Does group life buffer the effects of cumulative stress on long-term physical and mental health?

by

Nathan Sanghe

BSc. Hons., Queen's University, 2020

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

in

THE FACULTY OF GRADUATE AND POSTDOCTURAL STUDIES

(Kinesiology)

THE UNIVERSITY OF BRITISH COLUMBIA

(Vancouver)

June 2023

The following individuals certify that they have read, and recommend to the Faculty of Graduate and Postdoctoral Studies for acceptance, a thesis entitled:

Does group life buffer the effects of cumulative stress on long-term physical and mental health?

Submitted by Nathan Sanghe in partial fulfillment of the requirements for the degree of Master of Science in Kinesiology

Examining Committee:

Mark Beauchamp, Professor, Kinesiology, UBC

Supervisor

Eli Puterman, Assistant Professor, Kinesiology, UBC

Supervisory Committee Member

Nancy Sin, Associate Professor, Psychology, UBC

Supervisory Committee Member

Abstract

Multiple group membership has been found to positively contribute to individuals' health and wellbeing (Lam et al., 2018), but little research has examined the potential buffering effects of group involvement on the negative impacts of stress. The purpose of my Master's thesis research was to address this gap in the literature by exploring the moderating effects of social and sport group involvement on the relationship between cumulative stress and long-term physical and mental health outcomes. Data were collected from 3682 aged adults (aged 20-74 years), across approximately 10 years, as part of the longitudinal Midlife in the United States (MIDUS) study. Measures of group life and cumulative stress were assessed between 2004-2009 as part of MIDUS 2 (operationalized as 'Time 1'), and a range of physical and mental health measures were assessed between 2013-2019 as part of MIDUS 3 (operationalized as 'Time 2'). Cumulative stress at Time 1 was significantly and positively correlated with Time 2 mental health outcomes, including general affective distress ($r = 0.30$), self-reported affective disorder ($r = 0.17$), and affective diagnosis based on self-report ($r = 0.19$). Additionally, a positive, albeit very weak, correlation was observed between Time 1 cumulative stress and Time 2 physical health ($r = 0.07$). Group involvement did not significantly moderate the relationship between stress and any of the health outcomes examined in this study (general affective distress, $\beta = 0.028$, $p = 0.18$; self-reported affective disorder, $\beta = 0.005$, $p = 0.60$, affective diagnosis based on self-report, $\beta = 0.008$, $p = 0.47$. The same was true for physical health ($\beta = -0.003$, $p = 0.17$). These findings indicate that the protective effects of group life on health outcomes found in previous research was not observed within the MIDUS dataset, at least in relation to buffering against cumulative stress. Specifically, greater involvement in group life, observed within MIDUS 2, did not dampen the effects of cumulative stress in relation to either physical or mental health outcomes approximately 10 years later.

Lay Summary

Stress has notably been attributed to poor health. Finding ways to reduce stress is essential in order to lessen the burden it has on both mental and physical health outcomes. Using data from the Midlife in the United States (MIDUS) study, the research reported in this thesis examined whether involvement in social or sport groups protects effect against the effects of cumulative life stress, assessed among participants between 2004-2009, in relation to a range of mental and physical health outcomes assessed several years later (between 2013-2019). While cumulative life stress was prospectively related to different markers of physical and mental health over time, greater involvement in groups did not buffer against the prospective effects of stress over time.

Preface

All of the work presented in this thesis was conducted in the Psychology of Exercise, Health, and Physical Activity Laboratory at the University of British Columbia, Vancouver campus. The data that were used in this research derived from the public use file from the Midlife in the United States (MIDUS) study. Access to this file was obtained online through the MIDUS Colectica Portal.

I was the lead investigator on this study. With help from the Supervisory Author, Dr. Mark Beauchamp, and my committee members Drs. Eli Puterman and Nancy Sin, I was responsible for all major areas of topic formation, data analysis, and preparation of this manuscript.

The data presented in this manuscript are anticipated to produced one peer-reviewed publication. No publications have been submitted to date.

Table of Contents

List of Tables

Acknowledgements

I would like to express gratitude to my supervisor, Dr. Mark Beauchamp, for his unwavering support and dedication throughout this entire process. He has taught me the value of having a relentless work ethic and to always persevere. I will forever be grateful for what I have learned under his guidance. I also send my thanks to my committee (Drs. Puterman and Sin) for their thoughtful feedback that has led to a stronger and more insightful research project.

To the rest of the PEHPA lab, thank you for your advice and support throughout this process. Particular acknowledgement goes to Colin for all your help in the statistics department, I couldn't have done this without your help.

A sincere thank you to my family. They have and continue to give me the support and love to accomplish anything I put my mind to.

A final dedication goes to my Babaji and Grandad, who supported me throughout this journey but, regrettably, couldn't be present to witness its completion. I deeply miss you both, and I dedicate this work and degree to you both.

Introduction

In response to a psychological or physical stressor stimuli, an activated neuroendocrine response, facilitated by the autonomic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis, provokes both adaptive and deleterious health outcomes. On one hand, the psychological and physiological response to acute stressors may be adaptive and typically do not impose a burden on health. These adaptive mechanisms evoke physiological, psychological, and behavioural responses in order to manage the stressor accordingly. In contrast, chronic, uncontrolled exposure to stressors may cause major detrimental health outcomes and influence disease progression (Kemeny, 2003; Salleh, 2008). Repeated and/or prolonged exposure to stress-invoking stimuli correlate with impaired immune system function, damage neurons in the hippocampal brain regions and promote the development and/or progression of chronic diseases such as depression, anxiety, diabetes, cardiovascular disease, and cancer (Boomershine, Wang, & Zwilling, 2001; Jetten et al., 2017).

