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How the Use of Redemption Versus Contamination Sequences in the Telling of Life Stories Is Associated with Health Related Outcomes in Midlife Adults

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Abstract

There is a long history in psychological science of studying the negative sequelae that follow exposure to traumatic or other adverse life events. A large body of evidence has accumulated showing that individuals who have experienced major adversity are at higher risk for both mental and physical illness. However, while it is certainly true that some individuals experience these deleterious outcomes following adversity, the majority of individuals appear to be resilient to adversity. Moreover, some people even demonstrate personal growth following the experience. These observations have given rise to an interest in understanding how people make meaning out of threatening experiences, as well as the mechanisms through which people display resilience and even growth following adversity.

Relevant to this, a mounting body of research coming out of personality and narrative psychology has argued that a person's identity is formed by developing an autobiographical life narrative that reconstructs past experiences, acknowledges the present, and projects into the future. This is called "narrative identity." These life stories are not objective recounts of past experiences; rather, they are insights into who individuals views themselves as now. As such, individuals have some ability to shape past adversities insofar as they are able to choose how they will ultimately narrate the experience and incorporate it into their own sense of identity.

There are two major types of scenes that come up in life stories that have importance to how one fares in the face of adversity. Some people develop stories of redemption, where negative experiences are transformed into something positive. Conversely, some people narrate stories of contamination, where positive experiences are subsequently ruined by something negative. The use of redemptive imagery in the life story is positively associated with indicators of psychological well-being, whereas the use of contamination sequences is negatively

associated with well-being. However, whether redemption and contamination narrations are associated with physical health remains unknown.

To address this, I report on data drawn from a larger longitudinal study of midlife American adults. At the baseline visit, participants underwent an extensive life story interview and completed various questionnaires. Five years later they underwent the same procedure. Within a year of this second visit, they filled out additional questionnaires related to mood, well-being, and health, and also had their blood drawn to assess cardiometabolic health indicators. For metabolic properties, height, weight, waist circumference, blood pressure, total cholesterol, and glycosylated hemoglobin (a method of assessing average plasma glucose levels over the past 3 months) were assessed at the time of the study visit, and these variables were used to index metabolic syndrome related components and risk. At that time, serum was also frozen to allow for future batch testing of inflammatory proteins and markers. From this serum, C-reactive protein (CRP) and lipoprotein-associated phospholipase A_2 (Lp-PLA_2) were quantified in all participants to assess markers of general systemic inflammation (CRP) as well vasculature specific inflammation (Lp-PLA_2). Additionally, a panel of inflammatory chemical messengers – called cytokines – were assayed as broader indicators of peripheral inflammatory activity. These cytokines were interleukin-6 (IL-6), interleukin-8 (IL-8), interleukin-10 (IL-10), interferon-γ (IFNγ), and tumor necrosis factor α (TNFα). Levels of the various inflammatory markers and cytokines (CRP, Lp-PLA_2, IL-6, IL-8, IL-10, IFNγ, and TNFα) were *z*-scored and summed to create a composite inflammatory variable.

This study has three overarching sets of hypotheses, as well as one set of exploratory hypotheses. First, the usage of redemptive sequences in individuals' life stories should be associated with better cardiometabolic health outcomes. This should be evidenced by better

subjective self-reported health, fewer components related to metabolic syndrome as well as lower metabolic risk, lower levels of CRP, less risk of having a CRP value falling in the "high cardiovascular risk" category, and lower levels of composite inflammation. Second, the usage of contamination sequences in individuals' life stories should be associated with poorer cardiometabolic health outcomes. This should be evidenced by worse subjective self-reported health, more components related to metabolic syndrome as well as higher metabolic risk, higher levels of CRP, more risk of having a CRP value falling in the "high cardiovascular risk" category, and higher levels of composite inflammation. Third, there should be an interaction between the presence of redemption sequences and the presence of contamination sequences in predicting the various cardiometabolic health outcomes. Specifically, individuals who report contamination sequences without also reporting redemption sequences should fare worse on the various outcomes than individuals who report both contamination and redemption sequences. Finally, I will explore whether redemption and contamination reported during FLSA Time 1 (approximately 5 to 6 years before the current study visit) versus redemption and contamination reported during FLSA Time 5 (approximately 6 to 12 months before the current study visit) are better or worse predictors of the differences in the various cardiometabolic health outcomes. I do not have any *a priori* expectations as to what these analyses should yield. As such, these analyses should be considered exploratory with the main purpose of guiding future research.

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List of Abbreviations

ATP III	=	National Cholesterol Education Panel/Adult Treatment Panel III
BDI-II	=	Beck Depression Inventory, 2nd Edition
BMI	=	body mass index
CAT	=	Cognitive Adaptation Theory
CI	=	confidence interval
cm	=	centimeters
CRP	=	C-reactive protein
dL	=	deciliters
FLSA	=	Foley Longitudinal Study of Adulthood
HDL	=	high density lipoprotein
HIV	=	human immunodeficiency virus
HSP	=	Healthy Stories Project
ICC	=	intraclass correlation coefficient
IFI	=	incremental fit index (Bollen's)
IFNγ	=	interferon-γ
IL-6	=	interleukin-6
IL-8	=	interleukin-8
IL-10	=	interleukin-10
L	=	liters
LDL	=	low density lipoprotein
Lp-PLA_2	=	lipoprotein-associated phospholipase A_2
M	=	mean
MFI	=	McDonald's Fit Index
mg	=	milligrams
mL	=	milliliters
mmHg	=	millimeters of mercury
ng	=	nanograms
OR	=	odds ratio
pg	=	picograms
RCF	=	relative centrifugal force
RMSEA	=	root mean-square error of approximation
SD	=	standard deviation
SES	=	socioeconomic status
TNFα	=	tumor necrosis factor α

Dedication

With love and gratitude to my parents, George M. Murphy, PhD, and Eileen M. Murphy, MA, my mother-in-law, B. Randy Goodman, JD, my "Sister Law," Kristen L. Brewer, JD, my aunt, Sr. Mary Ann MacRae, MB BCh BAO, MPH, and my wife, Sarah E. Victor, MA (and soon to be PhD). To borrow from the words of Sir Isaac Newton (1676), if I have accomplished anything, it is by standing on the shoulders of giants.

Finally, I dedicate not only my work, but also my future to the memory of my beloved big brother, Sean P. M. Murphy, JD. He passed away unexpectedly on October 16, 2014, at the age of 32, just two weeks after I proposed this dissertation project. He was a brilliant man of rare integrity and wit who constantly set the bar for me growing up and will forever remain my role model. While illness cut his life tragically short, I hope that my own life story will ultimately honor both his memory and his legacy.

Michael Murphy

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Introduction

The history of humanity is punctuated regularly by hardship and tragedy. Where the iniquities of the world come from and whether humanity is intrinsically good or evil has been debated by philosophers, theologians, and other commentators on the human condition throughout the centuries, and these fundamental questions will almost assuredly never yield universally satisfying answers. However, there are important related questions about the human condition that do lend themselves to empirical study. For example, what typically happens to people who have had bad things happen to them? How do they understand the "why" of what has happened? What do they do to try to adapt? Do they try to draw meaning out of the misery and misfortune? And relatedly, how do people differ in the ways they experience hardships? Are there certain ways of understanding suffering that may be more helpful than others? In this dissertation, I will review some of the ways that social scientists have tried to address these questions so far. Then, based on this work, I will report on a study that further explored these questions by examining how the myriad ways that people understand suffering were associated with physical health in midlife American adults.

Ruination versus Resilience in the Face of Adversity

Psychological science has historically been largely concerned with the ways that hardships can damage a person and lead to pathology (Bonanno, Papa, & O'Neill, 2001; Seligman & Csikszentmihalyi, 2000). Unsurprisingly, there are a whole host of documented negative psychological outcomes that people have experienced following adverse life events. Individuals who experience some form of trauma such as war, natural disasters, acts of terror, or rape are at higher risk for developing posttraumatic stress disorder (Brewin, Andrews, Rose, & Kirk, 1999; Kilpatrick & Acierno, 2003; Neria, Nandi, & Galea, 2008; Resnick, Kilpatrick,

Dansky, Saunders, & Best, 1993), substance abuse problems (De Bellis, 2002; Stewart, 1996), and depression and anxiety (Grant, Beck, Marques, Palyo, & Clapp, 2008). Losing a loved one, whether through death or separation, increases an individual's risk for experiencing depression (Kendler, Hettema, Butera, Gardner, & Prescott, 2003; Prigerson et al., 1997). People who are victims of violent crimes are more likely to develop a pervasive anxiety disorder (Brewin et al., 1999; Kilpatrick & Acierno, 2003). More generally, undergoing any type of major negative life event increases a person's risk for subsequent psychopathology (Ensel & Lin, 1991; Kendler et al., 2003; Kessler, 1997). Furthermore, these sorts of psychiatric morbidities are all themselves risk factors for self-harm (Hawton, Saunders, Topiwala, & Haw, 2013) and suicide (Nock & Kessler, 2006). Additionally, there are major social and economic consequences, both at the individual and the societal levels, resulting from these assorted psychological illnesses (Kessler et al., 2011; Prince et al., 2007). These observations are made even more worrisome by the evidence that most individuals will experience some major negative event or traumatic episode at some point during their lives (Ozer, Best, Lipsey, & Weiss, 2003). Thus it is not without reasonable cause that psychologists have focused so much energy and attention on the range of harmful sequelae following major adversities.

Outside of the more traditional domains of psychological science, there is also an abundance of evidence linking life adversity to negative physical health processes and outcomes (Cohen, Janicki-Deverts, & Miller, 2007). Experimental work with animals and observational studies of humans have provided compelling evidence that psychologically threatening experiences and conditions are related to cardiometabolic lifestyle illnesses such as atherosclerosis (Barnett, Spence, Manuck, & Jennings, 1997), hypertension (Matthews et al., 2004), metabolic syndrome (Vitaliano et al., 2002), diabetes (Pickup, 2004), myocardial

infarction (Rosengren et al., 2004), stroke (May et al., 2002), and cardiovascular disease (Rozanski, Blumenthal, & Kaplan, 1999). According to the Center for Disease Control and Prevention in the United States (Heron, 2013), these diseases accounted for around a third of the annual mortality rate in the country in 2010 (with all of the cancers combined accounting for the next largest proportion of deaths), and a seminal overview of the topic referred to obesity, diabetes, and cardiovascular disease as "the greatest current threat to global human health and welfare" (Hotamisligil, 2006). Thus understanding what factors play a role in the pathogenesis and progression of these diseases is of paramount interest to public health officials.

At the same time, it is a timeless human belief that suffering and adversity may be overcome, and even lead to personal growth, redemption, and triumph. This theme has long been the topic of writers, theologians, and philosophers, with religious texts, philosophical discourse, and libraries full of novels telling stories of the human struggle and ability to triumph over suffering and evil (Tedeschi & Calhoun, 1995). However, the idea that many people show resilience and possibly even growth in the face of adversity is a relatively new topic in psychological science. While it is certainly true that some people experience significant hardship and distress following major negative life events, the vast majority of people do not (Bonanno, 2004). Relatedly, while psychologically distressing experiences increase disease risk, the majority of people who experience major adversity do not get sick (Cohen & Hamrick, 2003). Indeed, numerous lines of evidence have now documented that people can show astonishing resilience and growth when faced with a multitude of threatening events (Tedeschi & Calhoun, 2004; Updegraff & Taylor, 2000). For example, having cancer (Carver & Antoni, 2004; Park, Edmondson, Fenster, & Blank, 2008; Taylor, Lichtman, & Wood, 1984), being positive for human immunodeficiency virus (Taylor, Kemeny, Reed, Bower, & Gruenewald, 2000),

managing heart disease (Affleck, Tennen, Croog, & Levine, 1987; Sheikh, 2004), being the victim of a violent crime or domestic abuse (Cobb, Tedeschi, Calhoun, & Cann, 2006; Tedeschi, 1999), experiencing combat (Elder & Clipp, 1989), being a refugee (Berger & Weiss, 2003), caring for a disabled child (King, Scollon, Ramsey, & Williams, 2000), and losing a child or other loved one (Bogensperger & Lueger-Schuster, 2014; Bonanno et al., 2001) have all been shown to have the ability to bring about some type of growth-related transformation in people's lives.

This is not to say that for individuals who report growth, negative and traumatic situations are not still highly aversive or distressing, Likewise, it is not the case that individuals who report growth also report happiness regarding their suffering. Rather, in these growth scenarios, from the ashes of the bad, people were able to find something to hold onto that resulted in feelings of having been enhanced in someway. These enhancements often take the form of feeling more self-reliant, being more self-aware, disclosing more to others and feeling more emotional expressiveness in relationships, and having a changed overall philosophy about life (Tedeschi & Calhoun, 1995). Taken together, such observations have resulted in a flourishing interest in the concepts of meaning making (Park, 2010) and posttraumatic growth (Tedeschi & Calhoun, 2004), as well as the development of mechanistic theories about how people adapt to, overcome, and grow from distressing situations, and how these adaptions may benefit both mental and physical health and well-being (e.g., McAdams, 2006b; Taylor, 1983; Taylor & Broffman, 2011).

What accounts for these divergences in outcomes following adversity? Major experiences of life adversity are, at their core, highly threatening to the person experiencing them. They disrupt an individual's understanding of how the world works, shattering closely held beliefs

about the way things should be (Park, 2010; Tedeschi & Calhoun, 2004). According to Joseph and Linley (2005), following such a disruption, an individual is left with three possible trajectories. (1) The individual can assimilate the negative experience into his or her previously held worldview in an attempt to return to baseline psychological status. Or, instead, (2) the negative sequelae following a distressing experience may prove too much to adjust to, and the individual may subsequently develop a new worldview that is more negative and distressing than previously held. Such a change in understanding the world may lead to pathology. Alternatively, (3) the individual may be able to accommodate and integrate the experience into a new worldview that draws personal meaning from the adversity in a life affirming way. This is referred to as growth. Which arc a person follows is influenced by a host of psychosocial, contextual, and genetic factors.

One model that has been particularly influential in the understanding of what trajectory an individual will track following adversity is Cognitive Adaptation Theory (CAT; Taylor, 1983). Under this theory, psychological adjustment to threatening life experiences, along with subsequent benefits to both mental and physical health, is a function of three processes: meaning making, sense of mastery, and self-enhancement. This model holds that individuals who successfully adapt to and grow from negative experiences show evidence of overly optimistic reality distortions surrounding their experience. That is, individuals maintain a set of positive illusions about the negative experience that tend toward over-exaggerating their sense of mastery and self-efficacy, enhancing their self-esteem, and providing for an overly optimistic outlook of the future (Taylor & Brown, 1988; Taylor, Kemeny, Reed, & Aspinwall, 1991; Taylor et al., 2000). These positive illusions have been shown to not only have benefits for psychological well-being, but may also be protective against deleterious disease processes. For example,

numerous studies have documented that among individuals who were positive for human immunodeficiency virus (HIV), those who were able to maintain positive illusions about their illness prospectively experienced fewer HIV-related symptoms and complications, even after adjusting for a variety of potential social, demographic, and health-related confounders (for a review, see Taylor et al., 2000). Furthermore, in a longitudinal study of adult men and women who had undergone an angioplasty and were being treated for coronary artery disease, patients who were better able to maintain a positive outlook on life, feel more in control of their lives, and have more general positive perceptions about themselves were less likely to experience a new cardiac event during a four year post-surgery follow-up period (Helgeson, 2003).

It is important to note that these adaptive sorts of positive illusions are not completely out of touch with reality such that they belie immutable facts. Rather, they are chosen (whether consciously or not) ways of interpreting information about a threatening situation that focuses on aspects that can be subjectively reconstructed in a positive light, rather than focusing on the alternative (Taylor, 1983). The ability to successfully develop such positive illusions in the face of aversive life experiences is a function of a variety of individual psychological differences. In general, being more cognitively flexible (Bonanno, Papa, Lalande, Westphal, & Coifman, 2004), higher in dispositional optimism, religiousness, and use of positive reappraisal (Prati & Pietrantoni, 2009), hardy (Kobasa, Maddi, & Kahn, 1982), and higher in agreeableness, extraversion, openness to experiences, and conscientious, and lower in neuroticism (Linley & Joseph, 2004) are all predictive of being better able to adapt and grow following adversity.

While born out of different traditions and theoretical frameworks, Cognitive Adaptation Theory shares some interesting parallels to theory developed in personality psychology about how individuals develop their sense of identity from constructing stories about their lives

(McAdams, 1993). In this framework, a person's identity is a function of the stories he or she chooses to create and tell about his or her life. These stories present a reconstruction of the past, an acknowledgement of the present, and a projection into the future. When experiencing an adverse life event, a person has choices in how he or she goes about constructing a story that explains the event and its impact on the person's life. As discussed by Pals and McAdams (2004), one option is to attempt to minimize the effect of the event on the self by distancing the self from what happened and avoiding integrating it into the person's sense of identity. This option is unlikely to produce adaptation and growth following a major adverse life experience. At best, it may allow the person to return to his or her baseline. At worst, it can result in intrusive negative emotions, general distress, and even psychiatric pathology. Another option that individuals have following adversity is to spend time looking for meaning in the experience by embracing the negativity of what happened and describing the distress and emotional pain that followed. But the story cannot end there. As detailed by Pals (2006) and Pals and McAdams (2004), the individual must then construct a narrative as to how the negative experience provided a turning point for something new to take place, for positive transformation resulting in feeling enhanced in some way. Such a narration gives the individual the ability to shape the meaning that he or she derived from the negative experience, even after the negative experience has occurred.

Using narration to make meaning out of past negative experiences draws on a concept referred to as "interpretive control" (McAdams & Bowman, 2001). Interpretive control is a secondary control (versus primary control) mechanism that was originally born out of the locus of control literature and is defined as "the ability to interpret events so as to better understand and accept them" (p. 11; Rothbaum, Weisz, & Synder, 1982). Whereas primary control refers to

an individual's concrete belief (whether real or not) that he or she has the ability to *influence the outcome* of a given situation as it is being experienced, secondary control refers to an individual's ability to *influence the self* so as to better accommodate and adapt to an adverse situation that has typically already occurred or that is ongoing and chronic (Morling & Evered, 2006). Secondary control processes such as interpretive control are generally considered adaptive as they allow a person to utilize internal cognitive processes such as cognitive reappraisal, positive thinking, self-encouragement, and acceptance so as to learn how to control the self's understanding of an adverse experience, which can ultimately engender meaning making and positive growth (Morling & Evered, 2006; Taylor & Broffman, 2011). Furthermore, in discussing their lifespan theory of control, Heckhausen, Wrosch, and Shultz (2010) argue that secondary control processes can additionally help individuals minimize losses in primary control and increase current levels of primary control. In line with this, Poulin and Heckhausen (2007) found that secondary control processes were protective against motivational losses following the experience of major stressful life events, protecting individuals' beliefs in their ability to control the environment in a self-enhancing manner in the future. As such, secondary control processes such as interpretive control are separate but related constructs to the idea of primary control (which is often simply referred to as "control") as has been most typically conceptualized in the literature on life stress and health (Skinner, 1996).

Though primary control has more frequently been the topic of study concerning adverse life experiences and health related outcomes, Armor and Taylor (1998) note that in circumstances where a person is, for whatever reason, unable or unwilling to make use of primary control mechanisms, being able to later exert secondary control mechanisms such as interpretive control may offer a similar degree of protection insofar as it still provides tools for

eventually developing a sense of mastery over the adverse experience. Consistent with this notion, there is evidence that making use of secondary control processes is prospectively associated with better self-reported health as well as actual longevity in older adults (Chipperfield et al., 2012), improved quality of life and metabolic control among adolescents with Type I diabetes (Edgar & Skinner, 2003; Jaser & White, 2011), and fewer symptoms of anxiety, depression, and somatic complaints among patients with chronic pain (Compas, 2006; Thomsen et al., 2002). Additionally, secondary control strategies, including interpretive control, may even result in more improved health-related outcomes and better well-being compared to primary control strategies, at least among youth and older adults dealing with a chronic illness (Chipperfield et al., 2012; Compas, Jaser, Dunn, & Rodriguez, 2012). Furthermore, use of interpretive control specifically has also been shown to be related to greater sense of well-being among adults discussing negative life events (Lilgendahl & McAdams, 2011). More generally, being able to form life stories that describe how good rises up out of bad experiences has been linked to enhanced well-being, self-esteem, sense of coherence, and life satisfaction, as well as fewer depressive symptoms (McAdams, Reynolds, Lewis, Patten, & Bowman, 2001).

It is important to note that while theory about adaptations to negative life experiences derived from narrative psychology shares similarities with Cognitive Adaptation Theory, it was not developed to be in competition with CAT. Indeed, while the narrative approach draws on some similar themes, it examines them in light of a person's ongoing and ever-evolving life story and the construction of one's sense of identity. Nonetheless, there is reasonable conceptual overlap between research on making meaning out of negative experiences in narrative psychology and CAT. For example, spending time looking for meaning in a bad experience and allowing oneself to consider the distress and pain involved is akin to the process of meaning

making as described in CAT (e.g., Singer, 2004). Furthermore, using narrative to learn to understand how a bad experience was a turning point allowing for a positive transformation or redemption to take place out of the ashes of the adversity is also similar to how self-enhancement is conceptualized under CAT (e.g., Benish-Weisman et al., 2014). Finally, using narrative to exert interpretative control over an experience shares parallels with the description of how sense of mastery, a component of CAT, develops. Furthermore, interpretive control is similarly associated with improved psychological well-being, and may even act to further an individual's broader sense of mastery (Ryff & Essex, 1992). As such, while CAT was not developed with narrative psychological approaches in mind, it nonetheless offers some theoretical scaffolding for considering how the construction of life narratives may allow for positive growth following adversity, especially with regards to how individual differences in life narrative construction may be linked to physical health outcomes.

Taken together, the human tendency to create life stories that define an individual's identity provides a compelling framework for studying how people make sense out of adversity, and why some people whither while others flourish. However, to expand on this concept further, it is first necessary to discuss in more detail what the life story is, where it comes from, and how it provides insight into a person's identity. Indeed, as the next section discusses, how someone constructs his or her life story is an integral part of who that individual is as a whole person (McAdams, 1985).

The Person as Actor, Agent, and Author

As human beings, we each share numerous broad similarities with everyone else. Such deep-seated similarities have been forged over the eons by evolutionary processes within our species (Buss, 1995). Yet we are clearly not all the same. There are innumerable differences

between individuals in thoughts and behaviors. Such variations in how people think and behave have likely developed as adaptations to differential environmental demands experienced by diverse individuals in our evolutionary history (Buss, 1991; MacDonald, 1995). That is, neither our world nor our historical circumstances are homogenous; certain characteristic ways of thinking and behaving that provide an individual with an adaptive advantage in one environment may not be useful or may even be harmful in a different context (Nettle, 2006; Wolf, van Doorn, Leimar, & Weissing, 2007). Thus, who we are as individuals and what type of personality we develop are variations on an evolutionary template, situated within a specific culture and historical period (McAdams & Olson, 2009; McAdams & Pals, 2006).

Based on this perspective, a person's personality and how he or she is likely to react and behave in the world is a function of three interrelated concepts (Kluckhohn & Murray, 1953; McAdams & Pals, 2006). The first concept consists of the evolutionary template for humanity, or those broad characteristics that are expressed widely across our species, such as dietary preferences and the desire to find romantic/sexual partners. Second is the basic variations on this template, or the individual differences that exist within these broad personal characteristics, such as how outgoing and social or how careful and thoughtful someone is. Third is the specific features of people's lives beyond these broad characteristics – their hopes, dreams, goals, and projects – that drive them, instill them with individuality, and, ultimately, give rise to a sense of self-identity. These domains provide an overarching structure for conceptualizing where personality comes from. But what is personality, and how does it develop? Addressing these questions is central to understanding how we as humans differ and why we develop unique identities.

A modern, integrative framework for explaining the lifespan development and expression of human personality and identity that has engendered significant currency within psychological science is the actor-agent-author model (McAdams, 2013; McAdams & Cox, 2010; McAdams & Pals, 2006). This model posits that human personality is a function of three different psychological layers that develop at different points (and speeds) during a person's lifespan. The first layer, the actor, becomes discernable during infancy and involves the broad characteristics of the individual that tend to be relatively consistent across situations and over time. This layer deals with what people are generally like. Are they social or reserved, friendly or hostile, cautious or blithe? The second layer, the agent, starts to become manifest during childhood and encompasses the goals and motivations of the individual. This layer begins to capture who people are by addressing what matters to them. What sort of goals do they set? What projects are important to them? What are they striving toward in the future? The third and final layer, the author, begins to develop during adolescence. This layer is where an individual begins to construct a narrative of his or her life that provides a reconstruction of the past, an understanding of the present, and a projection of the imagined future. This layer further captures who people are and also how they believe they came to be that way. It is within this layer that an individual develops a "narrative identity" that provides a sense of continuity and purpose to life and begins to answer the classic existential question "who am I?" (McAdams & McLean, 2013; Singer, 2004).

It is important to note that these layers do not supplant each other, nor are they mutually exclusive. One does not cease to be an actor as one becomes an agent, nor does one cease to be an actor and an agent as one develops as an author. Rather, each layer represents an added advancement in the complexity of an individual's psychological makeup (McAdams & Cox,

2010). Furthermore, while all three layers change over time, they do not do so at the same rate, time, or complexity. Generally, the broad characteristics that describe the actor begin to stabilize during adolescence, though they do also continue to show patterns of change through the adult years (Costa & McCrae, 2006; Mroczek & Spiro, 2003; Roberts & Mroczek, 2008; Roberts, Walton, & Viechtbauer, 2006). Conversely, the goals and motivations of the agent, and the way the author narrates his or her life can change markedly over the lifespan (McAdams & Olson, 2009). The remaining portion of this section fills in more details about the levels of the actor-agent-author model.

