Relation between Family Strain and Depressive Symptoms in Middle-aged Adults: The Moderating Effect of Self-compassion

by

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ABSTRACT

Interpersonal strain is linked with depressive symptoms in middle-aged adults. Self-compassion is an emerging resilience construct that may be advantageous in navigating relationship strain by helping individuals respond to emotions in a kind and nonjudgmental way. Although theory and empirical evidence suggests that self-compassion is protective against the impact of stress on mental health outcomes, many studies have not investigated how self-compassion operates in the context of relationship strain. In addition, few studies have examined psychological or physiological mechanisms by which self-compassion protects against mental health outcomes, depression in particular. Thus, this study examined 1) the extent to which trait self-compassion buffers the relation between family strain and depressive symptoms, and 2) whether these buffering effects are mediated by hope and inflammatory processes (IL-6) in a sample of 762 middle-aged, community-dwelling adults. Results from structural equation models indicated that family strain was unrelated to depressive symptoms and the relation was not moderated by self-compassion. Hope, but not IL-6, mediated the relation between family strain and depressive symptoms and the indirect effect was not conditional on levels of self-compassion. Taken together, the findings suggest that family strain may lead individuals to experience less hope and subsequent increases in depressive symptoms, and further, that a self-compassionate attitude does not affect this relation. Implications for future self-compassion interventions are discussed.

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Depression is the leading cause of disability for adults in the U.S. (World Health Organization, 2008), affecting roughly 6.7% of individuals each year (Center for Behavioral Health Statistics and Quality, 2017). Direct and indirect costs of depression amount to around $210 billion annually (Greenberg, Fournier, Sisitsky, Pike, & Kessler, 2015). Among the factors that can elevate the risk of developing depressive symptoms are life events and circumstances that are stressful (McEwen, 2004). Although definitions of stress vary, a commonly used framework suggests that stress occurs when an individual encounters a situation or circumstance where the demands of the situation exceed an individual's resources needed to meet those demands (Butler, 1993). Among the most stressful circumstances individuals experience are ongoing interpersonal conflicts, which can lead to damaging mental health outcomes, including depression (Avison & Turner, 1988; Finch, Okun, Barrera, Zautra, & Reich, 1989; Whisman, 2007). It has been postulated that relationship conflict is the most common form of chronic stress/strain, and perhaps the most enduring one because of the significance individuals place on their social roles (Pearlin, 1989).

To develop a fuller understanding of the relation between interpersonal stress and depression, effort has been directed toward identifying mediators and moderators of the relation. With regard to moderators, some evidence suggests that the link between chronic interpersonal stress and depression varies considerably between individuals. Research has largely focused on risk factors that predispose individuals to depressive symptoms (Dobson & Dozois, 2008). What is less understood is how individual differences in one or more resilience factors may attenuate the detrimental effects of interpersonal stress on depressive symptoms (Zautra, Hall, & Murray, 2008). In

particular, those with strong social-emotional regulation skills may be most capable of navigating interpersonal strain, and one resilience concept that is associated with these skills is self-compassion (Neff, 2009). Self-compassion involves treating oneself kindly amidst the presence of challenges and suffering (Neff, 2003a), and thus is relevant for the suffering associated with social relations. There is also evidence to suggest that the relation between interpersonal stress and depression is mediated through both dysfunctional cognitive styles (Dozois & Beck, 2008) and physiological processes (McEwen, 1998).

How might self-compassion moderate the relationship strain – depression relation? Self-compassion may help individuals cope with relationship strain through its effects on cognitive styles and physiological stress responses (Allen & Leary, 2010; Breines et al., 2014). Self-compassion has been theorized to benefit relationships through enhanced connection and social support and is associated with positive relationship behavior, all of which have been shown to buffer the effects of relational stress on mental and physical health (Santini, Koyanagi, Tyrovolas, Mason, & Haro, 2015). Moreover, self-compassion has been found to mitigate the acute inflammatory effects of a social evaluation stressor (Dickerson & Kemeny, 2004). However, there is little empirical evidence of the protective effects of self-compassion against the health repercussions of chronic interpersonal stressors like relationship strain, and the mechanisms by which it influences mental health, depressive symptoms in particular.

Gaining knowledge regarding the role of self-compassion as a protective factor against relational strain may lend empirical support for self-compassion as an emerging resilience concept and inform the growing number of self-compassion interventions

(Gilbert & Proctor, 2006; Neff & Germer, 2013; Raes, 2010). This study examined 1) the extent to which trait self-compassion buffers the relation between interpersonal stress and depressive symptoms (See Figure 1), and 2) whether these buffering effects are mediated by psychological and inflammatory mechanisms in a sample of middle-aged, community-dwelling adults (See Figures 2 & 3).

Interpersonal Strain and Depressive Symptoms

Research literature suggests that social processes and interpersonal experiences play a major role in the occurrence and maintenance of depressive symptoms (Allen & Knight, 2005), with stressful interpersonal relations being among the most reliable predictors of depression (Kendler, Hettema, Butera, Gardner, & Prescott, 2003). Sustained interpersonal stressors, termed interpersonal strain, represent a chronic stressor that can impair psychological functioning (Krause & Rook, 2003; Schuster, Kessler, & Aseltine, 1990; Umberson & Montez, 2010). Interpersonal strain involves dissatisfaction or conflict with family, friends, spouses, and/or work colleagues (Walden & Lachman, 2000). Sources of relationship strain stem from disagreements and interpersonal confrontation within the dyad or group, as well as external stressors like financial events and work stress (Canary, Cupach, & Messman, 1995), which can lead to chronic negative repercussions for relationships, such as ongoing criticism, rejection, and antagonism (Krause & Rook, 2003). Cross-sectional, population-based studies of healthy adults consistently find that conflicts with social group members predict psychological distress (Fiori, Antonucci, & Cortina, 2006; Li & Liang, 2007; Marum, Clench-Aas, Nes, & Raanaas, 2014; Santini et al., 2015; Schuster et al., 1990). Likewise, research suggests that relationship strain has a stable and cumulative effect on mental health symptoms

(Umberson & Montez, 2010), with longitudinal data indicating that strain is positively correlated with depression levels four years later among middle-aged adults (Frone, Russell, & Cooper, 1997) and six years later among older adults (Krause & Rook, 2003).

Evidence is mixed as to whether strain stemming from particular kinds of social relationships, such as those with family, friends, spouses, or work colleagues differentially predict distress (Walden & Lachman, 2000). Family strain may be especially prevalent, given that the majority of individuals identify with a family group in some capacity. Researchers have presumed that family strain might be uniquely challenging because family members are not chosen, whereas friends and spouses are independently selected, thus characterizing family strain as more enduring and difficult to eliminate (Krause & Rook, 2003; Yang, Schorpp, & Mullan Harris, 2014).

These findings suggest that interpersonal strain, such as conflict with family, has short and long-term negative influences on depressive symptoms in adults. Unsurprisingly, conflict is an inevitable part of interpersonal relationships, especially as interdependence increases (Braiker & Kelly, 1979), but while some individuals experience depressive symptoms as a result of relationship strain, others do not. Therefore, individual resilience factors may buffer the negative effects of interpersonal stress on depressive symptoms, and the resilience factors that are most compelling theoretically are those that help an individual dampen emotional reactivity in the face of interpersonal strain (Seppala, Rossomando, & Doty, 2013). Thus, individuals who are able to navigate socio-emotional interactions and perceive them as less stressful may be the most adept at coping with interpersonal strain. Self-compassion is an emerging

resilience concept that is associated with reduced self-critical processes and stress reactivity.

Self-Compassion as a Resilience Factor

Self-compassion, as defined by Neff (2003a), relates to the larger construct of compassion. Compassion is described as having a deep connection and awareness of the suffering of others. A compassionate individual moves toward the pain of others, reflecting an intention to reduce suffering, rather than avoid it. Compassion also involves acceptance of others mistakes and wrongdoings as elements of the shared human experience. Self-compassion directs these same feelings toward the self. Theory suggests that those who are compassionate to others should also possess self-compassion (Neff, 2003a), but research findings show that compassion and self-compassion do not completely overlap, and in some cases, are only weakly related (Gilbert, McEwan, Matos, & Rivis, 2011). People are much more compassionate to others than towards themselves, especially in Western society and those in helping professions (Egan, Mantzios, & Jackson, 2017; Neff, Pisitsungkagarn, & Hsieh, 2008).

Self-compassion involves three related components: *self-kindness*, *common humanity*, and *mindfulness*. Through *self-kindness*, people respond with gentleness, rather than criticism, guilt, or judgment when faced with difficult situations and mental states (Neff, 2003a). *Common humanity* refers to viewing all experiences, whether positive or negative, as part of the larger human experience, helping to remove feelings of isolation and fostering connection with others. *Mindfulness* involves viewing thoughts and feelings as they are without over-identifying with them.

When approaching difficult circumstances with self-compassion, individuals are thought to experience each of these three components (Neff, 2003a). For example, approaching challenges from a mindful, nonjudgmental stance can allow space for individuals to respond with self-kindness as opposed to automatically responding with criticism. Individuals who are naturally inclined to be self-compassionate are able to judge themselves less harshly and have more accurate self-evaluations compared to individuals who do not have a self-compassionate disposition (Leary, Tate, Adams, Allen, & Hancock, 2007). In addition, highly self-compassionate individuals seem to evaluate their efforts less on the outcome of situations, in theory because they are able to maintain positive self-views regardless of poor results (Neff, Hsieh, & Dejitterat, 2005). Self-compassion has also been described as something that can be trained (Barnard & Curry, 2011). A study in which young adults received a self-compassion induction found that self-compassion interventions can increase attitudes of self-kindness among those who have low dispositional self-compassion, but little improvement in those who are already self-compassionate (Leary et al., 2007).

Having compassion for oneself allows for the recognition of the shared human experience, experiences that include joy and suffering. This recognition of an interconnected self helps individuals feel connection and compassion for others (Seppala et al., 2013). Despite self-compassion being a foundational principle of compassion in relationships (Salzberg, 2017), little research has been devoted to understanding the role that self-compassion plays in relationship strain. Correlational studies find that higher trait self-compassion is associated with more positive relationship behavior (i.e., care, control, autonomy, relatedness, less verbal aggression) in couples (Neff & Beretvas,

2013), relationship satisfaction for women and men high in conscientiousness (Baker & McNulty, 2011), and closeness and social support among roommates (Crocker & Canevello, 2008). The impact of self-compassion on the relationship strain – depression link has yet to be examined.

Self-Compassion and the Family Strain – Depression Relation

Self-compassion enhances connection with others through shared joy and suffering (Neff, 2003b). Theoretically, self-compassion proposes that when stressors arise, people have a tendency to consider their own experiences as unique, which can lead to feelings of loneliness (Neff, 2003a). A study of adolescents and young adults found that those who perceived their experiences as unique and not in congruence with others were more likely to experience depressive symptoms and were also less self-compassionate (Neff & McGehee, 2010). A self-compassionate attitude combats these feelings through recognition that events, thoughts, and emotions are both inevitable for everyone and also in constant flux (Neff, 2003a). A self-compassionate response towards family strain may entail a recognition that family conflicts are inevitable not only in one's own life, but also in the lives of all humans, allowing for an awareness of a connected, rather than separate self (Neff, 2008). Family strain may evoke shame about one's involvement in the cause of the conflict, but self-kindness and common humanity may help to attenuate feelings of self-criticism over family strain. Directing kindness towards oneself for making mistakes and acknowledging that everyone has conflicts can further ease judgment and self-blame (Neff, 2003a). Utilizing internal coping resources, like self-compassion, to soothe oneself when external social resources are conflictual would be valuable in the context of family strain.

Self-compassion has been examined as both a stable trait and a momentary feeling in evaluation of its benefits for mental health. Trait self-compassion is negatively associated with depressive symptoms (Körner et al., 2015; Muris & Petrocchi, 2017; Neff, Kirkpatrick, & Rude, 2007), self-criticism, rumination (Neff, et al., 2007), and anxiety (Neff, Hsieh, & Dejitterat, 2005; Svendsen et al., 2016), and positively associated with emotional flexibility (Svendsen et al., 2016) in cross-sectional studies.

Experimental findings suggest that trait and momentary self-compassion can modulate the effects of relational strain on psychological outcomes. In one of the few studies in this area, young adults were first assessed for trait self-compassion, and then were randomly assigned to either a state self-compassion induction, state self-esteem induction, a neutral induction, or no induction control group (Leary et al., 2007). Next, all participants were instructed to imagine distressing social situations. After inducing self-compassion, those low in trait self-compassion were buffered against negative self-feelings when imagining distressing social situations (Leary et al., 2007). The induction did not appear to affect individuals who were already high in trait self-compassion. In addition, the self-compassion induction was associated with the smallest increases in negative affect compared to individuals who received a self-esteem induction and the control group. Interestingly, young adults in the self-compassion induction group were also the most likely to attribute the cause of the negative social event to the kind of person they themselves were. In contrast to other groups however, there was almost no relation between attributing an event to one's character and negative affect. In this study, trait self-compassion did not predict severity or frequency of negative interactions reported, suggesting that those who are self-compassionate do not experience a lack of

negative interactions, but rather, may be more balanced when such situations arise. Therefore, individuals should not vary in the extent to which family strain is present in their lives based on differences in self-compassion. However, self-compassion may allow individuals to take responsibility for their role in negative social interactions without experiencing detrimental levels of negative affect. This acceptance may help self-compassionate individuals to remain resilient during interpersonal strain.

In another experimental study, young adults engaged in a dialogue wherein they were asked to voice a situation in which they were self-critical (Neff et al., 2007). The participant was then led in a Gestalt two-chair dialogue (guided by a counselor) by responding to their critical self with a voice that showed self-compassion. The dialogue proceeded until a resolution was complete. Three weeks later, participants self-reported increases in optimism, positive affect, and happiness compared to their self-ratings one week prior to the two-chair dialogue. These and other studies (Gilbert & Proctor, 2006; Neff & Germer, 2013) provide preliminary evidence that trait and momentary self-compassion can protect against self-criticism and negative psychological repercussions from strain (MacBeth & Gumley, 2012), but its effects on the relational strain – depression link require further investigation.