The influence of stressors on health is affected by the type, quantity, and persistence of the stressors along with the psychosocial resources available that allow for individuals to deal with those stimuli/events (Schneiderman, Ironson & Siegel, 2005). Psychosocial resources refer to individual personality and social relationship factors that both individually and collectively support psychological and physical health (Taylor, 2011; Wiley et al., 2018). Examples of psychosocial resources include self-esteem, optimism, active coping skills, and social support. Specifically, psychosocial resources can help individuals appraise stressful situations in more benign ways (i.e., reducing the perceived threat of an otherwise harmful situation). Furthermore, these resources help individuals handle the challenging, threatening, and/or taxing events they encounter during the active presence and aftermath of a stressor (Taylor, 2011). In sum,

psychosocial resources can substantively influence how individuals *appraise* and *manage* stressful events and/or stimuli.

A sense of connection and shared social identity (through group involvement) has the capacity to improve individual health and wellbeing by fostering an increase in available psychosocial resources (Haslam et al., 2018). Much of the focus of psychological models and research has been on understanding the psychology of individuals (i.e., 'I' and 'me') and fails to appreciate the importance of psychological factors associated with group life for one's health that derives from a sense of 'we' or 'us. Group involvement potentially allows individuals to identify and utilize essential psychosocial resources in times of need, namely during moments of stress. Despite extensive evidence that exposure to multiple stressors leads to detrimental mental and physical health outcomes, little research has sought to understand the potential of group involvement to buffer the effects of stress on long-term health outcomes. With this in mind, the overall purpose of my Master's thesis research project was to investigate the moderating effect of group involvement with regard to the relationship between cumulative stress and physical and mental health outcomes in a national sample of adults from the United States of America.

Stressors and the Stress Response

A widely embraced definition of psychological stress comes from the cognitive motivational relational theory of stress, in which Lazarus and Folkman (1984) define "psychological stress [as] a particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well being" (p. 19). When people evaluate potential stressors (or stress stimuli), this can elicit different emotions and reactions based on their cognitive appraisal of those stressors. Lazarus and Folkman describe cognitive appraisal as "categorizing an encounter, and its various facets,

with respect to its significance for well-being" (Lazarus & Folkman, 1984, p. 31). Specifically, stress is operationalized as a process that begins with a stressor (i.e., a stimulus that is perceived during an event or within one's thoughts) which is then appraised in terms of whether the stressor is perceived to be neutral, challenging, or threatening (Lazarus & Folkman, 1984). There are two forms of cognitive appraisal: (1) *primary appraisal* in which a person evaluates whether he/she has anything at stake in this particular encounter or if the stressor poses a threat (i.e., harm or benefit to ones mental/physical wellbeing), and (2) *secondary appraisal*, in which a person perceives that they have the requisite resources or coping strategies at their disposal to address the above threats or challenges (Folkman et al., 1986). Stressors exist in many different forms and present themselves across almost all facets of daily life. The physical environment (excessive heat/cold, weather, traffic), social relationships (conflict, loneliness, lack of social support, aggression with others), financial problems (taxes, bills, expenses), life events (marriage, children, work/unemployment, death, illness), lifestyle choices (lack of sleep, alcohol, drugs, diet, time management), and physiological disturbances (i.e., onset of health concerns, pregnancy, injury) are all examples of stressors that people face in their daily lives that may cause an ensuing stress response (Epel et al., 2018).

Although they arise in many forms, Epel and colleagues (2018) define four main types of stressors based on different durations of stressor presence: (1) Acute stressors, (2) Daily events/hassles, (3) Life events, and (4) Chronic stressors. Acute stressors are defined as intense short term-exposures to a single event or stimuli (e.g., giving a public talk). Daily events (frequently called "hassles") are minor inconveniences that occur regularly such as being in a rush, arguments with others, or addressing deadlines for work/school. Life events are episodic and are quite often time-limited such as being fired (i.e., employment termination), the ending of

a relationship, or experiencing a major accident of some form. These brief events can have long-term consequences that vary depending on the severity of the event and how the event is dealt with by the individual. Finally, long term stressors that are present for an extended period of time are categorized as chronic stressors. The definitive duration required for a stressor to be considered as chronic varies but typically requires a minimum exposure of 6 months to one year (Epel, 2018).

Research has consistently indicated that exposure to multiple stressors, or repeated exposure to the same stressor over time, far exceed the consequences of a single stressor (Evans et al., 2013; Turner & Floyd, 1995). In addition, in recent years, research has indicated that when all of the stressors that people experience across multiple domains of life are accumulated into a composite measure of *cumulative stress* (McLoughin et al., 2020; Slopen et al., 2018), this is prospectively related to depleted physical and mental health outcomes (Block et al., 2009; Morton et al., 2012; Reading et al., 2016; Slopen et al., 2012, 2013; Slopen & Williams, 2014; Sternthal et al., 2011).

Physiological Stress Response

The paraventricular nucleus area of the hypothalamus, the anterior lobe of the pituitary gland and the adrenal gland collectively form the hypothalamic-pituitary-adrenal (HPA) axis (Smith & Vale, 2006). The HPA axis, along with autonomic nervous system (ANS) activation and stress-related cognitions and emotions, ignite a neurosympathetic and hormonal response to both acute and chronic stressors in a manner that aims to level out any altered homeostatic changes within the human body. Homeostasis is known as our stable internal state and self regulates itself to adjust in circumstances of changing external conditions (Billman, 2020). The concept of homeostasis explains how human beings are able to maintain steady internal

conditions in hostile external environments that would otherwise alter our internal stability. Homeostatic fluctuations can therefore also encompass a positive, adaptive set of mechanisms that can enhance survival, anticipate future challenges, and deal with these challenges (McEwen & Akil, 2020).