Actor. The actor layer of personality is the most basic layer, and it refers to the characteristic ways individuals think about themselves and present themselves to the world (McAdams, 2013). Humans are a highly social species. In a seminal review on the topic, Baumeister and Leary (1995) concluded that the need to belong is one of the most fundamental human motivations. This dovetails with evolutionary theory that contends that our ancestors had to be able to rely heavily on one another for survival. From this perspective, being able to work with other people and being able to interpret behaviors of other people would have been highly adaptive in the evolutionary landscape (Buss, 2008). That is, if our evolutionary ancestors had not been able to determine at least to some degree the extent to which others tended to exhibit various socially informative features (e.g., reliability, cognitive flexibility, friendliness, anxiety, honesty, etc.), it would have been extremely challenging to form coherent and useful social groups, presumably making survival much more difficult. Along similar lines, to be able to participate in these important social groups, it would also be necessary to be able to understand one's own behavioral tendencies and to shape one's behaviors very early in life based on feedback from the social environment (McAdams, 2013). Consistent with this, evidence has

demonstrated that humans do indeed have the ability to identify how they characteristically tend to behave, to attend to the characteristic behaviors of others, and to adjust their own behaviors appropriately (Mehl, Gosling, & Pennebaker, 2006).

These characteristic behaviors that actors draw on and observe in others are captured by a taxonomy of traits. Traits are the relatively stable ways in which people, as actors, tend to behave on average, across situations (McCrae & Costa, 1997). The precursors to traits, called temperaments, begin to appear early in life, during childhood. Temperaments refer to individual differences in the ways children react and self-regulate in terms of mood, activity, and attention (Rothbart & Bates, 2006). Temperament is likely primarily rooted in genetic factors; however, it is also shaped to some degree by experiences and interactions with others during childhood (Shiner & DeYoung, 2013).

As youth make their way from childhood to adolescence, their temperaments form into a relatively stable trait structure (McAdams, 2013)[1]. This structure is commonly referred to as the Five Factor Model, or simply the Big Five (John, Naumann, & Soto, 2008; McCrae & John, 1992). The traits that are encompassed by the five factors are labeled agreeableness, conscientiousness, extraversion (sometimes called surgency), neuroticism (sometimes called emotional stability), and openness to experience (sometimes called intellect). Each of these traits has a bipolar structure, ranging from low to high. The expression of these traits appears across a wide age range and throughout numerous cultures, suggesting that they specify a near universal

[1] Historically, temperaments were relegated to the domain of childhood research and traits to the domain of adulthood research. However, whether temperaments and traits are truly distinct from each other is a matter of debate. The distinction may be more of a disciplinary one, and the two constructs may actually be referring to the same or similar underlying structures (Shiner & DeYoung, 2013).

description of the myriad characteristic ways that people present themselves to the world and are observed by others in the world (Caspi, Roberts, & Shiner, 2005; McCrae & Costa, 1997). Personality traits provide a broad, incomplete sketch of what a person is generally like (McAdams & Pals, 2006). That is, they offer basic insights into what kind of person someone generally is based on recognizable, easily described dimensions. However, they do not address who a person is, nor do they necessarily map well onto what a person will do in any specific given situation (Mischel, 2004). To be able to understand this, it is necessary to also focus on individual differences in what motivates people. Nonetheless, there is no question that traits on their own are important predictors of numerous life outcomes such as career achievement, relationship attainment and maintenance, health, and mortality (Chapman, Lyness, & Duberstein, 2007; Hogan & Ones, 1997; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007).

Agent. The agent layer of personality refers to the agency individuals exercise in their lives, or the personal goals and motives that drive people and inform their decision making and commitment to various life projects (McAdams, 2013). The idea of human agency is ancient and has been expressed in various ways by philosophers over the centuries (Wiggins, 1991). However, the actual term "agency" was first used by Bakan (1966), who defined it in terms of an individual's drive to protect, assert, and expand the self. As such, the agent takes conscious, purposeful actions to control and direct his or her life toward some desired outcome (Bandura, 2006). While the broad characteristics, or traits, that define the social actor remain relatively stable over the lifespan, context matters for the agent, and the types of goals one constructs can change dramatically across situations and time (Hooker & McAdams, 2003). Likewise, while the characteristics that define the social actor address what kind of person an individual generally is,

the goals and projects that the agent pursues begin to explain who a person is as an individual (McAdams & Pals, 2006).

As described by McAdams (2013), being an agent requires not only behaving in a goal directed manner (i.e., displaying agency), but also recognizing that one has goals. In this framework, while infants are quite capable of behaving in a goal directed manner when trying to, for example, get a caregiver's attention for food or comfort, they do not understand themselves as being agents. The understanding that one has goals and that other people also have their own goals begins to develop during childhood with the internalization of theory of mind, which has occurred for most children by the time they are approximately four years old. In this application of theory of mind, children understand that people around them have internalized mental states and act in ways to achieve certain ends. In recognizing this in others, they also recognize it in themselves (Wellman, 1993).

One's sense of agency continues to develop as an individual matures and provides the individual with the basis for forming an identity (McAdams & Cox, 2010). Indeed, being an agent requires projecting into the future an idea of the "possible selves" a person wishes to become and striving to accomplish such ends (Markus & Nurius, 1986; McAdams, 2013). Of course, goals can be in conflict with each other, and for any number of reasons, many "possible selves" are not obtainable. However, the psychologically mature agent sets new goals that are possible and extricates him or herself from goals that are not obtainable, while still acknowledging these lost "possible selves" (King & Hicks, 2007).

It is also important to note that agency is commonly discussed alongside a related concept called communion. Agency is about a person's focus on his or her selfhood and how he or she stands as an individual compared to others. Communion, on the other hand, is about a person's

focus on others (Wiggins, 1991). Agency and communion are typically conceptualized as orthogonal constructs, though there is some evidence that they may be correlated on a within person basis (Roche, Pincus, Hyde, Conroy, & Ram, 2013). Acting with agency is not synonymous with being an agent. Rather, the terms "agency" and "communion" indicate types of orientations in goal seeking. An agent can have both agentic (i.e., self-focused) and communal (i.e., other-focused) goals.

Author. The final layer of personality is that of the author. People are prone to attempt to seek meaning in what has happened in their lives, and to use these gained meanings to better understand the self. This is not done through characterizing oneself based on traits (actor) or by setting and working to obtain goals (agent). Rather, this is accomplished through constructing and telling stories about one's life (Baumeister & Newman, 1994). Thus, the last to form, most complex personality layer – the author – reflects the tendency people have to tell evolving, self-defining stories about their lives. The author reconstructs his or her past, acknowledges the present, and imagines the future so as to provide a sense of meaning to his or her life. This constructed story is referred to as "narrative identity" (McAdams, 2013; McAdams & McLean, 2013; Singer, 2004). These life stories are not objectively veridical reports (though they are certainly likely to be grounded in truth), but rather represent an amalgamation of what details of the life story a person chooses to include, and how that person has come to understand and remember the details that make up the story at a specific point in the person's life (McAdams et al., 2001). As such, while a person may not always be able to control what events he or she experiences during life, the individual does have some ability to shape what past events mean for that person's current life and possible future.

The skills necessary to begin authoring life stories do not start to crystalize until adolescence (McAdams, 1985). At this point, individuals have begun to develop the psychological complexity necessary to start to recognize and speak of coherent connections between events and what meaning these connections hold to them (Habermas & Silveira, 2008). This process of drawing connections between past episodic events and merging them with the present is called autobiographical reasoning, and it is a necessary component of being able to construct meaningful life stories (Habermas & Bluck, 2000). However, the adolescent author is not fully formed. Indeed, individuals' ability to engage in autobiographical reasoning and to author life stories continues to mature across the lifespan as people develop more nuanced perspectives on their lives (Pasupathi & Mansour, 2006).

There are numerous individual differences in the way people construct stories and the types of stories they tell (McAdams & Pals, 2006). As such, there are numerous individual differences in the types of meanings individuals draw from various life episodes, and these differences in meaning making result in differences in the way people understand their very identity. As people's understanding of who they are changes, so too does the way they narrate their life, creating a reciprocal feedback loop (McLean, Pasupathi, & Pals, 2007). In light of this, an individual's narrative identity is an important determinant of how a person experiences the world and the type of life outcomes a person encounters. Indeed, the many ways in which people narrate their life are linked to reliable differences in psychological maturity and well-being across the lifespan (Bauer & McAdams, 2004; Pals, 2006). While many different types of experiences contribute to the life story and narrative identity, the ways in which people narrate and come to understand adversity is likely to be a particularly important predictor of health and

well-being (Pals & McAdams, 2004). This idea will be discussed further in the following section.

Adversity and Narrative Identity: Stories of Contamination versus Redemption

As previously mentioned, the life story is not a historical account of factual material; rather, it is a powerful indicator of who a person believes he or she is in the here and now. When people recount their life stories, they are providing a window into who they see themselves as and the types of personal meanings they make out of events that they have reconstructed in their mind. These stories offer insight into individuals' very identity as human beings, situated within the bounds that their culture gives them for storytelling (McAdams & Pals, 2006; Singer, 2004). That is, they are telling their own "personal myth" about who they are and how they came to be that way (McAdams, 1993). Experiencing some form of adversity is a reality of life, even for the most fortunate of people, and there are many distinctive types of stories people can tell that emphasize different ways that the adversity they have undergone has informed their self-identity. Two particularly noteworthy prototypical scripts of "personal myths" that come up when American adults recount their life stories are ones where something bad is transformed into something good, called redemption, and where something good is transformed into something bad, called contamination (McAdams, Diamond, Aubin, & Mansfield, 1997; Tomkins, 1986).

As defined by McAdams (McAdams, 2006a, 2006b) the experience of redemption is about being delivered from suffering and pain to some more enhanced state of being. Life narratives that discuss a redemptive scene begin with the person talking about having been in some negative state – such as divorce, loss of a loved one, a diagnosis with a chronic and debilitating medical condition – that subsequently was transformed into something positive. These types of sequences give an individual the ability to shape how experiences define the self

and move the person's story forward by allowing for a more advanced understanding of the self resulting from personal growth and development (McAdams & Bowman, 2001). Redemption experiences are coded from interviews where the coder takes into account the meaning the narrator took away from the experience. That is, a redemption story is not necessarily one where something bad gave way to a clear, objectively good outcome such as being poor and than gaining material wealth (though it could be). Rather, it is a story where the narrator ascribes something personally meaningful that is positive, even if there was no material manifestation of this positivity (McAdams, 2006a). For example, gaining new insights into an individual's strength of character after going through a difficult divorce, or learning to better savor precious moments with family after developing a major medical condition could both be redemptive scenes, even though the objective "facts" of the scenes are themselves something that the typical person might construe as being distressing.

The opposite of the redemptive scene is the contamination scene (McAdams & Bowman, 2001). In this sequence, something that was once good is tainted or disrupted by some negative experience. For example, an individual might describe having had a wonderful family life that was all ruined after discovering his or her spouse was having an affair. Unlike in the redemption sequence, nothing is learned from this, and nothing positive comes from it. As such, while redemption sequences move a person's story forward, contamination sequences impede and stagnate the story, taking away opportunities for growth and development, often leaving the individual without hope (McAdams & Bowman, 2001).

Consistent with this theory, evidence suggests that redemption stories are generally associated with positive psychological outcomes, whereas contamination narrations are associated with negative outcomes. For example, in one study, the use of redemption imagery in

telling one's life story was positively associated with enhanced self-esteem, self-coherence, and life-satisfaction, and negatively associated with depressive symptoms; the opposite associations were found for contamination sequences (McAdams et al., 2001). In a separate study, tendencies to interpret life events positively, on average, were associated with enhanced well-being. Conversely, tendencies to interpret life events negatively, on average, were associated with poorer well-being (Lilgendahl & McAdams, 2011). These associations persisted even after controlling for lower level personality traits as well as demographic factors. To date, no studies have examined whether the use of redemption versus contamination sequences are related to physical health, though as will be discussed later, there is reason to believe that they might be.

Redemption scenes make up part of what McAdams (2006a) refers to as the "redemptive self." The prototypical "redemptive self" involves feeling that the protagonist of the story experienced some sort of early blessing, and noticing that others were not as fortunate. The narrator still experiences pain and suffering (i.e., while he or she feels blessed, his or her life is not without adversity), but these experiences tend to be redeemed. Interestingly, both redemption and contamination stories can be construed as strategies for dealing with aversive life experiences (McAdams & Bowman, 2001), and, as discussed earlier, these strategies, along with their related psychological outcomes, bear several notable parallels to Cognitive Adaptation Theory (Taylor, 1983). Under CAT, being able to create and maintain positive illusions that consist of believing that one is better off than the facts alone would suggest following an adverse experience, and comparing oneself favorably to others in a similar situation who are not doing as well are both linked to better mental and physical health outcomes (Taylor, 1983; Taylor & Brown, 1988; Taylor et al., 1991; Taylor et al., 2000). In line with this, forming a narrative identify rife with redemptive imagery may be construed as successfully being able to maintain

positive illusions in the face of something bad that has happened. Redemptive stories are a psychological construal. While it is certainly the case that some redemptive stories really do involve something objectively bad becoming objectively good, in many situations the move from bad to good is done through shaping one's interpretation of the event (i.e., regaining a sense of mastery over an event under CAT) by searching for psychological meanings (i.e., meaning making under CAT) that favor transformation and growth (i.e., self-enhancement under CAT) following a distressing experience. This suggests a possible scenario whereby the use of redemption stories in one's life story should generally be associated with similar physical health benefits as the use of positive illusions. The following section will explore this idea further, first by overviewing the mechanisms through which adversity is able to "get under the skin" to affect health, and then by considering how themes of redemption and contamination may play a role in shaping health outcomes following adversity.

Adversity, Physical Health, and the Life Story

Before discussing the physiological mechanisms through which adversity may impact health and the ways in which elements of the life story such as redemption and contamination might modulate this, it is important to consider what is meant by "adversity." Work on redemption and contamination in the life story as described by McAdams and colleagues (e.g., McAdams, 2006a, 2006b; McAdams & Bowman, 2001; McAdams et al., 1997; McAdams et al., 2001) has followed a standardized coding system for these sequences. In this coding system, a redemption sequence is defined as "a demonstrably 'bad' or emotionally negative event or circumstance [that] leads to a demonstrably 'good' or emotionally positive outcome" (p. 1; McAdams, 1999). Examples of negative events or circumstances given under this system include scenes where the narrator reports having experienced:

> "fear, terror, sadness, grief, anguish, guilt, shame, humiliation, anger, distress, or any of a large number of explicitly negative affective states. Also relevant would be physical pain, injury, and sickness. In other cases, the author may not explicitly describe a negative feeling, but the event itself is an especially negative one – e.g., death of a friend, divorce, major failure, poverty, addiction, broken relationship, being fired from one's job… The coder should be relatively conservative here. Minor setbacks (e.g., misplacing one's purse, waiting in line, getting a less-than-stellar grade on an exam) and mild negative states (e.g., feeling nervous at the beginning of a competitive event, feeling uncertain about one's skills, lacking direction in life) should not count" (p. 2; McAdams, 1999).

This coding guide allows for a wide range of experiences to fall under the umbrella of being emotionally negative. An important conceptual feature of the life story is that the narrations people construct to explain who they are, where they came from, and where they are going are a crafted psychological construal, and together, they form a cohesive identity. When a person describes a past event, it is not a historically accurate account of something that remains firmly in the past (nor is it intended to be), but rather is a reconstructed experience that provides the person with meaning and uniqueness in the present. As such, if a person includes a redemption sequence that took place twenty years prior to the interview in his or her life story, it means that this experience remains (or at some point became) an important part of his or her identity in the present. Thus, the emotionally negative episode that forms the basis of the redemption or contamination sequence can have originated as either a discrete event (such as the death of a loved one or the loss of a job), or a chronic circumstance (such as living with a disability or being impoverished); however, regardless of the duration of the original event, the meaning of the event has to continue to have relevance to the person in the present. Based on this, when I refer to adversity in this manuscript, I am referring to any significant emotionally negative, distressing, or stressful scene or circumstance from the life story. I will argue that redemption sequences may have the ability to cut short the negative sequelae following experiences of

adversity. Conversely, contamination sequences are situations when some past adversity continues to maliciously thrive within an individual.

There are many potential pathways through which experiences of adversity can "get under the skin" to affect health. One obvious route is through changes in health behaviors. Indeed, adversity is associated with heightened risk for a participating in a variety of risky or deleterious behaviors (Taylor, Repetti, & Seeman, 1997). These include poor diet, increased caloric intake, not sleeping enough, smoking, excessive alcohol and other drug use, and poor exercise habits. Such poor health behaviors increase an individual's risk for developing obesity, high blood pressure, insulin resistance, high blood glucose, and abnormal lipid profiles. In turn, this cluster of risk factors is widely implicated in the pathogenesis and progression of lifestyle diseases that develop slowly over decades and increase rapidly in incidence during midlife, such as metabolic syndrome, diabetes, and cardiovascular disease (Bjorntorp, 1997; Hu et al., 2001; Khot et al., 2003; Wellen & Hotamisligil, 2005). However, while health behaviors play important roles in shaping future disease risk, they do not explain the entire adversity-health story. Indeed, adversity also has the ability to directly modulate physiological systems related to disease risk, with over-activation and dysregulation of the immune system leading to a chronic inflammatory state being one of the main pathways implicated in disease risk (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Padgett & Glaser, 2003; Pickup, 2004; Rozanski et al., 1999; Schneiderman, Ironson, & Siegel, 2005).

How do adverse experiences get inside the body directly? People are constantly monitoring their environment and attending to their surroundings. Potential threats are evaluated by the hypothalamus (which also receives emotional input from other regions of the brain, including the frontal cortex and amygdala) in the brain (Steptoe & Kivimaki, 2012). In the

presence of something perceived to be threatening, two critical response systems are activated: the sympathetic-adrenal medullary (SAM) and hypothalamic-pituitary-adrenal (HPA) axes (Padgett & Glaser, 2003). These systems initiate a cascade of chemical messengers that orchestrate the body's stress response, preparing it to deal with the threat.

When an individual perceives the presence of a threat, the sympathetic nervous system (SNS), a component of the SAM axis responsible for the "fight or flight" response, stimulates the adrenals to produce the catecholamines epinephrine and norepinephrine (Guyton & Hall, 1996). These hormones act within seconds to prepare the body to deal with a variety of challenges. This includes increasing heart rate and blood pressure so as to supply skeletal muscles and the brain with more blood, allowing blood to clot faster in anticipation of injury, raising glucose production in the liver for added energy, and breaking down fat cells (via a process called lipolysis) into free fatty acids for additional energy (Schneiderman et al., 2005). At the same time, the hypothalamus releases corticotropin releasing factor, which singles the pituitary to release adrenocorticotropin. This pathway ultimately signals the adrenal cortex to release the glucocorticoid cortisol (Padgett & Glaser, 2003). In the relatively immediate aftermath following exposure to a threat, cortisol acts in tandem with catecholamines to promote lipolysis and conversion of glycogen to glucose, thus providing the individual with added energy to deal with the threat (Schneiderman et al., 2005).

In addition to providing necessary energy for dealing with a threat, the output from the HPA and SAM axes cues and regulates the immune system to prepare to deal with potential damages that may occur as a result of the threatening stimuli (Padgett & Glaser, 2003). Immune cells have receptors to receive messages from both the HPA and the SAM axes. Catecholamines signal innate immune cells such as monocytes/macrophages to be released from lymph tissue and

the spleen and enter the bloodstream (Schneiderman et al., 2005). From there, these cells make their way to areas likely to be the target of damage and initiate an inflammatory process that fends off pathogens in the case of a breach. These immune cells also have receptors for output from the HPA axis, of which cortisol is the paramount hormone in this process. Following the end of the threatening event, cortisol binds with immune cell receptors and silences the inflammatory response, ultimately helping to return the system back to its resting state (Sternberg, Chrousos, Wilder, & Gold, 1992).

This process from threat detection and appraisal to increased energy output, redirected use of resources, and an upregulated immune response is normal and highly adaptive. It provides the body excellent defenses against environmental danger. It is also energy intensive and shuts down resting restorative processes as they are not useful during an acute threat. This does not normally pose much concern for the individual, as the body has excellent counter-regulatory systems that end this response once it is no longer needed. That being said, the organismic response to threatening stimuli evolved to deal with acute threats to life and limb (Hotamisligil, 2006). However, there are many threatening experiences that are not so short-lived, or that have aftereffects that persist long after the initial stimuli has ended (e.g., poverty, loneliness, negative social evaluations and ostracism, being a victim of abuse, combat etc.). These adversities can repeatedly activate the stress response, ultimately resulting in dysregulation of the system at multiple levels. The consequences of this dysregulation are manifest in a variety of ways that are deleterious to cardiometabolic health (McEwen, 2008). For example, constant activation of the SAM axis puts strain on the cardiovascular system (Schneiderman et al., 2005). Over time, repeatedly raising blood pressure results in vasculature muscles thickening, elevating resting blood pressure. This in turn results in the heart doing more work resulting in hypertrophy of

components of the muscle. This persistent increase in blood pressure can eventually damage arteries and dislodge plaques, which can result in myocardial infarction and death (Roman et al., 2007).

Furthermore, constant activation of the immune system can result in immune cells becoming desensitized to the anti-inflammatory regulatory effects of cortisol (Miller, Cohen, & Ritchey, 2002). This can lead to persistent background inflammation, which takes the form of chronically elevated pro-inflammatory cytokines (Wellen & Hotamisligil, 2005). Long-term exposure to pro-inflammatory cytokines is strongly implicated in the incidence and progression of metabolic diseases, particularly in the context of obesity (Hotamisligil, 2006; Nathan, 2008; Nathan & Ding, 2010). Unfortunately, long-term exposure to these cytokines as well as output from the SNS also can play a role in the development of obesity by reducing sensitivity to insulin, and being obese in turn further increases insulin resistance (Garruti, Cotecchia, Giampetruzzi, Giorgino, & Giorgino, 2008; Nathan, 2008). Increased resistance to insulin results in rising levels of blood glucose. Chronically heightened blood glucose levels can lead to changes in red blood cells that ultimately result in the accumulation of cholesterol in the bloodstream and contribute to plaque formation throughout the cardiovascular system (Selvin et al., 2004). Additionally, exposure to heightened levels of blood glucose can result in nerve damage over time. Furthermore, visceral body fat in obesity acts as an inflammatory factory. For example, macrophages are taken up by adipose tissue and produce pro-inflammatory messengers. Additionally, macrophages store lipids and can become atherosclerotic foam cells, which play a role in the formation of atherosclerotic plaques (Wellen & Hotamisligil, 2005). If these plaques rupture and block an important artery, myocardial infarction may occur, followed swiftly by death depending on the location of the blockage.

Taken together, persistent activation of the body's stress systems through experiences with adversity can result in dysregulation of key systems intended for reducing collateral damage caused by chronic exposure to stress related hormones. This, coupled with deleterious changes in health behaviors that can occur following the exposure to adversity, poses potentially ruinous consequences to an individual's health and well-being. However, as previously discussed, many people who face major life adversities do not show evidence of maladaptation, nor do they get sick. As I have alluded to, the ways in which people narrate adversity in their life stories may provide important insights as to what trajectory a person will follow after having faced adversity.

How might constructing redemptive stories where something negative is transformed to something positive buffer against the potentially harmful consequences of experiencing the original adversity? It is likely that the meaning making process itself is unpleasant, resulting in initial declines in psychological well-being (Frazier et al., 2009). However, for the individual that successfully constructs a redemptive sequence, this is a temporary state, which gives way to developing interpretive control over the story, and ultimately transformation and growth. Utilizing interpretive control over the adversity is likely to have ameliorating effects, as evidence from the life stress literature suggests that controllability of a stressor is an important characteristic of whether or not the event has a lasting physiological impact (Taylor et al., 1997). While interpretive control is a secondary control process that is different from the idea of control as reflecting how much influence an individual has (whether real or not) over a given outcome, as discussed earlier, there is evidence in support of the notion that changing how one later interprets adversity through the use of secondary control processes may similarly buffer against the ill effects of the adversity (Armor & Taylor, 1998; Chipperfield et al., 2012). Indeed, this is consistent with evidence drawing on Cognitive Adaptation Theory that has shown that overly

optimistic beliefs of personal mastery promote adaptation following aversive life experiences (Helgeson, 2003; Taylor et al., 1991; Taylor et al., 2000). Physiologically, gaining a sense of interpretive control may ultimately interrupt the ability of the adversity to continuously be experienced as salient and threatening even after the actual event has ended. If this is the case, then one would expect the physiological stress systems to be able to return to baseline status before dysregulation is able to occur.

In addition to the possibility that gaining a sense of interpretive control over a past adversity has the ability to put the breaks on the stress response, the transformation and experience of redemption that completes the redemption sequence is also likely to have advantageous effects on adapting to a past life adversity. By incorporating this transformation to redemption into a person's life story, that person is identifying that he or she has been enhanced and made better in some way as a result of some negative experience. Once again, following evidence from research on Cognitive Adaptation Theory, viewing oneself in a more positive light as a result of having undergone some negative experience is likely to be adaptive and protective (Taylor & Broffman, 2011). Physiologically, once a negative event has been transformed into something positive and life affirming, the original event no longer holds the same type of threat. Without the presence or appraisal of a lingering threat, there should be no more physiological arousal to bring about negative health outcomes.