The literature on self-compassion as a resilience resource in the face of interpersonal strain is nascent. Correlational and intervention studies have found that trait and momentary self-compassion improve psychological symptoms and regulate emotions during stressful events, but the moderating effect of trait self-compassion on distress from relational strain remains to be clarified. Also limited in the literature is empirical evidence for mechanisms by which self-compassion impacts psychological symptoms,

like depression. One way to better understand the potential mechanisms by which self-compassion relates to the stress – depression link is to identify potential mediators. Efforts to understand the process whereby interpersonal strain contributes to depression have identified two key mediators: cognitive styles and physiological processes.

Cognitive Styles Linking Interpersonal Strain and Depression

The mechanisms by which interpersonal strain contributes to depression has been explained by several key domains of functioning, including perceived social support, cognitive styles, personality factors, behavioral factors, and physiological processes (Umberson & Montez, 2010). These mechanisms work individually and also in concert in the strain – depression relation (Umberson, Crosnoe, & Reczek, 2010). Of the mechanisms that have been explored, cognitive styles and physiological processes appear especially relevant in mediating the impact of stressful interpersonal experiences (Lakey, Tardiff, & Drew, 1994; Thoits, 2011; Yang et al., 2014). For example, cognitive styles are shown to explain the relation between negative social interactions and depressive symptoms more than perceived social support (Lakey et al., 1994). In addition, there is substantial evidence that chronic psychosocial stress is a major predictor of physiological dysregulation, specifically immune dysregulation (Yang et al., 2014).

Interpersonal strain and cognitive styles. Experiences of interpersonal strain can contribute to maladaptive cognitive styles. This may be due to the high value placed on inclusion and social cohesion (Thoits, 2011). For example, in constructing valued relationships with others, humans must consider not only how to engage with others, but also how others will engage with the self (Gilbert, 1992 as cited in Allen & Knight, 2005). This attention to the way others perceive the self increases self-focused thinking

and self-criticism (Allen & Knight, 2005). When conflict arises, the consideration of self and other can bring about concern with one's own success, acceptability, purpose, and significance as seen by the other person (Allen & Knight, 2005; Van Tongeren, Hill, Krause, Ironson, & Pargament, 2017). Relationships that foster insecurity and self-criticism can directly challenge a person's sense of self-worth and agency (Lakey et al., 1994), creating negative cognitions about the self (Krause & Rook, 2003; Neff & McGehee, 2010) and restricting social and internal resources of care (Allen & Knight, 2005). In turn, these negative attitudes can be detrimental to mental health. Studies find that some of the primary processes associated with depression include poor emotional regulation and decreased motivation (Pyszczynski & Greenberg, 1986; Tomarken & Keener, 1998).

Cognitive styles and depression. Cognitive styles associated with the regulation of emotional and motivational tendencies are key processes mediating the risk for depression (Tomarken & Keener, 1998). Specifically, individuals at low risk for depression attend to positive stimuli, downplay negative stimuli, and sustain motivation towards future goals despite experiencing stressors. In contrast, high-risk individuals tend to pay more attention to negative stimuli, ignore positive stimuli, and display decreased motivation towards valued goals (Allen & Badcock, 2003). There appears to be a neurobiological correlate for these findings with individuals at low risk for depression showing increased left prefrontal cortex activation and individuals at high risk for depression showing decreased left prefrontal cortex activation (Tomarken & Keener, 1998). These cognitive styles that influence attention to environmental stimuli and

motivation are complimentary to processes involved in social-cognitive theories of depression (Allen & Badcock, 2003).

Hope as a cognitive style mediating the strain-depression relation. Theories suggest that depression stemming from social rejection or threat is mediated by cognitive styles that fail to protect the individual. According to the social risk model of depression (Allen & Badcock, 2003), individuals experiencing social threats or limited social resources tend to restrict exposure to positive stimuli and seek out reassurance from others in an effort to conserve integrity and avoid future damaging social situations. For those at risk for depression, seeking reassurance from others does not always alleviate distress due to rumination of self-critical thoughts (Joiner & Metalsky, 2001). The perpetuation of shame and self-criticism results in individuals engaging in fewer social behaviors, limiting opportunities for positive affect reinforcement, and consequently, perpetuating depressive symptoms (Allen & Badcock, 2003). These social-cognitive models of depression are corroborated by findings showing that self-focused processes are devoid of perspective-taking, empathy, and prosocial action (Eisenberg et al., 1994). Taken together, individuals who are able to regulate emotion and maintain motivation may be better able to remain resilient following interpersonal strain, which may result in fewer depressive symptoms. From a conceptual standpoint, among potential mediating factors of interpersonal strain and depression, hope presents as a likely contributor. The relation between hope and depression has been examined extensively (Snyder, 2004).

Hope represents a cognitive style of perceiving that one has the capacity to reach goals through sustained motivation and plans (Snyder, 1994). According to Snyder (1994), hope is comprised of three related dimensions: goals, agency, and pathways. The

goals dimension refers to having self-relevant goals. According to hope theory, goals drive human behavior, and thus, having goals is essential to hopeful thinking (Snyder, 2002). Agency is the primary activator of hope in that it pushes individuals towards goals and values (Arnau, Rosen, Finch, Rhudy, & Fortunato, 2007). Agency represents a cognitive pattern that facilitates in the development of goals and the motivational endurance to meet goals. Pathways is described as believing in one's ability to remain flexible and develop a variety of ways to meet goals, even in the face of obstacles. Hope appears related to the cognitive processes involved in risk for depression, namely regulation of emotional and motivational states. Individuals who are hopeful are less likely to experience depressive symptoms (Chang, 2003; Chang & Banks, 2007; Chang & DeSimone, 2001), and more likely to have positive self-views (Umphrey & Sherblom, 2014). The hopelessness theory of depression identifies hope as a primary predictor of certain subtypes of depression (Abramson, Metalsky, & Alloy, 1989). Feelings of hopelessness are purported to be caused by negative life events, which enact feelings of hopelessness, and contribute to depressive symptoms. Relatedly, hopelessness is a common characteristic of individuals presenting with depression (American Psychiatric Association, 2013), suggesting that hope may be especially important in the etiology of depressive symptoms.

Studies show that hope's effect on depression may be related to differences in rumination. Among college students, low levels of hope and high rumination tendencies were associated with more depressive symptoms (Sun, Tan, Fan, & Tsui, 2014). The negative relations between hope and rumination make sense conceptually, given that hope emphasizes sustained commitment to goals, even if the route to the goals changes.

Those with less hope may be unable to find alternate ways to reach goals when faced with challenges due to excessive ruminative tendencies. This idea aligns with hopelessness theory, which suggests that those who are hopeless are likely to place importance on the outcome of the stressor, which can initiate rumination about the outcome and impact the ability to attend to other valued goals and actions (Abramson et al., 1989). Longitudinal findings suggest a unidirectional relation of hope with depression, such that hope predicts later depression (after 1 month), but that depression has no longitudinal effect on hope (Arnau et al., 2007). Although hope has traditionally been thought of as a trait, emerging interventions suggest that hope is a malleable construct, much like many cognitive processes that mediate depressive symptoms (Cheavens, Feldman, Gum, Michael, & Snyder, 2006). Hope is seen as a contextual experience, meaning that the availability of hope can fluctuate. The presence of hope in moments of stress is likely dependent upon cognitive processes (Folkman, 2010). Although hope has yet to be examined in relation with interpersonal strain, there is evidence of hope's negative relation to other stressors (Folkman, 2010; Korner, 1970; Yarcheski, Mahon, & Yarcheski, 2011) including caregiver stress (Granek et al., 2013), pain (Pulvers, & Hood, 2013), terminal illness (Sachs, Kolva, Pessin, Rosenfeld, & Breitbart, 2013), daily stress (Ong, Edwards, & Bergeman, 2006), and hostility (Kwon & Hugelshofer, 2010). Given this evidence, hope may represent a cognitive style that mediates the interpersonal strain and depressive symptoms relation due to its influence on emotional regulation and motivation.

A question that arises is whether there are dispositional factors that determine the effect of interpersonal strain on subsequent cognitive styles, like hope. A consistent

theme across theories of interpersonal strain and resulting cognitive styles is the primary need to seek validation and reassurance from others. Research finds that reassurance seeking may be a vulnerability factor for depression (Joiner & Metalsky, 2001). Since an aim of reassurance seeking is to measure self-worth (Coyne, 1976), it is plausible to hypothesize that differences in the way individuals value and accept themselves may determine whether individuals employ adaptive or maladaptive cognitive styles, like hope, following interpersonal strain. The theory of hopelessness depression suggests that inferences about the self (i.e., self-worth, personality, abilities) are likely to determine the development of hopelessness (Abramson et al., 1989). Self-compassion represents an attitude whereby the individual relates to the self in a kind, connected, and nonjudgmental way. Compassion for self and other is also related to activation of the left prefrontal cortex, the same region activated in individuals at low-risk for depression (Lutz, Greischar, Rawlings, Richard, & Davidson, 2004). Therefore, self-compassion may serve as a resilience resource against depression that engenders internal self-worth and hopefulness in the face of interpersonal strain.

Self-compassion as a moderator of the strain – hope relation. Those who are more self-compassionate may be protected from maladaptive cognitive styles resulting from interpersonal strain. For example, there is a degree of uncontrollability in interpersonal relations due to the inability to predict or control the actions of others. Self-compassion may be particularly helpful in the context of relationship strain, since it diminishes the need to control the outcome of the situation (Neff, 2008). Rather than basing one's worth on the success or failure of a relationship, the individual is able to understand that they can only control their own reactions. Self-compassion can reduce the

amount of time spent on dwelling on failures, redirect focus towards valued actions in the future (Neff, Hsieh, & Dejitterat, 2005), increase willingness to accept responsibility for negative relationship events, and take action to engage with event consequences, rather than change or avoid them (Allen & Leary, 2010). Self-compassion may modify cognitive styles, like hope, that have been shown to mediate the stress and depression relation.

Self-compassion has been associated with a variety of cognitive styles that are proposed to explain its effect on reducing the impact of stressful events. Neff's (2003a) early theorization was that trait self-compassion should enhance well-being and decrease depression by reducing experiences of shame, loneliness, over-identification, emotional avoidance coping, and heighten experiences of positive affect, valued action, accurate self-appraisal, and emotional approach coping. Empirical support for these theorized mechanisms is sparse, however (Raes, 2010), and largely focuses on self-compassion's effect on acute stressors.

In theory, self-compassion may help individuals sustain positive cognitive styles, like hope, in the face of interpersonal strain, but this has not been empirically examined. Those who are self-compassionate may approach family strain with less self-criticism and judgment and sustain hope that the relationship may be repaired in the future, or that despite the poor relationship, the individual can still treat themselves with kindness, experience positive affect, and move towards valued actions (Snyder, 2002). Importantly, self-compassion does not represent an excessive form of positivity in the absence of negativity (Neff, Rude, & Kirkpatrick, 2007). Rather, self-compassion helps individuals remain nonjudgmental and resilient in the face of stressful events. Following the

framework of the social risk model of depression, self-compassion should attenuate interpersonal strain by reducing self-criticism over social exchanges and enhancing greater internal resources of care. Such attitudes may influence depressive symptomatology by sustaining hopeful views of the future. Cross-sectional research finds that self-compassion predicts hope in adult populations, and further, that hope mediates the relation between self-compassion and well-being (Yang, Zhang, & Kou, 2016). To date, research has not tested the relation between self-compassion and hope in the face of chronic stressors, like relationship strain.

The link between family strain and depression may be explained by maladaptive cognitive styles that engender limited hope for the future. Theoretically, self-compassion should moderate these cognitive styles, and serve as a resilience resource in the face of familial strain. In addition to studying psychological mechanisms of the interpersonal strain – depression relation, literature indicates that dysfunctional physiological processes are also essential mechanisms by which interpersonal strain influences depression.

Physiological Processes Linking Interpersonal Strain and Depression

Interpersonal strain can also lead to dysfunction in physiological processes. In the presence of chronic or repeated stressors, the body's physiological systems can become dysregulated overtime, such that multiple systems attempt to accommodate the chronic environmental strains to maintain stability and homeostasis (Juster, McEwen, & Lupien, 2010). The shift toward a new, constant state of physiological dysregulation has been termed "allostasis," and the physiological indicators of allostasis have been termed "allostatic load" (McEwen, 1998). Regulatory biomarkers are measured to determine the extent of an individual's allostatic load across multiple systems, and include

neuroendocrine, immune, metabolic, cardiovascular and respiratory, and anthropometric parameters. Each system contributes unique functions in the regulation of the sympathetic-adrenal-medullary (SAM) and hypothalamic-pituitary adrenal (HPA) axes. Together, they are thought to provide a holistic indication of an individual's adaptation to ongoing stress (Robertson, Beveridge, & Bromley, 2017). Among the systems that contribute to depression, immune function is especially important (Miller, Maletic, & Raison, 2009), and inflammation is a key component of immune function and risk factor by which stress mediates overall mental health (Rosenblat, Cha, Mansur, & McIntyre, 2014).

Inflammation is an early response in the immune defense process that helps to prevent and limit infection. When functioning efficiently, inflammatory responses help combat common colds (Cohen et al., 2012), heal wounds, and protect the gut microbiome (Kau, Ahern, Griffin, Goodman, & Gordon, 2012). Physical and psychological stressors can impact inflammation (Minihane et al., 2015). If stressors are continuous, the body's inflammatory processes remain activated, which can be detrimental to mental health. The relation between inflammation and mental health is explained by disruption of many pathways including the hypothalamic-pituitary-adrenal (HPA) axis, glucocorticoid resistance (Mohr & Pelletier, 2006), serotonin production, microglial function, and impaired neuroplasticity (Rosenblat et al., 2014).

During extended periods of inflammation, inflammatory messengers called cytokines are produced by white blood cells and help regulate the extent of inflammation. Some cytokines, like interleukin-6 (IL-6), are pro-inflammatory and proliferate in the bloodstream to produce pro-inflammatory proteins, further exacerbating inflammation in

response to stress (Graham, Christian, & Kiecolt-Glaser, 2006). Inflammation is often measured through blood samples, with IL-6 being the most prevalent pro-inflammatory cytokine (Hänsel, Hong, Cámara, & von Känel, 2010). Elevations in IL-6 appear to be an important mediator in the course of the chronic stress – depression relation. Several chronic stressors have been purported to increase levels of IL-6, including interpersonal strain.