As an intricate and robust homeostatic mechanism, the HPA axis is modulated by particular brain signalling systems through neurotransmitters, and thus exhibits an observable relationship with the central nervous system (Stephens, 2012). The HPA axis response to stress begins in the paraventricular nucleus (PVN) of the hypothalamus which releases two hormones into the blood stream called corticotropin-releasing factor (CRF) and arginine vasopressin (Stephens, 2012). Both of these hormones derive from the hypothalamus, enter the hypophysial portal vessels, and stimulate the anterior pituitary gland to produce and secrete adrenocorticotropic hormone (ACTH) into the bloodstream destined for the adrenal glands atop the kidneys. This activation of the adrenal glands is the final step of the hormone releasing pathway as they control the production and release of cortisol into the bloodstream. Cortisol is a naturally occurring steroid hormone and influences the following organ systems, all of which contain glucocorticoid receptors within the tissue that can bind cortisol; these include nervous, immune, cardiovascular, respiratory, reproductive, musculoskeletal, and integumentary (skin) systems (Thau et al., 2021). Through its interaction with these systems and its diurnal release schedule, cortisol plays a vital role in ordinary daily functioning as it maintains blood glucose levels, controls inflammation and ensures organs such as the brain and neuromuscular system receive enough energy to function properly (Hannibal & Bishop, 2014). In addition to its crucial role in normal daily functions, cortisol levels surge during the stress response to provide enough energy and substrate for the body and to maximize resources available for the body's ensuing

response to a stressor (Hannibal, 2014). This adaptive response is key to ensuring survival during acute responses, but excessive and prolonged cortisol secretion can have detrimental physical and psychological effects (Hannibal, 2014).

In conjunction with hormone production stimulated by the HPA axis, the ANS has a direct role in the physical stress response. Through synaptic transmissions between the two branches of the autonomic nervous system, the parasympathetic and sympathetic nervous system, the ANS promotes swift physiological changes within the body through neurological signals and outputs (Rotenberg & McGrath, 2016). The parasympathetic branch within the ANS is often referred to as the "rest or digest" phase of living that is responsible for control over our homeostasis at rest. On the other hand, the sympathetic nervous system is often referred to as the "fight or flight" response that controls our body's responses to a perceived threat or challenge. Generally, the ANS response to stress causes an activation in the sympathetic nervous system and inhibition of the parasympathetic nervous system (Ziegler, 2004). The ANS activation of the sympathetic nervous system prepares human bodies for action by increasing blood supply to the neuromuscular systems, increasing blood glucose levels, stimulating production of adrenaline and decreasing digestive system activity to retain nutrient levels at a higher threshold (Ziegler, 2004). Despite the key role that the ANS response plays in stress management, chronic stress due to continuous stressor exposure can lead to the continuous activation of the sympathetic nervous system with no counteraction from the parasympathetic nervous system. This outcome often leads to detrimental physiological and psychological outcomes described below (Won & Kim, 2016).

Effects of Stress on Health

Physical Health

While exposure to stressors is inevitable for humans throughout their lives, there are large interindividual differences in how stressors affect health and wellbeing (Rohleder, 2016). Despite these noted differences, a growing number of studies have found stress to be associated with the development and advancement of numerous diseases such as coronary heart disease (CHD), cancer, and type 2 diabetes (Dhabhar, 2018; Marik & Bellomo, 2013; Rohleder, 2016; Rosengren et al., 2004).

CHD is the most common form of disease affecting the heart and, according to researchers involved in the Global Burden of Disease project, was responsible for roughly 20% of deaths across Europe in 2016 (Townsend et al., 2019). Several meta-analyses have examined the effects of work-related stress in relation to the increased risk of coronary heart disease (Belkic et al., 2004; Hemingway & Marmot, 1999; Kivimäki et al., 2006). The study by Kivimäki and colleagues offers unique quantitative estimates that showed employees' exposed to high job strain exhibited a 50% increased risk of CHD diagnosis when compared to those who did not experience this form of work-related stress. Of the included 14 studies within this meta-analysis, after controlling for age and sex, a relative risk ratio of 1.43 was found to exist for those with high work strain and the onset of CHD when compared to those with low work strain. Expanding on the impact work stress has on increasing the risk of CHD, INTERHEART, a large, standardized case-control study (Yusuf et al., 2004) examined long-term stress (previous 12 months) in 15,152 patients with myocardial infarction and 14,820 control participants. Yusuf et al. operationalized stress in this study in the form of a psychosocial index composed of the following parameters: presence of depression, low locus of control, perceived stress at work/home, financial stress, and major life events. Interestingly, the researchers focused on recruiting participants from across the globe and were able to include participants from 52

different countries to derive a diversified sample. The results revealed that exposure to increased levels of the aforementioned stressors, increased the presence of depressive symptoms and a low locus of control increased the risk of acute myocardial infarction (a symptom of CHD) when controlling for smoking, diabetes, hypertension, and obesity as covariates. In light of the fact that psychosocial factors displayed consistently adverse effects on heart health in participants from across the globe, this points to the universally debilitative effects of stress on human functioning (Yusuf, 2006). Notably, stress actively contributes toward the onset and exacerbation of chronic health conditions such as coronary heart disease and myocardial infarction. Furthermore, sound evidence also exists linking stressors and the stress response with cancer progression and metastasis (Moreno-Smith, 2010).

Despite evidence highlighting the effects of stress on the *progression* of cancer, the ability for stress to *initiate* cancer is quite controversial and limited evidence exists in support of this relationship (Duijts et al., 2003; Geyer, 1991; Moreno-Smith, 2010). Inflammation is often linked in the development and progression of cancer and is brought on by proinflammatory cytokines, chemokines, adhesion molecules, and inflammatory enzymes (Singh et al., 2019). Despite the therapeutic benefits of acute inflammation such as increased blood flow to injured/infected tissues to advance the healing process along with increased immunosurveillance, chronic inflammation has been shown to enhance tumour cell survival, promote tumour proliferation, and increase metastatic spread (Multhoff, Molls & Radons, 2012). Furthermore, pathological inflammation can lead to tumour angiogenesis, metastasis, and chemoresistance (Singh, 2019). A meta-analysis assessed the inflammatory response in humans under acute psychological stress in laboratory conditions (Steptoe, Hamer & Chida, 2007), in which the majority of studies included measurement of inflammatory markers such as Interleukin-6 (IL-6)

and Interleukin-1 β (IL-1 β). Specifically, Interleukin-6 is a proinflammatory cytokine that is released by various cells including macrophages during the immune response but also by cancerous tumour cells within the tumour microenvironment (Masjedi et al., 2018). Interleukin-6 is involved in the proliferation and differentiation of cells in a large number of cancers (Kumari et al., 2016). Increasing levels of IL-6 are indicative of aggressive tumour growth and subsequent inadequate responses to cancer therapies such as radiation and chemotherapy (Kumari, 2016). It also plays a role in the tumorigenesis process by regulating cell apoptosis, metabolism and angiogenesis within the tumour microenvironment (Masjedi, 2018). Within the 30 studies that were included within this meta-analysis (Steptoe et al., 2007), robust effects were found for increased levels of circulating IL-6 ($r = 0.19$) and IL-1β ($r = 0.58$) following exposure to acute stress.