The case of how contamination sequences may be aversely associated with health outcomes can be construed in a relatively straightforward manner. As described earlier, in a contamination sequence, something good gives way to something bad, and that which was good is lost. The individual instead integrates the soiled memory into his or her identity. As such, contamination can be viewed as the incorporation of some adversity that is never resolved or

transformed. In essence, the person continues to feel and live the ramifications of this adversity. Indeed, it may even be possible that the adversity is made even worse as it stole away something viewed as precious or happy. As such, there are no narrative breaks to prevent the reconstructed memory of the adversity from being relived continuously, or from continuously regretting what was lost, which itself is associated with poorer psychological well-being (King & Hicks, 2007). Given this, it is plausible that having a narrative identity sullied by contamination sequences may put an individual at risk for repeated and chronic activation of physiologic stress systems and the negative consequences to health and well-being that result.

Project Hypotheses

To examine the issues discussed above, I conducted a study of 61 midlife American adults. In this project, I studied the associations between individuals' use of redemption and contamination scenes as narrated in their life stories and indicators cardiometabolic health. These participants were drawn from a larger 9-year longitudinal study on life stories and were a little past halfway through that study at the time that they participated in the current project. During the baseline and fifth annual visit for the larger study, participants underwent an extensive life story interview that was subsequently coded for redemption and contamination scenes. Blood and other health data were collected in the year following the fifth time point. Using these data, I hypothesized the following:

First, participants who described redemption sequences in their life stories should show evidence of better cardiometabolic health-related outcomes than participants who did not. This should be indexed by better self-reported health, fewer components related to metabolic syndrome, lower overall metabolic risk, lower levels of inflammation, and lower levels of C-

reactive protein (CRP), a biomarker of inflammation that is often assessed clinically as it is associated with cardiovascular disease risk.

Second, participants who described contamination sequences in their life stories should show evidence of worse cardiometabolic health-related outcomes than participants who did not. This should be indexed by poorer self-reported health, more components related to metabolic syndrome, higher overall metabolic risk, higher levels of inflammation generally, and higher levels of CRP specifically.

Third, redemption and contamination sequences are not mutually exclusive within a single life story. Indeed, it is not uncommon for participants to tell stories about their lives that contain a mixture of both types of sequences. As such, I also examined whether there was an interaction between having described a redemption scene and having described a contamination scene in predicting the various cardiometabolic health-related outcomes. I hypothesized that among individuals who reported contamination, also having reported redemption should be associated with better outcomes compared to individuals who did not also report redemption.

Fourth, data on redemption and contamination sequences were first obtained approximately 5 to 6 years prior to the current study and then again approximately 6 to 12 months prior to the current study. I do not have specific hypotheses regarding whether data obtained at one session should more strongly predict health related outcomes than data obtained at the other session. It is possible that redemption and contamination sequences described during the first wave of data collection will more strongly associate with current cardiometabolic health insofar as these experiences may have had more time to affect downstream processes that alter physical health properties. Alternatively, it is possible that redemption and contamination sequences described at the more recent wave of data collection will more strongly associate with

current cardiometabolic health insofar as these scenes occurred more proximally to the health assessment, and thus may be temporally more potent contributors to health. These analyses are exploratory and the results should be considered as guides for generating hypotheses in the future.

Methods

Participants

Participants for the current study, called the Healthy Stories Project (HSP), were drawn from the Foley Longitudinal Study of Adulthood (FLSA), a larger ongoing 9-year longitudinal study of personality development and generativity among 163 midlife Caucasian and African American adult males and females. The FLSA sample was recruited from the Chicago area between 2008 and 2009 and has been followed annually since then. Recruitment was done through the use of flyers, bulletin board postings, and advertisements at community centers, public libraries, local newspapers, religious institutions, and grocery stores. At study entry (alternatively referred to in this manuscript as baseline, FLSA Time 1 or just simply Time 1), participants were between the ages of 55 and 58. The sample contained a slightly higher number of Caucasians (n = 90) than African Americans (n = 73), and a higher number of women (n = 105) than men (n = 58). All members of the FLSA cohort still enrolled following the fifth year of the study (n = 149) were eligible to enroll in the current project.

After their fifth annual visit (referred to in this manuscript as FLSA Time 5 or just simply Time 5), individuals currently enrolled in FLSA were invited via email to participate in HSP by the project coordinator of FLSA. They were informed that their decision regarding participation would not affect their status in FLSA. Those who decided to contact me with interest in participating were given an overview of the study. I explained to them that the current project was related to FLSA and that I would be merging the data I collected with the data they provided for FLSA. A total of 71 individuals contacted me with interest in the study. Of these prospective participants, a total of 61 ultimately agreed to participate. Of the 10 individuals who initially contacted me regarding participation but did not ultimately join the study, 3 did not respond to

my attempts to contact them after their initial email expressing interest in the study, 5 were not available to participate in the study until well after it was scheduled to end, and 2 ultimately changed their minds about participating due specifically to anxiety related to having their blood drawn. The study was approved by the Institutional Review Board at Northwestern University (Study Identification: STU00089004), and all participants provided written, informed consent at study entry.

Many of the variables measured in the current study were not available in the full FLSA dataset to allow for an exhaustive comparison of potential differences between the two samples. Nonetheless, I did compare HSP participants to members of FLSA who did not participate in HSP on gender (male or female), race (Caucasian or African American), annual gross family income (range = 1 – 13; 1 = "*under $25,000*," 7 = "*$150,00 – $174,999*," 13 = "*over $300,000*"), self-reported subjective overall health (range = 1 – 5; 1 = "*poor health*," 2 = "*fair health*," 3 = "*good health*," 4 = "*very good health*," 5 = "*excellent health*"), self-reported history of physical health conditions (range = 0 – 28; conditions reported on were allergies, arthritis, asthma/bronchitis/emphysema, autoimmune disorders, cancer, chronic pain, chronic sleeping problems, dental health, diabetes, gall bladder trouble, hearing problems, heart condition, high blood pressure, high cholesterol, human immunodeficiency virus, migraine headaches, neurological disorders, persistent skin trouble, sciatica, speech problems, stroke, thyroid disease, trouble with varicose veins, tuberculosis, ulcer, urinary/bladder problems, and vision problems), self-reported history of mental health conditions (range = 0 – 7; conditions reported on were alcoholism, anxiety disorders, bipolar disorder, depression, eating disorders, schizophrenia, and substance abuse), and depressive symptoms (measured using the Beck Depression Inventory, 2nd Edition; range = 0 – 63; Beck, Steer, & Brown, 1996). All of these variables were assessed

at the baseline visit for FLSA. Reported *p*-values are all two-tailed, and *p*-values associated with χ^2-tests are exact.

There were no significant group differences in gender distribution, $\chi^2(1) = 1.24$, $p = .311$, racial distribution, $\chi^2(1) = 0.18$, $p = .745$, age, $N_{HSP} = 61$, $M_{HSP} = 56.05$, $SD_{HSP} = 1.07$, $N_{FLSA} = 97$, $M_{FLSA} = 55.86$, $SD_{FLSA} = 0.98$, $t(156) = 1.17$, $p = .245$, $d = 0.19$, annual family income, $N_{HSP} = 61$, $M_{HSP} = 6.13$, $SD_{HSP} = 4.59$, $N_{FLSA} = 102$, $M_{FLSA} = 5.24$, $SD_{FLSA} = 4.05$, $t(161) = 1.30$, $p = .196$, $d = 0.21$, number of mental health conditions $N_{HSP} = 60$, $M_{HSP} = 0.35$, $SD_{HSP} = 0.58$, $N_{FLSA} = 102$, $M_{FLSA} = 0.41$, $SD_{FLSA} = 0.71$, $t(160) = 0.57$, $p = .568$, $d = 0.09$, or depressive symptoms on the BDI-II, $N_{HSP} = 60$, $M_{HSP} = 7.08$, $SD_{HSP} = 6.25$, $N_{FLSA} = 99$, $M_{FLSA} = 7.17$, $SD_{FLSA} = 6.41$, $t(157) = 0.09$, $p = .932$, $d = 0.01$. Individuals who participated in HSP reported fewer physical health conditions at the baseline FLSA visit than FLSA members who did not participate in HSP, $N_{HSP} = 60$, $M_{HSP} = 3.43$, $SD_{HSP} = 2.47$, $N_{FLSA} = 102$, $M_{FLSA} = 4.20$, $SD_{FLSA} = 2.61$, $t(160) = 1.83$, $p = .069$, $d = 0.30$. Consistent with this, HSP participants also reported subjectively feeling in better overall health than FLSA members who did not participate in HSP, $N_{HSP} = 61$, $M_{HSP} = 3.92$, $SD_{HSP} = 0.82$, $N_{FLSA} = 102$, $M_{FLSA} = 3.29$, $SD_{FLSA} = 1.01$, $t(161) = 4.08$, $p < .001$, $d = 0.66$. Taken together, the HSP sample appears similar to the larger sample it was drawn from in basic demographics and mental health status, but the HSP sample may have been somewhat more physically healthy than the sample of FLSA members who did not participate in HSP.

Procedures

Participants in the HSP study attended a single session at our laboratory at Northwestern University. All study sessions took place within 12 months of participants' FLSA Time 5 visit and were conducted by me. Upon arriving at our laboratory, I provided participants with a copy of the study consent form and asked them to spend a few minutes reading it over. I then

discussed any questions they had prior to signing the form. I also reiterated to participants that their study data would be kept confidential and de-identified except in specific situations in which I would be required to report information furnished to me under mandated reporting laws (i.e., if the participant told me that he or she planned to hurt him or herself or if the participant disclosed the abuse of a child or dependent adult). After this, I took a sample of venous blood from participants via standard venipuncture practices. I next brought participants to a private interview room where they filled out questionnaires on a computer about their socioeconomic status, health practices and history, and mood. Following this, I assessed participants' resting blood pressure, height, weight, and waist circumference. I provided individuals with a $50 prepaid Visa gift card as an honorarium to thank them for their participation and also gave them $5 to cover parking or public transportation costs. Finally, I mailed participants feedback relevant to their current health, including information about their height, weight, blood pressure, glycosylated hemoglobin percentages (a measure that taps into "average" blood sugar levels over the past three months), and total cholesterol levels.

Questionnaires

Demographics. Participants provided basic demographic information on their age, gender (male or female), and ethnicity (Caucasian or African American). The original FLSA sample was restricted to being between 55 and 58 years old at study entry. As such, the range of ages represented in the HSP sample was also relatively narrow, with all participants being between 59 and 63 years old (M = 61.05 years, SD = 1.01 years) at the time of their visit to our laboratory.

Socioeconomic Status. Participants filled out questionnaires regarding indicators of socioeconomic status (SES) via a standard, widely used measure (MacArthur Sociodemographic

Questionnaire; MacArthur Research Network on Socioeconomic Status and Health). Specifically, participants provided information about the highest academic degree they had obtained (range = 0 – 6; 0 = "*less than high school*," 1 = "*high school degree or equivalent*," 2 = "*associate's degree*," 3 = "*bachelor's degree*," 4 = "*master's degree*," 5 = "*doctoral degree*," 6 = "*professional degree, e.g., MD, JD, DDS, etc.*"). If participants were in a committed relationship, they also provided information about what their partner's highest obtained academic degree was. I used whichever academic degree was higher between the participant and his or her partner as the final indicator of education-related SES. Next, participants provided an estimate of what their total gross family income was for the past year (range = 1 – 9; 1 = "*less than $5,000*," 2 = "*$5,000 – $11,999*," 3 = "*$12,000 – $15,999*," 4 = "$16,000 – $24,999," 5 = "*$25,000 – $34,999*," 6 = "*$35,000 – $49,999*," 7 = "*$50,000 – $74,999*," 8 = "*$75,000 – $99,999*," 9 = "*$100,000 or greater*"). Finally, to understand each individual's general wealth, participants indicated how long they could live at their current address and standard of living if they lost all current sources of income (range = 1 – 5; 1 = "*less than 1 month*," 2 = "*1 – 2 months*," 3 = "*3 – 6 months*," 4 = "*7 – 12 months*," 5 = "*more than 1 year*"). These three measures of SES were all intercorrelated. Specifically, the correlation between educational obtainment and family income was $r(59) = .58$, $p < .001$, 90% CI for $r = [.42, .71]$, the correlation between educational obtainment and wealth was $r(59) = .44$, $p < .001$, 90% CI for $r = [.27, .60]$, and the correlation between family income and wealth was $r(59) = .51$, $p < .001$, 90% CI for $r = [.32, .68]$. As such, I computed *z*-scores for each participant on each of these three variables and then added them together to create a composite indicator of SES, with higher values indicating higher SES.

Depressive Symptoms. Though not always, depression and inflammation often co-vary together (Glassman & Miller, 2007; Slavich & Irwin, 2014). To be able to account for this

possibility in the current study, participants disclosed the magnitude of depressive symptoms they had experienced over the past two weeks prior to the study visit by filling out the Beck Depression Inventory, 2nd edition (BDI-II; Beck et al., 1996). This scale consists of 21 items that assess depressive symptoms such as sadness, pessimism, guilt, and anhedonia. For each type of symptom, participants responded on a 4-point Likert scale (range = 0 – 3). For example, for the symptom "sadness," participants selected one of the following statements that they believed most accurately reflected their experience over the past two weeks: 0 = "*I do not feel sad*," 1 = "*I feel sad much of the time*," 2 = "*I am sad all the time*," 3 = "*I am so sad or unhappy that I can't stand it*." I summed responses on the BDI-II. Final scores fell between 0 and 63 with higher values indicating greater depressive symptomatology. The BDI-II is based on the original BDI (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), a widely used measure of depressive symptoms that has been extensively validated, but has been updated to provide a superior factor structure, allowing for subgroups of symptom profiles to be assessed. Internal consistency within the current sample was excellent (Cronbach's $\alpha = .89$). Additionally, scores on the BDI-II demonstrated a high degree of stability between when they were measured at the baseline visit of FLSA and when they were subsequently assessed again during the HSP study visit approximately 6 years later, $r(58) = .76$, $p < .001$, 90% CI for $r = [.62, .85]$. This consistency between scores is not surprising as this is not a clinical sample, nor is it a treatment study. Furthermore, the average BDI-II score at FLSA baseline was 7.08 ($SD = 6.25$), and the average BDI-II score obtained during the HSP study visit was 5.67 ($SD = 6.10$). According to the BDI-II scoring manual, values under 9 are consistent with "minimal depression," suggesting that depressive symptoms in the current sample were stable at least in part because they were bounded by a floor effect.

Health Behaviors. Participants responded to questions about current tobacco and alcohol use as well as prescription and over-the-counter drug use. These instruments have been used extensively in past research and have demonstrated excellent reliability and validity (e.g., Miller, Cohen, & Herbert, 1999).

To quantify current tobacco use, participants reported on how many cigarettes, cigars, and bowls of tobacco they smoked in a typical week. I used this information to estimate how many units of tobacco (defined as one cigarette) participants used during a typical week. None of the participants endorsed bowl tobacco use, so I only had to convert cigars to cigarette units. While cigars can vary greatly in size and tobacco content, epidemiological evidence suggests that, on average, individuals who smoke 5 cigars per day have similar health risks as individuals who smoke 20 cigarettes per day (Shopland, 1998). As such, for the purposes of creating a composite variable reflecting units of tobacco products used in a typical week, I considered one cigar to be the equivalent of four cigarettes. I thus quantified tobacco use as the total number of cigarettes plus the total number of cigars multiplied by four that individuals endorsed using in a typical week. Smoking rates were very low in the current sample. Eighty-nine percent of participants reported that they did not use tobacco at all, and only 8% of participants reported smoking a pack or more of cigarettes during a typical week (i.e., $\geq$ 20 cigarettes per week). The associations between tobacco use and various negative health consequences has been shown to be dose dependent, with infrequent, light smokers having substantially less risk for the myriad deleterious outcomes associated with smoking compared to heavier smokers (Office of the Surgeon General of the United States of America, 2014; Schane, Glantz, & Ling, 2009). As such, it is unclear what, if any, implications the small amount of tobacco use reported in the HSP

sample has for physiological outcomes measured in this study, and there is too little variability in smoking within this sample to reliably probe this issue.

To quantify alcohol use, participants reported how many drinks they consumed separately on a typical weekday and weekend day. The instructions on the survey informed participants that one drink is defined as one 12-ounce bottle of beer, one 5-ounce glass of wine, or one shot of hard liquor. Participants then reported separately on how many days of the week and weekend they typically consumed alcohol. I multiplied the number of drinks participants consumed on a typical weekday or weekend day by the number of days during the week and the weekend they typically drank to provide an estimate of total number of alcoholic drinks consumed during a normal week. Alcohol use in the sample was low; 69% of the sample reported that they did not consume alcohol at all, and only 5% of the sample reported typically consuming more than 10 drinks in a week. The association between alcohol use and negative health consequences is dose dependent and J-shaped, with moderate drinkers potentially enjoying some health benefits from drinking, and the largest health risks being conferred by regular heavy drinking or infrequent binge drinking (Room, Babor, & Rehm, 2005). As such, alcohol consumption is unlikely to be a particularly relevant confounder or mediator in understanding associations between life experiences and physiological outcomes within the current sample.

Finally, participants listed all prescription and over-the-counter medications they were currently taking. Overall, medications fell into one of the six following classes: lipid-lowering agents (e.g., statins such as atorvastatin), anti-hypertensive agents (e.g., diuretics such as furosemide), anti-hyperglycemic agents (e.g., biguanidines such as metformin), anti-inflammatory agents (e.g., nonsteroidal anti-inflammatory drugs such as ibuprofen), psychiatric agents (e.g., selective serotonin re-uptake inhibitors such as fluoxetine), and other miscellaneous

medications (e.g., antihistamines such as fexofenadine, non-anti-inflammatory analgesics such as acetaminophen, and supplements such as calcium and multivitamins). For the purposes of the current project, I constructed a binary variable reflecting whether or not participants used medications for managing or treating cardiometabolic health-related conditions. Specifically, I assigned participants who used lipid-lowering (n = 12), anti-hypertensive (n = 17), or anti-hyperglycemic (n = 7) agents a value of 1; everyone else received a value of 0 on this variable. I chose these specific classes of medications because they are some of the most commonly prescribed drugs to midlife adults and they also modulate the various health outcomes of interest in this study (Qato et al., 2008). A total of 21 participants (34% of the sample) reported currently using one or more of these classes of medications.

Self-Reported Health. Subjective self-reported health is associated with actual morbidity and mortality, independent of a variety of potential confounding variables including age, socioeconomic status, health behaviors, actual diagnosed illnesses, functional status, depression, and cognitive function (DeSalvo, Bloser, Reynolds, He, & Muntner, 2006). As such, participants answered three questions designed to understand how they felt about their recent health. Participants indicated on a scale of 1 ("*very poor*") to 6 ("*excellent*") how they would rate their health over the past month. They then indicated how much their physical health had limited their ability to engage in normal daily physical activities on a scale of 1 ("*could not do physical activities*") to 5 ("*not at all*"). Finally, they indicated how much their physical health had limited their ability to work, either at home or away from home on a scale of 1 ("*could not do daily work*") to 5 ("*not at all*"). These three measures of subjective self-reported health were all intercorrelated. Specifically, the correlation between overall health and physical activity limitations due to health was $r(59) = .57$, $p < .001$, 90% CI for r = [.34, .73], the correlation

between overall health and work limitations due to health was $r(59) = .63$, $p < .001$, 90% CI for $r = [.47, .75]$, and the correlation between physical activity limitations due to health and work limitations due to health was was $r(59) = .73$, $p < .001$, 90% CI for $r = [.55, .84]$. As such, I *z*-scored participants' responses on each of these three variables and added these scores together to create a composite indicator of subjective self-reported health, with higher values indicating better subjective physical health.

Participants also self-reported as to whether or not a doctor had ever told them they had any of a variety of different health conditions. Specifically, participants reported on allergies, lifetime history of angina, angina within the past 12 months, arthritis, asthma, lifetime history of cancer, cancer within the past 12 months, chronic bronchitis, chronic infectious illnesses, diabetes, emphysema, heart failure, lifetime history of hypertension, current hypertension, kidney problems, liver problems, myocardial infarction, neurological conditions, lifetime history of stroke, stroke within the past 12 months, and thyroid problems. Refer to Table 2 for the breakdown of how many participants reported having at some point been diagnosed with these various conditions. These health conditions were not ultimately evaluated as potential covariates due to low or non-existent base rates of relevant illnesses. For example, no participants reported having ever had myocardial infarction (i.e., a heart attack), and only one participant reported having had a stroke (though not in the past 12 months). While a larger number of participants reported having at some point been diagnosed with potentially relevant conditions to cardiometabolic health such as diabetes ($n = 12$) and current hypertension ($n = 13$), the majority of these participants were also on medications to treat these illnesses. Additionally, there were participants who did not report such diagnoses but who were nonetheless currently taking medications used for treating these illnesses. This suggests that some individuals either were not

aware of their disease status, or were being prophylactically treated by their doctors due to having prodromal features of these diseases or other risk factors. As such, in this sample, evaluating cardiometabolic medication use likely better captures the potential confounding influences of disease status compared to evaluating self-reported diagnosis history.

Table 1

Self-reported health conditions (N = 61)

Health Condition	n	%
Allergies	24	39.3
Angina (Lifetime History)	1	1.6
Angina (Past 12 Months)	0	0
Arthritis	27	44.3
Osteoarthritis	15	24.6
Rheumatoid Arthritis	1	1.6
Unknown Arthritis	11	18.0
Asthma	5	8.2
Cancer (Lifetime History)	13	21.3
Breast Cancer	4	6.6
Prostate Cancer	2	3.3
Skin Cancer	5	8.2
Other Cancer	2	3.3
Cancer (Past 12 Months)	1	1.6
Chronic Bronchitis	3	4.9
Chronic Infectious Illnesses	3	4.9
Hepatitis	1	1.6
Herpes	1	1.6
HIV	1	1.6
Diabetes	12	19.7
Emphysema	0	0
Heart Failure	0	0
Hypertension (Current)	13	21.3
Hypertension (Lifetime History)	23	37.7
Kidney Problems	3	4.9
Liver Problems	1	1.6
Myocardial Infarction	0	0
Neurological Conditions	1	1.6
Stroke (Lifetime History)	1	1.6
Stroke (Past 12 Months)	0	0
Thyroid Problems	9	14.8

Note: HIV = Human Immunodeficiency Virus

Life Story Interview

As participants in FLSA, individuals in this study had been administered the Life Story Interview (McAdams, 1985, 1993) at FLSA Years 1 and 5. The Life Story Interview is a semi-structured interview where the respondent is guided through the telling of his or her life story. The story is not all-inclusive, but rather asks the participant to think of a subset of key episodes from their life across a variety of domains. For this study, the episodes I examined were (1) a particularly positive memory from anytime, (2) a particularly negative memory from anytime, (3) a turning point in life, (4) a particularly positive childhood memory, (5) a particularly negative childhood memory, (6) a particularly vivid adult memory, (7) a time when the participant had shown wisdom, (8) a religious/spiritual experience, (9) the participant's greatest loss, (10) the participant's greatest regret/failure, and (11) the participant's biggest life challenge. I coded these scenes for the constructs redemption and contamination following criteria that I describe below. To examine the reliability of my codes, a fellow graduate student who has extensive prior experience coding these constructs additionally coded a subset of 13 interviews (11%) for redemption and contamination.

Coding Redemption. To code for redemption sequences, I followed standard procedures provided by the Foley Center for the Study of Lives at Northwestern University. Specifically, I read through each individual's transcribed life story interview from FLSA Years 1 and 5 to identify scenes where the participant described something "bad" being followed by something "good." I assigned each scene that contained redemptive imagery a value of 1. I then further scored scenes that contained redemptive imagery for descriptions of enhanced agency (a scene where the narrator mentioned having received enhanced personal power in addition to whatever "good" outcome followed the previous negative state), enhanced communion (a scene where the

narrator mentioned an enhancement of some aspect of his or her social life in addition to whatever "good" outcome followed the previously negative state), and ultimate concerns (a scene where the narrator expressed coming to face with some fundamental existential issue). Based on these criteria, each scene could be assigned a total of up to 4 points, with higher numbers reflecting more redemption imagery.

An example of a redemption sequence was provided by a participant who I will refer to by the pseudonym Ted. When asked to describe a turning point in his life, Ted talked about a time when he discovered his now ex-wife had been cheating on him. He narrated that he confronted his ex-wife after discovering that she had been cheating on him, and this confrontation eventually lead to their divorce. Ted told the interviewer that this episode had left him feeling sad and skeptical about close relationships. However, Ted went on to talk about how one of his friends eventually convinced him to give dating a try again. He took his friend's advice and ultimately met a woman who he fell in love with and eventually married. He reported that they have a happy marriage and that the whole experience taught him that life does not end just because something bad happens. As such, the negative experiences of learning that his ex-wife had cheated on him and his divorce were eventually transformed by the positive experience of meeting, falling in love with, and marrying a new woman.

Reliability of these codes was only moderate (*ICC* = .60). This likely reflects the complexity of coding a complex construct from long, highly varied narratives. To better understand this lower reliability, I probed the discrepancies between my codes and the codes furnished by the other graduate student on the 13 cases we both coded. Qualitatively, it appeared that my coding approach may have been generally more conservative than the other coder, and thus I may have underestimated the total number of redemption sequences in the sample. This

modest reliability for the redemption codes may reduce statistical power to detect real associations between redemption and the various outcome variables of interest.