Interpersonal strain and physiological processes. Theories linking interpersonal stress and dysfunctional physiological processes suggest that social stressors initiate physiological reactions in the body. Two theories elucidating this relation are social self-preservation (SSPT; Dickerson, Gruenewald, & Kemeny, 2004a) and social mentality theory (SMT; Gilbert, 2005). These theories propose that social threats evoke feelings of shame and self-criticism, feelings which can be evolutionarily adaptive in preserving the self from future social interactions, at least in the short term. These theories compliment the social risk model of depression, but also acknowledge the physiological toll of social stressors. For example, SSPT asserts that threats to the social self can provoke feelings of shame and self-criticism, which mediate physiological changes, like increased levels of inflammation and cortisol (Dickerson et al., 2004a). Similarly, SMT states that self-criticism activates self-protecting, sympathetic nervous system mechanisms in the face of threats (Gilbert, 2005 as cited in Neff et al., 2018). Experimental and correlational studies provide some support for the effect of social strain on physiological processes.

Experimental findings indicate that stressors involving social evaluation activate the HPA axis as measured by increased cortisol output (Dickerson & Kemeny, 2004), but

these studies leave questions regarding the role of chronic social stressors on inflammation. Several correlational studies find that chronic social conflict and social strain is associated with increased inflammation (Kiecolt-Glaser, Gouin, & Hantsoo, 2010; Kiecolt-Glaser et al., 2018; Rohleder, 2014). For example, high social strain (Yang et al., 2014) and marital discord (Kiecolt-Glaser et al., 2005; Kiecolt-Glaser et al., 2010; Kiecolt-Glaser et al., 2018) predict increased cytokine production across age groups. Similarly, negative social events like caregiver stress are predictive of higher concentrations of IL-6 (Kiecolt-Glaser et al., 2003; Lutgendorf et al., 1999).

Evidence suggests that acute social stressors increase inflammation in the short term, and some studies find that chronic social stress, like marital discord, is also associated with inflammation. The literature examining this topic describes inflammation as an outcome of social strain. However, inflammation is more aptly described as a risk factor for other mental and physical health problems (Rosenblat et al., 2014). To date, it is unclear whether the inflammation resulting from interpersonal strain is related to subsequent depressive symptoms.

Inflammation and depression. The physiology of mood disorders like depression has historically been viewed from the perspective of neurotransmitter activity. There has since been a resurgence in the literature surrounding inflammation as a physiological initiator and indicator of depression (Rosenblat et al., 2014). Studies find associational and causal links between inflammation and depressed states.

Extended inflammation is strongly related to depressive symptoms among individuals with chronic illnesses and among physically healthy individuals experiencing chronic psychosocial stressors (Rosenblat et al., 2014). Cross-sectional studies find that

levels of IL-6 are elevated among individuals experiencing more mood symptoms and mood disorders like major depressive (Miller & Raison, 2016) and bipolar disorder (Fillman, Sinclair, Fung, Webster, & Weickert, 2014). Longitudinal studies find that inflammation is associated with subsequent depressive symptoms between 1 and 12 years later (Valkanova, Ebmeier, & Allan, 2013).

Experimental studies reliably show that manipulating levels of inflammation can initiate illness related behaviors in animal and human studies. For example, introducing the body to endotoxins and stressful states increases levels of IL-6 and other cytokines, which in turn increase illness behavior, such as fatigue, appetite changes, and depressive symptoms in animals and humans (Davis et al., 2008; Fleger, Haroon, & Miller, 2015). Alternatively, research finds that reducing inflammatory states with anti-inflammatory therapies can decrease depressed mood (Rosenblat et al., 2014). Depressed mood has been associated with auto-immune disorders, such as rheumatoid arthritis and inflammatory bowel disease. Treating these patients with anti-inflammatory therapies has been shown to reduce depressive symptoms. Together, the findings demonstrate a bidirectional relation between inflammation and depressive symptoms.

Numerous studies find that enhanced social support serves as a protective factor in the face of stress and subsequent mental and physical health symptoms (Cohen & Wills, 1985). However, when the source of stress is due to interpersonal strain among social support members, individuals may need to activate internal resources of care to help manage the inflammatory consequences of interpersonal strain. Exploratory research suggests that self-compassion plays a role in modulating such stress-induced immune responses (Pace et al., 2009).

Self-compassion as a moderator of the strain – IL-6 relation. Early research into self-compassion speculated that compassion towards the self regulates the stress response and activates self-soothing mechanisms (Gilbert, 1989 as cited in Neff et al., 2007). Social mentality theory states that self-criticism increases sympathetic nervous system activity and that self-compassion can serve to counteract this threat response by activating self-soothing, parasympathetic mechanisms (Gilbert, 2005 as cited in Neff et al., 2018). Self-compassion meets critical and threatening situations with self-care. Just as parasympathetic and sympathetic systems interact in stressful situations, so, too, do self-criticism and self-compassion (Neff et al., 2018). Recent research finds that a self-compassionate attitude can attenuate the impact of acute stress on regulating systems as measured by decreased cortisol output, increased heart rate variability (Arch et al., 2014; Svendsen et al., 2016), and decreased inflammation (Breines et al., 2014; Pace et al., 2009).

Studies relating self-compassion and inflammation focus on the role of self-compassion in mitigating the negative effects of acute social evaluative threat, the stress that arises when one is being evaluated by others. Self-compassion may act as a buffer against experiences of feeling threatened or self-critical, which could dampen the negative impact of social stressors on physiological mediators of mental health (Breines et al., 2014). For example, one study found that inducing thoughts of shame through written accounts of shameful experiences resulted in elevations of pro-inflammatory cytokine activity compared to a control writing group (Dickerson, Kemeny, Aziz, Kim, & Fahey, 2004). Researchers have also investigated whether self-compassion would serve as a protective factor against interpersonal stressors. Following exposure to a social

evaluative stressor, researchers found that those higher in self-compassion had lower IL-6 levels one day after the stressor (Breines et al., 2014) and lower salivary alpha-amylase levels, which measures sympathetic nervous system activation, the following two days after the stressor (Breines et al., 2015). These findings suggest that self-compassion may combat the effects of acute social evaluative threat on inflammation. No research has examined whether self-compassion moderates the chronic strain – inflammation relation.

Self-compassion is proposed to buffer the effects of stress on mental health via psychological and physiological mechanisms, although the extent to which it assists in coping with chronic stressors remains largely theoretical. Eastern and Western approaches to psychology have proposed many factors linking self-compassion to well-being, but the empirical tests of mechanisms are sparse (Raes, 2010). Understanding how self-compassion may influence mental health outcomes following chronic stressors is crucial for developing the theoretical foundation for testing self-compassion interventions.

Proposed Model and Hypotheses

Family strain is an important chronic stressor and has been shown to influence depressive symptoms in adults (Frone et al., 1997; Krause & Rook, 2003; Umberson & Montez, 2010). Negative interpersonal relationships are more strongly predictive of distress compared to other major life stressors (Ballas & Dorling, 2007). Theory and evidence suggest that possessing a positive, nonjudgmental view of oneself may help individuals combat the repercussions of acute stressful events, but little work has focused on whether and how the effects of chronic relationship strain on depressive symptoms are moderated by self-compassion. This study investigated whether relations between current

family strain and depression are moderated by self-compassion. Next, this study evaluated whether the buffering effects of self-compassion are explained by hope and levels of inflammation in a sample of middle-aged, community-dwelling adults.

Self-compassion is an emerging resilience concept that may be especially relevant for buffering effects of family strain on mental health. Self-compassion directly addresses cognitive patterns that relate to socio-emotional regulation (Allen & Knight, 2005). The features of self-kindness, common humanity, and mindfulness interact to dampen feelings of isolation, enhance connection with others, and allow space for intentional actions, which may be helpful during chronic family strain. Thus, it was hypothesized that self-compassion will buffer the effect of family strain on depressive symptoms in adults.

Hypothesis 1: Self-compassion will buffer the effect of family strain on depressive symptoms in adults, such that the relation between strain and depressive symptoms will be smaller in magnitude among those higher versus lower in self-compassion.

Although empirical research is accumulating, there is less understanding of how self-compassion influences the strain-depression relation in adults. Therefore, this study also investigated whether hope explains this relation. It was hypothesized that self-compassion will moderate the family strain-depression relation through enhancements in hope.

Hypothesis 2: Self-compassion will moderate the family strain-depression relation via increased hope. Specifically, the weaker strain—depressive symptoms relation among individual who are higher versus lower in self-compassion will be partially explained by their more hopeful cognitive styles.

Self-compassion is theorized to counteract threats that cause self-criticism and shame with physiological self-soothing mechanisms and positive regard for oneself (Gilbert, 2005). Burgeoning research indicates that self-compassion dampens inflammatory responses following acute, social evaluative stress (Breines et al., 2014). An additional aim of this study is to expand this finding to investigate the moderating effect of self-compassion on depressive symptoms related to a chronic stressor, family strain. Self-compassion is hypothesized to moderate the family strain-depression link through reductions in inflammation, as measured by IL-6.

Hypothesis 3: Self-compassion will moderate the family strain-depression link through reductions in inflammation, as measured by IL-6. Specifically, the weaker strain—depressive symptoms relation among individual who are higher versus lower in self-compassion will be partially explained by their lower levels of IL-6.

Methods

Participants

Participants for the current study were drawn among individuals enrolled in the ASULive study, a study of risk and resilience factors in middle-aged adults living in the Phoenix metropolitan area. Eligibility criteria included: 1) being fluent in English and/or Spanish, 2) aged 40-65 years, and 3) residing within one of 20 Census tracts within the Phoenix metropolitan area. The communities were selected to reflect the racial, age, and economic diversity of the region. Exclusion criteria included presence of physical, cognitive, or psychiatric impairment that would prevent participation in the project. Nine hundred and fifteen participants were initially recruited through mailings and informational flyers and enrolled in the study, but 110 participants dropped out before

beginning study activities. Of the 805 participants who began study activities, 762 provided information regarding family strain and mental health, 623 provided blood samples for assessment of IL-6, and 538 provided follow-up information regarding mental health.

Procedure

Study procedures were approved by the Institutional Review Board at Arizona State University and participants completed informed consent prior to enrollment. Participants were initially screened by phone. Once admitted into the study, participants completed a series of initial self-report questionnaires containing questions on demographic characteristics (i.e., age, gender, ethnicity), social relationship quality (e.g., family strain), and personal risk and resilience characteristics (i.e., self-compassion, hope). Second, they engaged in a phone interview during which they were asked about their physical and emotional health. Next, participants engaged in a home visit conducted by a research nurse who collected blood samples for assessment of physiological parameters, including IL-6 levels. Finally, at least six months following their initial phone interview, a follow-up assessment was conducted with participants who were able to be contacted via phone and agreed to provide information about their emotional health over the past six months.

Family strain, self-compassion, hope, and baseline depressive symptoms were assessed at the initial assessment, IL-6 was assessed at the home visit, and depressive symptoms were re-assessed at follow-up.

Measures

Family Strain. Current family strain was assessed by creating a latent factor of items from the Family Strain subscale from the MIDUS-1 Study (MIDUS; Schuster et al., 1990; See Appendix A) and the Negative Social Ties Scale (NST; Finch, Okun, Barrera, Zautra, & Reich, 1989; See Appendix A). Two family strain items from the MIDUS ("How often do members of your family make too many demands on you?" and "How often do they make you feel tense?") were answered on a Likert scale from 1 (Not at all) to 4 (A lot). Higher scores indicated greater family strain. The two MIDUS-1 items have an internal consistency in this sample of $\alpha = .67$. Four items from the NST asked participants to indicate the extent to which statements apply to current family members, such as, "How often are they critical of your behavior?" and "How often do they provoke feelings of conflict and anger?" and were answered on the same Likert scale as the MIDUS. Scores range from 1 to 4 with higher scores indicating greater family strain. The four NST items have good internal consistency in this sample ($\alpha = .82$).

Self-Compassion. Nine items from the Self-Compassion Scale (SCS; Neff, 2003b; See Appendix B) were completed by participants and a latent factor was created using factor scores. The original scale contains 26 questions asking participants to indicate how often they do the following things, such as being kind to oneself when experiencing suffering and tolerance of one's flaws and inadequacies. Questions are rated on a Likert scale from 1 (Almost Never) to 5 (Almost Always). The SCS contains six subscales consisting of self-kindness/self-judgment, common humanity/isolation, and mindfulness/over identification. Total scores are gathered by calculating the mean of the responses. This study used nine items from the self-kindness and mindfulness subscales. Scores range

from 1 to 5 with higher scores indicating higher levels of self-compassion. The SCS has high internal consistency in this sample (α = .90).

Hope. Hope was measured using the eighteen items from the Revised Trait Hope Scale (THS-R; Shorey & Snyder, 2004; See Appendix C). A latent variable was constructed using the factor scores from the eighteen items. Questions ask participants to indicate whether statements apply to them on a Likert scale from 1 (False) to 8 (True). Items include questions such as, "I have found that I can overcome challenges" and "I'm good at coming up with solutions". The THS-R contains three subscales: Goals, Agency, and Pathways. Total scores are calculated by obtaining the mean of all eighteen items. The scores range from 1 to 8 with higher scores indicating greater trait hope. The THS-R had excellent internal consistency in this sample (α = .92).

Inflammation. Inflammation was indexed by IL-6, which has been frequently associated with relationships and systemic inflammation (Kiecolt-Glaser et al., 2010). Blood samples collected during the home visit were transported on ice to the laboratory, and centrifuged to yield plasma, which was aliquoted and frozen at -80° F until shipment. Samples were then shipped frozen to University of California, Los Angeles, were the plasma samples were assessed to determine IL-6 values. Detailed procedures of the collection of inflammation biomarkers followed that of Davis et al. (2018). Since this study focuses on the effects of chronic inflammation, cases will be excluded if IL-6 exceeds 10pg/mL, as this indicates acute illness. IL-6 values are log transformed to address non-normality of the distribution. Higher scores indicate greater presence of the inflammatory cytokine IL-6 in the bloodstream.