Norepinephrine (NE), a well-known stress hormone and neurotransmitter, has also been shown to upregulate levels of IL-6 in oral cancer patients which increases the severity of the cancer and promotes its proliferation and metastasis into surrounding tissues (Bernabé et al., 2011). Bernabé et al. mixed levels of NE that simulated concentrations of the hormone present during times of physiological stress in a cell culture containing oral squamous cell carcinoma (OSCC) cells. Results of the study revealed notable increases of IL-6 mRNA expression and production within the assay. With the introduction of NE, there was a 5-fold increase in IL-6 secretion from cancerous cells after one hour, 3.7-fold increase after six hours and a 3.2-fold increase after twenty-four hours. Although Bernabé's et al' research mainly comprised in-vitro assays opposed to in-vivo, the results indicate that NE presence at concentrations compatible with physiological stress levels can upregulate IL-6 expression and secretion from OSCC cells and affect tumour progression (Bernabé, 2011). Stress induced production of the IL-6

proinflammatory cytokine thus appears to be implicated in the progression and differentiation of cancer along with reduced therapy efficacy.

Proinflammatory cytokines, specifically IL-6 and tumour necrosis factor alpha (TNF-α), play a crucial role in the induction of another chronic physical health illness inline with insulin resistance, particularly diabetes mellitus (Afrisham et al., 2019). The stress response progresses into the downstream release of cortisol, which in turn, causes the mobilization of glucose into the blood stream to increase sugar levels. This is essential in the "fight or flight" response to ensure the body has enough energy available to feed our essential organ systems. Continual activation of the HPA axis, often due to increased levels of stress, causes heightened blood glucose levels and the onset of hyperglycaemia (Marik & Bellomo, 2013). For those living with diabetes mellitus (T2 Diabetes), high blood glucose levels are already prevalent within their bodies. Type 2 diabetes is an insulin resistant condition with associated beta-cell dysfunction (Goyal & Jialal, 2021). Upon initial onset of type 2 diabetes, increased insulin secretion occurs to maintain glucose levels in the normal range. As beta-cells change and alter their structure and function, insulin secretion decreases and glucose levels within the blood rise drastically. Paired with increased levels of stress, these extremely high levels of glucose within the bloodstream can lead to glucose toxicity, and more specifically, complications within pancreatic beta cells and vascular endothelial cells (Campos, 2012). This obstruction with pancreatic beta cells further decreases insulin release and exacerbates the hyperglycemic state as insulin typically causes reuptake of glucose into the tissue.

Despite implementing interventions targeting known risk factors such as obesity, physical inactivity, diet quality, smoking, hypertension, and abnormal cholesterol levels (Murea et al., 2012), the incidence of diabetes continues to rise. In order to examine the relationship between

perceived stress and type 2 diabetes onset, Harris and colleagues (2017) utilized data from the Australian Longitudinal Study on Women's Health. A significant relationship was found to exist for the total effect of perceived stress on type 2 diabetes onset, in particular, among women experiencing moderate to high levels of perceived stress (OR = 2.33) relative to women with no perceived stress. Furthermore, women experiencing low perceived stress exhibited an increase in type 2 diabetes onset as well (OR = 1.56) relative to those identified as not experiencing stress. As mentioned in the section above on the relations between stress and cancer, chronic stress levels directly activate the innate immune system, which, in turn, stimulate the production of proinflammatory cytokines such as IL-6. The IL-6 proinflammatory cytokine, along with the TNF-α cytokine, induce insulin resistance through the dysregulation of the insulin signalling pathway and are considered predictive markers for the development of diabetes mellitus (Afrisham, 2019). Through the phosphorylation of the insulin receptor substrate-1-associated proteins, proinflammatory cytokines can inhibit the binding and signalling of insulin which further exacerbates the reduced response to insulin in type 2 diabetes patients (Mattacks & Pond, 1999).

Mental Health

In addition to the adverse effects of stress on physical health conditions such as CHD, cancer and diabetes, stress plays a substantial role in the onset and exacerbation of numerous mental health conditions such as depression, anxiety, and disorders related to trauma (Schneiderman, Ironson & Siegel, 2008). Mazure (1998) conducted a review analyzing the associations between major adverse life events and major depressive disorder onset and found that more than 80% of community cases of major depressive disorders were preceded by a severe adverse life event. This relationship between stressful life experience and subsequent onset of

major depression has led to further research in the hopes of understanding the biological and cognitive factors that result in specific mental health disorders such as depression and anxiety.