Coding Contamination. To code for contamination sequences, I followed standard procedures made available by the Foley Center. Specifically, contamination was defined by scenes where the participant expressed that something good gave way to something bad. An example of a contamination sequence was provided by a participant who I will refer to by the pseudonym Sally. When describing a low point in her life, Sally talked about how a job that she had really enjoyed and found meaning in turned toxic due to issues that developed with a colleague. Sally took care to discuss how much she enjoyed her job prior to this conflict, and how the conflict had subsequently made her job difficult and unpleasant for her to do. She went on to talk about how the experience revealed to her that she was psychologically more fragile than she had previously thought. As such, the "good" memory of a happy job was spoiled by the subsequent "bad" experience that Sally had with her colleague, as well as Sally's realization that she was not as psychologically strong as she had previously believed. This variable was simply coded as present or absent. Reliability for these codes was higher than for redemption (*ICC* = .78) and consistent with the reliability of contamination codes described in previously published research (e.g., McAdams et al., 2001).

While it is theoretically possible for participants to have received a redemption score as high as 44 and a contamination score as high as 11, at both FLSA Years 1 and 5, the majority of participants received scores of 2 or fewer for both redemption and contamination imagery. Specifically, for the FLSA Year 1 visit, 82% of the sample received a redemption score of 2 or less, and 95% of the sample received a contamination score of 2 or less. For the FLSA Year 5 visit, 87% of the sample received a redemption score of 2 or less, and 90% of the sample

received a contamination score of 2 or less. Due to the general lack of scores greater than 2, it did not make sense to attempt to model associations between redemption/contamination and health related outcomes using continuous scores for redemption and contamination. Instead, I created four binary variables that indicated whether or not redemption and contamination imagery had been present across any of the 11 scenes from the interviews for both FLSA Years 1 and 5. I also used these binary variables to create interaction terms for FLSA Time 1 redemption × contamination and FLSA Time 5 redemption × contamination. These binary variables and interaction terms were used for all of the data analyses reported in this manuscript.

Physical Health Indicators

Physical Characteristics. Obesity and central adiposity are major risk factors for various chronic illness such as diabetes and cardiovascular disease and are also lifestyle factors associated with adverse experiences (Mokdad et al., 2003; Nathan & Ding, 2010). To evaluate obesity, I assessed participants' height using a standard stadiometer and their weight using an electronic scale (Tanita BF-350 Body Composition Analyzer; Tanita Corporation). I then used these height and weight measurements to calculate body mass index (BMI) by dividing each participant's weight in kilograms by their height in meters squared. The mean BMI in the current sample was 29.4 (*SD* = 5.9, range = 19.9 – 43.9). A BMI between 18.5 and 24.9 is generally considered to indicate that a person is of normal weight, a value between 25 and 29.9 is generally considered to indicate that a person is overweight, and a value of 30 or higher is generally considered to indicate that a person is obese (Kopelman, 2000). Based on these criteria, 36% of the current sample was overweight, and an additional 41% of the sample was obese (i.e., 77% of the sample was either overweight or obese). However, BMI is not necessarily always reflective of an individual's adiposity, as it is also higher in people with increased lean muscle mass. Thus,

to assess central adiposity somewhat more directly, I also measured participants' waist circumferences using a standard tape measure. The mean waist circumference for women in the current sample was 89.6cm (*SD* = 17.2cm, range = 27.5cm – 132.1cm), and the mean waist circumference for men was 106.3cm (*SD* = 18.1cm, range = 83.8cm – 148.6cm). Waist circumferences greater than 88cm for women and 102cm for men are generally accepted as cut-off values for being at high risk of deleterious cardiometabolic conditions (Kopelman, 2000). Based on these criteria, 49% of the current sample had a waist circumference in the "high risk" category. Although I provided information on BMI in this section, I did so mainly for descriptive purposes as it is still one of the most commonly used metrics for identifying overweight and obese individuals in clinical settings. However, in statistical models that adjusted for adiposity, I entered the waist circumference variable rather than the BMI variable, as waist circumference tends to be a better indicator of adiposity as well as a better predictor of deleterious health outcomes associated with obesity than BMI (Janssen, Katzmarzyk, & Ross, 2004).

Resting Blood Pressure. Blood pressure refers to the amount of force exerted upon the walls of arteries (expressed in millimeters of mercury, mmHg), and is divided into two categories: systolic and diastolic blood pressure. Systolic blood pressure refers to the pressure on arterial walls that occurs when the heart muscle contracts (i.e., the maximum pressure that occurs when the heart beats). Conversely, diastolic blood pressure refers to the pressure on arterial walls between heart beats (i.e., the minimum pressure that occurs while the heart is at rest). Chronically elevated blood pressure, referred to as hypertension, is a major risk factor for poor cardiovascular outcomes such as myocardial infarction and stroke (Chobanian et al., 2003). As such, I measured each participant's resting blood pressure. To do so, participants first sat quietly and relaxed for five minutes. Next, I took four blood pressure readings spaced two minutes apart

using an automatic blood pressure device (Dinamap Pro 100; Critikon Corporation). I dropped the first reading and took the average of the remaining three readings to quantify each participant's resting systolic and diastolic blood pressures. Prior research supports that this protocol provides data consistent with gold standard 24-hour ambulatory blood pressure monitors, suggesting that it yields a reliable estimate of resting blood pressure (Beckett & Godwin, 2005). Specifically, this study showed that correlations between systolic and diastolic blood pressure taken using an automated blood pressure device in the manner described above and 24-hour ambulatory blood pressure were .57 and .61 respectively. For comparison, this study only found correlations of .15 and .32 between systolic and diastolic blood pressure readings averaged across three visits to a doctor's office and readings taken from 24-hour ambulatory monitoring.

Blood Draw. I drew 12mL of peripheral venous blood via venipuncture of the antecubital fossa (i.e., interior elbow) from each participant during the study session[2]. I then used these samples to assess a number of physiological processes related to cardiovascular risk and diabetes. I chose these endpoints as they are responsible for the largest proportion of morbidity and mortality in the United States and become increasingly common as individuals age (Heron, 2013). Furthermore, as detailed earlier, these outcomes have associations with experiences of adversity. The outcomes I assessed were as follows:

Metabolic Health Indicators. I obtained values for each participant's glycosylated hemoglobin percentage (also known as hemoglobin $A1_C$% or $HbA1_C$%) and total serum

[2] Of the 61 participants in this study, I was unable to to obtain blood from 1 individual. A second experienced venipuncture technician from a nearby clinic also tried to obtain blood from this individual without success.

cholesterol at the time of the study visit. Glycosylated hemoglobin percentage provides a measure of "average" blood sugar levels over the previous three months that does not require an individual to be fasting prior to the blood draw. Specifically, as blood sugar levels increase (as occurs with hyperglycemia and unmanaged diabetes), more and more glucose molecules bind themselves to hemoglobin proteins in red blood cells. The percentage of hemoglobin proteins that have bound glucose increases predictably when blood sugar levels remain chronically elevated. As such, glycosylated hemoglobin is often used in clinical settings to determine diabetes risk and track diabetes progression and management, and it is also associated with cardiovascular health related outcomes (Goldstein et al., 2004). Total cholesterol is also routinely quantified in clinical settings, and it reflects an individual's lipid status, with higher values being generally associated with increased cardiovascular risk (Ezzati et al., 2002).

Glycosylated hemoglobin was assessed in whole blood. For this, I drew 4mL of venous blood into an EDTA Vacutainer tube (BD Biosciences, Mississuaga, ON). I then stored samples in our laboratory refrigerator at 4° Celsius until they were ready to be analyzed. A courier transported the samples to a local hospital laboratory on a weekly basis where they were assessed using standardized assays. Normal values for glycosylated hemoglobin range from 4.4% – 5.6%. Values between 5.7% and 6.4% suggest an increased risk for diabetes, and values above 6.5% are considered diagnostic of diabetes (Goldstein et al., 2004). In addition to not having data on this variable from the one individual who I could not obtain a blood sample from, presumably random errors that occurred at the local hospital laboratory further resulted in missing data from an additional five participants. As such, data on glycosylated hemoglobin was only available for 55 participants.

Total cholesterol was assessed in serum. For this, I drew 8mL of blood into a serum-separating Vacutainer tube (BD Biosciences, Mississuaga, ON). I then centrifuged the tube within 30 – 120 minutes of the blood draw at 1200 RCF for 10 minutes. Next, I drew a 500 microliter aliquot of serum from the centrifuged tube and stored the sample at -30° Celsius in a freezer in our laboratory until analysis. A courier transported samples to a local hospital laboratory on a weekly basis where they were assessed using standardized assays. Normal ranges for total cholesterol are 120 – 200mg/dL. Data on this variable were missing for one participant who I was unable to obtain a blood sample from.

The historical clinical gold standards for assessing blood glucose and cholesterol are to take fasting measures (though, evidence suggests that glycosylated hemoglobin may be just as informative as fasting blood glucose, if not more so; Goldstein et al., 2004). If a person is fasting, then current levels of blood glucose can be estimated with reliable reference ranges for understanding diabetes risk. Likewise, if a person is fasting, the breakdown of types of cholesterol can be estimated. This is useful as the type of cholesterol matters for health, with higher levels of low density lipoproteins (LDL; often referred to as “bad cholesterol”) and lower levels of high density lipoproteins (HDL; often referred to as “good cholesterol”) conferring greater cardiometabolic risk. While it thus would have been more ideal to collect fasting samples, it was simply not logistically feasible. Many participants in this study were in the workforce and were only available to come in for a study session during the late afternoon or evening. As such, requiring participants to not eat for 10 – 12 hours prior to the blood draw, as would be required for fasting tests, would have been overly burdensome on participants and likely would have made recruiting a reasonably sized sample untenable.

Metabolic Syndrome Risk. Metabolic syndrome refers to a cluster of clinical features that are associated with increased risk for cardiovascular morbidity and mortality (Lakka et al., 2002; Lambert et al., 2011; Melamed, Shirom, Toker, Berliner, & Shapira, 2006; Mendez, Goldberg, & McCabe, 2010). Scientists, doctors, and other public health experts have proposed a number of definitions over the years to describe metabolic syndrome, but in general, the core components include elevated blood sugar (referred to as hyperglycemia), poorer lipids status (referred to as dyslipidemia), increased central adiposity, and elevated blood pressure (Eckel, Grundy, & Zimmet, 2005). The most commonly employed definition of metabolic syndrome was developed by the National Cholesterol Education Panel/Adult Treatment Panel III (ATP III) and is utilized by the American Heart Association (Grundy, Brewer, Cleeman, Smith, & Lenfant, 2004). Following the criteria established by the ATP III, a person can receive a diagnosis of metabolic syndrome if he or she presents with any three of the following five possible components: (1) fasting blood sugar greater than 100 mg/dL or use of medications for managing blood sugar levels, (2) triglyceride levels greater than 150 mg/dL, (3) high-density lipoprotein (i.e., HDL cholesterol) levels less than 40mg/dL for men or less than 50mg/dL for women, (4) a waist circumference of greater than 88cm for women or greater than 102cm for men, and (5) a resting systolic blood pressure greater than 130mmHg, or a resting diastolic blood pressure greater than 85mmHg, or use of medications for managing blood pressure.

In order to reduce the total number of outcome variables being predicted in the current study, and to examine a clinically relevant outcome, I summed up the number of components of metabolic syndrome that participants presented with at the time of their HSP study visit. However, as described earlier, fasting blood draws were not feasible for this study. As such, I was unable to determine an actual diagnosis of metabolic syndrome. Instead, I used the available

data to evaluate components related to metabolic syndrome that may suggest increased risk of being eligible for a diagnosis of metabolic syndrome. Specifically, in place of fasting blood sugar, I counted glycosylated hemoglobin percentages greater than or equal to 5.7% as indicating positive hyperglycemia status. This cutoff comes from prior research investigating the use of glycosylated hemoglobin percentages in place of fasting blood sugar levels for determining metabolic syndrome status (Ong et al., 2010). Likewise, as I could not quantify participants' fasting triglyceride or high-density lipoprotein levels, I counted a total cholesterol level greater than or equal to 200mg/dL as indicating possible positive dyslipidemia status. Following these criteria, participants could have between zero and four components of metabolic syndrome risk. The mean number of metabolic syndrome related components in the current sample was 1.65 (SD = 1.11, range = 0 – 4).

Composite Metabolic Risk. In addition to tallying the available components of metabolic syndrome, I also computed a variable for composite metabolic risk using the continuous distributions of each of the collected components of metabolic syndrome. To do so, I summed the z-scores for participants' waist circumference (computed separately for men and women), systolic blood pressure, diastolic blood pressure, total cholesterol, glycosylated hemoglobin percentage, blood sugar medication use status, and blood pressure medication use status. Higher positive values on this variable indicate relatively greater metabolic risk; lower negative values indicate relatively lower metabolic risk. The mean value for this composite index was -0.16 (SD = 3.61, range = -7.61 – 8.84). Unsurprisingly, composite metabolic risk was strongly positively associated with the number of components related to metabolic syndrome participants presented with, $r(53) = .86$, $p < .001$, 90% CI for r = [.80, .90].

Inflammation. As discussed earlier, adversity is associated with dysregulation of the immune system which in turn can result in persistent background inflammation (Miller, Chen, & Cole, 2009). This can be evidenced by a variety of easily assessable biological markers, including increased levels of the acute phase protein C-reactive protein (CRP), the enzyme lipoprotein-associated phospholipase A_2 (Lp-PLA_2), and a variety of chemical messengers known as inflammatory cytokines. As such, serum levels of the inflammatory indicators CRP and Lp-PLA_2, as well as the inflammatory cytokines interleukin-6 (IL-6), interleukin-8 (IL-8), interleukin-10 (IL-10), interferon-γ (IFNγ), and tumor necrosis factor α (TNFα) were assessed using standard procedures discussed below.

C-reactive protein is an acute phase protein produced by the liver that rises rapidly as a non-specific response to tissue damage, infection, and other inflammation prompting threats (Pepys & Hirschfield, 2003). It is not an inflammatory protein in of itself, but rather CRP follows a predictable surge during the inflammatory response and thus is commonly used in both research and clinical settings as a marker of systemic ongoing inflammation. Indeed, epidemiological evidence has linked circulating CRP levels to future cardiovascular disease risk (Ridker, Hennekens, Buring, & Rifai, 2000). Values greater than 10mg/L are generally time-limited and suggestive of acute infection, rendering such values as clinically non-useful for determining cardiovascular risk. However, individuals who have a CRP value between 3mg/L and 10mg/L ("high risk") are at approximately 35 – 75% higher risk for incident heart disease than individuals who have a CRP value of less than 1mg/L ("low risk"), and individuals who have a CRP value between 1mg/L and 3mg/L ("average risk") are at approximately 10 – 30% higher risk for incident heart disease than individuals who have a CRP value of less than 1mg/L (Buckley, Fu, Freeman, Rogers, & Helfand, 2009).

In the current study, CRP was quantified in participants' serum. For this, I drew 8ml of blood into a serum-separating Vacutainer tube (BD Biosciences, Mississuaga, ON). I then centrifuged the tube within 30 – 120 minutes of the blood draw at 1200 RCF for 10 minutes. Next, I drew a 1 mL aliquot of serum from the tube and stored it in our laboratory freezer at -30° Celsius until the end of the study when samples were analyzed in batch. Assays were run by a local hospital laboratory following standard protocols that can detect CRP values as low as 0.19 mg/L. Participants who had an undetectably low value for CRP were entered into the data file as 0.19 mg/L (n = 3). Conversely, I dropped CRP values greater than 10 mg/L (n = 3). Of the remaining 57 participants, a total of 19 individuals had a CRP value in the "low risk" range (i.e., < 1mg/L), 25 individuals had a CRP value in the "average risk" range (i.e., 1mg/L – 3mg/L), and 13 individuals had a CRP value in the "high risk" range (i.e., 3mg/L – 10mg/L). I used these CRP data to generate two variables. The first was simply a continuous variable reflecting the serum levels of CRP for each participant. The second variable was a binary variable indicating whether an individual had a CRP value below (coded as 0) versus above (coded as 1) 3mg/L.

Lipoprotein-associated phospholipase A_2 is an enzyme released primarily by macrophages that is bound to low density lipoproteins in circulation (Oei et al., 2005). Heightened levels of Lp-PLA_2 are a risk factor for cardiovascular disease and ischemic stroke independent of other risk factors such as CRP and cholesterol levels, with each one standard deviation increase in Lp-PLA_2 being associated with approximately a 10 – 15% higher risk of heart disease (The Lp-PLA_2 Studies Collaboration, 2010). Unlike CRP, Lp-PLA_2 is not sensitive to acute infection, but rather appears to be a more specific marker of vascular inflammation due to atherosclerotic plaques (Ballantyne et al., 2005; Oei et al., 2005). Clinically, Lp-PLA_2 values less than 200 ng/mL are considered "low risk," values between 200ng/mL and 235ng/mL are

considered "average risk," and values greater than 235ng/mL are considered "high risk" for cardiovascular disease and stroke (Davidson et al., 2008).

In the current study, Lp-PLA_2 was measured in serum. For this, I drew 8ml of blood into a serum-separating Vacutainer tube (BD Biosciences, Mississauga, ON) and centrifuged the tube within 30 – 120 minutes of the blood draw at 1200 RCF for 10 minutes. Next, I drew a 0.5 mL aliquot of serum from the tube and stored it in our laboratory freezer at -30° Celsius until the end of the study. After the study was complete, I batch analyzed the samples in duplicate using commercially available enzyme-linked immunosorbent assay kits (DPLG70; R&D Systems, Minneapolis, MN) on an ELx808 Absorbance Reader (BioTek, Winooski, VT), which have a minimum detection threshold of 0.074 ng/mL and a manufacturer reported inter- and intraassay variability of less than 10% and 7% respectively. The average intraassay coefficient of variation in the current sample was 3.61% (*SD* = 2.41%, range = 0.00% – 9.69%). Lp-PLA_2 values in the current sample were overall lower than expected, especially for a midlife population with a high proportion of obese individuals (*M* = 112.91 ng/mL, *SD* = 37.17 ng/mL, range = 50.25ng/mL – 214.44ng/mL), with only two participants having a value in the "average risk" range (i.e., 200ng/mL – 235ng/mL) and no participants having a value in the "high risk" range (i.e., > 235ng/mL). It is unclear why values were so low, though the prevalent use of medications for managing metabolic and cardiovascular risk factors (especially the use of statins) in the current sample may have resulted in therapeutically lowered levels of Lp-PLA_2 in people who might be expected to have otherwise had higher values based on clustering of other risk factors (Albert, Glynn, Wolfert, & Ridker, 2005). Due to the lack of clinically relevant variability in Lp-PLA_2 values in the current sample, I did not analyze them on their own, but rather used them to help generate a composite inflammatory variable that will be described below.

Serum levels of the inflammatory cytokines interleukin-6, interleukin-8, interleukin-10, interferon-γ, and tumor necrosis factor α were measured in the current study using a MesoScale Discovery multiplexing instrument (SECTOR Imager 2400, Gaithersburg, MD) with the commercially available Human Pro-Inflammatory 7-Plex Kit (MesoScale Diagnostics, Rockville, MD)[3]. For this protocol, I drew 8ml of blood into a serum-separating Vacutainer tube (BD Biosciences, Mississuaga, ON). I then centrifuged the tube within 30 – 120 minutes of the blood draw at 1200 RCF for 10 minutes. Next, I drew a 0.5 mL aliquot of serum and stored it at -30° Celsius until the end of the study. After the study was complete, I batch analyzed the samples in duplicate following instructions provided by the assay kit manufacturer. The manufacturer reports an average inter- and intraassay variability of less than 13.0% across cytokines. The minimum detection threshold for each cytokine was 0.18pg/mL for IL-6, 0.10pg/mL for IL-8, 0.57pg/mL for IL-10, 0.80pg/mL for IFNγ, and 0.28mg/mL for TNFα. In the current sample, the average intraassay coefficients of variation were 3.60% (*SD* = 2.12%, range = 0.00% – 8.39%) for IL-6, 3.30% (*SD* = 2.28%, range = 0.01% – 8.60%) for IL-8, 5.29% (*SD* = 2.67%, range = 0.00% – 9.85%) for IL-10, 5.19% (*SD* = 2.89%, range = 0.05% – 10.17%) for IFNγ, and 3.80% (*SD* = 2.09%, range = 0.24% – 9.10%) for TNFα.

Finally, in order to reduce the number of regressions being modeled, and in recognition that the assessed inflammatory cytokines tend to co-vary both together and with CRP and Lp-PLA_2, I created a composite inflammatory variable by *z*-scoring and then summing values for CRP, Lp-PLA_2, IL-6, IL-8, IL-10, IFNγ, and TNFα. To justify the creation of this composite

[3] This assay also quantifies serum levels of interleukin-1β and interleukin-12p70, however levels of these cytokines in the current sample were too low to be reliably assessed for most individuals.

variable, I ran a confirmatory factor analysis using the statistical software R (R Core Team, 2015) and the R package "lavaan" for structural equation modelling (Rosseel, 2012) with each of the seven inflammatory variables loading onto a single latent variable for inflammation. The model fit the data, $\chi^2(14) = 16.69$, $p = .270$. Fit indices based off of both the covariance matrix and the observed variable means also indicated reasonable fit, especially considering the small sample size, Bollen's *IFI* = 0.90, *MFI* = 0.98, *RMSEA* = 0.06. I also modeled CRP separately in my analyses as it is a clinically relevant marker of cardiovascular risk on its own. As discussed above, I did not model Lp-PLA_2 separately, however, due to lack of variability outside of the "low risk" range.

Data Analysis

Basic Approach. I analyzed data for this study using the statistical software package SPSS (Version 23; IBM Corporation, 2014), as well as the SPSS macro "Process" for probing regression interactions (Hayes, 2013). To test my hypotheses, I evaluated a series of either multiple linear or binary logistic regression models. Analyses were based on all available data for each set of models. All *p*-values presented are two-tailed and confidence intervals were set at 90%. This was an exploratory pilot study for guiding future research in this area. As such, I set α at the more liberal level of .10 rather than the conventional .05 level. Raising α to .10 increases the risk of making a Type I error (i.e., incorrectly rejecting a true null hypothesis); thus, the results from this study should be interpreted with caution and considered as more of a preliminary roadmap for future research in this area to replicate and extend.

There were six main outcomes that I modeled: (1) self-reported health, (2) the number of components related to metabolic syndrome participants presented with, (3) composite metabolic risk, (4) CRP levels, (5) whether or not participants had a CRP value in the "high risk" range

(i.e., 3mg/L – 10mg/L), and (6) the composite inflammatory variable. Outcomes 1, 2, 3, 4 and 6 were predicted using multiple linear regression. Outcome 5 was predicted using binary logistic regression. For each outcome, I analyzed in separate models both (1) the associations between FLSA Time 1 redemption, contamination, and the interaction between redemption and contamination and the given outcome variable of interest, and (2) the associations between FLSA Time 5 redemption, contamination, and the interaction between redemption and contamination and the given outcome variable of interest[4, 5].

Model Building and Adjustments for Covariates. I analyzed all outcome variables of interest in a series of four progressively more comprehensive models, labeled Model 0 – Model 3. Model 0 refers to the base model with no adjustment for covariates. In this model, the outcome variable of interest was simply predicted by Time 1 or 5 redemption, Time 1 or 5 contamination, and the interaction between Time 1 or 5 redemption and Time 1 or 5 contamination. Model 1

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[4] I also analyzed whether or not changes in the presence or absence of redemption/contamination imagery from FLSA Time 1 to Time 5 predicted any of the outcomes of interest. However, these analyses did not yield anything noteworthy. As such, in the interest of keeping the total number of analyses reported on to a more reasonable number, I have omitted these results.

[5] Another analytic option would be to average the Time 1 and Time 5 values of both codes, based on the notion that such an approach could result in a more stable, psychometrically optimal evaluation of people's tendencies toward telling redemption and contamination stories. However, this approach would actually provide less information than looking at Time 1 and Time 5 models separately. Specifically, in this formulation, a person could have one of three values for both redemption and contamination: 0, 0.5, and 1. A value of 0 would indicate that redemption/contamination was not reported at either Time 1 or Time 5, a value of 0.5 would indicate that redemption/contamination was reported at one of the two time points (but would not reflect which time point), and a value of 1 would indicate that redemption/contamination was reported at both time points. This approach does not actually offer improved resolution for understanding potentially differential associations between life story codes and health based on timing. The more optimal way to conduct such an analysis would involve creating four dummy codes to capture all the different combinations of ways people could report redemption and contamination throughout the study. However, the resulting full-factorial model would involve a 4-way interaction. Unfortunately, with only 61 participants such an analysis is not feasible.

adjusted Model 0 for the demographic covariates gender, race, and composite socioeconomic status (in the interest of parsimony, age was not included as a covariate because participants were all similar in age by study design). Model 2 further adjusted Model 1 for the personal characteristic variables depressive symptoms (from the BDI-II), waist circumference, and the use of prescription medications for managing either lipids, blood sugar, or hypertension (except in analyses looking at components of the metabolic syndrome and composite metabolic risk as outcomes; in these analyses, waist circumference and medication use were not included as covariates as they are themselves part of metabolic syndrome). I centered the variables for gender, race, socioeconomic status, depressive symptoms, waist circumference, and prescription medication around a mean of 0. While mean centering binary variables such as gender, race, and medication use may at first seem unintuitive, it is an accepted statistical practice that allows for examining the nature of intercepts and regression slopes regardless of individual differences in the various binary variables (for further discussions about centering binary variables, see Bryk & Raudenbush, 2002; Hox, 2010). Finally, Model 3 adjusted Model 2 additionally for the presence of redemption and contamination at the other FLSA time point. That is, in models predicting the given outcome variables of interest as a function of Time 1 redemption, contamination, and the interaction term, Model 3 further adjusted for the presence of Time 5 redemption and contamination. Likewise, in models predicting the given outcome variables of interest as a function of Time 5 redemption, contamination, and the interaction term, Model 3 further adjusted for the presence of Time 1 redemption and contamination. Thus, this model provides an answer to the question: what is the association between redemption and contamination at one time point and the outcome variable of interest, independent of whether or not the participant reported redemption and contamination imagery at the other time point? All figures presented in this

manuscript are based on the regression estimates furnished by Model 3. Likewise, all results described in the results section of this manuscript are from Model 3. However, full results for Models 0 – 2 (as well as Model 3) for the various regressions can be found in Tables 4 – 15.