Depressive Symptoms. Depressive symptoms were assessed by creating a latent factor using factor scores from the seven items of the Mental Health Inventory (MHI-D; Veit & Ware, 1983; See Appendix D), which measures psychological distress and well-being in general populations. The questions ask participants to indicate how often they experienced various emotions and behaviors on a scale from 1 (All of the time) to 6 (None of the time). Examples of the seven items in this scale include, "Have you felt like crying?", "Have you felt lonely?", and "How you been in low or very low spirits?" Total score was computed as the mean of all seven items. Scores ranged between 1 and 6, with higher scores indicating greater well-being. The MHI had good internal consistency for this sample ($\alpha = .90$).

Control Variables. Control variables included demographic variables, anti-depressant medication, and depressive symptoms at initial study assessment. The demographic variables of age, gender, were assessed via self-report questionnaires. Participants also indicated whether or not they used anti-depressant medication by answering (1 = Yes; 0 = No). Participants were not asked to specify what they were taking anti-depressant medication for.

Data Analytic Plan

Descriptive statistics and correlations were derived using IBM SPSS Statistics 24. Hypotheses were tested using *Mplus* Version 7.1. *Mplus* uses full information maximum likelihood (FIML) estimation (Muthén & Muthén, 1998-2012), which estimates parameters and standard errors using all available data. This creates parameter estimates and standard errors robust to data assumed to be missing at random (Enders, 2010). Family strain, hope, and IL-6 were mean-centered, and age, gender, anti-depressant

medication, and initial depressive symptoms scores were included as covariates. Factor scores were calculated for family strain, self-compassion, hope, and depressive symptoms for use in regression analyses. *Mplus* creates factor scores using the maximum a posteriori method, which predicts the location of measured scores on the latent factor (Muthén & Muthén, 1998-2012). A measurement model was tested to examine the relations between the latent factors (family strain, self-compassion, hope, and depressive symptoms) and their measured variables (Anderson & Gerbing, 1988). Following the measurement model, structural models were tested to determine the relations among the latent variables and measured variable (IL-6). Model fit was tested according to recommendations set out by Hooper, Coughlan, and Mullen (2008) using χ^2, root mean square of the association (RMSEA), comparative fit index (CFI), and standardized root mean square residual (SRMR) with the following criteria to indicate good model fit: $\chi^2 = \chi^2$/df between 5 and 2; RMSEA < .08; CFI $\geq$ 0.95; SRMR < .05. Significance level for all analyses was set at $p < .05$.

Primary Analyses. To test Hypothesis 1, Model 1 included the latent factors of family strain, self-compassion, and their interaction (path c_3) as predictors of depressive symptoms. It was hypothesized that trait self-compassion would moderate the relation of family strain and depressive symptoms, such that the relation between strain and depressive symptoms would be weaker for those high versus low in self-compassion (Hypothesis 1; See Figure 1).

To test Hypothesis 2, Model 2 tested the first order (*a* path) conditional effect of self-compassion on the indirect effect of hope on the family strain - depressive symptoms relation (Hypothesis 2; See Figure 2). This hypothesis was tested using moderated

mediation analyses. To determine conditional indirect effects of the family strain X self-compassion relation with depressive symptoms, the bias-corrected bootstrap confidence interval for the product of paths a_3 and b_1 set out by Hayes (2012) will be used. SEM was conducted to determine if hope is a mediator of strain X self-compassion – depressive symptom relation. Hypothesis 2 tested the conditional indirect paths from the family strain X self-compassion interaction to hope (path a_3) to depressive symptoms (path b_1) and tested the presence of moderated mediation by determining the bias-corrected bootstrap confidence interval for the product of paths a_3 and b_1. It was hypothesized that the interaction between family strain and self-compassion would predict less hope, which would predict more depressive symptoms.

For moderated mediation (ω) to occur, the moderation of the residual direct effect of family strain should be reduced compared to the moderation of the overall effect of family strain on depressive symptoms (Hayes, 2015; Muller, Judd, & Yzerbyt, 2005), such that c_3' in Hypothesis 2 should be smaller in absolute value than c_3 in Hypothesis 1.

$\omega = (a_3$ *family strain X self-compassion)*$(b_1$ *hope)* $= c_3$ *family strain X self-compassion -* c_3' *family strain X self-compassion*

For Hypothesis 3, Model 3 tested the conditional indirect paths from family strain X self-compassion interaction to hope and IL-6 to depressive symptoms (Hypothesis 3; See Figure 3). The evidence of moderated mediation follows that from Hypothesis 2, such that c'_3 in Hypothesis 3 should be smaller in absolute value than c_3 in Hypothesis 1.

Exploratory Analyses. The fourth hypothesis tested the second order (*b* path) conditional effect of self-compassion on the indirect effect of hope on the family strain – depressive symptoms relation (Hypothesis 4; See Figure 4). Hypothesis 4 tested the conditional

indirect paths from family strain (path a_1) to the hope X self-compassion interaction (path b_2) and tested the presence of moderated mediation by determining the bias-corrected bootstrap confidence interval for the product of paths a_1 and b_2. It was hypothesized that family strain would predict less hope and the interaction between hope and self-compassion would be negatively related to depressive symptoms.

Hypothesis 5 tested whether the first and second order conditional indirect effect of self-compassion on the indirect effect of hope on the family strain – depressive symptoms relation differs as a function of gender (3-way latent variable interaction; Hypothesis 5; See Figure 5). This hypothesis was tested using dual moderated-mediation analyses, wherein gender was added as a moderator of the family strain X self-compassion interaction from Model 2 as well as the hope X self-compassion interaction from Model 4. Evidence of dual moderated mediation was determined by calculating the bias-corrected bootstrap confidence interval for the product of paths a_3, a_4, and b_1 for dual moderated mediation on the *a* path and the product of paths a_1, b_2, and b_3 for dual moderated mediation on the *b* path. The literature on gender differences of self-compassion is mixed and therefore, no hypotheses were made as to whether the conditional indirect effects would differ among males and females in the sample.

The sixth hypothesis examined a model where self-compassion served as the mediator of the strain-depressive symptoms relation, and hope served as the moderator (Hypothesis 6; See Figure 6). Although in the present study, self-compassion was hypothesized to operate as a moderator, there is evidence that self-compassion may also serve as a mediator. Studies find that self-compassion mediates the relation between rumination and depression (Raes, 2010). If self-compassion were a mediator, family

strain should exert its influence on depressive symptoms via self-compassion. Differences in self-compassion should account for the association between family strain and depressive symptoms. To determine whether self-compassion serves as a mediator, exploratory moderated mediation analyses were conducted in which self-compassion replaced hope as a mediator, and hope replaced self-compassion as a moderator of the *a* path, and then the *b* path. It was hypothesized that family strain would predict less self-compassion and the interaction between self-compassion and hope would be negatively related to depressive symptoms. The evidence of moderated mediation follows that of Hypotheses 2-4.

Results

Sample Characteristics

Table 1 displays the demographic characteristics of the sample. The 762 participants were predominately female (54.5%), middle-aged (M = 53.51, SD = 7.25), and did not endorse taking antidepressant medication (80.4%). The ethnic composition of the sample was 68.4% Non-Hispanic White, 23.6% Hispanic, 2.4% Black/African American, 1.4% Asian, and .8% American Indian/Alaska Native. Table 2 depicts the descriptive statistics and intercorrelations among study variables. Average family strain was 2.20 (SD = .66), indicating that participants experienced instances of family strain rarely. Participants reported above average levels of self-compassion compared to similar community samples (M = 3.60, SD = .75; Neff & Pommier, 2013). Mean scores for hope and IL-6 were 5.85 (SD = 1.09; Range 2.50 to 8) and .51 (SD = .73; Range .22 to 72.63), respectively, indicating above average levels of hope and average levels of IL-6 compared to community samples (Cheavens, Feldman, Gum, Michael, & Snyder, 2006;

Singh-Manoux et al., 2014). At the beginning of the study, the average level of depressive symptoms was 1.78 (*SD* = .88; Range 1 to 6). At follow-up, which occurred around 20 months later, on average, the mean level of depressive symptoms was 1.75 (*SD* = .87; Range 1 to 5.43).

Factor Analytic Results

The six items from the two family strain scales were analyzed through exploratory factor analysis to determine whether they adequately reflect a single construct of family strain. *Mplus* Version 7.1 was used to conduct exploratory and confirmatory factor analyses (EFA and CFA, respectively). The EFA was performed with half of the data selected at random (*n* = 379) using maximum likelihood estimation and GEOMIN (oblique) factor rotation, which increases interpretability of factors and allows items to correlate. Model fit was tested according to recommendations set out by Hooper et al. (2008), using χ^2, root mean square of the association (RMSEA), comparative fit index (CFI), and standardized root mean square residual (SRMR). Based on these factors, the single factor model was selected because fit statistics indicated a good model fit [$\chi^2(9)$ = 77.321, RMSEA = .142 [90% CI = .113 - .171], CFI = .934, SRMR = .044]. The two-factor model was not retained because it yielded an eigenvalue of less than 1 and only fit one item to the second factor. Next, a confirmatory factor analysis was conducted using the remaining half of the data (*n* = 383), employing the same GEOMIN factor rotation and maximum likelihood estimation and restricting the six items to a single factor. The CFA for the one-factor model demonstrated adequate fit statistics, $\chi^2(9)$ = 169.173, RMSEA = .153 [90% CI = .133 - .173], CFI = .921, SRMR = .046. The standardized parameter results for the final one factor model are presented in Table 3. All factor

loadings were greater than .58. The six items representing the family strain had good reliability in the total sample, Cronbach's α = .87.

Measurement Model Results

A measurement model was conducted to determine the relations between latent variables and their observed variable indicators (See Table 4; Anderson & Gerbing, 1988). Next, and overall measurement model was calculated, which included family strain, self-compassion, hope, and depressive symptoms. Indices demonstrated adequate fit with the data (RMSEA = .065 [90% CI = .063 - .068], CFI = .930).

Structural Model Results

Next, a series of structural models were examined to test the hypothesized paths among the latent and observed variables. Hypothesis 1 measured the moderating effect of self-compassion on the family strain-depressive symptoms relation, controlling for age, gender, initial depressive symptoms, and anti-depressant medication. Results from Model 1 are depicted in Table 5: Model 1. Findings indicated no relation between family strain and depressive symptoms, β = .057, *SE* = .07, *p* = .390, and no family strain X self-compassion interaction effect, β = -.128, *SE* = .09, *p* = .137. Together, the predictors explained 35.6% of the variance in depressive symptoms. Model 1 fit indices were as follows: $\chi^2(2)$ = 21.513, RMSEA = .113 [90% CI = .073 - .159], CFI = .938, SRMR = .022. Based on model fit criteria, Model 1 had adequate fit with the data.

Hypothesis 2 measured the first order conditional indirect effect of hope on the family strain-depressive symptoms relation as a function of self-compassion, controlling for age, gender, initial depressive symptoms, and anti-depressant medication. Findings from Model 2 are depicted in Table 5: Model 2, and Figure 7. Findings indicated that

hope mediated the family strain – depressive symptoms relation, such that family strain was negatively associated with hope (a_1 path), and hope was negatively associated with changes in depressive symptoms (b_1 path) at all levels of self-compassion, β = .026, *SE* = .01, $p < .05$. However, contrary to prediction, the indirect effect (via hope) was not significantly different as a function of self-compassion, β = .020, *SE* = .02 $p = .194$ (a_3b_1). Specifically, the effect of family strain on hope was not different as a function of self-compassion. Together, the predictors in Model 2 accounted for 38.4% of the variance in depressive symptoms. Model 2 fit indices were as follows: $\chi^2(2) = 21.152$, RMSEA = .112 [90% CI = .072 - .158], CFI = .969, SRMR = .022. These indices suggest that this model has good fit with the data.

Hypothesis 3 measured the first order conditional indirect effect of IL-6 on the family strain – depressive symptoms relation as a function of self-compassion, controlling for age, gender, initial depressive symptoms, and anti-depressant medication. Results from Model 3 are depicted in Table 5: Model 3, and Figure 8. Findings indicated that inflammation, as measured by IL-6, did not mediate the family strain-depressive symptoms relation, β = .001, *SE* = .01, $p = .773$. The relation did not differ as a function of self-compassion (a_4b_1), β = -.001, *SE* = .01, $p = 804$. Model 3 fit indices indicate poor fit with the data: $\chi^2(11) = 342.471$, RMSEA = .199 [90% CI = .181 - .217], CFI = .500, SRMR = .103.

Exploratory Analyses

The first exploratory analysis tested whether self-compassion moderated the *b* path of the moderated mediation model. Model 4 measured the second order conditional indirect effect of hope on the family strain-depressive symptoms relation as a function of

self-compassion, controlling for covariates. Findings are depicted in Table 5: Model 4, and in Figure 9. Model 4 indicated that hope mediated the family strain – depressive symptoms relation, such that family strain was negatively related to hope (a_1 path), and hope was negatively associated with changes in depressive symptoms (b_1 path) at all levels of self-compassion, β = .045, *SE* = .02, $p < .001$. The indirect effect of family strain on depressive symptoms through hope as a function of self-compassion was marginally significant, β = -.028, *SE* = .01, $p = .05$ (a_1b_2). This suggests that as family strain increases, hope decreases, and depressive symptoms increase. However, the effect of decreased hope on increased depressive symptoms appeared weaker at increasing levels of self-compassion, but not at high levels (+1 SD) of self-compassion (see Figure 10). Together, the predictors in Model 4 accounted for 39.7% of the variance in depressive symptoms. Model 4 fit indices were as follows, $\chi^2(8) = 227.157$, RMSEA = .190 [90% CI = .169 - .211], CFI = .678, SRMR = .060, and indicate poor model fit.

The second exploratory analysis tested whether the moderated mediation effect examined in Models 2 and 4 varied by gender. Findings are depicted in Table 6. Dual moderated mediation analyses were used to compare differences in first and second order conditional indirect effects based on gender, controlling for covariates. These analyses were performed using the PROCESS macro in SPSS (Version 23; Hayes, 2012). Results of the dual moderated mediation on the *a* path indicated that males and females did not differ in the extent to which self-compassion buffered them from the indirect effect of hope on the family strain – depressive symptoms relation, β = .006, *SE* = .03, $p > .05$ (See Table 5: Model 5). Results of the dual moderated mediation on the *b* path were also not significant, β = -.016, *SE* = .05, $p > .05$ (See Table 5: Model 6).