To understand the extent to which accumulated adversity and stress contribute to the subsequent onset of depression and anxiety, Turner and Lloyd (2004) conducted a community-based study assessing exposure of lifetime stressors and the observed onset of several psychiatric health conditions. These psychiatric health conditions were depression, dysthymia, generalized anxiety disorder, social phobia, panic disorder, alcohol abuse and dependence, drug abuse and dependence, posttraumatic stress disorder, and/or antisocial personality. Through the use of interviews, either in person or over the phone, individuals shared their own personal experiences with a diverse range of lifetime stressors that were then compiled into a cumulative life adversity score consisting of 33 different items. Turner and Lloyd defined two forms of adversity being 'distal' and 'proximal'. Distal adversity represents the long-term effects of exposure to adverse events whereas proximal adversity is characterized as the immediate consequences of specific adverse events (Turner & Lloyd, 1995; Turner & Lloyd, 2004; Whitesell et al., 2007). Both proximal and distal adversity can precede the onset of mental illness and/or dependence symptoms with each form of adversity potentially contributing to future health problems. A strong relationship was observed between the presence of adverse life events and the increased risk of a depressive or anxiety disorder. To illustrate, the researchers used odds ratios to compare the risk of disorder onset at high versus low levels of adversity exposure. They found that individuals who had experienced a higher number of distal adversities (earlier in life) had double the odds of developing a disorder than those who had experienced fewer adversities, when controlling for proximal (more recent) adversities. In addition, individuals who had experienced a higher number of proximal adversities had a 53% higher odds of developing a disorder when

controlling for distal adversities. The researchers suggested that the number and timing of adversities experienced in a person's life significantly impact their risk of developing a mental health disorder; in particular, depression and/or anxiety.

The effects of chronic stress also affect areas of the brain through physical modifications of neural networks and volume variations of certain brain areas (Mariotti, 2015). Specifically, Lucassen and colleagues (2014) reported that stress affects areas of the prefrontal cortex and limbic system that exhibit neuronal plasticity (changing connection) via dendritic atrophy. Specifically, humans living with long-term occupational stress showed reduced gray matter of the dorsolateral prefrontal cortex (DL-PFC) and the anterior cingulate cortex (ACC) (Blix et al., 2013; Lucassen et al., 2014). The ACC is responsible for higher-level functioning such as attention allocation, impulse control, emotion regulation, and decision making along with regulating autonomic and endocrine functions (Devinsky, Morrell & Vogt, 1995). The prefrontal cortex region modulates social behaviour, controls aspects of speech and language, facilitates planning, decision making and has a role in short-term memory (Pizzagalli & Roberts, 2021). Alterations in neural plasticity in these regions are complex pathophysiological processes and can, on the one hand, be caused by depression, or on the other hand, be induced by stressors and contribute to the onset and development of depression (Liu et al., 2017). This research supports the idea that cognitive and biological stress reactivity may contribute independently to symptoms of depression.

Can Group Life Mitigate the Effects of Stress?

The *social identity approach* (SIA) to health is comprised of two intertwined, but distinct, social psychological theories: social identity theory (SIT; Tajfel & Turner, 1979) and self-categorization theory (SCT; Turner & Reynolds, 2012). This approach acknowledges that

individuals can describe themselves, and behave, not only as individuals (i.e., "I' and "me") but also as group members (i.e., "we" and "us") (Stevens et al., 2017). The social identity approach to health is encompassed within a *sociopsychobio* framework (Haslem et al., 2019). In contrast to George Engel's (1977) case for a *biopsychosocial* model of health, the sociopsychobio model presents the same three key elements of the biopsychosocial model (biology, psychology and social interactions) as dynamic and interdependent rather than privileging the effects of biological factors on the remaining elements of psychology and social interaction. Haslam and colleagues (2019) contrasted Engel's fixation on the biological aspect of health with the sociopsychobio framework that considers social interactions (or social group life) and psychology not as an appendage to biology, but rather as a basis for collective experiences that shape one another.

Social identity theory posits that a person's sense of who they are is based on their in-group memberships (Tajfel, 1979). This theory was conceptualized by Henri Tajfel and focuses on intergroup relations and how identification with ingroups in contrast with outgroups is able to shape behaviour. The origins of this theory go back to Tajfel's study in 1971 where schoolboys were asked to assign points to two laboratory groups, one to which they belonged and one to which they did not. Researchers assigned groups (based on arbitrary criteria) and study participants did not receive any compensation or information about future individual beneficiaries. The findings of this study highlighted strong ingroup favouritism and outgroup rejection as the boys tended to assign points to a greater extent within their own groups (Tajfel 1971). This finding illustrated that the act of categorization (i.e., a perceived association within a particular group, even on the basis of minimal or no criteria) encourages ingroup favouritism and outgroup rejection**.** Tajfel concluded that people develop social identities based on the various

social groups to which they belong. Tajfel and Turner (1979) also described a need and drive for "positive distinctiveness" in which individuals wish to achieve or maintain self-esteem by differentiating their ingroup from comparable outgroups. Social identities not only affect individual behaviour in isolation but also structures the way in which individuals engage, interact, and help one another (Haslam et al., 2018). Specifically, behaviours and actions that serve to advance the interests of group members are observed as they see the person(s) in need of help or support not as "other(s)", but rather as "one of us" (Haslam, 2018).

As an illustration of the power of social identification, Levine and colleagues (2005) conducted a study to observe the intergroup rivalries between soccer fans in hopes of examining the role of social identity in helping behaviours. Researchers recruited Manchester United fans and asked them to complete a questionnaire identifying what team they support, how long they have supported them, how often they watch their team play and how they feel about the successes and failures of the team. To avoid participants identifying that Manchester United supporters were of focus in this experiment, researchers asked participants to write down the team they support at the beginning of the questionnaire in a blank field. Upon completion, participants were asked to walk a short distance to another building to watch a video of compiled football matches and observe team and crowd behaviours. As participants were walking to the subsequent location, a jogger wearing either (1) a Manchester United jersey, (2) a Liverpool FC jersey, or (3) an ordinary and unbranded shirt, tripped and fell, displaying pain in front of the participant. Results supported the researchers' hypothesis in so far as the injured stranger wearing a Manchester United jersey (i.e., the ingroup) was more likely to be helped than if they wore a Liverpool FC jersey (i.e., the outgroup) or an unbranded jersey. Social identity theory provides an explanation for the inter-group relations that drive behaviour both through a shared

social identity and positive distinctiveness when comparing one's ingroup with other outgroups. However, social identity theory does not explicate to the same extent the antecedents of social identity and social identification *within* groups (Haslam et al., 2018), and so it is to self-categorization to which I now turn.