Results

Descriptive Information

Participants in this study were on average just over 61 years old at the time of the HSP visit (*SD* = 1.01, range = 59 – 63). Of the 61 participants, 24 (39.3%) were male and 37 (60.7%) were female. Additionally, 34 participants (55.7%) identified as Caucasian and 27 (44.3%) identified as African American. While this sample tended toward being higher in socioeconomic status (SES) on average, it was also socioeconomically diverse. The median family annual income was $75,000 – $99,999, and the range of income represented in the sample went from $5,000 – $11,999 to $100,000 and greater. The median educational degree participants had achieved was a bachelor degree, and the range of degrees represented in the sample went from high school diploma to doctoral/professional degrees. Finally, the median amount of time participants indicated they could survive off of savings at their current standard of living if all sources of income were to go away (i.e., a measure of wealth) was more than a year, and the range of savings represented in the sample went from less than a month to more than a year. Composite SES was associated with participant race, with African American participants tending to be lower in SES and Caucasian participants tending to be higher in SES, $r(59) = -.51$, $p < .001$, 90% CI for $r = [-.66, -.35]$. Full descriptive information for this sample can be found in Table 2. Additionally, simple bivariate associations between all the study predictor, outcome, and covariate variables can be found in Table 3.

Table 2

Descriptive characteristics of the sample (N = 61)

Variable	n	Mean	SD	Median	Range
Age (years)		61.05	1.01	61	59 – 63
Caucasian participants (number)	34				
African American participants (number)	27				
Male participants (number)	24				
Female participants (number)	37				
Highest degree obtained by either participant or participant's partner [a]		3.38	1.39	3	1 – 6
Combined annual family income (United States Dollars) [b]		7.07	2.07	8	2 – 9
Length of time that participant could survive off of savings with no income [c]		3.84	1.45	5	1 – 5
Composite socioeconomic status (SES) measure ($Z_{education} + Z_{income} + Z_{savings}$)		0.00	2.46	0.7	-5.4 – 3.6
Beck Depression Inventory II (BDI-II; 0 – 63)		5.67	6.10	4	0 – 29
Self-reported health (SRH; higher is better; 1 – 6) [d]		4.74	1.00	5	2 – 6
Physical activity limitations due to health (PAL; higher is better; 1 – 5) [e]		4.38	0.82	5	2 – 5
Work limitations due to health (WL; higher is better; 1 – 5) [f]		4.66	0.75	5	2 – 5
Composite of self-reported health measures (higher is better; $Z_{SRH} + Z_{PAL} + Z_{WL}$)		0.00	2.62	0.5	-9.2 – 2.5
Number of participants taking drugs to manage lipids	12				
Number of participants taking drugs to manage blood pressure	17				
Number of participants taking drugs to manage blood sugar	7				
Number of participants taking either lipid, blood pressure, or blood sugar drugs	24				
Number of alcoholic drinks in a typical week		2.56	4.93	0	0 – 23
Number of cigarettes smoked in a typical week		4.95	18.08	0	0 – 100
Waist circumference (cm)		96.14	19.25	94	27.5 – 148.6
Systolic blood pressure (mmHg)		123.11	16.51	120	96 – 170
Diastolic blood pressure (mmHg)		71.38	8.35	71	54 – 92
Total cholesterol (mg/dL)		196.50	35.29	196	108 – 284
Glycosylated hemoglobin (HbA1c; %)		5.64	0.69	5.4	4.7 – 8.6
Composite metabolic risk [g]		-0.16	3.61	-0.78	-7.6 – 8.8
Number of features of metabolic syndrome present (0 – 4) [h]		1.65	1.11	1	0 – 4
C-reactive protein (CRP; mg/L)		2.36	2.24	1.6	0.2 – 8.5
Lipoprotein-associated phospholipase A_2 (Lp-PLA_2; ng/mL)		112.91	37.17	111.2	50.3 – 214.4
Interferon gamma (IFNγ; pg/mL)		1.90	3.05	1.09	0.3 – 20.0

Tumor necrosis factor alpha (TNFα; pg/mL)	3.10	1.00	3.0	1.0 – 6.1
Interleukin-6 (IL-6; pg/mL)	1.21	0.72	1.0	0.3 – 4.3
Interleukin-8 (IL-8; pg/mL)	16.03	6.72	15.1	5.4 – 32.4
Interleukin-10 (IL-10; pg/mL)	2.62	5.01	1.6	0.6 – 33.7
Composite inflammation ($Z_{CRP} + Z_{Lp\text{-}PLA2} + Z_{IFN\gamma} + Z_{TNF\alpha} + Z_{IL\text{-}6} + Z_{IL\text{-}8} + Z_{IL\text{-}10}$)	0.00	3.07	-0.2	-5.07 – 7.00
Number of participants with at least one redemption sequence at time 1	40			
Number of participants with at least one contamination sequence at time 1	36			
Number of participants with at least one redemption sequence at time 5	31			
Number of participants with at least one contamination sequence at time 5	32			

[a] Categories for educational degree obtainment were 0 = Less than high school, 1 = High school diploma or GED, 2 = Associate degree, 3 = Bachelor's degree, 4 = Master's degree, 5 = Doctorate, 6 = Professional degree (e.g., MD, DDS, JD).

[b] Categories for family income were 1 = less than $5,000, 2 = $5,000 – $11,999, 3 = $12,000 – $15,999, 4 = $16,000 – $24,999, 5 = $24,000 – $34,999, 6 = $35,000 – $49,000, 7 = $50,000 – $74,999, 8 = $75,000 – $99,999, 9 = $100,000 and greater.

[c] Categories for how long participant could maintain current standard of living if all sources of income were lost were 1 = Less than 1 month, 2 = 1 – 2 months, 3 = 3 – 6 months, 4 = 7 – 12 months, 5 = More than a year.

[d] Categories for self-reported health over past 4-weeks were 1 = Very poor, 2 = Poor, 3 = Fair, 4 = Good, 5 = Very good, 6 = Excellent.

[e] Categories for physical activity limitations over past 4-weeks due to health were 1 = Could not do physical activities, 2 = Impaired quite a lot, 3 = Somewhat impaired, 4 = Impaired very little, 5 = Not at all impaired

[f] Categories for limitations on ability to work over past 4-weeks due to health were 1 = Could not do daily work, 2 = Impaired quite a lot, 3 = Impaired some, 4 = A little bit impaired, 5 = Not at all impaired

[g] Composite metabolic risk was calculated by summing *z*-scores for waist circumference (calculated separately for men and women), systolic blood pressure, diastolic blood pressure, total cholesterol, glycosylated hemoglobin, blood sugar medication use, and hypertension mediation use.

[h] Criteria for metabolic syndrome were based on guidelines outlined by the American Heart Association. These include 1) a larger waist circumference, 2) higher fasting triglyceride levels, 3) lower fasting HDL cholesterol levels, 4) elevated blood pressure or use of blood pressure medication, and 5) higher fasting blood sugar levels or use of blood sugar medication. Metabolic syndrome is assumed if an individual presents with at least three of these criteria simultaneously. However, because fasting blood draws were not possible in the current study, I used the following criteria as indicators of the elements of metabolic syndrome: 1) a waist circumference > 102cm for men or > 88cm for women, 2) total cholesterol ≥ 200 mg/dL, 3) either a glycosylated hemoglobin amount ≥ 5.7% or use of medications for controlling blood sugar, 4) either a systolic blood pressure ≥ 130 mmHg, or a diastolic blood pressure ≥ 85 mmHg, or use of medications for managing hypertension.

Table 3

Bivariate associations between all predictor, outcome, and covariate variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Gender	1															
2. Race	0.18	1														
3. Composite SES	-0.09	-.51**	1													
4. BDI-II	-0.05	-.22†	-.25*	1												
5. Self-Reported Health	.24†	-.22†	.43**	-.29*	1											
6. Waist Circumference	-.43**	0.11	-0.07	0.02	-.34**	1										
7. Medication Use	-.24†	.23†	-0.15	-0.01	-.28*	.36**	1									
8. Met. Syn. Components	-0.05	.34**	-0.20	0.01	-.32*	.55**	.55**	1								
9. Metabolic Risk	-0.13	.42**	-.24†	0.06	-.31*	.62**	.55**	.86**	1							
10. Composite Inflammation	0.05	0.07	-0.12	0.14	-0.19	0.19	0.02	0.06	0.04	1						
11. Serum CRP Levels	0.01	0.04	-0.19	0.12	-0.19	0.15	0.14	.24†	0.18	.53**	1					
12. CRP Binary Risk	0.00	0.04	-0.22	0.17	-0.15	0.14	0.10	.28*	.24†	.38**	.89**	1				
13. Time 1 Redemption	-0.09	-0.12	-0.05	0.10	0.09	-0.06	0.09	-0.14	-0.08	-0.16	-.23†	-0.15	1			
14. Time 1 Contamination	0.15	0.00	-0.11	-0.19	0.04	-0.10	-0.08	-0.06	-0.06	0.16	0.12	0.11	-0.04	1		
15. Time 5 Redemption	0.03	0.11	-.33*	-0.09	0.11	-0.17	0.01	-0.09	-0.17	0.00	-0.13	-0.15	0.02	0.03	1	
16. Time 5 Contamination	-0.08	-0.06	-.23†	0.12	-0.06	-0.15	-0.16	-0.14	-0.29	0.11	-0.17	-0.17	-0.02	-0.08	.30*	1

Note: SES = Socioeconomic Status; BDI = Beck Depression Inventory; Met. Syn. = Metabolic Syndrome; CRP = C-Reactive Protein

$^{\dagger}p < .10$; $^{*}p < .05$; $^{**}p < .01$

Self-Reported Health

Time 1 Redemption and Contamination. I did not find evidence for an association between the presence of redemption imagery at FLSA Time 1 and participants' self-reported health during the HSP study visit as estimated in Model 3, $\beta = 0.22$, $b = 1.22$, $SE = 1.14$, $p = .290$, 90% CI for $b = [-0.69, 3.13]$. Likewise, I did not find evidence for an association between the presence of contamination imagery at FLSA Time 1 and participants' self-reported health during the HSP study visit as estimated in Model 3, $\beta = 0.10$, $b = 0.51$, $SE = 1.20$, $p = .672$, 90% CI for $b = [-1.49, 2.51]$. Finally, I did not find evidence for an interaction between the presence of redemption and contamination imagery at FLSA Time 1 predicting participants' self-reported health during the HSP study visit as estimated in Model 3, $\beta = -0.13$, $b = -0.69$, $SE = 1.51$, $p = .650$, 90% CI for $b = [-3.22, 1.84]$. Refer to Table 4 for the full results from Models 0 – 3.

Table 4

Regression results for modeling self-reported health as a function of Redemption and Contamination at Time 1

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	-1.09 (0.94)	.251		[-2.65, 0.48]	-1.15 (0.88)	.197		[-2.63, 0.33]
Time 1 Redemption	1.40 (1.14)	.224	0.26	[-0.50, 3.30]	1.46 (1.09)	.188	0.27	[-0.37, 0.75]
Time 1 Contamination	1.23 (1.19)	.307	0.23	[-0.76, 3.22]	1.03 (1.12)	.365	0.19	[-0.85, 2.91]
Time 1 Redemption × Time 1 Contamination	-1.47 (1.46)	.318	-0.28	[-3.92, 0.97]	-1.08 (1.40)	.443	-0.20	[-3.43, 1.26]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	-0.51 (0.98)	.609		[-2.15, 1.14]	-0.93 (1.09)	.399		[-2.75, 0.90]
Time 1 Redemption	1.52 (1.06)	.159	0.28	[-0.26, 3.30]	1.22 (1.14)	.290	0.22	[-0.69, 3.13]
Time 1 Contamination	0.79 (1.13)	.490	0.15	[-1.11, 2.69]	0.51 (1.20)	.672	0.10	[-1.49, 2.51]
Time 1 Redemption × Time 1 Contamination	-1.23 (1.39)	.383	-0.23	[-3.56, 1.11]	-0.69 (1.51)	.650	-0.13	[-3.22, 1.84]

Note: CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for the personal characteristic variables depressive symptoms (BDI-II), waist circumference, and the use of prescription medications for managing either lipids, blood sugar, or hypertension.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 5 Redemption and Time 5 Contamination.

Time 5 Redemption and Contamination. I did not find evidence for an association between the presence of redemption imagery at FLSA Time 5 and participants' self-reported health during the HSP study visit as estimated in Model 3, β = -0.01, b = -0.03, SE = 0.92, p = .975, 90% CI for b = [-1.57, 1.51]. Likewise, I did not find evidence for an association between the presence of contamination imagery at FLSA Time 5 and participants' self-reported health during the HSP study visit as estimated in Model 3, β = -0.25, b = -1.34, SE = 0.92, p = .154, 90% CI for b = [-2.89, 0.21]. However, the interaction between the presence of redemption and contamination imagery at FLSA Time 5 was significantly associated with differences in self-reported health during the HSP study visit as estimated in Model 3 (see Figure 1), β = 0.42, b = 2.32, SE = 1.28, p = .075, 90% CI for b = [0.18, 4.47]. Simple slopes analyses indicated that among individuals who reported contamination imagery at Time 5, those that did not also report redemption imagery had poorer self-reported health than those who did report redemption imagery, b = 2.29, SE = 0.91, p = .015, 90% CI for b = [0.78, 3.81]. Conversely, among individuals who did not report contamination imagery at Time 5, there was no difference in self-reported health regardless of whether or not participants reported redemption imagery, b = -0.03, SE = 0.92, p = .975, 90% CI for b = [-1.57, 1.51]. Refer to Table 5 for the full results from Models 0 – 3.

Table 5

Regression results for modeling self-reported health as a function of Redemption and Contamination at Time 5

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	0.21 (0.63)	.745		[-0.84, 1.25]	-0.43 (0.54)	.428		[-1.34, 0.47]
Time 5 Redemption	-0.09 (1.05)	.933	-0.02	[-1.84, 1.66]	0.50 (0.89)	.573	0.10	[-0.98, 1.99]
Time 5 Contamination	-1.27 (1.02)	.218	0.24	[-2.97, 0.43]	-0.83 (0.85)	.334	-0.16	[-2.26, 0.60]
Time 5 Redemption × Time 5 Contamination	1.51 (1.44)	.299	1.51	[-0.90, 3.92]	1.82 (1.21)	.138	0.33	[-0.20, 3.85]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	0.27 (0.66)	.682		[-0.83, 1.37]	-0.29 (0.96)	.766		[-1.90, 1.33]
Time 5 Redemption	-0.11 (0.91)	.905	-0.02	[-1.63, 1.41]	-0.03 (0.92)	.975	-0.01	[-1.57, 1.51]
Time 5 Contamination	-1.47 (0.91)	.111	-0.28	[-2.99, 0.05]	-1.34 (0.92)	.154	-0.25	[-2.89, 0.21]
Time 5 Redemption × Time 5 Contamination	2.47 (1.26)	.055	0.45	[0.36, 4.57]	2.32 (1.28)	.075	0.42	[0.18, 4.47]

Note: CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for the personal characteristic variables depressive symptoms (BDI-II), waist circumference, and the use of prescription medications for managing either lipids, blood sugar, or hypertension.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 1 Redemption and Time 1 Contamination.

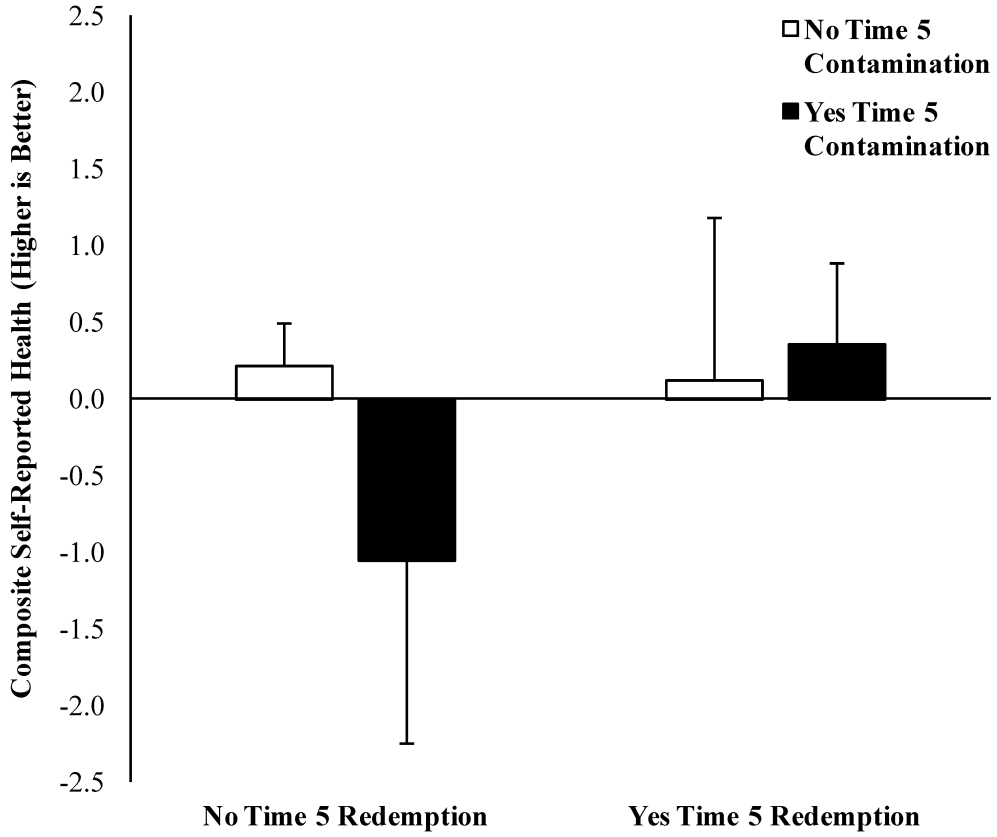

Figure 1. Mean differences in the composite measure of self-reported health as a function of the interaction between having experienced redemption at Time 5 and having experienced contamination at Time 5 from Model 3, $b = 2.32$, $SE = 1.28$, $p = .075$, 90% CI for $b = [0.18, 4.47]$. Simple slopes analyses indicated that among individuals who experienced contamination at Time 5, those that did not also experience redemption had poorer self-reported health than those who did experience redemption, $b = 2.29$, $SE = 0.91$, $p = .015$, 90% CI for $b = [0.78, 3.81]$. Conversely, among individuals who did not experience contamination at Time 5, there was no difference in self-reported health regardless of whether participants had experienced redemption, $b = -0.03$, $SE = 0.92$, $p = .975$, 90% CI for $b = [-1.57, 1.51]$. Error bars represent 1 standard error.

Metabolic Syndrome Risk Components

Time 1 Redemption and Contamination. I did not find evidence for an association between the presence of redemption imagery at FLSA Time 1 and the number of components related to metabolic syndrome participants presented with during the HSP study visit as estimated in Model 3, $\beta = 0.23$, $b = 0.56$, $SE = 0.59$, $p = .346$, 90% CI for b = [-0.43, 1.54]. Likewise, I did not find evidence for an association between the presence of contamination imagery at FLSA Time 1 and the number of components related to metabolic syndrome participants presented with during the HSP study visit as estimated in Model 3, $\beta = 0.37$, $b = 0.85$, $SE = 0.64$, $p = .191$, 90% CI for b = [-0.23, 1.93]. However, the interaction between the presence of redemption and contamination imagery at FLSA Time 1 showed a trend toward being associated with differences in the number of components related to metabolic syndrome participants presented with during the HSP study visit as estimated in Model 3 (see Figure 2), $\beta = -1.64$, $b = -1.25$, $SE = 0.77$, $p = .108$, 90% CI for b = [-2.54, 0.03]. Simple slopes analyses indicated that among individuals who reported contamination imagery at Time 1, those that did not also report redemption imagery trended toward having more components related to metabolic syndrome than those who did report redemption imagery, $b = -0.70$, $SE = 0.43$, $p = .109$, 90% CI for b = [-1.41, 0.02]. Conversely, among individuals who did not report contamination imagery at Time 1, there was no difference in number of components related to metabolic syndrome regardless of whether or not participants reported redemption imagery, $b = 0.56$, $SE = 0.59$, $p = .346$, 90% CI for b = [-0.43, 1.54]. Refer to Table 6 for the full results from Models 0 – 3.

Table 6

Regression results for modeling components of metabolic syndrome as a function of Redemption and Contamination at Time 1.

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	2.00 (0.43)	< .001		[1.28, 2.72]	1.57 (0.43)	.001		[0.86, 2.29]
Time 1 Redemption	-0.44 (0.51)	.397	-0.19	[-1.30, 0.42]	0.18 (0.52)	.733	0.08	[-0.69, 1.05]
Time 1 Contamination	-0.27 (0.55)	.620	-0.12	[-1.19, 0.64]	0.45 (0.57)	.434	0.20	[-0.50, 1.40]
Time 1 Redemption × Time 1 Contamination	0.28 (0.66)	.678	0.12	[-0.83, 1.38]	-0.71 (0.69)	.305	-0.32	[-1.87, 0.44]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	1.43 (0.50)	.006		[0.59, 2.27]	1.65 (0.51)	.002		[0.80, 2.51]
Time 1 Redemption	0.21 (0.53)	.691	0.09	[-0.67, 1.09]	0.56 (0.59)	.346	0.23	[-0.43, 1.54]
Time 1 Contamination	0.54 (0.59)	.369	0.24	[-0.46, 1.53]	0.85 (0.64)	.191	0.37	[-0.23, 1.93]
Time 1 Redemption × Time 1 Contamination	-0.77 (0.70)	.279	-0.34	[-1.94, 0.41]	-1.25 (0.77)	.108	-1.64	[-2.54, 0.03]

Note: CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for depressive symptoms (BDI-II). Unlike the other analyses, the covariates waist circumference and medication use were not entered in models related to metabolic syndrome as they are components of metabolic syndrome.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 5 Redemption and Time 5 Contamination.

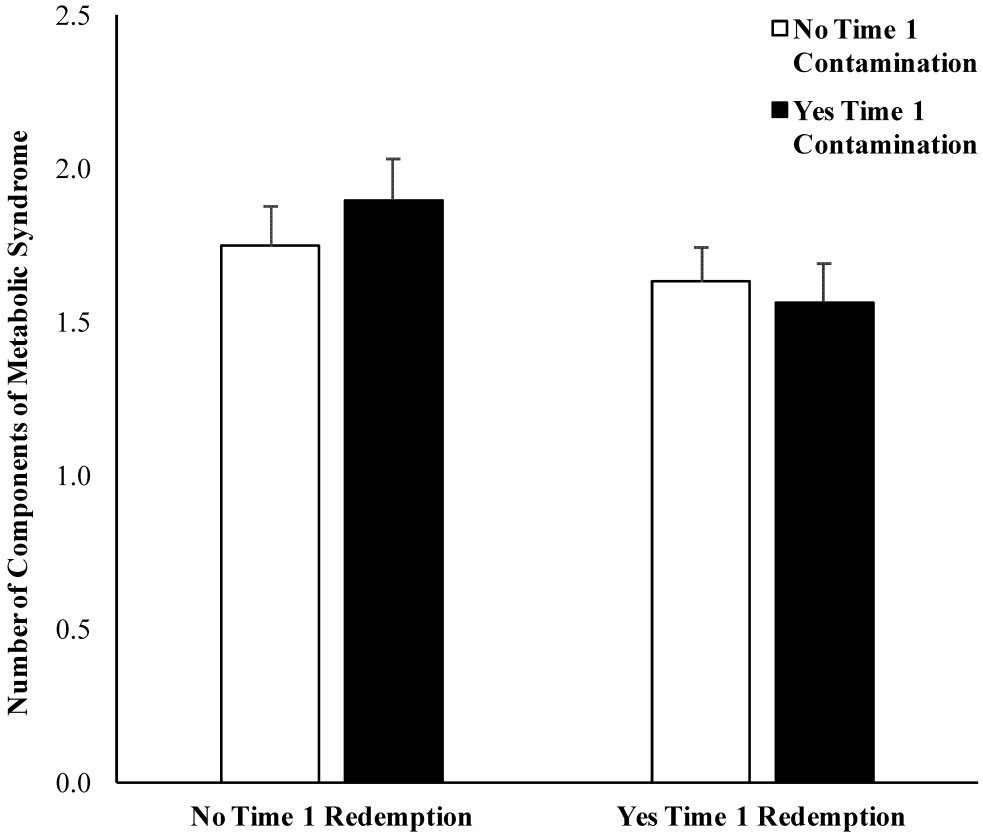

Figure 2. Mean differences in the number of components related to metabolic syndrome participants had as a function of the interaction between having experienced redemption at Time 1 and having experienced contamination at Time 1 from Model 2, b = -1.25, SE = 0.77, p = .108, 90% CI for b = [-2.55, 0.03]. Simple slopes analyses indicated that among individuals who reported contamination imagery at Time 1, those that did not also report redemption imagery trended toward having more components related to metabolic syndrome than those who did report redemption imagery, b = -0.70, SE = 0.43, p = .109, 90% CI for b = [-1.41, 0.02]. Conversely, among individuals who did not report contamination imagery at Time 1, there was no difference in number of components related to metabolic syndrome regardless of whether or not participants reported redemption imagery, b = 0.56, SE = 0.59, p = .346, 90% CI for b = [-0.43, 1.54]. Error bars represent 1 standard error.