Table 7 displays the results of the first order (Model 7) and second order (Model 8) conditional indirect effects of self-compassion on the family strain-depressive symptoms relation as a function of hope, controlling for covariates. Results from Model 7 indicated that self-compassion did not mediate the family strain-depressive symptoms relation, β = .007, *SE* = .01, *p* = .983 (a_3b_1). The relation did not differ as a function of hope (see Table 6). The fit indices for Model 7 demonstrated good fit: $\chi^2(2) = 18.284$, RMSEA = .103 [90% CI = .063 - .149], CFI = .974, SRMR = .020. Results from Model 8 indicate that self-compassion did not mediate the family strain – depressive symptoms relation, β = -.001, *SE* = .01 *p* = .878. However, moderated mediation analyses found a conditional indirect effect of self-compassion as a function of hope (a_1b_2). Specifically, self-compassion had a negative indirect effect on the family strain-depressive symptoms relation only at high levels of hope (+1 SD), such that as family strain increased, self-compassion decreased, and depressive symptoms decreased only when individuals had high hope (see Figure 11). The predictors in Model 8 accounted for 39.6% of the variance in depressive symptoms. Fit indices for Model 8 were as follows: $\chi^2(5) = 172.925$, RMSEA = .210 [90% CI = .184 - .237], CFI = .734, SRMR = .057, indicating poor fit.

Discussion

This study examined whether and how self-compassion moderates the relation between family strain and subsequent depressive symptoms in a sample of middle-aged community-dwelling adults. Specifically, it tested the extent to which self-compassion buffered the relation between family strain and depressive symptoms, to what degree the effect was mediated by hope and IL-6, and whether these indirect effects were conditional on levels of self-compassion. Findings showed that there was no direct effect

of family strain on depressive symptoms, nor did self-compassion buffer the relation. Hope, but not IL-6, mediated the relation between family strain and depressive symptoms, but the indirect effects were not conditional on levels of self-compassion.

Self-compassion as moderator of family strain – depressive symptoms relation. A primary hypothesis of the current study was that family strain would be related to subsequent increases in depressive symptoms, and that this relation would be weaker for those high in self-compassion. However, findings revealed no direct relation between family strain and depressive symptoms and no conditional effect of self-compassion. The current findings with regard to the strain – depressive symptoms relation contrast with those of previous research, which has found that relationship strain is associated with later depressive symptoms in middle-aged and older adults (Frone et al., 1997; Krause & Rook, 2003). Other studies have found that relationship strain predicted depressive symptoms four years later in middle-aged adults (Frone et al., 1997) and six years later in older adults (Krause & Rook, 2003). An important distinction is that previous studies measured several sources of strain, unlike the current study. For example, Frone et al., (1997) found that the impact of family strain on work life predicted depressive symptoms in middle-aged adults four years later. In the current study, there was no indication of how family strain impacted other areas of life. In addition, Krause and Rook (2003) modeled relationship strain as a latent factor comprised of strain with friends, children, and other relatives. In contrast, the current study only reported on family strain. Thus, it is possible that strain from many sources, or more intense strain, may better predict depressive symptoms than a single source of relationship strain.

Previous studies suggest that individuals who are able to regulate emotions during socio-emotional interactions may be most adept at navigating interpersonal strain (Seppala et al., 2013). The present study hypothesized that self-compassion would buffer the family strain – depressive symptoms relation based on prior research, which finds that self-compassionate individuals display less verbal aggression towards their partners, have greater relationship satisfaction, and report greater closeness and social support (Baker & McNulty, 2011; Crocker & Canevello, 2008; Neff & Beretvas, 2013). In addition, theories of self-compassion suggest that self-compassion should help reduce feelings of shame and increase connection with others (Neff, 2003a; Neff, 2008). No research had examined self-compassion as a moderator of the relationship strain – depressive symptoms relation. Current results showed no conditional effect of self-compassion on the family strain – depressive symptoms relation, contrary to hypotheses. This result suggests that self-compassion may not be protective against the psychological effects of family strain. One reason for this may be due to a discrepancy between the type of stress and the support available. According to the matching hypothesis, support against stress should be matched according to the stress domain, the type of support, and the type of stressor (Cohen & Willis, 1985). Self-compassion is a unique form of stress-buffering in that support is self-generated. Although an awareness of the connection to others' suffering is a part of self-compassion, it may not be adequate, according to the matching hypothesis, because support for oneself may not satisfy the support desired from one's family. It also may not provide the emotional or instrumental support needed during family strain. There may be other explanations for the lack of an interaction effect of self-compassion, such as the presence of a different form of support that was unaccounted for

in this study. For example, Schuster et al., (1990) found that, among middle-aged adults, the relation between negative family interactions and depressive symptoms was buffered by positive interactions with family members. The present study did not examine the presence of family support, but future studies testing the stress-buffering effects of self-compassion may want to consider the effects of social support in order to isolate the impact of self-compassionate responding.

Finally, this study only used the kindness and mindfulness subscales of the Self-Compassion Scale (SCS; Neff, 2003a). This decision was made to reduce participant burden, but the absence of the common humanity subscale may be contributing to the lack of conditional effects. A study examining depressive symptoms in a community sample of German adults found that the individual self-compassionate subscales alone did not protect against depressive symptoms (Körner et al., 2015). Only the composite score of self-kindness, mindfulness, and common humanity were found to buffer depressive symptoms among individuals with low and high levels of self-coldness. Thus, this study may have been unable to capture conditional effects due to the exclusion of common humanity, a key component in self-compassion.

Hope as a mediator of the family strain – depressive symptoms relation. A second hypothesis of the current study was that hope would serve as a cognitive style that would mediate the family strain – depressive symptoms relation. The hypothesis was based on the social risk model of depression, which indicates that the link between social strain and depression is driven by ruminative thinking styles and reduced social interactions (Allen & Badcock, 2003). Additionally, the hopelessness theory of depression suggests that one common subtype of depression is due to an experience of

hopelessness following a negative event or stressor (Abramson et al., 1989). This study proposed that hope may regulate the emotions and motivations during interpersonal strain and explain subsequent depressive symptoms. Results found that hope partially mediated the relation between family strain and depressive symptoms, such that more family strain was related to less hope, and less hope was related to subsequent increases in depressive symptoms. To date, no studies have looked at the indirect effect of hope on the family strain - depressive symptoms relation. From the perspective of the social risk model and hopelessness depression theories, the stress of family strain may reduce hope through provoking feelings of self-criticism and reducing motivation to engage in social behaviors. This reduction in hope may have driven participants to experience depressive symptoms over time.

Research finds that individuals attribute stressors to different causal factors based on the level of importance of that stressor and whether the stressor impacts a person's goals and values. Stress presents adults with opportunities to actively pursue valued goals, reevaluate goals, or create new goals (Brandtstädter, 1999). Setting unrealistic goals or being inflexible in altering one's goal pursuits is a risk factor for depression (Coyne & Gotlib, 1983; Karoly, 1999; Lecci, Okun, & Karoly, 1994). These processes are reflective of facets involved in hope, namely having self-relevant goals, having agency to meet those goals, and remaining flexible in developing a variety of ways to meet goals in the face of obstacles (Snyder, 2002). It is possible that the experience of family strain forces individuals to confront initial goals and ideas regarding family, which can fuel feelings of hopelessness, making it difficult to reevaluate goals, and lead to subsequent depression. One direction for future research would be to test whether the

negative relation between family strain and hope can be explained by increased self-criticism, reduced motivation for social engagement, and changes in goal pursuits.

Conditional effects of self-compassion. A third hypothesis of the current study was that the indirect effect of hope would be conditional on levels of self-compassion. Two models were tested to determine whether self-compassion moderated the family strain – hope relation (Model 2, *a* path) or the hope – depressive symptoms relation (Model 4, *b* path). In Model 2, self-compassion was proposed to buffer family strain and help to sustain hope, which would be related to fewer changes in depressive symptoms. In Model 4, family strain was proposed to reduce hope, and self-compassion was proposed to buffer the hope – depressive symptoms relation. Results showed no first order conditional indirect effect but pointed to the possibility of a second order conditional indirect effect of hope on the family strain – depressive symptoms relation as a function of self-compassion. However, poor model fit for the second order conditional indirect effect precludes further interpretation of these results. The lack of a buffering effect of self-compassion on both the *a* and *b* paths is inconsistent with theories of self-compassion, which state that self-compassion should help individuals relate to negative events (family strain) and emotions (hopelessness) from a nonjudgmental perspective, offer kindness to oneself, and recognize one's connection to humanity (Neff, 2003a). For example, one study found that self-compassion was related to fewer hopelessness depressive symptoms in adolescents and young adults (Zhou, Chen, Liu, Lu, & Su, 2013). The lack of a moderating relation in the current study, as noted above, may be explained by exclusion of the common humanity subscale. Researchers have found that the self-compassion subscales may have differential effects in buffering the relation

between cognitive vulnerabilities and depressive symptoms. For example, among Chinese adults, the mindfulness and self-kindness subscales buffered the relation between autonomous and self-critical thinking styles and depression. However, the common humanity subscale only buffered the relation between self-criticism and depressive symptoms (Wong & Mak, 2013). Thus, the exclusion of the common humanity subscale may have reduced response rates for some self-critical individuals.

Given that previous research has found that self-compassion buffers the relation between more severe stressors and physical/psychological distress, it is possible that family strain and hope may not have been potent enough to evoke a need for self-compassion in this sample. For instance, one theory of self-compassion proposes that self-compassion enacts self-soothing mechanisms in the presence of threat (Gilbert, 2005). It is possible that family strain and reduced hope were not acute enough to elicit a threat response.

IL-6 as a mediator of the family strain – depressive symptoms relation. A fourth hypothesis of the current study was that IL-6 would serve as an indicator of a physiological inflammatory process that would mediate the family strain – depressive symptoms relation. Results showed that IL-6 did not mediate the family strain – depressive symptoms relation. This is inconsistent with social self-preservation and social mentality theories, which propose that social threats elicit shame and self-criticism and resulting physiological effects, such as increased levels of inflammation (Dickerson et al., 2004; Gilbert, 2005). Empirical evidence finds that high social strain predicts increased cytokine production across age groups (Kiecolt-Glaser et al., 2003; Lutgendorf et al., 1999) and a wealth of evidence links increased inflammation to depressive symptoms as

well (Rosenblat et al., 2014). Both Kiecolt-Glaser et al. (2003) and Lutgendorf et al. (1999) found increased IL-6 production among caregivers of dementia and Alzheimer's patients. Thus, family strain may not have been sufficiently provocative to elicit an inflammatory response in this sample, and may explain the lack of significant findings. In addition, this community sample of middle-aged adults was relatively healthy and had low levels of IL-6 and depressive symptoms at the time of assessment.

Conditional effects of self-compassion. A fifth hypothesis of the current study was that self-compassion would moderate the indirect effect of IL-6 in the family strain – depressive symptoms relation. Contrary to hypotheses, the model had poor fit with the data and there was no conditional indirect effect of family strain on depressive symptoms through IL-6 as a function of self-compassion. This result does not align with experimental research, which finds that self-compassion buffers the relation between acute social evaluative threat and subsequent, immediate inflammation (Breines et al., 2014). This result is likely explained by differences in experimental versus naturalistic settings. Experimental studies are likely to find increased inflammation as a result of manipulation within a controlled environment, which increases power to detect effects of acute inflammation versus chronic levels accrued over time. Experimental findings are difficult to generalize to chronic stressors encountered in daily life, which may have broader health implications. This study tested existing levels of inflammation in a relatively healthy sample, introducing the possibility of many sources of error.

This was the first study to examine the effects of self-compassion on the relation between chronic interpersonal strain, inflammation, and subsequent depressive symptoms. Previous research had only examined these relations under acute social

stressors. Although the general level of health in the sample, reflected in part by the relatively low average levels of IL-6, may explain these findings, it is also possible that current measures of self-compassion explain processes under acute stressors better than chronic stressors. However, given evidence that self-compassion is protective against distress from chronic health problems (Costa & Pinto-Gouveia, 2013; Pinto-Gouveia, Duarte, Matos, & Fráguas, 2014; Sirois, Molnar, & Hirsch, 2015; Wren et al., 2012) and other age-related chronic stressors (Herriot, Wrosch, & Gouin, 2018), the truncated levels of IL-6 is a more likely explanation. That said, this does raise a question of how self-compassion operates under low stress conditions, when there is less suffering.

Exploratory Analyses

Conditional indirect effects as a function of gender. In addition to testing formal hypotheses, this study explored several additional mediated and moderated effects of the strain –depressive symptoms relation. First, this study sought to determine whether there were variations in the way self-compassion buffered men and women from the indirect effect of hope on the family strain-depressive symptoms relation. No differences in the conditional indirect effect as a function of gender emerged in the findings. This result is unsurprising, given the small and non-significant conditional indirect effects. Research finds mixed evidence for gender differences in levels of self-compassion (Yarnell et al., 2015), but little research has been dedicated to understanding whether the processes linking self-compassion with outcomes differs among men and women. Baker and McNulty (2011) found that among heterosexual couples, women's self-compassion was associated with positive relationship behavior. However, for men in their sample, self-compassion was only beneficial for relationships when men were also high in

conscientiousness, indicating a willingness to accept mistakes. Among men low in conscientiousness, self-compassion was associated with poor relationship behavior. This suggests that self-compassion may have iatrogenic effects for certain subsets of men, but not for women. There are a few notable differences in this study compared to the present study. First, Baker and McNulty's (2011) study examined the impact of self-compassion on relationship behavior. In contrast, the present study tested whether the employment of self-compassion differs among men and women experiencing family strain. It is possible that the experience of family strain does not evoke the same motivation to correct for mistakes as does being in an intimate relationship. This interpretation should be considered with caution, however, since this study was unable to capture the extent to which participants felt responsibility for their role in family strain. Future work in this area should examine processes by which self-compassion differs among men and women in the context of different relationships.

