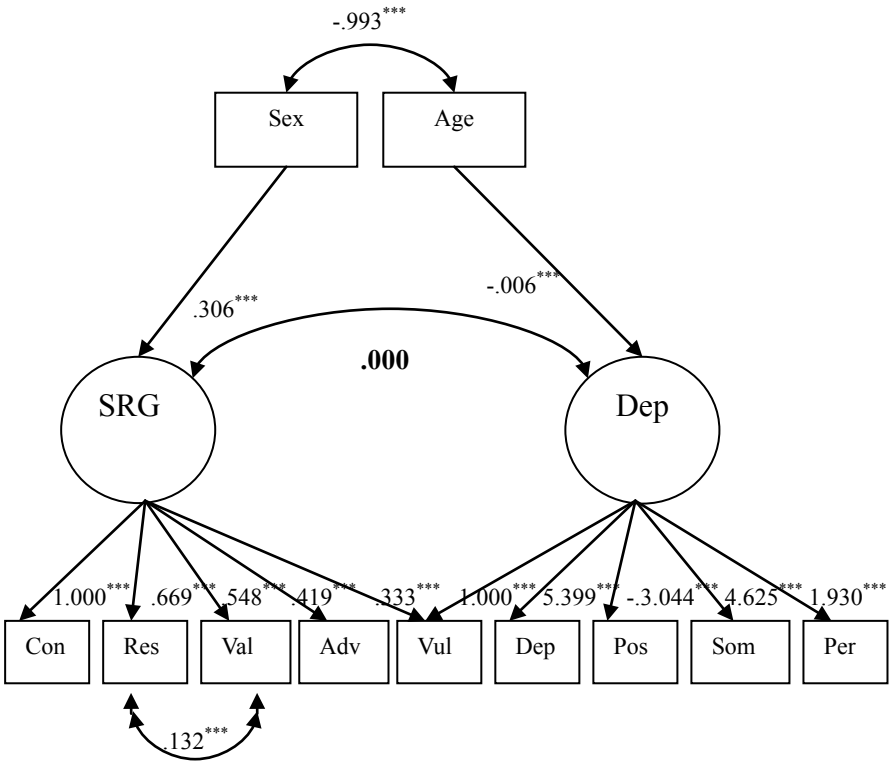
Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities; Dep = Depressed Affect; Pos = Positive Affect; Som = Somatic Complaints; Per = Interpersonal Problems.

Figure 2.5. Theoretical Model for Longitudinal Relations between SRG and Depressive Symptoms in High Stress Severity Group



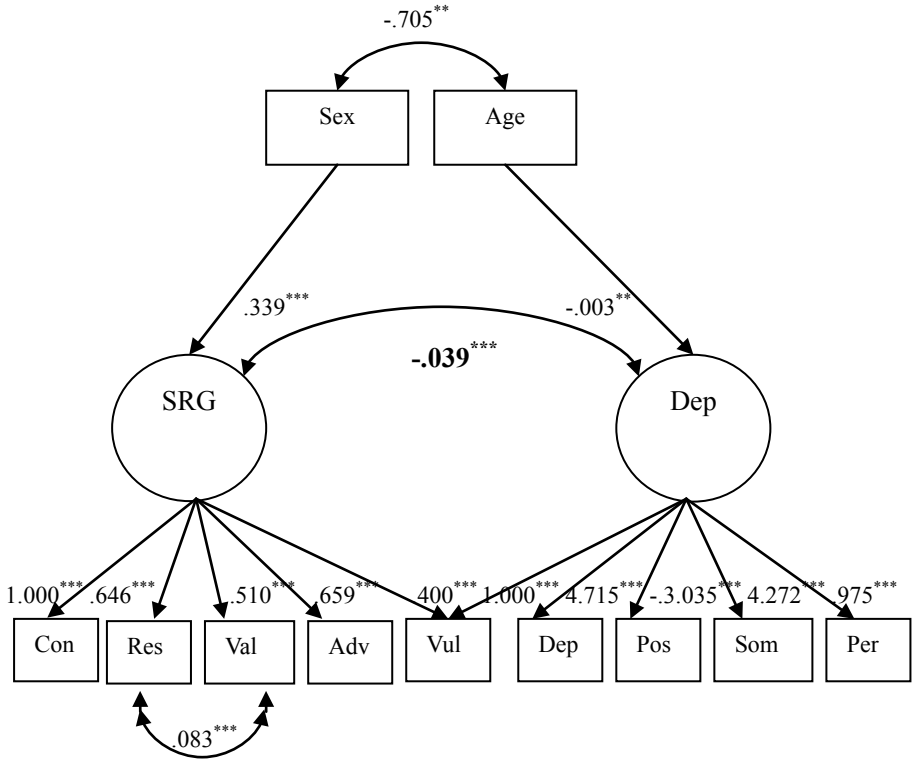
Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities; Dep = Depressed Affect; Pos = Positive Affect; Som = Somatic Complaints; Per = Interpersonal Problems.