Time 5 Redemption and Contamination. I found evidence for an association between the presence of redemption imagery at FLSA Time 5 and the number of components related to metabolic syndrome participants presented with during the HSP study visit as estimated in Model 3, β = -0.48, b = -1.03, SE = 0.39, p = .011, 90% CI for b = [-1.68, -0.37]. Reporting redemptive imagery at FLSA Time 5 was associated with fewer components related to metabolic syndrome. Likewise, I found evidence for an association between the presence of contamination imagery at FLSA Time 5 and the number of components related to metabolic syndrome participants presented with during the HSP study visit as estimated in Model 3, β = -0.47, b = -0.99, SE = 0.38, p = .012, 90% CI for b = [-1.63, -0.36]. This association was opposite of what I expected. Specifically, reporting contamination imagery at FLSA Time 5 was also associated with fewer components related to metabolic syndrome. Additionally, the interaction between the presence of redemption and contamination imagery at FLSA Time 5 predicted differences in the number of components related to metabolic syndrome participants presented with during the HSP study visit as estimated in Model 3 (see Figure 3), β = 0.79, b = 1.82, SE = 0.56, p = .002, 90% CI for b = [0.88, 2.76]. Simple slopes analyses indicated that among individuals who did not report contamination imagery at Time 5, those that did report redemption imagery had fewer components related to metabolic syndrome than those who did not report redemption imagery, b = -1.03, SE = 0.39, p = .012, 90% CI for b = [-1.68, 0.37]. Unexpectedly, among individuals who did report contamination imagery at Time 5, those that also reported redemption imagery had more components related to metabolic syndrome on average, b = 0.79, SE = 0.41, p = .061, 90% CI for b = [0.10, 1.49]. Refer to Table 7 for the full results from Models 0 – 3.

Table 7

Regression results for modeling components of metabolic syndrome as a function of Redemption and Contamination at Time 5.

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	2.00 (0.24)	< .001		[1.59, 2.41]	2.08 (0.23)	< .001		[1.69, 2.47]
Time 5 Redemption	-0.80 (0.41)	.055	-0.38	[-1.48, -0.12]	-1.00 (0.38)	.012	-0.47	[-1.64, -0.36]
Time 5 Contamination	-0.91 (0.40)	.026	-0.43	[-1.57, -0.25]	-0.92 (0.37)	.016	-0.43	[-1.53, -0.30]
Time 5 Redemption × Time 5 Contamination	1.47 (0.57)	.013	0.64	[0.52, 2.43]	1.64 (0.53)	.003	0.71	[0.75, 2.54]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	2.03 (0.28)	< .001		[1.55, 2.51]	2.20 (0.42)	< .001		[1.49, 2.91]
Time 5 Redemption	-0.99 (0.39)	.014	-0.46	[-1.64, -0.34]	-1.03 (0.39)	.011	-0.48	[-1.68, -0.37]
Time 5 Contamination	-0.93 (0.37)	.016	-0.44	[-1.55, -0.31]	-0.99 (0.38)	.012	-0.47	[-1.63, -0.36]
Time 5 Redemption × Time 5 Contamination	1.66 (0.54)	.003	0.72	[0.76, 2.57]	1.82 (0.56)	.002	0.79	[0.88, 2.76]

Note: CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for depressive symptoms (BDI-II). Unlike the other analyses, the covariates waist circumference and medication use were not entered in models related to metabolic syndrome as they are components of metabolic syndrome.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 1 Redemption and Time 1 Contamination.

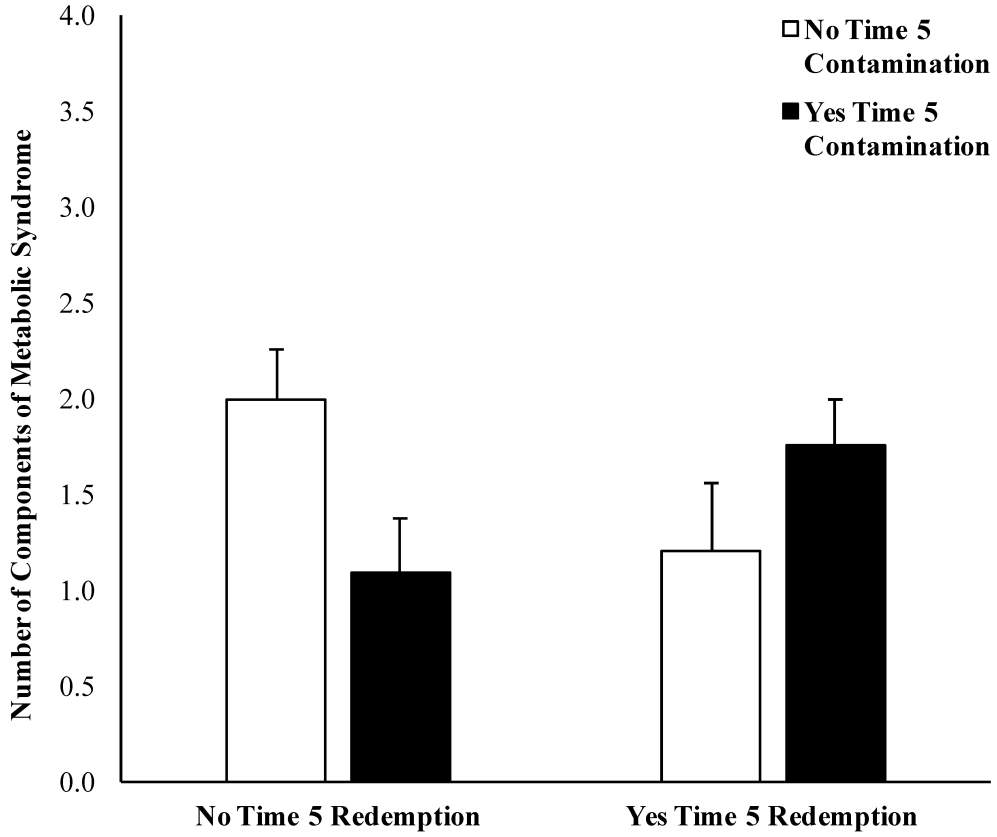

Figure 3. Mean differences in the number of components related to metabolic syndrome participants had as a function of the interaction between having experienced redemption at Time 5 and having experienced contamination at Time 5 from Model 3, $b = 1.82$, $SE = 0.56$, $p = .002$, 90% CI for $b = [0.88, 2.76]$. Simple slopes analyses indicated that among individuals who did not report contamination imagery at Time 5, those that did report redemption imagery had fewer components related to metabolic syndrome than those who did not report redemption imagery, $b = -1.03$, $SE = 0.39$, $p = .012$, 90% CI for $b = [-1.68, -0.37]$. Unexpectedly, among individuals who did report contamination imagery at Time 5, those that also reported redemption imagery had more components related to metabolic syndrome on average, $b = 0.79$, $SE = 0.41$, $p = .061$, 90% CI for $b = [0.10, 1.49]$. Error bars represent 1 standard error.

Composite Metabolic Risk

Time 1 Redemption and Contamination. I did not find evidence for an association between the presence of redemption imagery at FLSA Time 1 and participants' composite metabolic risk as estimated in Model 3, $\beta = 0.11$, $b = 0.82$, $SE = 1.81$, $p = .452$, 90% CI for b = [-2.23, 3.86]. Likewise, I did not find evidence for an association between the presence of contamination imagery at FLSA Time 1 and participants' composite metabolic risk as estimated in Model 3, $\beta = 0.18$, $b = 1.34$, $SE = 1.96$, $p = .500$, 90% CI for b = [-1.97, 4.63]. Finally, there was no evidence for an interaction between the presence of redemption and contamination imagery at FLSA Time 1 and participants' composite metabolic risk as estimated in Model 3, $\beta = -0.30$, $b = -2.18$, $SE = 2.29$, $p = .348$, 90% CI for b = [-6.03, 1.68]. Refer to Table 8 for the full results from Models 0 – 3.

Table 8

Regression results for modeling metabolic risk as a function of Redemption and Contamination at Time 1.

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	1.70 (1.49)	.259		[-0.80, 4.20]	0.58 (1.45)	.693		[-1.86, 3.01]
Time 1 Redemption	-2.18 (1.75)	.218	-0.28	[-5.11, 0.75]	-0.56 (1.72)	.748	-0.07	[-3.45, 2.33]
Time 1 Contamination	-2.26 (1.85)	.228	-0.31	[-5.37, 0.84]	-0.01 (1.86)	.997	-0.001	[-3.13, 3.11]
Time 1 Redemption × Time 1 Contamination	2.50 (2.21)	.263	0.34	[-1.20, 6.20]	-0.34 (2.23)	.880	-0.05	[-4.08, 3.40]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	0.12 (1.50)	.935		[-2.39, 2.64]	1.06 (1.61)	.659		[-1.64, 3.76]
Time 1 Redemption	-0.26 (1.74)	.881	-0.03	[-3.18, 2.65]	0.82 (1.81)	.452	0.11	[-2.23, 3.86]
Time 1 Contamination	0.66 (1.94)	.735	0.09	[-2.60, 3.92]	1.34 (1.96)	.500	0.18	[-1.97, 4.63]
Time 1 Redemption × Time 1 Contamination	-0.74 (2.25)	.743	-0.10	[-4.52, 3.03]	-2.18 (2.29)	.348	-0.30	[-6.03, 1.68]

Note: CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for depressive symptoms (BDI-II). Unlike the other analyses, the covariates waist circumference and medication use were not entered in models related to metabolic syndrome as they are components of metabolic syndrome.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 5 Redemption and Time 5 Contamination.

Time 5 Redemption and Contamination. I found evidence for an association between the presence of redemption imagery at FLSA Time 5 and participants' composite metabolic risk as estimated in Model 3, β = -0.34, b = -2.44, SE = 1.28, p = .064, 90% CI for b = [-4.59, -0.28]. Reporting redemptive imagery at FLSA Time 5 was associated with lower composite metabolic risk. Likewise, I found evidence for an association between the presence of contamination imagery at FLSA Time 5 and participants' composite metabolic risk as estimated in Model 3, β = -0.34, b = -3.66, SE = 1.21, p = .004, 90% CI for b = [-5.69, -1.63]. Similar to the results for components related to metabolic syndrome, this association was opposite of what I expected. Specifically, reporting contamination imagery at FLSA Time 5 was also associated with less metabolic risk. Additionally, the interaction between the presence of redemption and contamination imagery at FLSA Time 5 predicted differences in participants' metabolic risk as estimated in Model 3 (see Figure 4), β = 0.45, b = 3.54, SE = 1.82, p = .058, 90% CI for b = [0.48, 6.60]. Simple slopes analyses indicated that among individuals who did not report contamination imagery at Time 5, those that did report redemption imagery had lower metabolic risk than those who did not report redemption imagery, b = -2.44, SE = 1.28, p = .063, 90% CI for b = [-4.59, -0.28]. Conversely, among individuals who did report contamination imagery at Time 5, those that also reported redemption imagery had qualitatively more components of metabolic syndrome on average, though this association was not statistically significant, b = 1.11, SE = 1.34, p = .412, 90% CI for b = [-1.14, 3.35]. Refer to Table 9 for the full results from Models 0 – 3.

Table 9

Regression results for modeling metabolic risk as a function of Redemption and Contamination at Time 5.

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	1.50 (0.82)	.072		[0.13, 2.87]	1.95 (0.75)	.012		[0.70, 3.20]
Time 5 Redemption	-2.12 (1.41)	.140	-0.30	[-4.49, 0.25]	-2.51 (1.26)	.052	-0.35	[-4.63, -0.40]
Time 5 Contamination	-3.13 (1.33)	.022	-0.44	[-5.35, -0.91]	-3.36 (1.17)	.006	-0.47	[-5.33, -1.39]
Time 5 Redemption × Time 5 Contamination	2.72 (1.96)	.171	0.35	[-0.56, 6.01]	3.01 (1.74)	.091	0.39	[0.08, 5.93]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	1.89 (0.75)	.015		[0.63, 3.14]	2.60 (1.24)	.041		[0.52, 4.67]
Time 5 Redemption	-2.40 (1.27)	.064	-0.34	[-4.52, -0.28]	-2.44 (1.28)	.064	-0.34	[-4.59, -0.28]
Time 5 Contamination	-3.48 (1.18)	.005	-0.49	[-5.46, -1.50]	-3.66 (1.21)	.004	-0.51	[-5.69, -1.63]
Time 5 Redemption × Time 5 Contamination	3.24 (1.76)	.072	0.42	[-0.29, 6.19]	3.54 (1.82)	.058	0.45	[0.48, 6.60]

Note: CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for depressive symptoms (BDI-II). Unlike the other analyses, the covariates waist circumference and medication use were not entered in models related to metabolic syndrome as they are components of metabolic syndrome.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 1 Redemption and Time 1 Contamination.

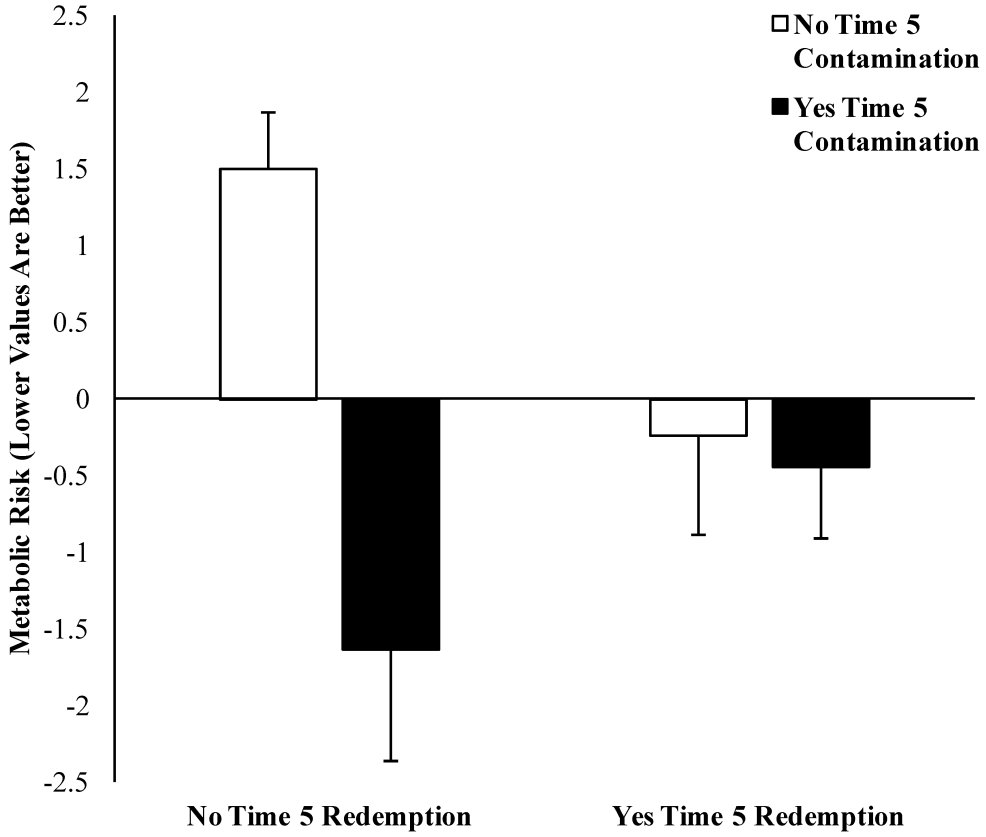

Figure 4. Mean differences in composite metabolic risk as a function of the interaction between having experienced redemption at Time 5 and having experienced contamination at Time 5 from Model 3, $b = 3.54$, $SE = 1.82$, $p = .058$, 90% CI for $b = [0.48, 6.60]$. Simple slopes analyses indicated that among individuals who did not report contamination imagery at Time 5, those that did report redemption imagery had lower composite metabolic risk than those who did not report redemption imagery, $b = -2.44$, $SE = 1.28$, $p = .063$, 90% CI for $b = [-4.59, -0.28]$. Conversely, similar to the unexpected findings for metabolic syndrome components, among individuals who did report contamination imagery at Time 5, those that also reported redemption imagery had qualitatively higher composite metabolic risk on average, though this association was not statistically significant, $b = 1.11$, $SE = 1.34$, $p = .412$, 90% CI for $b = [-1.14, 3.35]$. Error bars represent 1 standard error.

C-Reactive Protein Levels

Time 1 Redemption and Contamination. I did not find evidence for an association between the presence of redemption imagery at FLSA Time 1 and CRP levels at the time of the HSP study visit as estimated in Model 3, $\beta = 0.14$, $b = 0.69$, $SE = 1.15$, $p = .550$, 90% CI for b = [-1.23, 2.61]. However, I did find evidence for a significant association between the presence of contamination imagery at FLSA Time 1 and CRP levels, $\beta = 0.53$, $b = 2.42$, $SE = 1.23$, $p = .055$, 90% CI for b = [0.36, 4.48]. Specifically, individuals who reported contamination imagery at Time 1 had higher CRP levels than those who did not. Furthermore, the interaction between the presence of redemption and contamination imagery at FLSA Time 1 predicted differences in CRP levels as estimated in Model 3 (see Figure 5), $\beta = -0.69$, $b = -3.15$, $SE = 1.52$, $p = .044$, 90% CI for b = [-5.70, -0.60]. Simple slopes analyses indicated that among individuals who reported contamination at Time 1, those who also reported redemption had lower levels of CRP, $b = -2.46$, $SE = 0.85$, $p = .006$, 90% CI for b = [-3.89, -1.03]. Conversely, among individuals who did not report contamination at Time 1, there were no differences in CRP levels regardless of whether or not they reported experiencing redemption, $b = 0.69$, $SE = 1.14$, $p = .550$, 90% CI for b = [-1.23, 2.61]. Refer to Table 10 for the full results from Models 0 – 3.

Table 10

Regression results for modeling C-reactive protein as a function of Redemption and Contamination at Time 1.

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	1.97 (0.83)	.021		[0.59, 3.35]	1.90 (0.88)	.034		[0.44, 3.37]
Time 1 Redemption	0.11 (0.99)	.991	0.02	[-1.55, 1.77]	0.30 (1.06)	.776	0.07	[-1.48, 3.85]
Time 1 Contamination	1.74 (1.04)	.101	0.38	[0.00, 3.48]	1.96 (1.13)	.087	0.43	[0.08, 3.85]
Time 1 Redemption × Time 1 Contamination	-1.87 (1.26)	.145	-0.41	[-3.99, 0.25]	-2.34 (1.38)	.096	-0.51	[-4.65, -0.03]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	1.94 (0.90)	.037		[0.43, 3.46]	2.75 (0.99)	.008		[1.08, 4.42]
Time 1 Redemption	0.20 (1.09)	.852	0.04	[-1.62, 2.02]	0.69 (1.15)	.550	0.14	[-1.23, 2.61]
Time 1 Contamination	1.98 (1.19)	.103	0.44	[-0.02, 3.97]	2.42 (1.23)	.055	0.53	[0.36, 4.48]
Time 1 Redemption × Time 1 Contamination	-2.20 (1.44)	.134	-0.48	[-4.61, 0.22]	-3.15 (1.52)	.044	-0.69	[-5.70, -0.60]

Note: CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for the personal characteristic variables depressive symptoms (BDI-II), waist circumference, and the use of prescription medications for managing either lipids, blood sugar, or hypertension.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 5 Redemption and Time 5 Contamination.

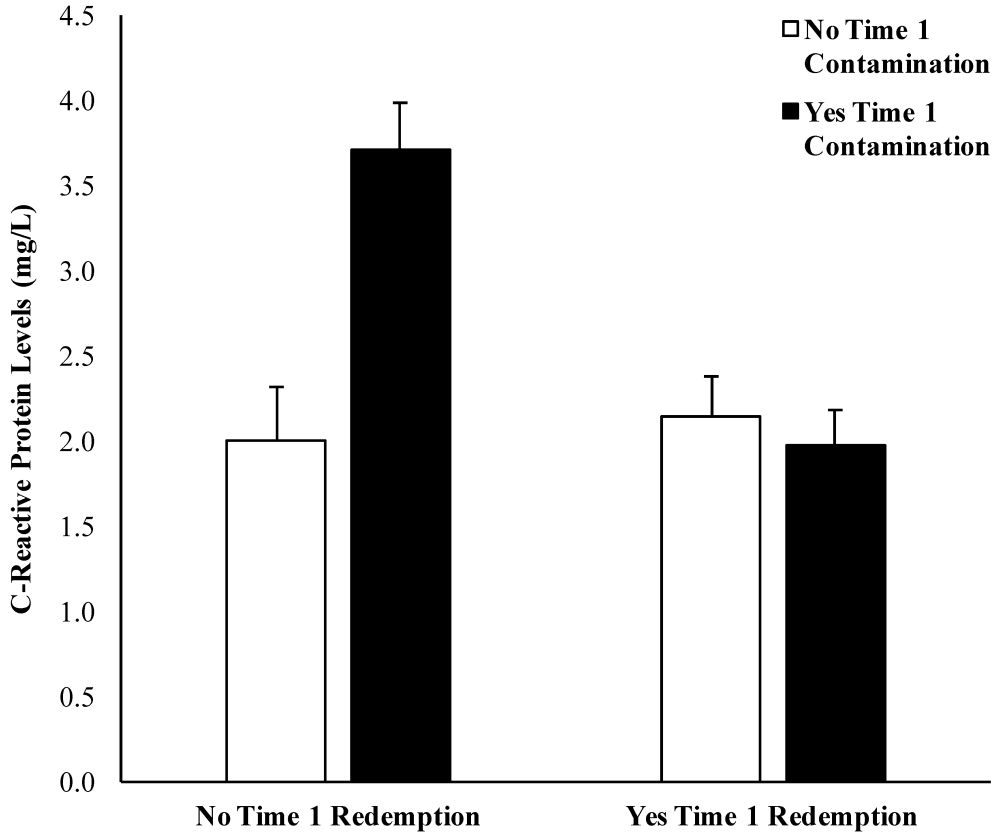

Figure 5. Mean differences in C-reactive protein (CRP) levels as a function of the interaction between having experienced redemption at Time 1 and having experienced contamination at Time 1 from Model 3, b = -3.15, SE = 1.52, p = .044, 90% CI for b = [-5.70, -0.60]. Simple slopes analyses indicated that among individuals who experienced contamination at Time 1, those who also experienced redemption had lower levels of CRP, b = -2.46, SE = 0.85, p = .006, 90% CI for b = [-3.89, -1.03]. Conversely, among individuals who did not report contamination at Time 1, there were no differences in CRP levels regardless of whether they reported experiencing redemption, b = 0.69, SE = 1.14, p = .550, 90% CI for b = [-1.23, 2.61]. Error bars represent 1 standard error.

Time 5 Redemption and Contamination. I did not find evidence for an association between the presence of redemption imagery at FLSA Time 5 and CRP levels at the time of the HSP study visit as estimated in Model 3, β = -0.13, b = -0.60, SE = 0.96, p = .535, 90% CI for b = [-2.21, 1.01]. Likewise, I did not find evidence for an association between the presence of contamination imagery at FLSA Time 5 and CRP levels, β = -0.16, b = -0.73, SE = 0.97, p = .453, 90% CI for b = [-2.36, 0.89]. Furthermore, the interaction between the presence of redemption and contamination imagery at Time 5 did not predict differences in the CRP levels as estimated in Model 10, β = -0.02, b = -0.11, SE = 1.36, p = .938, 90% CI for b = [-2.40, 2.18]. Refer to Table 11 for the full results from Models 0 – 3.

Table 11

Regression results for modeling C-reactive protein as a function of Redemption and Contamination at Time 5.

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	2.94 (0.57)	< .001		[1.99, 3.90]	3.19 (0.58)	< .001		[2.22, 4.17]
Time 5 Redemption	-0.44 (0.92)	.632	-0.10	[-1.98, 1.10]	-0.65 (0.93)	.489	-0.14	[-2.21, 0.91]
Time 5 Contamination	-0.55 (0.89)	.463	-0.15	[-2.16, 0.84]	-0.80 (0.89)	.368	-0.18	[-2.29, 0.68]
Time 5 Redemption × Time 5 Contamination	0.02 (1.26)	.985	0.01	[-2.09, 2.14]	-0.17 (1.26)	.893	-0.04	[-2.28, 1.94]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	3.04 (0.60)	< .001		[2.03, 4.06]	3.88 (0.92)	< .001		[2.34, 5.41]
Time 5 Redemption	-0.40 (0.97)	.682	-0.09	[-2.03, 1.23]	-0.60 (0.96)	.535	-0.13	[-2.21, 1.01]
Time 5 Contamination	-0.43 (0.97)	.657	-0.10	[-2.07, 1.20]	-0.73 (0.97)	.453	-0.16	[-2.36, 0.89]
Time 5 Redemption × Time 5 Contamination	-0.57 (1.37)	.680	-0.12	[-2.86, 1.73]	-0.11 (1.36)	.938	-0.02	[-2.40, 2.18]

Note: CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for the personal characteristic variables depressive symptoms (BDI-II), waist circumference, and the use of prescription medications for managing either lipids, blood sugar, or hypertension.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 1 Redemption and Time 1 Contamination.