Self-categorization theory (SCT) shares many of the same tenets as social identity theory, but more specifically, sheds light on the cognitive processes through which individuals categorize themselves within various social groups (Hornsey, 2008). As such, self-categorization theory seeks to understand how, when, and why we categorize ourselves as a part of certain groups as opposed to others (Reimer et al., 2020). According to this theoretical framework, people tend to shift their self-conception according to the salience of various interpersonal (i.e., as an individual group member), intergroup (i.e., a member of some groups but not others), and/or superordinate categorizations (Cargile, 2017; Turner & Reynolds, 2012). This gives rise to the core psychological processes articulated within SCT which correspond to the extent to which an identity is *salient* based on both *accessibility* and *fit* within a given context.

Accessibility (also referred to as perceived readiness) reflects an individual's past history, experiences, and values that they draw upon to categorise oneself and others. This accessibility arises through ongoing socialised processes with significant others (friends, family, workplace) or cultural surroundings (e.g., media influences, educational upbringing), and reflects a person's readiness to make use of a particular social categorization. The perceived *fit* of a category depends on both comparative and normative considerations (Turner et al., 2012). Comparative fit corresponds to the extent to which people perceived that they are similar to others within a given social group, when also contrasted against people within different social groups. For example, a young adult might strongly identify with being an exerciser if surrounded by other physically

active people with whom they perceive themselves to be similar. Normative fit reflects the behaviours of group members and, in particular, the extent to which the behaviours displayed by group members are consistent with what one might expect of that social group (Oakes et al., 1987). If a person perceived that the behaviours of members of a current group align with one's own behaviours, then normative fit exists. In sum, when people are exposed to various social identities (based on family, school, or social interactions) that are perceived to be salient, this leads them to identify more strongly with others that display those shared or similar social identities. This shared sense of social identification can act as a psychological resource to help individuals bolster their self-esteem, reduce any sense of loneliness, as well as improved their health and wellbeing (Jetten et al., 2017).

In recent years, a growing body of research has explored ways in which a person's social relationships, social networks, social support and other social identity-based resources derive beneficial health outcomes in the form of a "social cure" (Cruwys et al., 2013; Haslam et al., 2018; Haslam et al., 2014; Jetten et al., 2012). A critical contributor in the protective mechanisms of group involvement, and ensuing social cure for well-being, is the experience of social support that one experiences in a group context (Greenway et al., 2015). During stressful situations or individual turmoil, the increased exposure to social support – especially from perceived in-group members – is important in dealing with stressful situations in a manner that reduces psychological harm (Greenway, 2015). Furthermore, when support is received from ingroup members, communications may be perceived in a more constructive manner due to the shared identities between oneself and other group members (Levine & Thompson, 2004; Levine et al., 2005). Building on literature linking increased social support to positive health outcomes, Bucholz and colleagues (2014) observed the effect of low social support on health outcomes in

patients with acute myocardial infarction. Interestingly, this study analyzed social support at both the time of acute myocardial infarction and 12-months afterwards. Results showed patients with low social support presented with poorer mental health functioning and depressive symptoms at the initial time of onset when compared to those with moderate/high social support. The differences between those identified as receiving low versus moderate/high social support persisted at the 12-month follow up which resulted in continuing mental health (e.g., depression) disparities between the different groups. Despite these mental health differences, those in the low social support group did not differ with regard to their physical health when compared with the moderate and high social support groups.

Social group involvement allows for increased social support through connection and identification whilst also playing a key role in influencing individual self-esteem (Harris & Orth, 2019). Self-esteem is defined as an individual's overall sense of personal worth or value (Du et al., 2017). Self-esteem impacts one's decision-making processes, relationships, emotional health, and overall wellbeing (Orth & Robins, 2014). In line with social identity theorizing, group members protect and enhance the status of their in-group through the process of 'positive distinctiveness' in order to positively improve their self-esteem (Martiny & Rubin, 2016). Individuals find themselves striving to create, maintain, and protect the positivity of their social identity by increasing the status of their ingroup while discriminating against comparable out-groups through the means of *in-group biases* (Martiny & Rubin, 2016).

Personal self-esteem is a crucial aspect of psychological health and well-being as it influences how individuals perceive and interact with the world around them (Jetten et al., 2015). Low self-esteem has been shown to be a predictor of poor mental health outcomes (Zimmerman, 1999; Jetten et al., 2015). Jetten and colleagues (2015) conducted research consisting of three

different studies to evaluate the effects of group membership on personal self-esteem in different populations. Across all three studies, that included school children, older adults from China, and homeless adults living in China, increased group membership was positively correlated with personal self-esteem (Jetten et al, 2015). The results suggest that belonging to multiple groups can potentially improve personal self-esteem levels regardless of one's age, socioeconomic status, and/or ethnicity.

The importance of group membership is not only emphasized through its ability to increase levels of personal self-esteem but also through its relationship with psychological disorders such as depression. Specifically, Cruwys and colleagues (2013) evaluated the *protective* and *curative* abilities that social group membership has against the development and progression of depression. They found that having a greater number of group memberships was *protective* against the development of depression among an initially non-depressed sample, whilst also being *curative* of reduced depressive symptoms in those living with depression currently. Specifically, among those participants that reported a history of depression, 41% who reported no group membership experienced depressive symptoms 6 years later compared to only 15% of participants that were involved with 3 or more groups.