Figure 2.6. Cross-Sectional Relations between SRG and Depressive Symptoms in Low Stress Severity Group



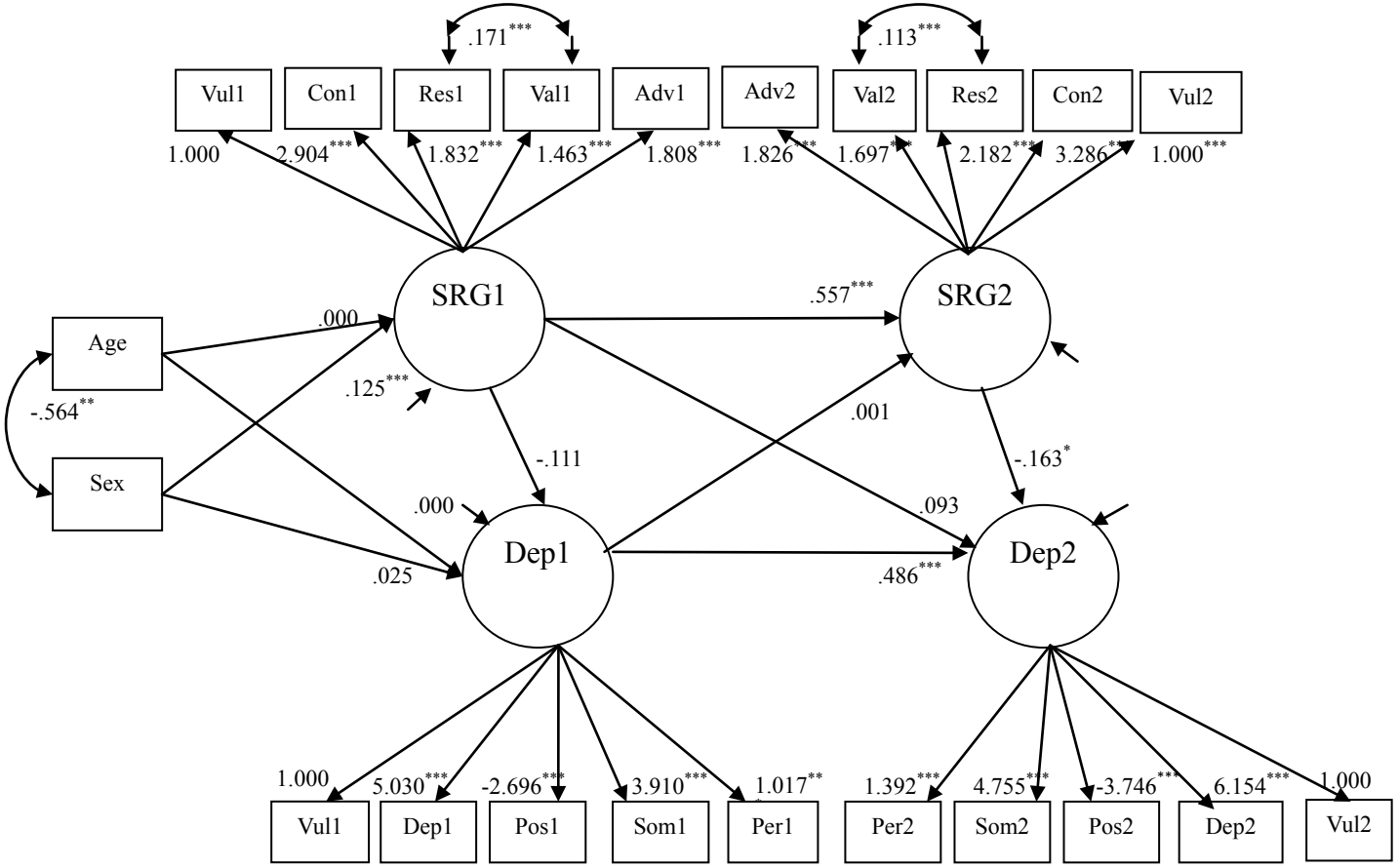
Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities; Dep = Depressed Affect; Pos = Positive Affect; Som = Somatic Complaints; Per = Interpersonal Problems.