Self-compassion as a mediator of the family strain-depressive symptoms relation. Finally, this study explored whether a model in which self-compassion served as the mediator and hope as the moderator provided a better fit of the data. Thus, self-compassion was tested as a mediator of the family strain – depressive symptoms relation. Inconsistent with hypotheses, there was no significant indirect effect of self-compassion. The first and second order conditional indirect effects of hope were also examined. Results found a significant second order conditional indirect effect, but poor model fit prohibits further explanation of this effect. Although the number of studies examining the indirect effects of self-compassion on the relation between stress and maladaptive mental health outcomes is small, these results conflict with the findings in this literature. Earlier

evidence indicates that self-compassion mediates the link between psychosocial stress and later emotional dysregulation (Vettese, Dyer, Li, & Wekerle, 2011) as well as links between self-criticism/rumination and psychological distress (Joeng & Turner, 2015; Przezdziecki et al., 2013; Raes, 2010). One possible explanation for the discrepancy between the current findings and those in the literature is the exclusion of the self-critical subscales of self-compassion (i.e., self-judgment, isolation, over identification) in the current study. A study of community adults found that the self-critical subscales explained more variance in depressive symptoms than the self-compassionate subscales (Körner et al., 2015). Additionally, a recent meta-analysis including clinical and non-clinical populations found that the self-critical subscales were more strongly linked to psychopathology than the self-compassionate subscales (Muris & Petrocchi, 2017). Although Körner and colleagues (2015) did not examine indirect effects, it is possible that one explanation for why self-compassion was not indirectly related to depressive symptoms may be due to missing subscales primarily associated with depressive symptoms.

Theoretical Considerations

When examining the current study within the broader literature linking self-compassion to health, one key consideration becomes readily evident: the nature of the samples employed in the majority of existing investigations. The majority of research that addresses the relation among self-compassion and depressive symptoms have used young adult and clinical samples (Krieger, Altenstein, Baettig, Doerig, & Holtforth, 2013; MacBeth & Gumley, 2012; Raes, 2010 & 2011). In contrast, this sample of community-dwelling middle-aged adults found no significant conditional, nor conditional indirect

effect of self-compassion on the relation between social strain and depressive symptoms. Important questions that need to be addressed include whether the construct of self-compassion is interpreted or endorsed differently as individuals age as well as whether self-compassion operates differently for clinical versus non-clinical populations.

Emerging research suggests that self-compassion, as it is currently measured may not operate the same way in middle-aged and older adult populations (Bratt & Fagerström, 2019), and that perhaps the question items are too abstract for older populations. Initial theories suggested that self-compassion increased with age, but existing research has found mixed results. Some factor analytic studies of the Self-Compassion Scale (SCS; Neff, 2003a) in middle-aged and older adult populations have been unable to replicate the original factor structure derived from young adult samples (Costa, Marôco, Pinto-Gouveia, Ferreira, & Castilho, 2016; López et al., 2015). Most of these studies have identified a two-factor structure consisting of self-compassionate and self-critical factors instead of Neff's six-factor correlated structure. It is possible that self-compassion may be interpreted differently in middle-aged adults, who perhaps approach personal suffering in a more basic compassionate vs. self-critical way as opposed to the nuanced model Neff theorized and validated with young adult populations. For example, middle-aged adults may respond to their own suffering in a kind versus unkind way as opposed to considering their experience in relation to universal suffering or being mindful of their emotional state. No studies have examined measurement invariance with age, but Neff et al. (2018) note that a forthcoming manuscript will address the issue of measurement invariance across age groups. It is also possible that group differences in self-compassion between young and middle-aged adults are explained by other reasons.

Researchers recognize a need for further inquiry on differences in self-compassion in young, middle-aged and older adults, but few provide theoretical guidance for why this difference may exist. One idea may be due to unique challenges at developmental stages that may or may not require self-compassionate responding as well as the accumulation of life experiences.

To understand the role of self-compassion as a stress-buffer requires that the nature and meaning of the stressor(s) to the individual be taken into account, since self-compassion is proposed to function when one experiences suffering (Neff, 2003a). Neff et al. (2018) found that individuals do not employ the elements of self-compassion in identical ways across all situations. Therefore, the moderating effect of self-compassion may differ depending on the stressors that are of primary concern to individuals at their developmental age. Middle adulthood is associated with different stressors than young adulthood (Brandtstädter & Greve, 1994; Heckhausen, 1997; Erikson, 1963). Young adults are primarily focused on identity development and establishing intimate connections with others. Adults have a generally stable identity and are focused on ways they can contribute to society by caring for others and living a meaningful life (Erikson, 1959). Thus, stressors eliciting a lack of purpose and meaning may be especially difficult at this life stage. In addition, declining health is another stressor that has traditionally been restricted to older adults, but in the last two decades the emergence of chronic health conditions (Wu & Green, 2000) in the population situates health as a major concern in middle adulthood (Ebner, Freund, & Baltes, 2006).

Surprisingly little research has looked into the moderating effects of self-compassion under varying stressful contexts. The vast majority of evidence finds that

self-compassion is a predictor of distress and well-being when middle-aged adults encounter health threats (Allen, Goldwasser, & Leary, 2012; Allen & Leary, 2010; Brion, Leary, & Drabkin, 2014; Costa & Pinto-Gouveia, 2011; Pinto-Gouveia et al. 2014; Terry, Leary, Mehta, & Henderson, 2013). No study has looked at whether self-compassion moderates the link between psychosocial stress and depressive symptoms in healthy, middle-aged adults. A cross-sectional study of healthy, middle-aged women found that self-compassion predicted attitudes about psychosocial loss and attitudes about physical changes related to aging (Brown, Bryant, Brown, Bei, & Judd, 2016). However, only attitudes of physical changes predicted mental health symptoms. Thus, one reason why self-compassion did not moderate or mediate the family strain-depressive symptom relation in this sample may be that middle-aged adults feel less threatened by social strain and more threatened by issues of physical health. The studies in which self-compassion mediates social stress and depressive symptoms have looked primarily at young adults. One reason for these relations in young adults may be that they are more likely to internalize psychosocial stress as something threatening to one's self-concept. This aligns with developmental perspectives detailing that young adulthood is a time where social connections are of primary focus and can dictate whether one will find a partner, a supportive community, or employment (Erikson, 1959; Nurmi, 1992; Nurmi, Poole, & Kalakoski, 1994). By comparison, middle-aged adults are likely to feel more secure about these aspects of their lives. Stress associated with physical health may be more threatening to adult's sense of self-concept and self-worth than psychosocial stress. Aging studies find that quality of life does not decline in adults until they experience serious health problems (Brandtstädter & Greve, 1994). Although further work is needed,

taking into account stressors associated with developmental age may be important for the future of self-compassion interventions and compassion-focused therapy with adult populations.

Theories purporting increased self-compassion with age state that adults may have higher levels of self-compassion based on the accumulation of experiences and an acceptance of life's challenges (Homan, 2016). Neff and Vonk (2009) theorized that self-compassion increases with age as individuals come to terms with their life. This is evidenced by empirical studies finding that as adults age, they are less inclined to seek control over external circumstances (Heckhausen, 1997). Thus, middle-aged adults may have greater self-compassion than young adults as a result of greater life experience in dealing with stressful events and an ability to interpret stressors differently.

A second important consideration between the present study and previous self-compassion research is the use of clinical samples. Self-compassion is theorized to operate in the presence of suffering. Therefore, self-compassion may be more strongly related to depressive symptoms in clinical samples due to the presence of more consistent and severe stressors, which are situations in which self-compassion and self-criticism are likely to occur. Raes (2011) sampled non-clinical young-adults and found that self-compassion explained a small amount of variation in reduced depressive symptoms over the course of five months with an effect size of $d = .29$. In contrast, studies of clinical populations find that the relation between self-compassion and psychopathology is large in magnitude ($d = 1.28$; MacBeth & Gumley, 2012). The current study only measured self-compassion during the initial assessment, but future studies should test whether explanatory and protective effects of self-compassion on the relation between stress and

depressive symptoms differ among clinical and non-clinical populations. This work would help to inform self-compassion interventions with clinical (Gilbert & Proctor, 2006) and non-clinical samples (Neff & Germer, 2013).

Given that self-compassion did not mediate the strain-depressive symptoms relation, it is interesting that hope, another positive psychological construct, did mediate this relation. The different indirect effects of hope and self-compassion suggest they may involve different processes or are relevant under different circumstances and populations.

There is extensive evidence showing strong links between hopelessness and depressive symptoms (Abramson et al., 1989), but fewer studies showing relations between self-compassion and depressive symptoms (Körner et al., 2015; Raes, 2010 & 2011; Zhang et al., 2018). Putting aside that research on self-compassion and depression is still emerging, there may be other reasons for this difference. Again, the interaction between type of stressor and developmental age may partially explain the divergent explanatory effects of self-compassion and hope in this sample. Social-cognitive theories of depression propose that stressors that threaten one's sense of self-worth and elicit shame can contribute to difficulties with emotional and motivational regulation, which can initiate depressive symptoms (Zhang et al., 2018). Moreover, developmental theorists suggest that stressors vary across the lifespan (Carstensen, 1992; Carstensen, Isaacowitz, & Charles, 1999). Thus, it is possible that different stressors threaten sense of self-worth as a result of developmental age, and hope may be more adaptive under certain stressors than self-compassion.

An attitude of hope involves the creation, acquisition, and reframing of valued goals when difficulties arise (Snyder, 2002). Hope has both a cognitive and behavioral

quality and is lowest when individuals feel they have limited personal control (Folkman, 2010). Self-compassion represents a form of hypo-egoic self-regulation in which the individual relinquishes control of their behavior and accepts flaws, inadequacies, mistakes, and events as part of the human experience (Leary & Guadagno, 2011). Those who are less self-compassionate may experience a hyper-egoic self, wherein stressors threaten one's self-concept. It appears that having hope involves a belief that one has control, but having self-compassion involves a relinquishing of control. Even though both are associated with well-being, their occurrence may depend on the extent to which individuals feel in control of the stressor or whether the stressor threatens their self-concept. In this study, reductions in hope mediated the relation between family strain and depressive symptoms, potentially signaling that people experienced a loss of control. Self-compassion may not have operated the same way because family strain did not threaten their self-concept. These hypotheses are purely speculative, but future studies should identify under what circumstances hope and self-compassion are present or whether they work in concert to influence the relation between stress and mental health.

Limitations and Strengths

Several methodological limitations should be kept in mind when interpreting these results. One major limitation of this study is the use of an incomplete measure of the Self-Compassion Scale. The absence of items from the self-critical dimension (i.e., self-judgment, isolation, over identification) make it difficult to generalize these results to other studies, which include items from all subscales and find that the self-critical items are primary drivers of depressive symptoms. A second limitation is that the mediators were measured very close in time to the predictors, which limits causal inference of the

findings. Nevertheless, these results are useful for investigating this theoretical model. A third limitation involves the use of a relatively healthy sample of middle-aged adults, resulting in a restricted range of levels of inflammation. Although the use of a large community-dwelling sample increases the generalizability of results, future studies examining physiological factors should sample populations that include more individuals with risk factors for health problems, including elevated inflammation. Finally, this study focused on a resilience factor, self-compassion, and did not address how vulnerability factors influence the relation between family strain and depressive symptoms. There is extensive research showing that risk factors, such as lack of perceived social support and financial stress moderate the social strain-depressive symptoms relation (Billings, Cronkite, & Moos, 1983; Teo, Choi, & Valenstein, 2013). A more comprehensive model should include both risk and protective factors.

This study also had a number of notable strengths. The study assessed a large, and diverse sample of participants within communities, suggesting these findings may generalize to similar community samples of middle-aged adults. Secondly, this study included a wide range of ages within middle-adulthood. Middle-adulthood represents an important developmental period that has not been a focus of study in the self-compassion literature (Bratt & Fagerström, 2019). A focus on healthy, middle-aged adults is needed because they face many stressors through their roles as caretakers, experiencing deaths in the family, and disturbances in their own lives, as well as the lives of their parents and children. This study also included a longitudinal design, allowing the ability to track depressive symptoms over time in a non-clinical sample. Other strengths of this study

include the use of structural equation modeling, which eliminated error variance in the models as well as the inclusion of self-report and physiological measures.

Future Directions

The findings of the current study point to several potential avenues for future work that can help shed light on when and how self-compassion may promote resilience to stress. First, evaluation of self-compassion related processes in the context of more severe stressors that more directly provoke self-blame and criticism may prove fruitful based on theory suggesting that self-compassion is evident in the presence of suffering. Second, future studies should take into account the contextual relevance of self-compassion across development. More investigation of the developmental trajectories of self-compassion, especially in adulthood, would be valuable for the implementation of self-compassion interventions in this aging population. This study did not examine age differences within middle adulthood, but there are likely to be differences between adults in their early forties (young middle-adulthood) and mid-sixties (older middle-adulthood). Adults in the early stages of middle-adulthood are likely to be balancing stressors associated with work, childcare, and family. Those in the later stages of middle-adulthood are likely dealing with issues of retirement, the launching of their children, and aging parents. These stressors may evoke different emotional needs and self-compassionate responding.

This study found that hope mediated the relation between family strain and depressive symptoms, suggesting that instilling hope in this population may protect individuals experiencing stress from subsequent depressive symptoms. There is inconsistent evidence of hope interventions decreasing psychological distress (Weis &

Speridakos, 2011), but other evidence-based positive psychology interventions (i.e., mindfulness, gratitude) may indirectly instill hope and psychological well-being. Understanding how self-compassion and hope interact with one another could enrich positive psychology interventions.

Conclusion

In summary, the results of this study suggest that the relation between family strain and depressive symptoms is not moderated by self-compassion. In addition, the results expand theories of hopelessness depression to a non-clinical sample of middle-aged adults, suggesting that hope may be an important process by which family strain impacts depressive symptoms.

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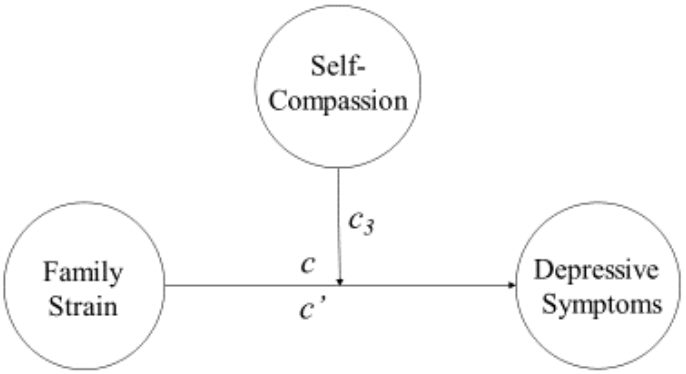

Figure 1. Model 1: Conceptual SEM model of the conditional effect of family strain on depressive symptoms for those high, average, and low in self-compassion.