C-Reactive Protein Risk Dichotomy

Time 1 Redemption and Contamination. I did not find evidence that reporting redemption imagery at FLSA Time 1 was associated with increased risk of having a CRP value greater than 3mg/L as estimated in Model 3, $b = 0.89$, $SE = 1.54$, $p = .564$, *Odds Ratio* (*OR*) = 2.43, 90% CI for *OR* = [0.19, 30.84]. However, I did find evidence that reporting contamination imagery at Time 1 was associated with increased risk of having a CRP value greater than 3mg/L as estimated in Model 3, $b = 3.00$, $SE = 1.68$, $p = .075$, $OR = 19.98$, 90% CI for *OR* = [1.26, 318.14]. Furthermore, the interaction between redemption and contamination imagery at Time 1 significantly predicted differential risk for whether or not participants had a CRP value greater than 3mg/L as estimated in Model 3 (see Figure 6), $b = -3.72$, $SE = 2.19$, $p = .089$, $OR = 0.02$, 90% CI for *OR* = [0.00, 0.89]. Simple slopes analyses indicated that among individuals who reported contamination imagery at Time 1, those that did not also report redemption imagery were significantly more likely to have a CRP value greater than 3mg/L than individuals who did report redemption imagery, $b = -2.83$, $SE = 1.30$, $p = .029$, $OR = 0.06$, 90% CI for *OR* = [0.02, 0.50]. Conversely, among individuals who did not report contamination imagery at Time 1, there was no difference in risk of having a CRP value greater than 3mg/L regardless of whether or not participants reported redemption imagery, $b = 0.89$, $SE = 1.54$, $p = .564$, $OR = 2.43$, 90% CI for *OR* = [0.19, 30.84]. Refer to Table 12 for the full results from Models 0 – 3.

Table 12

Logistic regression results for predicting whether or not CRP was > 3 as a function of Redemption and Contamination at Time 1.

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*OR*	90% CI for *OR*	*b (SE)*	*p*-value	*OR*	90% CI for *OR*
Intercept	-1.79 (1.08)	.097	0.17		-2.01 (1.17)	.084	0.13	
Time 1 Redemption	0.33 (1.26)	.796	1.39	[0.18, 10.92]	0.61 (1.37)	.658	1.83	[0.19, 17.35]
Time 1 Contamination	1.46 (1.23)	.236	4.29	[0.57, 32.34]	1.89 (1.39)	.172	6.64	[0.68, 64.87]
Time 1 Redemption × Time 1 Contamination	-1.49 (1.49)	.317	0.23	[0.02, 2.61]	-2.38 (1.78)	.181	0.09	[0.01, 1.73]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*OR*	90% CI for *OR*	*b (SE)*	*p*-value	*OR*	90% CI for *OR*
Intercept	-2.09 (1.21)	.086	0.12		-1.02 (1.27)	.421	0.36	
Time 1 Redemption	0.39 (1.42)	.785	1.47	[0.14, 15.23]	0.89 (1.54)	.564	2.43	[0.19, 30.84]
Time 1 Contamination	2.04 (1.47)	.164	7.69	[0.69, 85.58]	3.00 (1.68)	.075	19.98	[1.26, 318.14]
Time 1 Redemption × Time 1 Contamination	-2.12 (1.87)	.258	0.12	[0.01, 2.61]	-3.72 (2.19)	.089	0.02	[0.00, 0.89]

Note: CRP = C-reactive protein; CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for the personal characteristic variables depressive symptoms (BDI-II), waist circumference, and the use of prescription medications for managing either lipids, blood sugar, or hypertension.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 5 Redemption and Time 5 Contamination.

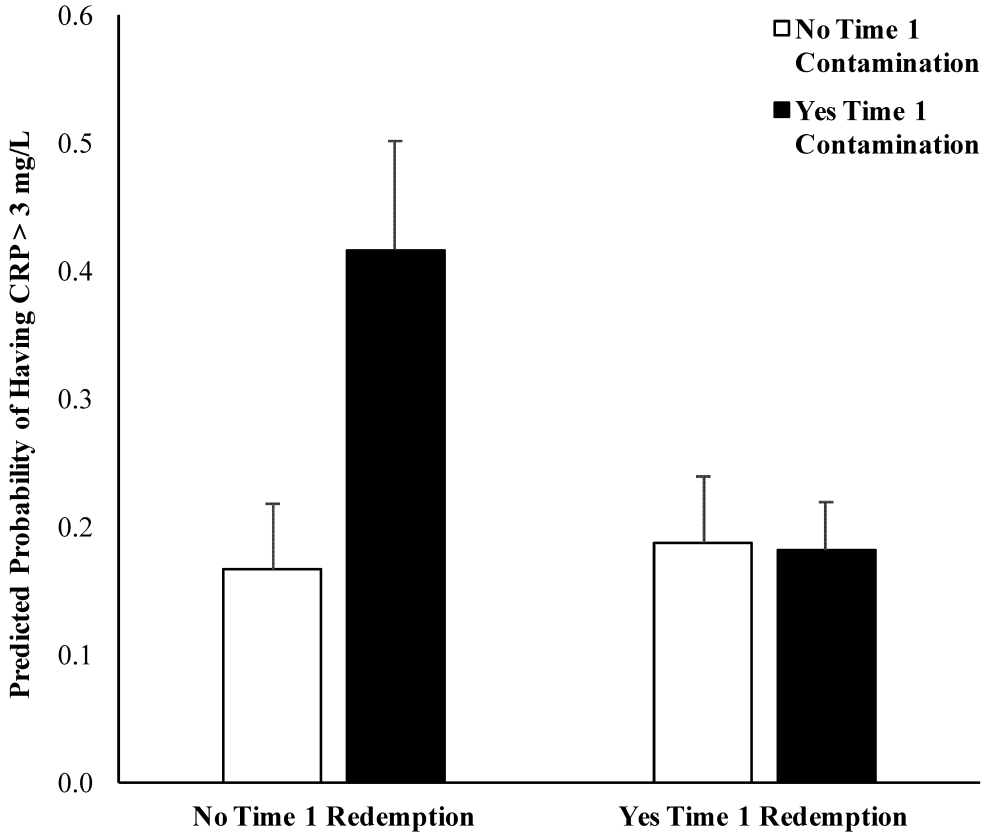

Figure 6. Predicted probability of having a C-reactive protein (CRP) value greater than 3mg/L (i.e., "high risk") as a function of the interaction between having experienced redemption at Time 1 and having experienced contamination at Time 1 from Model 3, b = -3.72, SE = 2.19, p = .089, OR = 0.02, 90% CI for OR = [0.00, 0.89]. Simple slopes analyses indicated that among individuals who reported contamination imagery at Time 1, those that did not also report redemption imagery were significantly more likely to have a CRP value greater than 3mg/L than individuals who did report redemption imagery, b = -2.83, SE = 1.30, p = .029, OR = 0.06, 90% CI for OR = [0.02, 0.50]. Conversely, among individuals who did not report contamination imagery at Time 1, there was no difference in risk of having a CRP value greater than 3mg/L regardless of whether or not participants reported redemption imagery, b = 0.89, SE = 1.54, p = .564, OR = 2.43, 90% CI for OR = [0.19, 30.84]. Error bars represent 1 standard error.

Time 5 Redemption and Contamination. I did not find evidence that reporting redemption imagery at FLSA Time 5 was associated with increased risk of having a CRP value greater than 3mg/L as estimated in Model 3, b = -1.52, SE = 1.15, p = .186, OR = 0.22, 90% CI for OR = [0.03, 1.45]. Likewise, I did not find evidence that reporting contamination imagery at Time 5 was associated with increased risk of having a CRP value greater than 3mg/L as estimated in Model 3, b = -2.09, SE = 1.45, p = .150, OR = 0.12, 90% CI for OR = [0.01, 1.35]. Furthermore, the interaction between the presence of redemption and contamination imagery at Time 5 did not predict differential risk for having a CRP value greater than 3mg/L as estimated in model 3, b = 1.48, SE = 1.80, p = .413, OR = 4.38, 90% CI for OR = [0.23, 85.05]. Refer to Table 13 for the full results from Models 0 – 3.

Table 13

Logistic regression results for predicting whether or not CRP was > 3 as a function of Redemption and Contamination at Time 5.

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*OR*	90% CI for *OR*	*b (SE)*	*p*-value	*OR*	90% CI for *OR*
Intercept	-0.88 (0.52)	.323	0.60		-0.21 (0.56)	.705	0.81	
Time 5 Redemption	-0.99 (0.94)	.354	0.42	[0.09, 1.97]	-1.31 (1.06)	.215	0.27	[0.05, 1.54]
Time 5 Contamination	0.71 (0.94)	.609	0.37	[0.08, 1.73]	-1.32 (1.00)	.188	0.27	[0.05, 1.39]
Time 5 Redemption × Time 5 Contamination	-0.51 (1.38)	.600	2.03	[0.21, 19.53]	0.44 (1.48)	.764	1.56	[0.14, 17.77]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*OR*	90% CI for *OR*	*b (SE)*	*p*-value	*OR*	90% CI for *OR*
Intercept	-0.32 (0.59)	.590	0.73		0.38 (1.03)	.716	1.46	
Time 5 Redemption	-1.15 (1.10)	.295	0.32	[0.05, 1.93]	-1.52 (1.15)	.186	0.22	[0.03, 1.45]
Time 5 Contamination	-1.21 (1.14)	.289	0.30	[0.05, 1.95]	-2.09 (1.45)	.150	0.12	[0.01, 1.35]
Time 5 Redemption × Time 5 Contamination	0.38 (1.61)	.812	1.47	[0.10, 20.67]	1.48 (1.80)	.413	4.38	[0.23, 85.05]

Note: CRP = C-reactive protein; CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for the personal characteristic variables depressive symptoms (BDI-II), waist circumference, and the use of prescription medications for managing either lipids, blood sugar, or hypertension.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 1 Redemption and Time 1 Contamination.

Composite Inflammatory Variable

Time 1 Redemption and Contamination. I did not find evidence for an association between the presence of redemption imagery at FLSA Time 1 and composite inflammatory levels at the time of the HSP study visit as estimated in Model 3, $\beta = 0.10$, $b = 0.67$, $SE = 1.67$, $p = .688$, 90% CI for $b = [-2.13, 3.48]$. However, I did find some evidence for an association between the presence of contamination imagery at FLSA Time 1 and composite inflammatory levels, $\beta = 0.48$, $b = 3.00$, $SE = 1.79$, $p = .100$, 90% CI for $b = [0.00, 6.00]$. Specifically, participants who reported contamination imagery tended to have higher levels of inflammation than those who did not (see Figure 7). The interaction between the presence of redemption and contamination imagery at Time 1 did not predict differences in composite inflammatory levels as estimated in Model 3, $\beta = -0.38$, $b = -2.40$, $SE = 2.21$, $p = .285$, 90% CI for $b = [-6.12, 1.32]$. Refer to Table 14 for the full results from Models 0 – 3.

Table 14

Regression results for modeling the inflammatory composite variable as a function of Redemption and Contamination at Time 1.

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	-0.89 (1.14)	.441		[-2.80, 1.03]	-1.18 (1.23)	.342		[-3.25, 0.88]
Time 1 Redemption	0.41 (1.37)	.766	0.06	[-1.89, 2.71]	0.90 (1.50)	.550	0.14	[-1.61, 3.42]
Time 1 Contamination	2.49 (1.44)	.090	0.40	[0.08, 4.90]	3.00 (1.59)	.064	0.48	[0.34, 5.66]
Time 1 Redemption × Time 1 Contamination	-2.26 (1.75)	.203	-0.36	[-5.19, 0.67]	-3.07 (1.94)	.121	-0.49	[-6.33, 0.19]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	-1.86 (1.39)	.188		[-4.19, 0.48]	-2.23 (1.57)	.163		[-4.87, 0.41]
Time 1 Redemption	0.77 (1.51)	.610	0.12	[-1.75, 3.30]	0.67 (1.67)	.688	0.10	[-2.13, 3.48]
Time 1 Contamination	3.08 (1.65)	.067	0.50	[0.32, 5.85]	3.00 (1.79)	.100	0.48	[0.00, 6.00]
Time 1 Redemption × Time 1 Contamination	-2.67 (2.00)	.188	-0.43	[-6.02, 0.68]	-2.40 (2.21)	.285	-0.38	[-6.12, 1.32]

Note: CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for the personal characteristic variables depressive symptoms (BDI-II), waist circumference, and the use of prescription medications for managing either lipids, blood sugar, or hypertension.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 5 Redemption and Time 5 Contamination.

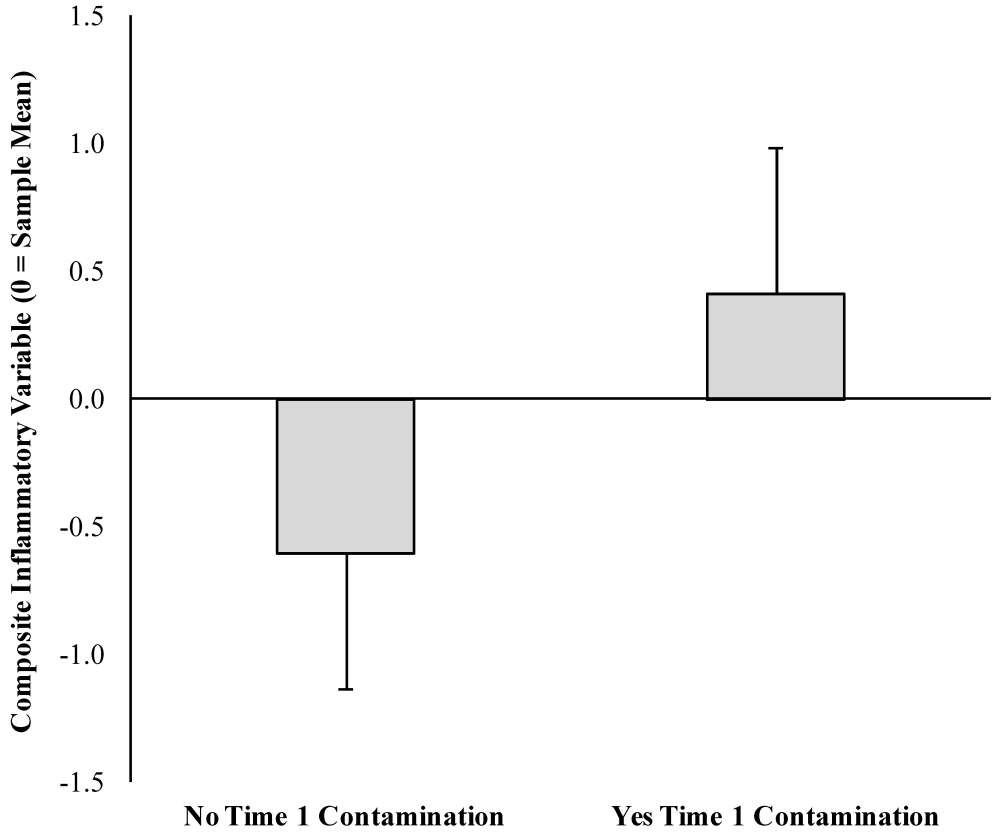

Figure 7. Mean differences in the composite inflammatory measure as a function of whether or not participants experienced contamination at Time 1 from Model 3 (b = 3.00, SE = 1.79, p = .100, 90% CI for b: 0.00 – 6.00). Participants who reported contamination imagery tended to have higher levels of inflammation than those who did not. Error bars represent 1 standard error.

Time 5 Redemption and Contamination. I did not find evidence for an association between the presence of redemption imagery at FLSA Time 5 and composite inflammatory levels at the time of the HSP visit as estimated in Model 3, $\beta = 0.06$, $b = 0.35$, $SE = 1.35$, $p = .798$, 90% CI for $b = [-1.92, 2.62]$. Likewise, I did not find evidence for an association between the presence of contamination imagery at FLSA Time 5 and composite inflammatory levels estimated in Model 3, $\beta = 0.19$, $b = 1.18$, $SE = 1.36$, $p = .391$, 90% CI for $b = [-1.11, 3.47]$. Furthermore, the interaction between the presence of redemption and contamination imagery at Time 5 did not predict differences in composite inflammatory levels as estimated in Model 3, $\beta = -0.06$, $b = -0.38$, $SE = 1.92$, $p = .846$, 90% CI for $b = [-3.60, 2.85]$. Refer to Table 15 for the full results from Models 0 – 3.

Table 15

Regression results for modeling the inflammatory composite variable as a function of Redemption and Contamination at Time 5.

	Model 0 [a]				Model 1 [b]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	-0.46 (0.79)	.560		[-1.78, 0.86]	-0.30 (0.83)	.720		[-1.69, 1.09]
Time 5 Redemption	0.27 (1.27)	.831	0.05	[-1.86, 2.40]	0.06 (1.33)	.963	0.01	[-2.17, 2.30]
Time 5 Contamination	1.15 (1.24)	.355	0.19	[-0.92, 3.22]	1.08 (1.27)	.400	0.18	[-1.05, 3.20]
Time 5 Redemption × Time 5 Contamination	-0.87 (1.75)	.619	-0.14	[-3.80, 2.05]	-0.88 (1.81)	.627	-0.14	[-3.91, 2.14]
	Model 2 [c]				Model 3 [d]			
Variable	*b (SE)*	*p*-value	*β*	90% CI for *b*	*b (SE)*	*p*-value	*β*	90% CI for *b*
Intercept	-0.95 (0.98)	.336		[-2.59, 0.69]	-1.45 (1.42)	.314		[-3.83, 0.94]
Time 5 Redemption	0.51 (1.36)	.710	0.08	[-1.77, 2.78]	0.35 (1.35)	.798	0.06	[-1.92, 2.62]
Time 5 Contamination	1.34 (1.36)	.328	0.22	[-0.94, 3.62]	1.18 (1.36)	.391	0.19	[-1.11, 3.47]
Time 5 Redemption × Time 5 Contamination	-0.88 (1.91)	.647	-0.14	[-4.08, 2.32]	-0.38 (1.92)	.846	-0.06	[-3.60, 2.85]

Note: CI = Confidence Interval

[a] Model 0 is the base model with no adjustment for covariates.

[b] Model 1 adjusts Model 0 for the demographic variables gender, race, and socioeconomic status.

[c] Model 2 additionally adjusts Model 1 for the personal characteristic variables depressive symptoms (BDI-II), waist circumference, and the use of prescription medications for managing either lipids, blood sugar, or hypertension.

[d] Model 3 is the full model. It includes everything from Model 2 and additionally adjusts for the presence of both Time 1 Redemption and Time 1 Contamination.

Summary of Results

Self-Reported Health. Individuals' levels of self-reported health varied significantly as a function of the interaction between FLSA Time 5 redemption and contamination. Specifically, among individuals who reported contamination at Time 5, those who did not also report redemption endorsed being in poorer self-reported health than those who did report redemption. There were no findings for self-reported health in models using the Time 1 redemption and contamination codes.

Metabolic Risk. There was modest evidence that the interaction between FLSA Time 1 redemption and contamination predicted differential outcomes when modeling the number of components related to metabolic syndrome risk, but not composite metabolic risk. Among individuals who reported contamination at Time 1, those that did not also report redemption had slightly more components related to metabolic syndrome risk than participants who did not report redemption. However, this observation did not extend to models of composite metabolic risk. Furthermore, this finding was not replicated when modeling the Time 5 narrative codes. Instead, in the Time 5 models, among individuals who reported contamination, those that also reported redemption had more components related to metabolic syndrome risk, as well as higher composite metabolic risk. These associations were contrary to what was hypothesized.

C-Reactive Protein. The evidence supports an interaction between FLSA Time 1 redemption and contamination in predicting levels of the inflammatory biomarker CRP, as well as in predicting who is above versus below the threshold for being considered "high risk" for cardiovascular disease based on CRP levels. Among individuals who reported contamination at Time 1, those who did not also report redemption tended to have higher levels of CRP and to be at higher risk for having a CRP value in the "high risk" range for cardiovascular disease

compared to individuals who did report redemption. These findings were not observed in models based on the FLSA Time 5 codes.

Composite Inflammation. There was no evidence suggesting an interaction between redemption and contamination in predicting composite inflammation at either FLSA Time 1 or Time 5. However, at FLSA Time 1, there was an independent association between contamination and composite inflammation. Specifically, all else being equal, individuals who reported contamination at Time 1 had more inflammation than individuals who did not report contamination at Time 1. This finding was not replicated in models of the Time 5 FLSA narrative codes.

Discussion

This dissertation was a pilot project designed to study whether two opposite ways that people may narrate experiences of adversity in their life stories were differentially associated with physical health-related outcomes in midlife American adults. Specifically, drawing on theory from the literature on posttraumatic growth (Tedeschi & Calhoun, 2004), meaning making (Park, 2010), Cognitive Adaptation Theory (Taylor, 1983; Taylor & Brown, 1988; Taylor et al., 2000), and narrative identity (McAdams & McLean, 2013), I examined whether individuals whose life stories contained redemption imagery – that is, a scene where some negative state was subsequently transformed into something positive – showed better indicators of cardiometabolic health. Additionally, I assessed whether individuals whose life stories contained contamination imagery – that is, a scene where some positive state was subsequently soiled by something negative – showed poorer indicators of cardiometabolic health. I also evaluated whether there was an interaction between reporting contamination and reporting redemption in predicting health outcomes. The life story data was collected approximately 5 to 6 years prior to when I assessed participants' health, when individuals were in their mid-to-late 50s, and again approximately 6 to 12 months prior to when I assessed health, when participants were in their early-to-mid 60s. To understand how redemption and contamination may be associated with physical health, I selected a range of cardiometabolic health indicators relevant to midlife adults living in a developed nation, including measures of overall self-reported health and general inflammation, as well as clinically relevant assessments of inflammation-mediated cardiac risk and components related to metabolic syndrome. To my knowledge, this is the first study to examine associations between these elements of a person's life story and cardiometabolic health-related outcomes.

I did not find any support for the hypothesis that reporting redemptive sequences in life stories, in and of itself, would be associated with improved physical health. That is, holding everything else constant, the presence of redemptive imagery in the life stories collected during FLSA Time 1 was not associated with participants' self-reported health, the number of components related to metabolic syndrome that participants presented with, composite metabolic risk, participants' CRP levels, whether or not participants had CRP levels that were in the "high risk" range for cardiovascular disease, and overall composite inflammation. Similarly, redemptive imagery reported at FLSA Time 5 was also not associated with self-reported health, components related to metabolic syndrome, composite metabolic risk, CRP levels, cardiovascular risk based on CRP levels, and overall composite inflammation.

Likewise, with one exception, I did not find support for the hypothesis that reporting contamination sequences in life stories, in and of itself, would be associated with poorer physical health. Holding everything else constant, the presence of contamination imagery in the life stories of participants collected during FLSA Time 1 was not associated with participants' self-reported health, the number of components related to metabolic syndrome that participants presented with, composite metabolic risk, participants' CRP levels, or whether or not participants had CRP levels that were in the "high risk" range for cardiovascular disease. There was, however, very modest evidence that individuals who reported a contamination sequence at FLSA Time 1 had higher overall composite inflammation than those who did not narrate a contamination sequence. With regards to FLSA Time 5, contamination imagery was not associated with self-reported health, components related to metabolic syndrome, composite metabolic risk, CRP levels, or cardiovascular risk based on CRP levels. Furthermore, the

observed association between FLSA Time 1 contamination and composite inflammation was not replicated using FLSA Time 5 assessments of contamination.

However, when considering the interaction between redemption and contamination in life stories, a different pattern emerged. Indeed, I found modest but reasonably consistent evidence for an interaction between the presence of redemption and contamination in predicting health-related outcomes. These findings generally indicated that individuals whose life stories contained contamination sequences tended to show better indicators of cardiometabolic health if their life stories also contained redemption sequences, and these associations tended to be more evident from the FLSA Time 1 data than the FLSA Time 5 data. Specifically, at FLSA Time 1, among individuals who reported contamination sequences, those that did not also report redemption sequences had more components related to metabolic syndrome, higher levels of CRP, and were also generally more likely to have a CRP value between 3 and 10mg/L, which is clinically considered to indicate "high risk" for cardiovascular disease. Likewise, at FLSA Time 5, among individuals who reported contamination sequences, those that did not also narrate redemption sequences reported being in overall worse subjective health. Furthermore, at FLSA Time 5, among individuals who did not report contamination, those that did report redemption had fewer components related to metabolic syndrome as well as lower composite metabolic risk scores. Unexpectedly, however, among individuals who did report contamination at FLSA Time 5, those who also reported redemption had more components related to metabolic syndrome compared to individuals who did not additionally report redemption. This finding was replicated for composite metabolic risk as well, though not in a statistically significant manner. It is unclear what might account for these findings. That these two unexpected findings corroborated each other is not surprising given the strong positive correlation between components related to

metabolic syndrome and composite metabolic risk (r = .86). Furthermore, given the small sample size and the number of analyses conducted, these associations may simply be spurious idiosyncrasies of these specific data.