Figure 2. 7. Cross-Sectional Relations between SRG and Depressive Symptoms in High Stress Severity Group



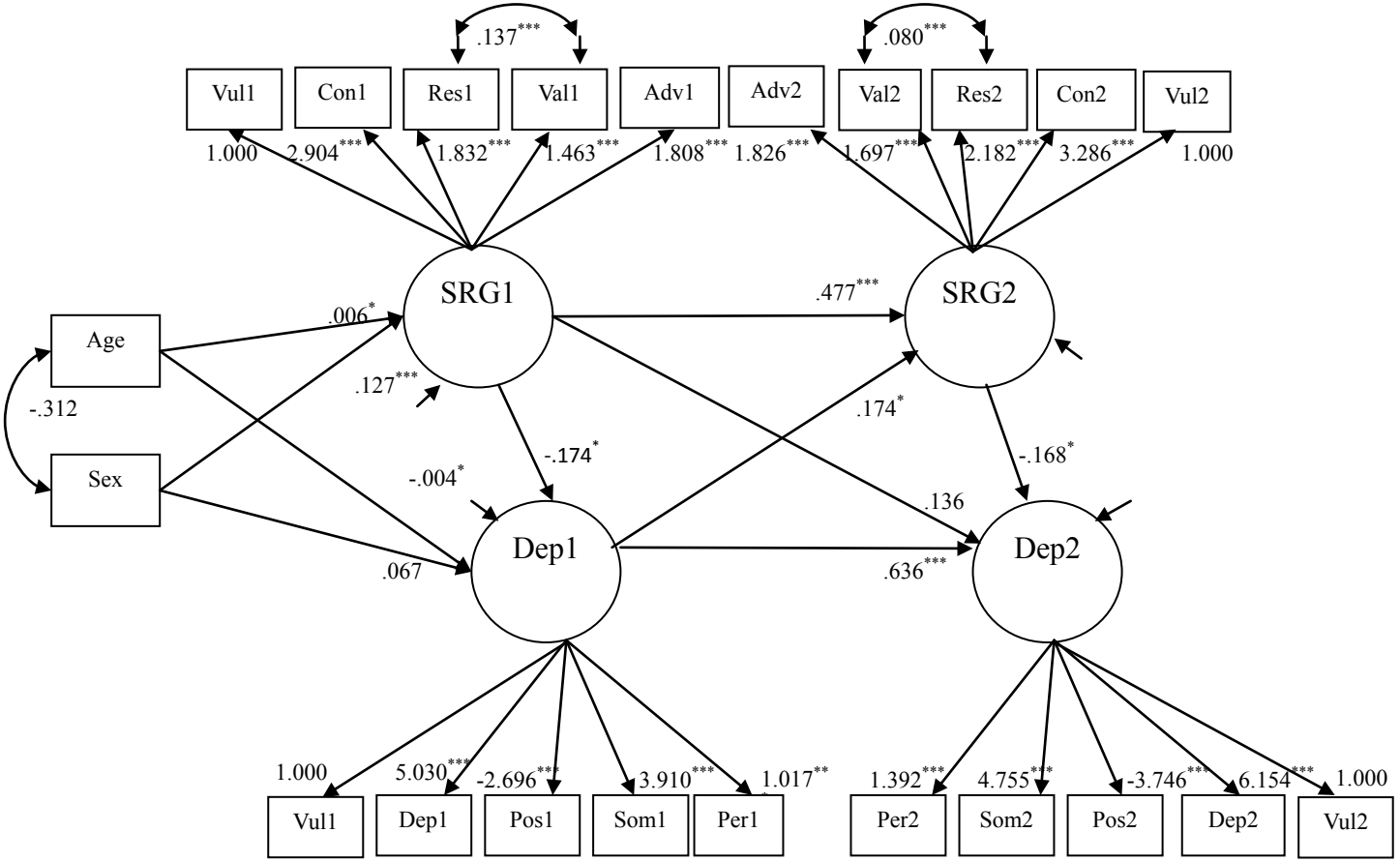
Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities; Dep = Depressed Affect; Pos = Positive Affect; Som = Somatic Complaints; Per = Interpersonal Problems.