Group Life and the Protective Effects against Stress

The natural physical and mental response to chronic and uncontrollable levels of stress has been implicated in numerous chronic health conditions such as cardiovascular disease, cancer, diabetes, and depression. Social relationships are critical components of human well-being as they fulfill both emotional and material needs that protect individuals from psychological harm and ensuing chronic illnesses. A recent body of social-psychological research has begun to focus on why and how social relationships and interactions relate to health

outcomes by focusing on the distinct benefits of *group* memberships (Haslam et al., 2018; Kyprianides & Brown, 2019). This literature has notably been underpinned by social identity theorizing to health to highlight the importance of an person's social identification with fellow group members. Social identification is associated with better adjustment, coping and wellbeing largely due to the fact that social identities satisfy basic psychological needs and provide the basis for productive engagement (Greenaway et al., 2015). These include the need to belong, the need for self-esteem, the need for control and the need for meaningful existence (Greenaway et al., 2015). As such, the social identity approach indicates that people gain a sense of increased self-esteem from their positive social identities within their ingroups (Kyprianides & Brown, 2019; Tajfel & Turner, 1979). Meaningful social identities also enhance social support, which in turn, enhance well-being (Haslam et al, 2018). When people identify and relate to one another through shared group memberships, they tend to give one another support and receive support more frequently (Kyprianides, 2019). Group members also "construe the support they receive more positively because they recognize fellow in-group members as 'one of us'" (Kyprianides, 2019, p. 3). Previous research has identified that social support and personal self-esteem operate as coping resources against the detrimental effects of social stressors in the form of a buffering mechanism (Gayman et al., 2013).

In a complementary line of research, Rosengren and colleagues (1993) examined the relations between stressful life events, social support, and mortality; mortality measures were based on deaths from coronary heart disease, cancer, alcohol related consumption, and non-specific/other sources. For those living with low emotional social support, the presence of stressful life events predicted an increased risk of mortality (Rosengren et al, 1993). Comparatively, there was no effect on mortality for stressful life events among those who

reported receiving high levels of emotional social support. In a similar regard, a more recent study by Heshizer and Knapp (2016) explored whether social support is able to alleviate work-related stress symptoms in teachers. Work stress was assessed by a measure of "actual danger" that tapped into the frequency of receiving, and self-reported risk of, threats within the workplace. Perceived social support was assessed in the form of organizational support, union support, and support from the principle, fellow teachers, and one's family. The researchers found that increased support from all groups (besides the union which showed no positive correlation) worked to alleviate work related stress symptoms.

To test the hypothesis that increased self-esteem has the ability to reduce an ensuing stress response in the face of a stressful task, Rector and Roger (1997) conducted the stroop test in order to simulate a high stress task for two different groups: a high self-esteem group and a neutral self-esteem group. These groups were created based on a fake personality test given to participants that was designed to manipulate their perceived self-esteem into a high category group or neutral category group. Furthermore, the researchers operationalized two different conditions being a low stress condition and a high stress condition. In both the high stress and low stress conditions, those who were allocated to the high self-esteem group showed superior performance and experienced less personal threat during the task**.** Across both the high and low stress conditions, the high self-esteem group reported a lower mean perceived subjective stress score while the neutral group reported a higher mean perceived subjective stress score. When taken together, the protective effects of psychosocial resources appear to have the potential to buffer against stressors and reduce the effects of chronic stress on the body and mind.

Purpose and Hypotheses

There is considerable evidence demonstrating the negative effects of stress on both mental and physical health outcomes (Kendler et al., 1999; McEwen, 2006). Despite this evidence, there is an absence of research examining the extent to which group involvement can moderate the relationship between stress and health outcomes. Given the potential implications for public health interventions, the purpose of this Master's thesis research was to investigate the extent to which group involvement buffers against the putative negative effects of cumulative stress on long-term mental and physical health outcomes. Two hypotheses guided the research: (1) Cumulative stress will prospectively predict the occurrence of mental and physical health outcomes, and (2) Increased levels of group involvement will mitigate the adverse effects of stress on long-term mental and physical health outcomes.

Method

Participants

Data were derived from the Midlife in the United States (MIDUS) study (https://www.midus.wisc.edu/). MIDUS researchers conducted a baseline study that ran from 1995-1997 that consisted of 7108 participants aged 25-75. Beyond the aforementioned baseline study that ran from 1995-1997, otherwise know as MIDUS 1 (M1), further data collection events included MIDUS 2 (M2) which ran from 2004-2009 along with MIDUS 3 (M3) which ran from 2013-2019. Of the initial sample of 7108 participants that participated in M1, 4963 participated in M2, and 3294 participated in M3 to provide a roughly 20-year range in data collection comprised of the same participants from the initial cohort. Only data derived from M2 and M3 were utilized in my thesis research as they were conducted most recently and contained variables of interest that were not included in M1 and were only initially introduced as part of M2. Data from M2 were operationalized as Time (T1) and data from M3 were operationalized as Time 2 (T2). These data consisted of participants from both the main longitudinal MIDUS study, as well as participants from the Milwaukee sub-study (Ryff et al., 2017), which represented a subsample of black participants who were recruited to diversify the original pool of participants.

The research subsumed within this thesis project included data from 3682 adults from around the United States (Mean age at T1 = 54.04 years, SD = 11.37). The inclusion criteria for participants enrolled in MIDUS included: being between the ages of 25 and 74 at the time of recruitment, the ability to read and write in English, residence in the United States, providing written consent, and completing a health screening questionnaire to ensure participants were physically and mentally capable of participating. Participants with severe mental or physical illness, or those with a history of substance abuse, were excluded from the study.

Overall, the sample at T1 was predominantly female (56.1%), currently employed (72.1%), living or residing with a partner (72.7%), and had children (87.1%). Furthermore, a vast majority of the participants identified as white (82.5%), and roughly half of the cohort had at least a high school education (46.6%) or a college degree (46.3%) (**See Table 1**).

Procedure

Within both M2 and M3 longitudinal follow up events, researchers conducted multiple projects covering an array of topics. Each project involved collecting data on variables from multiple fields of study, but only data derived from one project (with regard to both M2 and M3) were utilized in my Master's thesis project. This corresponded to The Survey Project, which was designed to investigate the role of behavioural, psychological, and social factors in contributing to age-related differences in physical and mental health.

The data derived from M2 and M3 in this study were procured online through the MIDUS Colectica Portal (https://midus.colectica.org/). As such, no institutional review board (IRB) was required prior to undertaking this project; parenthetically, the original MIDUS researchers received ethical approval through the University of Wisconsin Institutional Review Board and in doing so, ensured that all participants provided informed consent prior to providing data.