While these associations were generally modest, there were several findings that merit further attention. The strongest observed associations for the FLSA Time 1 moderation analyses were for the interaction between reporting redemption and contamination in predicting participants' CRP levels as well as participants' CRP-based cardiac risk category. Specifically, individuals who reported both contamination and redemption sequences at FLSA Time 1 had CRP values that were, on average, 47% lower than among individuals who reported contamination without redemption. Furthermore, the average CRP value within the group of individuals who reported contamination but not redemption at FLSA Time 1 was 3.71mg/L, which is well above the 3 mg/L cut-off for being considered at "high risk" for cardiovascular disease. This is in contrast to individuals who reported both contamination and redemption at FLSA Time 1, whose mean CRP values fell in the "average risk" category at 2.00mg/L. Consistent with this finding, individuals who reported both contamination and redemption at FLSA Time 1 were 56% less likely to have a CRP value falling in the "high risk" category than individuals who reported contamination without redemption.

These associations are of particular interest because CRP is an indicator of chronic inflammation and cardiovascular risk that is routinely used in clinical primary care settings (Ridker, 2007). Indeed, in a large prospective study of healthy midlife men being followed for incident cardiovascular related problems (Ridker, Cushman, Stampfer, Tracy, & Hennekens, 1997), individuals who had CRP values in the highest quartile within the sample (corresponding to serum CRP concentrations of $\geq$ 2.11mg/L) were nearly three times as likely to experience

myocardial infarction and nearly twice as likely to experience ischemic stroke during an 8-year follow-up period compared to individuals in the lowest quartile for CRP within the sample (corresponding to serum CRP concentrations of ≤ 0.55mg/L). This risk persisted even after controlling for a variety of relevant covariates, and was equally strong in non-smokers as in smokers. Similarly, in a prospective study of healthy midlife women being followed for incident cardiovascular related problems (Ridker et al., 2000), compared to individuals in the lowest quartile of CRP (median CRP = 0.60mg/L), individuals who had CRP values in the highest quartile within the sample (median CRP = 8.50mg/L) were over four times as likely to experience any type of cardiac event over a 3-year follow-up period, including death from coronary heart disease, nonfatal myocardial infarction or stroke, or requiring a coronary-bypass procedure. Likewise, women in the third highest CRP quartile within the sample (median CRP = 3.8mg/L) were over twice as likely to experience any type of cardiac event over follow-up compared to women in the lowest CRP quartile. Once again, these associations persisted after controlling for a host of potentially confounding variables. Taken together, these two studies highlight that CRP may provide useful information along side other measures related to cardiovascular health such as central adiposity and lipid status when assessing the risk for incident cardiac events among initially healthy midlife adult men and women.

Given the evidence for CRP-related cardiac risk, the finding that individuals who express any contamination imagery in their life story without also expressing redemptive imagery are significantly more likely to have a CRP value falling in the clinical category for "high risk" raises at least two substantive questions. First, do individuals who maintain a life story that contains contamination imagery but not redemption imagery actually go on to experience more cardiovascular events over time? Second, can individuals be trained to reframe aspects of their

life story so as to develop redemption sequences, and would such an intervention lower CRP levels and reduce the incidence of future cardiac events? While these are empirical questions that are not possible to address in the current study, psychotherapy has certainly been shown to be able to influence and modify how individuals frame their life stories, with observable benefits for mental health outcomes (Adler, 2012; Adler, Skalina, & McAdams, 2008). As such, it would be reasonable for future studies to examine whether similar therapy-triggered changes in how individuals narrate their lives might result in benefits for heath such as lowered CRP, and potentially lowered cardiac risk.

Another noteworthy finding from the moderation analyses was that individuals who reported both contamination and redemption at FLSA Time 5 had self-reported health scores that were, on average, 211% higher than among individuals who reported contamination without redemption. Self-report measures of overall health have repeatedly been shown to predict future morbidity and mortality, independent of a number of potential confounding variables, including physician ranked illness severity and actual assessed functional ability (DeSalvo et al., 2006). While the mechanisms through which self-report measures of health confer risk are still under investigation, some recent evidence suggests that people may actually be unconsciously sensitive to their own ability to resist disease, and lower self-reported health may thus be tapping into premorbid reduced immunocompetence (Cohen, Janicki-Deverts, & Doyle, 2015).

If it is the case that poorer self-reported health portends reduced immunocompetence, this might help explain the general lack of findings related to the more objective assessments of health at FLSA Time 5. That is, if self-reported health is a proxy for the early stages of reduced immunocompetence, then the lower self-reported health values found in individuals who reported contamination without redemption at FLSA Time 5 may suggest that these individuals

are in the early, premorbid stages of developing health problems. It is possible that, should these individuals be assessed again after more time has passed, more downstream consequences of immune system dysregulation may eventually manifest, such as increases in CRP. Consistent with this possibility, self-reported health at FLSA Time 1 (approximately 5 to 6 years prior to physical health assessments) coded such that higher values indicated better health was negatively associated with the inflammatory composite variable, $r(54) = -.25$, $p = .065$, 90% CI for $r = [-.49, -.04]$, as well as CRP, $r(54) = -.24$, $p = .076$, 90% CI for $r = [-.45, -.03]$, demonstrating in this sample the prospective relationship between self-reported health and physical indicators of inflammation. Likewise, self-reported health at FLSA Time 5 (approximately 6 to 12 months prior to physical health assessments) was also negatively associated with the inflammatory composite variable, $r(54) = -.28$, $p = .041$, 90% CI for $r = [-.47, -.06]$ and CRP, $r(54) = -.27$, $p = .048$, 90% CI for $r = [-.46, -.06]$. Taken together, individuals in the sample who had poorer self-reported health 5 to 6 years prior to health assessments as well as 6 to 12 months prior to health assessments were more likely to present at the HSP visit with higher levels of inflammation. Conversely, this explanation is at least partially contradicted by the lack of an interaction between redemption and contamination at FLSA Time 1 in predicting self-reported health at the time of the HSP study visit. Regardless, future studies should consider this possibility.

How might having redemption sequences integrated into people's life stories have protected individuals who also reported contamination from showing evidence of health consequences? Contamination sequences on their own are psychologically devastating. Indeed, in one study of adult men and women by McAdams, Reynolds, Lewis, Patten, and Bowman (2001), researchers found negative correlations between contamination sequences and life satisfaction ($r = -.40$, $p < .001$) and self-esteem ($r = -.46$, $p < .001$), and a positive correlation

between contamination sequences and depression ($r = .49$, $p < .001$). A subsequent study of adult men and women corroborated these findings with similarly sized correlations and additionally demonstrated that contamination sequences were predictive of both higher depressive symptoms and lower satisfaction with life even after statistically adjusting for the personality trait neuroticism (Adler, Kissel, & McAdams, 2006). Not surprisingly, given the links to depression, contamination sequences can leave individuals feeling hopeless (McAdams & Bowman, 2001). Hopelessness and depression, while interrelated, are also both independent risk factors for cardiovascular disease and premature mortality (Anda et al., 1993; Everson et al., 1996; Everson, Kaplan, Goldberg, Salonen, & Salonen, 1997). To the extent that contamination stories leave people feeling depressed and hopeless, their risk for health consequences may increase. However, if a person's life story also contains scenes of redemption, this may buffer against the negative consequences of contamination by giving the individual a hopeful personal example of adversity being overcome to draw on. In other words, the presence of redemptive scenes in a person's life story may offer the individual hope by reminding the person that although he or she may have experienced negative situations for which nothing good could ever be said, he or she also experienced adversity that he or she ultimately mastered and transformed into something self-enhancing and positive. Consistent with this possibility, a growing body of research suggests that experiencing some amount of adversity that can be overcome is more adaptive than experiencing no adversity at all or experiencing only insurmountable adversities, as these sorts of experiences can "toughen" an individual to better weather other negative experiences (Dienstbier, 1989; M. D. Seery, 2011; Mark D. Seery, Holman, & Silver, 2010; M. D. Seery, Leo, Holman, & Silver, 2010; M. D. Seery, Leo, Lupien, Kondrak, & Almonte, 2013).

As a potential counterargument to this explanation, it is also worth noting that the current sample had low depressive symptoms as indexed by the BDI-II, and that the interaction between redemption and contamination predicted health outcomes after statistically adjusting for depressive symptoms. Presumably, if current depressive symptoms mediated the association between reporting contamination and deleterious health outcomes, statistically controlling for differences in depression should have either partially or fully attenuated the findings. However, that this was not the case may be due to the nature of the sample. Specifically, the BDI-II has been shown to underestimate depressive symptoms in community samples (Hunt, Auriemma, & Cashaw, 2003). This may be due at least in part to the relatively large jump in symptom severity described in the items that make up the questionnaire. For example, the item "sadness" offers respondents the following options: 0 = "*I do not feel sad*," 1 = "*I feel sad much of the time*," 2 = "*I am sad all of the time*," 3 = "*I am so sad or unhappy that I can't stand it*." For participants to endorse even the low score of 1 would require these individuals to have felt sad for most of the previous two weeks. Conversely, in the Center for Epidemiological Studies Depression Scale (Radloff, 1977), another widely assessed measure of depressive symptoms intended for use in the general population, the item for "sadness" offers respondents much more temporally nuanced options, with a value of 1 indicating that the individual felt sad "*some or a little of the time (1 – 2 days)*." As such, the BDI-II may not have been the best measure of depressive symptoms for this sample.

What might account for the lack of independent associations between redemption and physical health? Redemption sequences are narrations of experiences of adversity that have subsequently been transformed into something positive. This transformation presumably either partially or completely attenuates the threatening nature of the original experience. As such,

independent benefits of constructing redemptive narrations on physical health might depend on when and how an individual constructs and incorporates the redemptive sequence into his or her narrative identity. When people engage in attempts to make meaning out of a negative situation, they often experience a temporary increase in distress that eventually comes back down once the episode has been processed (Frazier et al., 2009). A redemptive sequence by definition is an episode where the person has already finished the meaning making process surrounding the experience, at least as it pertains to the individual's life story at the time of narration (McAdams, 2006a). It is possible that individuals who ultimately construct redemptive stories about adversity would show temporary declines in some indicators of physical health that may accompany the increased distress of the meaning making process. But presumably, once the individual has identified the positive transformation and integrated the new enhanced self into her or her identity, there should be no more distress present related to the experience. As such, benefits to health might only be observable during a relatively short window, namely, from the actual occurrence of the negative event to the time an individual identifies positive value or meaning in the event, or shortly thereafter. Furthermore, if the person was already generally healthy to begin with, then there may not be substantial possible gains in physical health to be made from forming redemptive narratives independent of other factors. Consistent with this formulation, the current study group was drawn from a relatively healthy community sample of adults, and these data may be exhibiting a floor effect with regards to how much benefit to physical health constructing redemptive narratives, independent of other factors, was able to confer. It is possible that making use of redemption narratives may indeed be independently associated with improved health-related outcomes under certain circumstances, such as among individuals already dealing with a chronic illness. Indeed, much of the research linking meaning making and growth to improved

physical health indicators has been carried out in patient populations, particularly samples of individuals living with HIV (e.g., Taylor et al., 2000). Unfortunately, this hypothesis was not testable in the current study.

Finally, what might have accounted for the lack of independent associations between contamination and physical health? The general lack of independent findings for contamination narratives may simply have reflected that the interaction between contamination and redemption provided more useful information. Indeed, when probing the interaction between reporting redemption and reporting contamination, the evidence generally suggested that contamination in the absence of redemption was associated with poorer health outcomes than when both contamination and redemption were reported. That is, redemption appeared to have acted as a buffer against the deleterious consequences of contamination.

Limitations

This study was an exploratory pilot project designed to examine whether narrated scenes from life stories that contained redemption or contamination imagery were associated with cardiometabolic health-related outcomes. As a result, there are a number of limitations that merit consideration. First, the sample size for the current study was small. Small sample sizes reduce statistical power to be able to detect real associations, particularly when associations are relatively modest. This is especially problematic for the redemption × contamination moderation analyses, where the cell sizes within each subgroup are even smaller. Furthermore, the sample size limited my ability to investigate potential moderators and mediators of the associations between redemption, contamination, and health-related outcomes. For example, evidence suggests that both women (Linley & Joseph, 2004) and ethnic minorities (Stanton, Bower, & Lowe, 2006) tend to report more growth following adversity than men and Caucasians.

Furthermore, in a meta-analysis of benefit finding and growth conducted by Helgeson, Reynolds, and Tomich (2006), the authors found that growth related processes were more strongly associated with physical health in African Americans and women, compared to Caucasians and men. The authors speculated that minorities may generally have more experience with adversity, and thus may have had both more opportunities and reasons to attempt to look for positive outcomes following negative experiences. Regarding the gender-growth interactions, the evidence suggested that the stronger associations between growth and health in women were simply due to confounding by the type of adverse experience reported. Specifically, the majority of studies included in this meta-analysis looking at growth and health in women focused on women who were being treated for breast cancer, making it less clear as to the nature of the association between gender, growth, and health more broadly. Nonetheless, taken together, these findings suggest that race and possibly gender may be important theoretical moderators of the association between growth-related variables and health. However, in light of the sample size for this study, it was simply not statistically feasible to evaluate 3-way interactions in these data (e.g., redemption × contamination × gender).

Given the exploratory nature of this project, to increase statistical power, I raised the α-level used to guide statistical decision making to .10. While this does increase power, it also increases the risk of making a Type I error (i.e., incorrectly rejecting a true null hypothesis). Moreover, the majority of findings described in this manuscript as statistically significant had associated *p*-values in the .05 to .10 range. As such, while the findings for this project may suggest some starting points from which future researchers can build, from an inferential standpoint, they should be interpreted with caution pending replication. Furthermore, while these findings are intended to provide initial insight as to the potential magnitude of associations

between life narrative codes for redemption and contamination and indicators of cardiometabolic health, I caution against over-interpreting the practical significance of the observed associations. This may be especially true for the unadjusted, bivariate associations reported in Table 3, given how much the regression estimates change as more covariates are entered into the models, as well as the issue that codes from the life narrative do not exist independently of other factors, and the interaction of life narrative codes may be more important than any individual independent association. Additionally, due to the sample size, the correlation coefficients being estimated are not necessarily stable statistics, and even relatively small changes in the data can result in non-trivial differences between correlation coefficients. For example, the bivariate association between FLSA Time 1 redemption and serum CRP was r = -.23, suggesting that reporting redemption may be associated with lower CRP (and thus theoretically less inflammation and cardiovascular risk). In a large enough sample, this correlation magnitude may seem impressive given the complexity and nuance of life story codes. However, with a sample of 61, it is likely prudent to view the magnitude of this association with skepticism, at least until replication.

Additionally, the current study was both correlational and functionally cross-sectional in design. Furthermore, there were no assessments of physical health (other than a brief self-report measure) during the FLSA Time 1 or Time 5 visits. As such, while I statistically adjusted for some obvious potential confounds (e.g., gender, ethnicity, and socioeconomic status), it is not possible to make any causal claims about the nature of the association between redemption, contamination, and cardiometabolic health-related outcomes. Likewise, without knowing participants' baseline health status, I cannot rule out the possibility that differences in baseline health accounted for the associations between redemption, contamination, and health-related outcomes. For example, it is possible that individuals who were already in poorer health at FLSA

Time 1 were also more likely to narrate stories of contamination due to generally feeling less well. Alternatively, individuals who were in comparatively better health at FLSA Time 1 may have been more likely to tell redemptive stories due to feeling more well. In order to at least partially address this issue, I tested to see if there were differences in FLSA Time 1 and FLSA Time 5 self-reported health among individuals who reported redemption or contamination at FLSA Times 1 and 5. There was no evidence for significant differences in self-reported health as a function of these variables (all *p*s > .409). This offers some support for the notion that differences in health at the time of the FLSA study interviews may not have confounded current findings; however, self-reported health is only one index of health, and these findings to not preclude the possibility that there were differences in various other cardiometabolic health indicators that may be associated with experiences of redemption and contamination.

Relatedly, as the data I collected on health-related outcomes was cross-sectional, I was unable to examine the possibility that changes in the use of contamination and redemption imagery over time might be associated with subsequent changes in health-related outcomes (i.e., health trajectories). This is problematic because the various health parameters assessed in this study are all dynamic, and taking any single snapshot is unlikely to provide a particularly reliable window into an individual's actual health status. Likewise, the development of the life story is a lifespan process, with people's narratives changing, developing, and maturing over many years (Pasupathi & Mansour, 2006). To better understand these issues, future studies will need to (1) collect baseline health data alongside life narrative data, and (2) continue to collect health data alongside narrative data longitudinally over a diverse age range.

An additional interpretational challenge to the current study was that features of the initial adversity that participants narrated could be confounded by the various ways in which

individuals narrated the experience. For example, redemption imagery in the life story may simply be a proxy for less severe experiences of adversity. While this is ultimately an empirical question that would require careful interview-based assessments of stressful life experiences alongside life story narrations, there is some evidence suggesting that that the original experience of adversity may not generally be confounded by individuals' methods of narration. Specifically, in a study conducted by Lilgendahl and McAdams (2011), researchers investigated the valence of actual events narrated separately from how individuals interpreted the impact that these events had on their life stories. To do so, they coded the valence of narrated events on a 5-point scale ranging from 1 = "*very negative*" to 5 = "*very positive*." They found that the meaning participants drew from their past experiences predicted well-being even after controlling for the actual valence of the past events. For example, individuals in their study who tended to narrate how past experiences impacted growth in a more positive manner had higher well-being regardless of how negative the actual past event was.

Another limitation of the current study was that it was not able to tease apart the physiological mechanisms through which life story scenes such as redemption and contamination may affect cardiometabolic health. The data are suggestive of inflammation and deleterious metabolic properties potentially being associated with the use of redemption and contamination imagery, but the specific pathways remain unknown. For example, to assess inflammation, I used a composite of five different inflammatory messengers and two inflammatory biomarkers. I did this because data that come from biological assays involve noise, and the various inflammatory measures also all tend to be interrelated. As a result of this, especially in the context of having a small sample, it was not justifiable to look separately at different molecular pathways. Future

studies will be needed to replicate this general pattern of findings, and further unpack the specific biological mechanisms modulated by redemption and contamination narratives.

Relatedly, one of the central assumptions in my hypotheses was that narrating redemption transforms something that was once threatening into something that is no longer threatening that one has grown from, while narrating contamination implies that an individual is still being threatened by that life experience. However, I did not actually test these notions directly. Rather, I am inferring that these assumptions are likely to be true based on the observed associations between redemption, contamination, and health-related outcomes. Future studies should directly test these assumptions by collecting physiological data during an individual's life story interview. For example, it may be useful to examine whether outcomes like blood pressure or levels of catecholamines such as epinephrine and norepinephrine are more likely to rise when discussing scenes that are subsequently coded as contamination and stay stable (or perhaps decrease) when discussing scenes that subsequently are coded as redemption.

An additional limitation is that the reliability statistic for the redemption codes in the current study was fairly modest (*ICC* = .60). This may have impacted study results in several different ways. When I qualitatively examined the discrepancies between my redemption codes and the redemption codes furnished by the second, highly experienced, coder, it appeared that my codes were systematically more conservative. That is, the type of narration that I determined as meeting threshold for redemption appeared to capture only stories that involved a significantly more negative state being followed by a significantly more positive state than was reported in scenes that I did not code as redemption but the other coder did. As such, my power to detect associations may have been lowered and the associations observed may have been attenuated due to having a smaller pool of redemption scenes to work with, along with extra measurement error

introduced by the possibility of having actual redemption scenes incorrectly specified as non-redemptive. Conversely, it is possible that it is only the more "major" types of redemption scenes that are linked to health, and casting a wider net may have actually attenuated findings by watering down the pool of scenes being evaluated. While research on stressful life events generally suggests that associations with health are stronger when the experienced stressors are more severe (Monroe, 2008; Monroe & Simons, 1991; Monroe, Slavich, & Georgiades, 2009), it is important to remember that the data obtained from people's life stories are not intended to assess or reflect stressor occurrence or severity, and the scenes being coded are not designed to obtain objective information about past stressors. Rather, the life story data provides a window into who people view themselves to be. In these accounts, scenes that an interview designed to assess stressful life events might overlook as being relatively minor may hold significant personal meaning to the individual, and thus may have a major influence on how the person views his or her life and his or her very identity. This raises at least two issues for future studies to address. First, given the difficulty of coding long, varied interviews for complex constructs, future studies should take special care to train a team of coders to reliability, or should use consensus coding among trained raters to determine whether scenes contain redemption or contamination imagery. Second, future studies should consider examining whether or not there is some form of "threshold" for redemption and contamination sequences below which associations with physical health and well-being become unobservable.

Relatedly, in the current study, I did not unpack the content that made up the various redemption and contamination narratives. It is possible that not all redemption or contamination stories are equally impactful for physical health. As an illustrative example, the coding manual for redemption provided by the Foley Center lists five different themes that redemption

sequences tend to embody: sacrifice, recovery, growth, learning, and improvement (McAdams, 1999). Under the "learning" category, one of the examples listed is a mother-in-law hating a participant (negative state) being transformed into the participant working hard to be a good mother-in-law herself (positive state). Would this example of redemption be expected to confer health benefits or buffer against negative health consequences? It is certainly possible that this hypothetical participant's belief that her hardships with her own mother-in-law lead to her becoming a wonderful mother-in-law might be beneficial insofar as the pride and joy she experiences from being a good mother-in-law herself may outweigh any lingering hurt or resentment about her own relationship with her mother-in-law. Alternatively, her negative experiences with her mother-in-law may continue to be an ongoing source of psychological pain, and, it is possible that her own attempts to be a better mother-in-law are actually rooted in an anxious desire to not be judged by her own daughter- or son-in-law as being a bad mother-in-law. In this scenario, the redemptive transformation might not be enough to counter deleterious effects of the painful memories and current anxiety. Ultimately, whether or not diverse thematic categories of redemption or contamination are differentially associated with health-related outcomes is an empirical question that could be addressed by future studies.

Another potential limitation of the current study was the age of the sample. As people get older, they may develop certain expectations about unpleasant situations that they view as likely or inevitable to occur, such as bereavement and illness. While I did not assess this possibility in the current study, if true, it may have served to attenuate the magnitudes of the various observed associations. That is, if individuals have been mentally preparing for various bad things to happen simply as a function of aging, they may also have been altering their own narrative in a manner that reduces threat from these situations. Consistent with this formulation, while every

individual is asked to discuss the loss of a close loved one during their life story interview, many of these scenes did not end up meeting criteria for contamination. For example, many of the participants chose to talk about the death of a parent who was in his or her 80s or 90s. While that death still induced sorrow and grief, the participants often also acknowledged that their loved one had been suffering under illness for a long time, and that the death, while painful, was both inevitable and relieving. In comparison, an individual in his or her 20s may be less likely to take such a stoic view when a parent dies, and may thus be more likely to construct a narrative consistent with contamination. This possibility may also help further explain the lack of observed associations between the more recent FLSA Time 5 redemption and contamination codes and physical health. However, the broader literature on age-related adversity does not generally favor this idea; instead, a large body of evidence suggests that when older adults face unavoidable adversities, even relatively small ones, they often experience stronger declines in both physical and mental health compared to their younger counterparts (for an extensive review, see Charles & Carstensen, 2010).

Implications and Future Directions

Even acknowledging these limitations, this study provides a useful first step in understanding how narratives of redemption and contamination may be associated with physical health. The next step would be to replicate these findings in a larger sample where health measures are taken alongside of narrative measures from the beginning, or at the very least on two separate occasions with enough time between them to allow for biological variables to have sufficient opportunity to change. If the pattern of findings holds, there are a number of potential next steps to take. For example, it would be interesting to know whether or not differently themed redemption and contamination sequences confer similar benefits and risks. If not, what

categories of redemption are most beneficial to health, and what categories of contamination are most detrimental? It is also possible that the answer to this question will depend on any number of yet-to-be determined individual difference factors such as race, gender, lower-level personality variables, trait affect, social relationship quality, or goal-directedness.

Additionally, assuming the current study replicates in larger and different samples, it will be important to begin to search for the biological, behavioral, psychological, and environmental mediators that facilitate the transition from narrating redemption and contamination stories to differences in health-related outcomes. For example, understanding the pathways through which narrations of redemption buffer against the consequences of contamination may suggest novel intervention possibilities and clinical applications. Along these lines, it would be useful to know the extent to which an individual's ability to frame life experiences in a redemptive or a contaminative manner is modifiable. If these abilities can be modified, then it would be important to evaluate whether helping individuals learn to construct redemptive stories provides tangible health benefits, and whether there are certain situations or certain individuals for which such an intervention would be more tailored.

Conclusion

In conclusion, this study offers tentative evidence that people's use of redemption and contamination imagery in the telling of their life stories is predictive of differences in cardiometabolic health-related outcomes, as indexed by self-reported physical health, components related to metabolic syndrome, CRP levels, and CRP-determined cardiac risk. Generally speaking, in the telling of their life stories, individuals who reported contamination without redemption showed evidence of poorer cardiometabolic health compared to individuals who reported both contamination and redemption. These findings add to a growing body of

literature from personality and narrative psychology that has demonstrated redemption to be associated with better psychological outcomes and contamination to be associated with poorer psychological outcomes (Adler et al., 2006; McAdams, 2006b; McAdams & Bowman, 2001). Furthermore, it highlights another possible mechanism through which adversity can impact health: by altering our identities through shaping how we come to understand, narrate, and internalize the stories of our lives.

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