Figure 2.8. Longitudinal Relations between SRG and Depressive Symptoms in Low Stress Severity Group



Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities; Dep = Depressed Affect; Pos = Positive Affect; Som = Somatic Complaints; Per = Interpersonal Problems.

Figure 2. 9. Longitudinal Relations between SRG and Depressive Symptoms in High Stress Severity Group



Note. Con = Confidence; Res = Resources; Val = Values; Adv = Advantages; Vul = Vulnerabilities; Dep = Depressed Affect; Pos = Positive Affect; Som = Somatic Complaints; Per = Interpersonal Problems.

GENERAL CONCLUSION

Studying SRG is especially important for the existing stress literature. Given the high prevalence of stress among individual's lives, numerous studies have documented the effects of stress on physical and mental health. However, most of these studies primarily focus on the negative aspects of stress and ignore potential positive aspects. Therefore, the positive effects of stress such as SRG show us another side of stress and depict a more complete picture about stress. In addition, studies have showed that SRG has significant implications for physical and psychological health, with higher levels of SRG relating to lower levels of morbidity (Affleck et al., 1987; Moskowitz et al., 2008), fewer negative changes in biological markers (Bower et al., 1998; Milam, 2006), better well-being (Helgeson et al., 2006), and fewer depressive symptoms (Carver & Antoni, 2004). Given the significance of SRG, a growing number of studies have concentrated on potential factors that influence SRG as well as the association of SRG with other outcomes of stress. Studies are especially interested in how stress severity, coping strategies, and depressive symptoms are related to SRG. However, most of these studies are cross-sectional, and few use longitudinal designs to test their relations, especially the reciprocal relations over time.

Based on GRRs (Antonovsky, 1979, 1987), COR (Hobfoll, 1988), and DAM (Aldwin et al., 1996), the primary purpose of this dissertation was to examine longitudinal relations between SRG, stress severity, coping strategies, and depressive symptoms over two waves across five years. This dissertation is composed of two studies. The first study examined the reciprocal effects between stress severity and SRG, and the longitudinal mediating effects of coping on stress severity and SRG. To be more specific, it tested the effect of previous stress severity on subsequent SRG, as well as the effect of previous SRG on the stressfulness of subsequent episodes. Further, it examined how their potential reciprocal relations between stress severity and SRG were mediated by both PAC and NAC. The second study

concentrated on both within-time and cross-lagged effects between SRG and depressive symptoms, especially how their relations were stratified by stress severity.

Summary Results for Study 1

The first study did not find reciprocal cross-lagged relations between stress severity and SRG. Specifically, there were weakly positive within-time effects from stress severity to SRG, whereas no significant cross-lagged effects between stress severity and SRG were found.

However, when the coping strategies were taken into account, the relations between stress severity and SRG were changed. Stress severity no longer had direct positive effects on SRG within time; instead it had significantly indirect positive effects, mediated by both PAC and NAC. That is, the higher level of stress severity led to the use of more PAC and NAC, which resulted in higher levels of SRG within time.

The direct cross-lagged effects between stress severity and SRG were not significant, whereas the cross-lagged effect from stress severity at Time 1 to SRG at Time 2 was significant, partially mediated by PAC instead of NAC itself. The direct effect from PAC at Time 1 to SRG at Time 2 was negative, which yielded the indirect effect via PAC itself was negative, but the total indirect effects via other paths were positive. In addition, SRG at Time 1 had significant predictive effects on subsequent PAC rather than NAC.