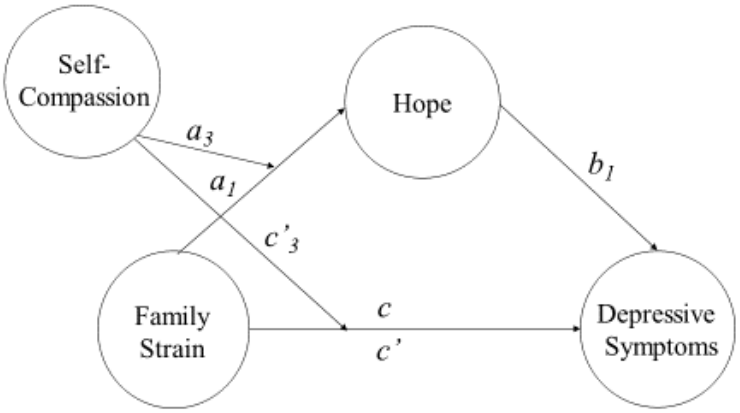

Figure 2. Model 2: Conceptual SEM model of the first order conditional indirect effect of family strain on depressive symptoms through hope for those high, average, and low in self-compassion.

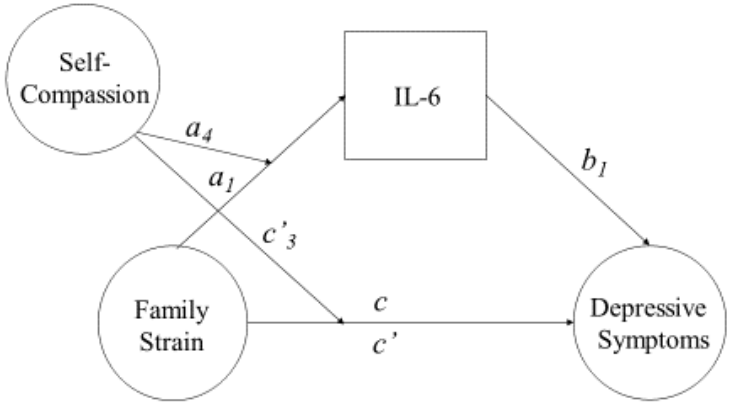

Figure 3. Model 3: Conceptual SEM model of the first order conditional indirect effect of family strain on depressive symptoms through IL-6 for those high, average, and low in self-compassion.

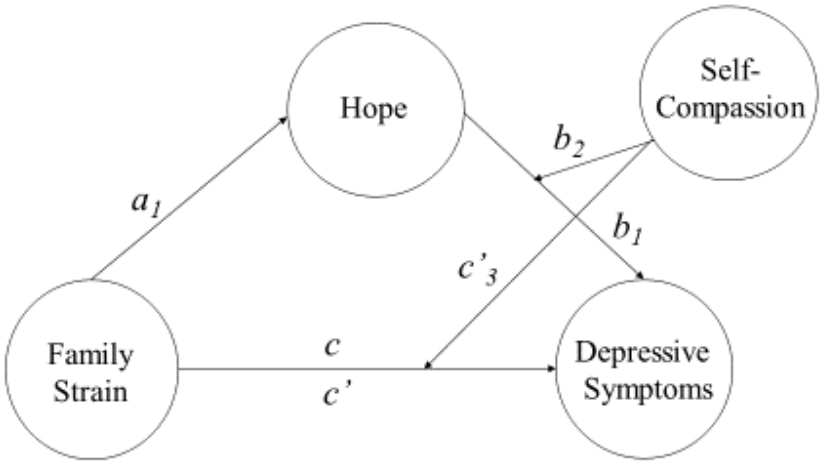

Figure 4. Model 4: Conceptual SEM model of the second order conditional indirect effect of family strain on depressive symptoms through hope for those high, average, and low in self-compassion.

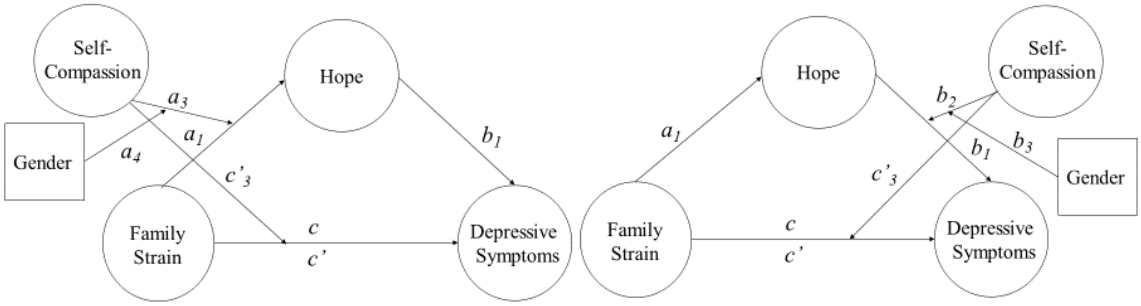

Figure 5. Model 5 & 6: Conceptual SEM model of the three-way interaction of family strain on depressive symptoms through hope as a function of self-compassion and gender.

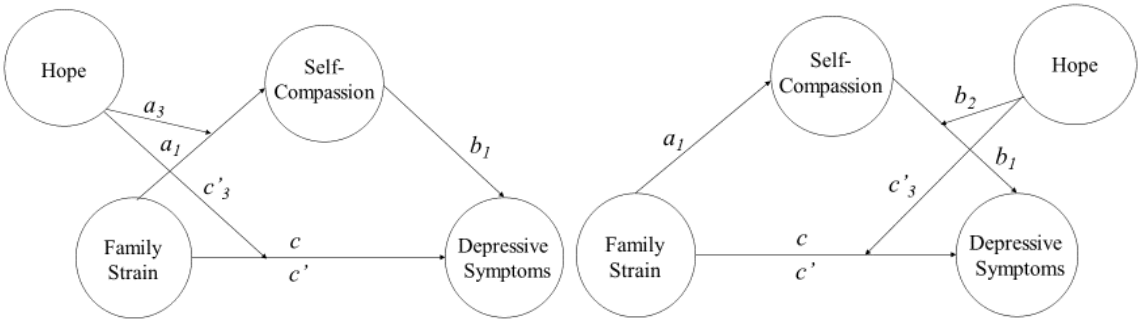

Figure 6. Model 7 & 8: Conceptual SEM model of the first and second order conditional indirect effect of family strain on depressive symptoms through self-compassion for those high, average, and low in hope.

Table 1.
Participant Demographics

Variable	**%***	***M(SD)***
Gender		
Male	45.4	
Female	54.6	
Race		
Non-Hispanic White	68.4	
Hispanic	23.6	
Black/African American	2.4	
Asian	1.4	
American Indian/Alaska Native	0.8	
Age		53.51 (7.25)
Initial Depressive Symptoms		1.79 (0.88)
Medication		
Taking antidepressants	19.6	
Not taking antidepressants	80.4	
Time to Follow-up		19.94 (11.15)

*Percentages may not equal 100% due to missing data

Table 2.
Means, Standard Deviations, and Intercorrelations of Study Variables (N = 762)

Variable	**Range**	***M(SD)***	*2*	*3*	*4*	*5*	*6*
1. Family Strain	1-4	2.20 (.67)	-.25**	-.26**	.08*	.33**	.25*
2. Self-Compassion	1-5	3.61 (.76)	-	.50**	-.01	-.30**	-.25**
3. Hope	1-8	5.86 (1.1)		-	-.16**	-.37**	-.38**
4. IL-6†	.22-72.63	.512 (.73)			-	.10*	.09*
5. Initial Depressive Symptoms	1-6	1.78 (.88)				-	.55**
6. Follow-Up Depressive Symptoms†	1-6	1.75 (.88)					-

*$p < .05$; ** $p < .01$ (two-tailed)
Note. † IL-6 ($N = 610$); Depressive Symptoms ($N = 524$).

Table 3.
Factor loadings for the Family Strain one-factor CFA solution

Family Strain Items	Factor 1
How often do they provoke feelings of conflict or anger?[a]	.798
How often do they make you feel tense?[b]	.797
How often are they critical of your behavior?[a]	.714
How often do they use you or take advantage of you?[a]	.697
How often do they break a promise of help, let you down, or neglect you?[a]	.666
How often do members of your family make too many demands on you?[b]	.582

Note. [a]Negative Social Ties, [b]Family Strain Subscale from MIDUS

Table 4.
Goodness-of-fit indices for each latent factor

Factor	χ^2	**RMSEA**	**CFI**	**SRMR**
Family Strain	(9) 169.173; $p < .05$	.153	.921	.046
Self-compassion	(27) 605.537; $p < .05$	.168	.843	.066
Hope	(135) 1382.980; $p < .05$	.110	.795	.065
Depressive Symptoms	(14) 95.566; $p < .05$	.105	.964	.031

Table 5.
Regression Models predicting Depressive Symptoms, Controlling for Initial Depressive Symptoms, Gender, Age, Antidepressant Medication ($N = 762$)

Model 1: Predictors include Family Strain, Self-Compassion, and Family Strain X Self-Compassion

Model 2: Testing Indirect Paths from Family Strain X Self-Compassion to Hope to Depressive Symptoms

Model 3: Testing Indirect Paths from Family Strain X Self-Compassion to IL-6† to Depressive Symptoms

Model 4: Testing Indirect Paths from Family Strain to Hope X Self-Compassion to Depressive Symptoms

Model	***b***	***(SE)***	**95% *CI***
Model 1			
Family Strain	.057	.07	-.073, .186
Self-Compassion	-.069	.04	-.147, .008
Family Strain X Self-Compassion	-.128	.09	-.297, -.044
Model 2			
Total Effect (*c*)	.056	.07	-.072, .184
Direct Effect (*c'*)	.029	.07	-.097, .157
Indirect Effect (*ab*) – High Self-Compassion	.041*	.02	.013, .091
Indirect Effect (*ab*) – Average Self-Compassion	.026*	.01	.006, .061
Indirect Effect (*ab*) – Low Self-compassion	.011	.02	-.018, .047
Index of Moderated Mediation (a_3b_1)	.020	.02	-.004, .060
Model 3			
Total Effect (*c*)	.058	.07	-.073, .187
Direct Effect (*c'*)	.056	.07	-.074, .186
Indirect Effect (*ab*) – High Self-Compassion	.000	.00	-.005, .014
Indirect Effect (*ab*) – Average Self-Compassion	.001	.01	-.005, .015
Indirect Effect (*ab*) – Low Self-Compassion	.002	.01	-.009, .020
Index of Moderated Mediation (a_4b_2)	-.001	.00	-.016, .005
Model 4			
Total Effect (*c*)	.080	.07	.171, .210
Direct Effect (*c'*)	.034	.07	.055, .161
Indirect Effect (*ab*) – High Self-Compassion	.025	.02	-.002, .071
Indirect Effect (*ab*) – Average Self-Compassion	.045**	.02	.019, .089
Indirect Effect (*ab*) – Low Self-compassion	.066**	.02	.030, .122
Index of Moderated Mediation (a_1b_2)	-.028	.01	-.064, -.007

† Log-transformed Observed Variable; *$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed); High Self-Compassion (1 SD above mean); Low Self-Compassion (1 SD below mean).

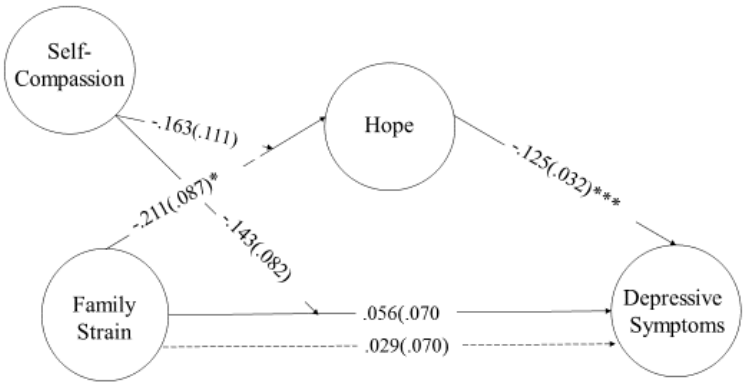

Figure 7. Model 2: Moderated Mediation Model Depicting the First Order Conditional Indirect Effect of Hope in the Relation between Family Strain and Depressive Symptoms as a Function of Self-Compassion (*p < .05, **p < .01, ***p < .001; two-tailed; dashed line depicts direct effect).

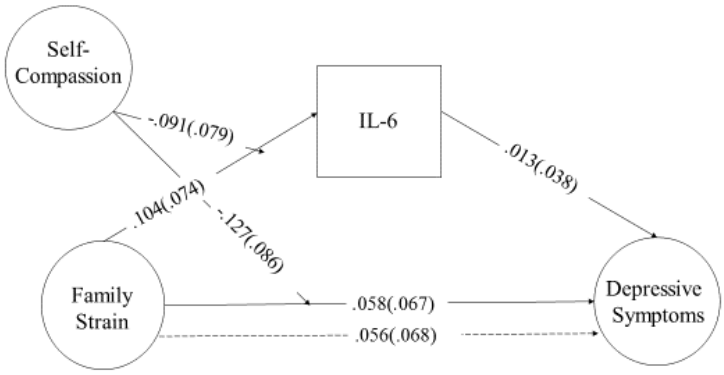

Figure 8. Model 3: Moderated Mediation Model Depicting the First Order Conditional Indirect Effect of IL-6 in the Relation between Family Strain and Depressive Symptoms as a Function of Self-Compassion (*p < .05, **p < .01, ***p < .001; two-tailed; dashed line depicts direct effect).