Taken together, these results indicated that positive relations between stress severity (Hobfoll, 1988) and SRG (Antonovsky, 1979) were supported, but neither Antonovsky's positive nor Hobfoll's (1988) negative cross-lagged effects were supported. In general, the arguments made by Aldwin et al. (1996) about contingent relations between stress severity and SRG by the mediator of coping strategies were mostly supported. Namely, if individuals used PAC to deal with their stressors, stress severity was much more likely to result in higher levels of SRG, which in turn led to more PAC subsequently. However, slightly different from

Aldwin et al.'s proposition, if individuals used more NAC to deal with their stressors, stress severity led to higher instead of lower levels of SRG within time. But, in general, the results of Study 1 supported Aldwin et al.'s contingent model of DAM.

Summary Results for Study 2

The second study primarily examined the stratified or moderated effect of stress severity on the relations between SRG and depressive symptoms. Besides the three theories of resource accumulation of GRRs (Antonovsky, 1979), resource depletion of COR (Hobfoll, 1988), and contingent model of DAM (Aldwin et al., 1996), the second study added two additional theories, which were Tedeschi and Calhoun's functional-descriptive model and Zautra's (2003) DMA. Specifically, we hypothesized that in the low stress severity group, there were no significant relations between SRG and depressive symptoms, while in the high stress severity group, there were negative relations between them.

Our hypotheses were first examined in cross-sectional sample. The result showed that there were significant differences in the relations between SRG and depressive symptoms in the low and high stress severity groups, with orthogonal (i.e., no relations) and unidimensional (i.e., negative) relations found, respectively. This finding provided strong support for Aldwin et al.'s (1996) DAM, Hobfoll's (1988; Hobfoll & Lilly, 1993) COR, and Zautra's (2003) DMA about the stratified or moderated effect of stress severity.

The stratified effect of stress severity, however, was not strongly supported by longitudinal sample. Specifically, the result showed that in the low stress severity group, the within-time effect from SRG to depressive symptoms was not significant, at least at Time 1, and the cross-lagged effects between SRG and depressive symptoms were not significant. In the high stress severity group, the within-time effect from SRG to depressive symptoms at both Time 1 and Time 2 were significantly negative, whereas the cross-lagged effect was significantly positive, at least from depressive symptoms at Time 1 to SRG at Time 2.

These results indicated that the relations between SRG and depressive symptoms were more complicated. Aldwin et al., Hobfoll, and Zautra's stratified effect of stress severity seemed to be supported by the non-significant within-time relations in the low stress severity group and significantly negative within-time relations in the high stress severity group. However, their arguments were not supported by the significantly positive rather than negative cross-lagged effect from depressive symptoms at Time 1 to SRG at Time 2 in the high stress severity group. Instead, the positive cross-lagged relation provided support to Tedeschi and Calhouns' (2004) motivating effect of depressive symptoms on SRG. In addition, the stratified effect of stress severity was not supported by the non-significant cross-lagged effect from SRG at Time 1 to depressive symptoms at Time 2 at both groups, which instead supported Zautra's argument of an orthogonal relation between SRG on depressive symptoms either in the low or high stress severity groups.

However, further analyses showed that the stratified relations between SRG and depressive symptoms across stress severity groups were not statistically significant. Therefore, our hypotheses were not fully supported by the longitudinal sample.

Connections between the Two Studies

Based on the theories of Antonovsky (1979), Hobfoll (1988), and Aldwin et al. (1996) (even though two additional theories of Tedeschi & Calhoun [2004] and Zaurta [2003] were examined in study 2), the two studies of this dissertation used two-wave longitudinal data to test two major questions in SRG literature. The first question examined the relations between stress severity, coping strategies, and SRG. After identifying their relations, another intriguing question that arose was the relations between SRG and people's adjustment such as psychological health. This led to the second question examined in this dissertation, regarding the relations between two common stress outcomes – SRG and depressive symptoms.

Unlike previous studies, this dissertation looked at not only the effect of stress severity and coping strategies on SRG cross-sectionally, but also the reciprocal relations longitudinally. In the same vein, this dissertation examined the reciprocal relations instead of unidirectional relations between SRG and depressive symptoms. Furthermore, it investigated how their reciprocal relations were stratified by stress severity. Generally, this dissertation supported Aldwin et al.'s (1996) DAM, although one part of the hypotheses in Study 2 was not fully supported, which might be because of limited power (i.e., sample size).

Limitations

Although the present studies made great contributions to the longitudinal relations between stress severity, coping, SRG, and depressive symptoms, there were some limitations. The first limitation is the homogenous sociodemographic characteristics of the sample. Specifically, both studies in this dissertation were performed using primarily young and middle-aged (from 30- to 50-year-old) European Americans, with at least a bachelor's degree and high levels of average family income. Given their characteristics, the results of this dissertation may not be applicable to other ethnic groups, or even within the same ethic group with much younger or older ages, less education, or lower levels of family income because SRG and depressive symptoms are not consistent across different sociodemographic characteristics (for reviews, see Lechner & Weaver, 2009; Lorant et al., 2003).

A second limitation is that this dissertation does not examine whether other possible confounding factors of personality account for the autocorrelations of stress severity, coping, SRG, and depressive symptoms. In both Study 1 and Study 2, the exploratory analyses only tested a factor of personality – neuroticism, and showed that the autocorrelations were not accounted for by neuroticism. Future studies need to further examine other possible personality confounders such as extraversion and conscientiousness.

The third limitation of this dissertation is that retrospective methods are used to assess SRG and depressive symptoms. It has been argued that retrospective report may not be a reliable method to capture respondents' SRG and depressive symptoms because it is more likely to produce response bias and distort (i.e., overestimate or underestimate) respondents' perceptions of their previous adverse events, and thus to report higher or lower levels of SRG and depressive symptoms than they should. Given this limitation, if it is possible, future studies should use prospective methods to examine SRG and depressive symptoms as well as their relations with stress severity and coping strategies.

The fourth limitation is that this dissertation mainly examined respondents' major life stressors instead of traumatic events (e.g., hurricane, sexual abuse, or other traumas). Thus, it is unknown the relations between stress severity, coping strategies, SRG, and depressive symptoms in a traumatic stress group. Given high prevalence and harm of trauma, future studies need to examine the relations in the traumatic stress group to determine whether the relations are different from the stress severity groups examined in this dissertation.

Clinical Implications

The findings from this dissertation have several implications for clinical programs. First, Study 1 results indicate that positive coping strategies are more useful to facilitate respondents' SRG levels. That is, when individuals experience adverse life events, it will be more effective if intervention programs specifically focus on improving positive coping strategies rather than negative coping strategies to promote SRG levels. Also, the increased level of SRG in turn will benefit subsequent positive coping strategies.

Second, SRG does not necessarily lead to decreased depressive symptoms. As clearly showed in the cross-sectional study of Study 2, SRG is related to a reduction of depressive symptoms only in the high stress severity group but not in the low stress severity group. Therefore, future clinical work should be sensitive to the relations between SRG and

depressive symptoms and be aware of the fact that utilizing people's SRG level to decrease their depressive symptoms may only work at certain stress severity levels.

Third, Study 2 also suggests that suffering depressive symptoms is not always meaningless but may be meaningful and worthwhile because previous depressive symptoms may promote subsequent experiences of SRG. On the basis of this finding, clinicians may consider guiding and helping individuals who suffer depressive symptoms do so meaningfully in order to motivate the development of SRG rather than decreasing depressive symptoms immediately.

Taken together, the results of this dissertation have significant implications for clinicians in improving individual's SRG levels and regarding the relations between stress severity, coping, SRG, and depressive symptoms. However, since this dissertation did not examine the implications for effectiveness of interventions, future research should apply the results of this dissertation to design an intervention model to enhance people's positive adaptation and to decrease their negative adjustment.

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APPENDIX

Questionnaire

Recent Low Points

Most people also have low points in their lives. Please circle the events which have been major low points for you and when they occurred.