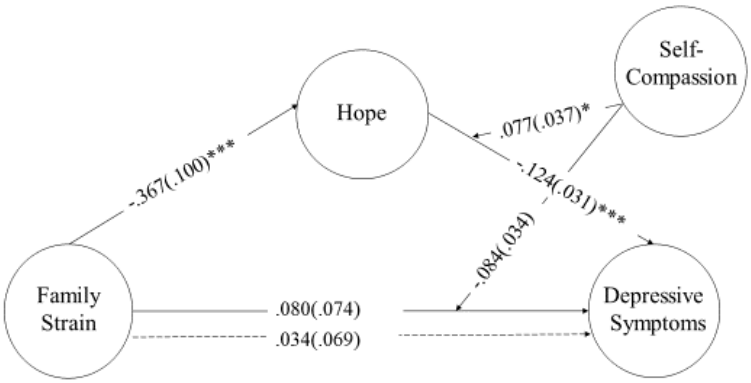

Figure 9. Model 4: Moderated Mediation Model Depicting the Second Order Conditional Indirect Effect of Hope in the Relation between Family Strain and Depressive Symptoms as a Function of Self-Compassion (*p < .05, **p < .01, ***p < .001; two-tailed; dashed line depicts direct effect).

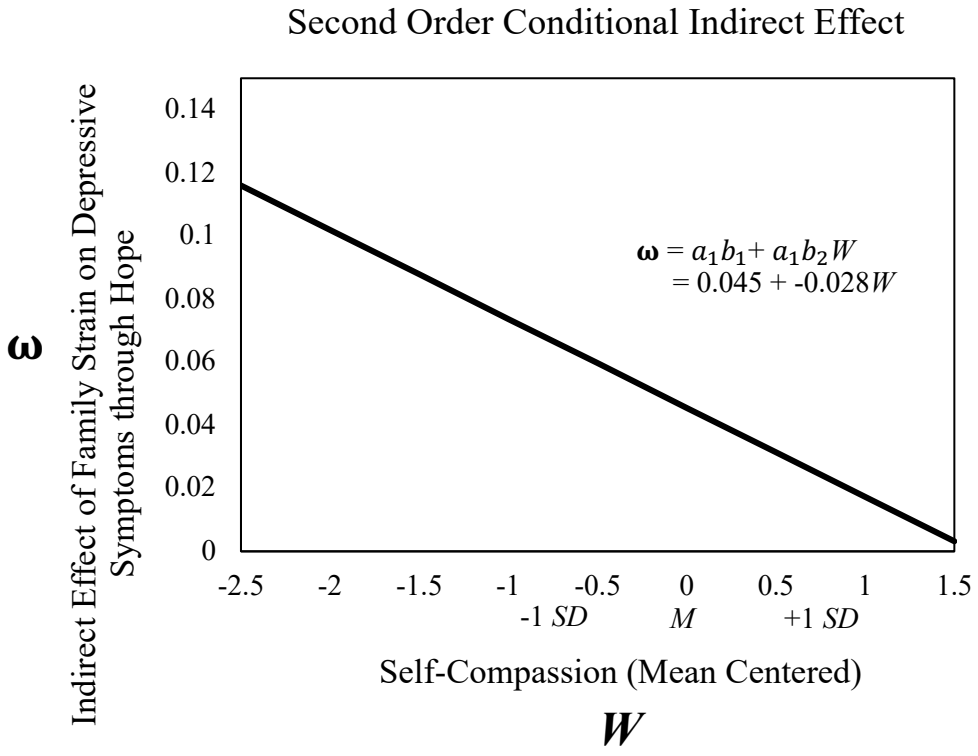

Figure 10. Graph displays the second order (*b* path) conditional indirect effect of hope on the family strain-depressive symptoms relation across values of self-compassion controlling for gender, age, initial depressive symptoms, and anti-depressant medication. The slope of the line is the weight in the function linking the indirect effect to self-compassion. Lower values of the indirect effect reflect weaker mediation effects.

Table 6.

Regression Models predicting Depressive Symptoms, Controlling for Initial Depressive Symptoms, Age, Antidepressant Medication

Model 5: Testing Indirect Paths from Family Strain X Self-Compassion X Gender to Hope to Depressive Symptoms (N = 522)

Model 6: Testing Indirect Paths from Family Strain to Hope X Self-Compassion X Gender to Depressive Symptoms (N = 522)

Model	***b***	***(SE)***	**95% *CI***
Model 5			
Direct Effect (*c'*)	.045	.06	-.072, .161
Conditional Moderated Mediation – Self-Compassion	-.051	.24	-.517, .414
Female (0)	.010	.02	-.031, .067
Male (1)	.017	.02	-.020, .073
Indirect Effect (*ab*) – High Self-Compassion	-.013	.04	-.094, .049
Female (0)	.045*	.03	.000, .123
Male (1)	.032	.02	-.006, .092
Indirect Effect (*ab*) – Average Self-Compassion	-.018	.03	-.079, .026
Female (0)	.038*	.02	.004, .091
Male (1)	.019	.02	-.012, .068
Indirect Effect (*ab*) – Low Self-Compassion	-.023	.04	-.104, .045
Female (0)	.030	.03	-.013, .087
Male (1)	.007	.03	-.047, .062
Index of Mediated Moderation Moderation ($a_3a_4b_1$)	.006	.03	-.059, .076
Model 6			
Direct Effect (*c'*)	.203**	.07	.074, .331
Conditional Moderated Mediation – Self-Compassion	.024	.06	-.099, .147
Female (0)	-.057	.04	-.132, .006
Male (1)	-.073*	.04	-.165, -.004
Indirect Effect (*ab*) – High Self-Compassion	-.013	.07	-.149, .118
Female (0)	.083*	.04	.012, .182
Male (1)	.069	.06	-.031, .189
Indirect Effect (*ab*) – Average Self-Compassion	-.002	.05	-.096, .099
Female (0)	.125*	.04	.060, .216
Male (1)	.124*	.04	.056, .226
Indirect Effect (*ab*) – Low Self-Compassion	.014	.06	-.096, .131
Female (0)	.168*	.05	.083, .284
Male (1)	.178*	.05	.099, .295
Index of Mediated Moderation Moderation ($a_1b_2b_3$)	-.016	.05	-.119, .081

*$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed); High Self-Compassion (1 SD above mean); Low Self-Compassion (1 SD below mean).

Table 7.

Regression Models predicting Depressive Symptoms, Controlling for Initial Depressive Symptoms, Gender, Age, Antidepressant Medication (N = 762)

Model 7: Testing Indirect Paths from Family Strain X Hope to Self-Compassion to Depressive Symptoms

Model 8: Testing Indirect Paths from Family Strain to Self-Compassion X Hope to Depressive Symptoms

Model	***b***	***(SE)***	**95% *CI***
Model 7			
Total Effect (*c*)	.016	.06	-.105, .139
Direct Effect (*c'*)	.016	.06	-.106, .139
Indirect Effect (*ab*) – High Hope	.000	.01	-.027, .022
Indirect Effect (*ab*) – Average Hope	.000	.01	-.017, .015
Indirect Effect (*ab*) – Low Hope	.000	.00	-.011, .009
Index of Moderated Mediation (a_3b_1)	.000	.00	-.009, .007
Model 8			
Total Effect (*c*)	.032	.07	-.093, .161
Direct Effect (*c'*)	.034	.07	-.091, .163
Indirect Effect (*ab*) – High Hope	-.028*	.01	-.063, -.007
Indirect Effect (*ab*) – Average Hope	-.002	.01	-.027, .020
Indirect Effect (*ab*) – Low Hope	.024	.02	-.008, .067
Index of Moderated Mediation (a_1b_2)	-.022*	.01	-.047, -.006

*$p < .05$, **$p < .01$, ***$p < .001$ (two-tailed); High Hope (1 SD above mean); Low Hope (1 SD below mean).

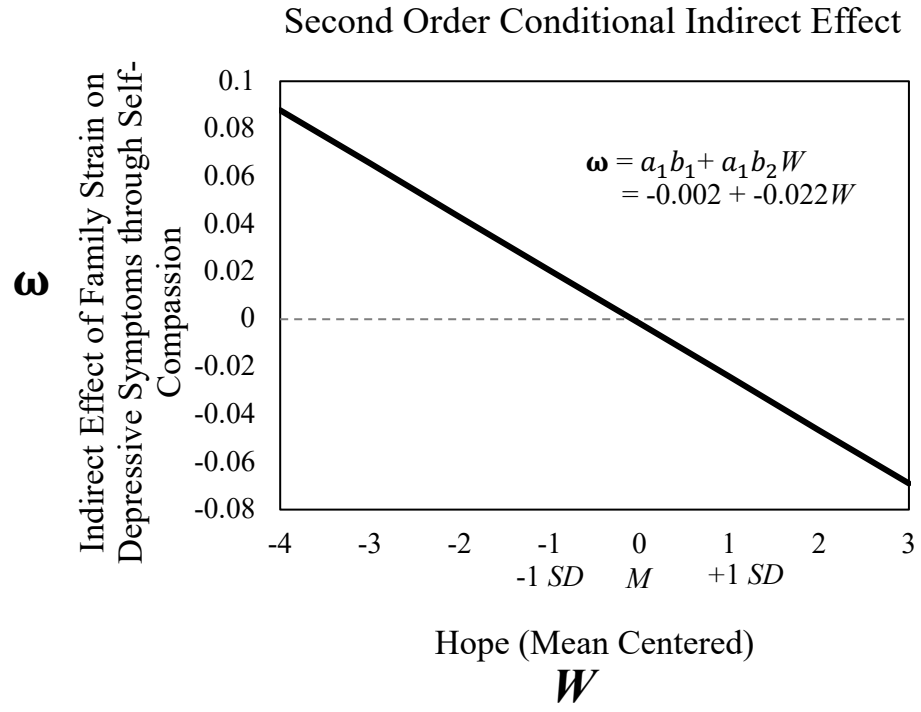

Figure 11. Graph displays the second order (*b* path) conditional indirect effect of self-compassion on the relation between family strain and depressive symptoms across values of hope controlling for gender, age, initial depressive symptoms, and anti-depressant medication. The slope of the line is the weight in the function linking the indirect effect to hope. Y-axis displays the strength of the indirect effect.

APPENDIX A

FAMILY STRAIN SCALE

Appendix A

Family Strain

Please circle the appropriate number for each question about your family.

Answer how often for each of these questions.	Never	Rarely	Sometimes	Often
a. Not including your spouse or partner, how often do members of your family make too many demands on you?	1	2	3	4
b. How often do they make you feel tense?	1	2	3	4
c. How often are they critical of your behavior?	1	2	3	4
d. How often do they use you or take advantage of you?	1	2	3	4
e. How often do they break a promise of help, let you down, or neglect you?	1	2	3	4
f. How often do they provoke feelings of conflict and anger?	1	2	3	4

APPENDIX B

SELF-COMPASSION SCALE

Appendix B

Self-Compassion

How often you do the following things?

		Almost never				Almost always
a.	I try to be loving towards myself when I'm feeling emotional pain.	1	2	3	4	5
b.	When something upsets me I try to keep my emotions in balance.	1	2	3	4	5
c.	When I'm going through a very hard time, I give myself the caring and tenderness I need.	1	2	3	4	5
d.	When something painful happens I try to take a balanced view of the situation.	1	2	3	4	5
e.	When I fail at something important to me I try to keep things in perspective.	1	2	3	4	5
f.	I'm kind to myself when I'm experiencing suffering.	1	2	3	4	5
g.	When I'm feeling down I try to approach my feelings with curiosity and openness.	1	2	3	4	5
h.	I'm tolerant of my own flaws and inadequacies.	1	2	3	4	5
i.	I try to be understanding and patient towards those aspects of my personality I don't like.	1	2	3	4	5

APPENDIX C

HOPE SCALE

Appendix C

Hope

How true or false is each statement about you, using the scale in the box below?

Definitely False	Mostly False	Somewhat False	Slightly False	Slightly True	Somewhat True	Mostly True	Definitely True
1	2	3	4	5	6	7	8

	False							True
a. I have trouble getting what I want in life.	1	2	3	4	5	6	7	8
b. I have found that I can overcome challenges.	1	2	3	4	5	6	7	8
c. I clearly define the goals that I pursue.	1	2	3	4	5	6	7	8
d. It is difficult to find ways to get what I want.	1	2	3	4	5	6	7	8
e. I can think of many ways to get out of a jam.	1	2	3	4	5	6	7	8
f. I have many goals that I am pursuing.	1	2	3	4	5	6	7	8
g. I prefer easy goals over hard goals.	1	2	3	4	5	6	7	8
h. I have what it takes to get the job done.	1	2	3	4	5	6	7	8
i. I have difficulty finding ways to solve problems.	1	2	3	4	5	6	7	8
j. I do not have very many goals.	1	2	3	4	5	6	7	8
k. I give up easily.	1	2	3	4	5	6	7	8
l. I'm not good at coming up with solutions.	1	2	3	4	5	6	7	8
m. I'm good at coming up with new ways to solve problems.	1	2	3	4	5	6	7	8
n. I'm successful at getting what I want.	1	2	3	4	5	6	7	8
o. I create alternate plans when blocked.	1	2	3	4	5	6	7	8

p.	I do not try hard enough to overcome challenges.	1	2	3	4	5	6	7	8
q.	I go after goals that are difficult and challenging.	1	2	3	4	5	6	7	8
r.	I do not care about the goals I am pursuing.	1	2	3	4	5	6	7	8

APPENDIX D

DEPRESSIVE SYMPTOMS SCALE

Appendix D

Depressive Symptoms

These next questions are about how you feel and how things have been with you during the past 4 weeks.

For each question, please give the one answer that comes closest to the way you have been feeling.

How much time during the past 4 weeks.

	All of the time	Most of the time	A good bit of the time	Some of the time	A little of the time	None of the time
Did you feel that you had nothing to look forward to?	☐	☐	☐	☐	☐	☐
Have you felt like crying?	☐	☐	☐	☐	☐	☐
Did you feel that nothing turned out for you the way you wanted it to?	☐	☐	☐	☐	☐	☐
Have you felt lonely?	☐	☐	☐	☐	☐	☐
Have you been moody and brooded about things?	☐	☐	☐	☐	☐	☐
Have you been in low or very low spirits?	☐	☐	☐	☐	☐	☐
Did you feel depressed?	☐	☐	☐	☐	☐	☐