0 = within the past year	**1 = within the past 5 years**	**2 = more than 5 years ago**
1. Marital/relationship problems	0	1	2
2. Problems with children	0	1	2
3. Career/work problems	0	1	2
4. Education/graduation	0	1	2
5. Physical health problems (self)	0	1	2
6. Mental health problems (self)	0	1	2
7. Social relations	0	1	2
8. Loneliness/isolation	0	1	2
9. Spiritual crisis	0	1	2
10. Lack of meaning/purpose in life	0	1	2
11. Parent's health	0	1	2
12. Spouse's health	0	1	2
13. Child's health	0	1	2
14. Military service/war	0	1	2
15. Natural disasters (e.g. earthquake)	0	1	2
16. Spouse's death	0	1	2
17. Finances	0	1	2
18. Separation/divorce	0	1	2
19. Spouse's problem(s)	0	1	2
20. Political/macrosocial events	0	1	2
21. Parent's death	0	1	2

0 = within the past year	**1 = within the past 5 years**	**2 = more than 5 years ago**
22. Death of a pet	0	1	2
23. Other bereavement	0	1	2
24. Separation from loved ones	0	1	2
25. Childlessness	0	1	2
26. Victim of a crime	0	1	2
27. Child's achievement problems	0	1	2
28. Child's marital problems	0	1	2
29. Grandchildren	0	1	2
30. Retirement	0	1	2
31. Caretaking for older relative	0	1	2
32. Other (explain) _____	0	1	2

Think of the most recent low point in your life. Write the number from the previous list here.

_____

In what year(s) did this low point occur? _____

Stress Severity

How stressful was this low point for you? By 'stressful"we mean how troubling or disturbing it was to you. Please circle the number which best describes this, where 1 = *not at all stressful*, and 7 = *most stressful thing ever experienced*.

1	2	3	4	5	6	7
Not at all stressful						Most stressful

The California Coping Inventory (CCI)

The following are strategies that people sometimes use to manage their problems and/or their negative emotions. For this particular low point (which you identified in question 16) please indicate to what extent you used each of these strategies. If these are not applicable to this problem, simply indicate that you did not use that strategy ('not at all').

0 = not at all	**1 = used a little**	**2 = used somewhat**	**3 = used a lot**

1. Accepted that this was a problem I had to deal with.	0	1	2	3
2. Asserted control over the situation.	0	1	2	3
3. Blamed others.	0	1	2	3
4. Took things one step at a time.	0	1	2	3
5. Complained to other people.	0	1	2	3
6. Decided nothing could be done.	0	1	2	3
7. Deferred action on problem until I got more information.	0	1	2	3
8. Tried to placate the other person(s).	0	1	2	3
9. Developed new skills or understanding.	0	1	2	3
10. Did something concrete to help others.	0	1	2	3
11. Directly confronted the person(s).	0	1	2	3
12. Decided it wasn't really my problem.	0	1	2	3
13. Distracted myself using TV, books, hobbies, work, etc.	0	1	2	3
14. Exercised to control stress.	0	1	2	3
15. Refused to worry about it too much.	0	1	2	3
16. Expressed hostility to the other person(s).	0	1	2	3
17. Focused on managing the problem.	0	1	2	3
18. Had a little something to calm myself down.	0	1	2	3
19. Prayed for guidance.	0	1	2	3

0 = not at all	**1 = used a little**	**2 = used somewhat**	**3 = used a lot**
20. Imagined ways of retaliating.	0	1	2	3
21. Isolated myself from others.	0	1	2	3
22. Knew that I would come up with something.	0	1	2	3
23. Lied to or withheld information from someone.	0	1	2	3
24. Looked for alternative solutions.	0	1	2	3
25. Trusted that others would do the right thing.	0	1	2	3
26. Kept my feelings to myself.	0	1	2	3
27. Prayed or meditated on the problem.	0	1	2	3
28. Tried to pretend that it just hadn't happened.	0	1	2	3
29. Provided emotional support to others.	0	1	2	3
30. Put my feelings on the "back burner".	0	1	2	3
31. Tried to make the other person(s) feel guilty.	0	1	2	3
32. Realized that it could have been worse.	0	1	2	3
33. Used drugs, alcohol or food to escape from situation.	0	1	2	3
34. Restrained action or suppressed my initial impulse.	0	1	2	3
35. Stewed about it.	0	1	2	3
36. Strengthened my ties to others.	0	1	2	3
37. Told myself to calm down.	0	1	2	3
38. Trusted in the Lord or a higher power.	0	1	2	3
39. Took time outs when I needed them.	0	1	2	3
40. Tried to forget about the problem.	0	1	2	3
41. Tried to get perspective on the problem.	0	1	2	3
42. Threw or punched things.	0	1	2	3
43. Tried to get the other person(s) to see my point of view.	0	1	2	3

0 = not at all	**1 = used a little**	**2 = used somewhat**	**3 = used a lot**
44. Trusted my instincts or intuitions about the problem.	0	1	2	3
45. Used prescription drugs to reduce anxiety or depression.	0	1	2	3
46. Was persistent or tried harder.	0	1	2	3
47. Was careful not to overextend myself.	0	1	2	3
48. Wished that the situation would just go away.	0	1	2	3
49. Yelled or cursed.	0	1	2	3
50. Other (explain)_____	0	1	2	3

Advantages

Were you able to turn any part of the situation to your advantage? (Circle all that apply.)

0. No
1. Yes, emotional well-being (e.g., pride, satisfaction)
2. Yes, tangible advantage/gain (including new coping skills)
3. Yes, developed new philosophy/attitude towards life
4. Yes, other (please explain) _____

Learn from the Low Point

The following statements reflect some of the things people learn from going through a low point. Please indicate the extent to which you have learned the following things.

0=Not at all	**1=a little**	**2=somewhat**	**3=a lot**
1. Family is very important to me.	0	1	2	3
2. Religion/spirituality is very important to me.	0	1	2	3
3. Taking care of myself is very important to me.	0	1	2	3
4. I had positive psychological resources (e.g., ability to cope).	0	1	2	3
5. I had positive physical resources (e.g., physical stamina).	0	1	2	3
6. I had positive social resources (e.g., good friends, neighbors, family).	0	1	2	3
7. There were some things about myself with which I was not happy.	0	1	2	3
8. I could stand on my own two feet.	0	1	2	3
9. My health prevented me from doing as much as I would have liked	0	1	2	3
10. Many people weren't as helpful as I would have liked.	0	1	2	3
11. New skills (e.g., I learned how to manage doctors.)	0	1	2	3
12. New, positive attitudes towards life.	0	1	2	3
13. There are some situations that I can't do anything about.	0	1	2	3
14. My longstanding values were a resource I could draw upon.	0	1	2	3
15. Other (please specify) _____	0	1	2	3

Center for Epidemiologic Studies Depression Scale (CESD)

In the past week, how often did you experience the following feelings?

0 = hardly ever or never	**1 = some of the time**	**2 = much or most of the time**
1. I did not feel like eating; my appetite was poor.	0	1	2
2. I felt depressed.	0	1	2
3. I felt everything I did was an effort.	0	1	2
4. My sleep was restless.	0	1	2
5. I was happy.	0	1	2
6. I felt lonely.	0	1	2
7. People were unfriendly.	0	1	2
8. I enjoyed life.	0	1	2
9. I felt sad.	0	1	2
10. I felt that people disliked me.	0	1	2
11. I could not "get going."	0	1	2

EPI-Q

Decide whether "yes" or "no" best corresponds to your character, feelings and actions. Do not spend too much time on each question. We are trying to determine your immediate reactions.

	No	Yes
1. Have you often got a restless feeling that you want something but do not know what?	0	1
2. Do you sometimes feel happy, sometimes sad, without any real reason?	0	1
3. Do you often make up your mind too late?	0	1
4. Have you often felt listless and tired for no good reason?	0	1
5. Are you often "lost in thought"?	0	1
6. Are you touchy about some things?	0	1
7. Do you sometimes get so restless that you cannot sit long in a chair?	0	1
8. Do you suffer from "nerves"?	0	1
9. Do you worry too long after an embarrassing experience?	0	1