Measures

Group Involvement

Group involvement was measured by asking participants in the MIDUS 2 Survey Project the following question: "In a typical month, about how many times do you attend the following? Meetings of sports or social groups." **(See Appendix).** Group involvement utilizing this variable

was operationalized by Creaven and colleagues (2020) in a study analyzing the effects of cardiovascular reactivity on social participation.

Cumulative Stress

Cumulative stress was measured by combining psychosocial stressors from eight different domains (Slopen et al., 2013). Data derived from the MIDUS study allows for the measurement of a *cumulative stress score* as participants were questioned about multiple domains of psychosocial stressors at repeated time points (Slopen et al., 2018). These eight domains include relationship stress, financial stress, work stress, work-family spillover, perceived deprivation, neighborhood stress, discrimination, and recent problems in the family. Slopen et al. (2018) described a step-by-step procedure for creating a cumulative stress measure, which allows for a comprehensive assessment of stress across multiple domains. Their approaches begin by creating composite scores separately for multiple stressor domains (based on the grouped categories of stressors noted above) and then grouping them together into one main composite score across four main steps. First, the measurements related to a specific domain of stress are transformed into z-scores, which provide a common scale for comparison. Second, these z-scores are added together within each specific stressor domain. In the third step, the resulting sum is standardized again into a z-score containing all the stress measures within each individual domain. Finally, once these individual domain scores are standardized, the next step is to create a cumulative stress score, by combining multiple types of stressors. This is done by summing the z-scores of each stressor domain and then standardizing this new sum into another z-score.

Relationship stress consisted of four measures, family strain (Cronbach's α=0.8), friend strain (α=0.79), marital risk (α=0.69), and spouse/partner strain (α=0.81) (Walen & Lachman,

2000). *Financial stress* was assessed using two measures that capture financial difficulties (α=0.79). *Work stress* consisted of measures of skill discretion (α=0.68), decision authority (α=0.85), demands (α=0.74), coworker non-support (α=0.74), supervisor non-support (α=0.87), risk of injury on the job, and job insecurity (Karasek, 1985). *Work-family spillover* was measured by negative work-to-family spillover (α=0.84) and negative family-to-work spillover (α=0.81) (Grzywacz, 2000). *Perceived deprivation* was assessed by the extent to which individuals are dissatisfied with their relative position or experiences in the domains of work opportunity (α=0.78), living environments (α=0.80), and ability to provide for one's children (α=0.69) (Ryff et al., 1999). *Neighbourhood stress* was assessed by inquiring about the safety and trust in one's neighbourhood (α=0.68) (Keyes, 1998). *Discrimination* included an inventory of major discrimination events and the everyday discrimination scale (α=0.97) (Kessler, Mickelson & Williams, 1999). A final measure that was operationalized by Slopen et al. (2013) but was not sourced from previous research corresponded to *past-year family problems* that measured financial, health, legal and relationship problems in the respondent's immediate family including parents, children and spouse (**See Appendix).** Scores derived from each of these measures within the cumulative stress score have been found to display acceptable internal consistencies, with Cronbach alpha coefficients ranging from 0.68 to 0.97 (Slopen et al., 2013).

Mental Health

Mental health was assessed using measures of general affective distress, affective diagnosis based on self-report, and self-reported affective disorder (**See Appendix)**. General affective distress was assessed as per the Non-Specific Psychological Distress Scale by Kessler and colleagues (2002). Specifically, participants answered how often during the previous 30 days they experienced emotions of worthlessness, hopelessness, nervousness, restlessness, or fidgety

feelings while also assessing two emotional states: (1) how much of the time everything was an effort, and (2) how often they were so sad that nothing could cheer them up. Previous Cronbach alpha coefficients reported during MIDUS 1 (0.86) and MIDUS 2 (0.83) showed good evidence for internal reliability of the general affective distress measure (Charles et al., 2013).

Affective diagnosis based on self report was assessed through participants meeting the criteria for one or more of the following: (1) Major depressive disorder, (2) Dysthymia (a chronic form of depression), or (3) Generalized anxiety disorder. Specifically, this measurement is derived from the Composite International Diagnostic Interview Short Form (CIDI-SF; Kessler et al., 1998). This measure was developed by the World Health Organization using criteria established in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition (DSM-IIIR;* American Psychiatric Association, 1987).

Finally, self-reported affective disorder was assessed with a single item that asked whether in the past 12 months participants had experienced or been treated for "anxiety, depression, or some other emotional disorder".

Physical Health

In each phase of the MIDUS studies, participants were asked (in The Survey Project) if they had experience of, or had been treated for, any of 15 different chronic health conditions during the previous 12 months. Additionally, participants were asked if they had experienced or been treated for cancer at any point in their life as this illness requires ongoing treatment from medical professionals. Overall, twenty-nine individual conditions were assessed in the survey, that were clustered into 15 chronic health condition categories. These chronic health categories include autoimmune disorder/lupus, cardiovascular conditions, diabetes or high blood pressure, digestive conditions, foot trouble, hay fever, gall bladder conditions, neurological conditions,

lung conditions, pain related conditions, skin trouble, thyroid disease, trouble with gums/mouth/teeth, urinary or bladder problems, and cancer. As per procedures implemented by Piazza and Colleagues (2013). Grouping of illnesses that are the same or similar was necessary in order to prevent these conditions from being measured multiple times within the same participant (**See Appendix).**

Covariates

Careful consideration was given to ensure that any findings were not attributed to confounding variables that have been known to previously influence physical and mental wellbeing (Piazza, 2013; Charles et al., 2013). In this study, education, age, and gender were operationalized as covariates in the analyses in which mental and physical health measures were operationalized as dependent measures (Piazza, 2013; Charles et al., 2013). Furthermore, in line with Lockwood and colleagues (2022), three dummy-coded variables were included as covariates in both the mental and physical health analyses. These variables were employment status, parental status, and partner status and are researcher created rather than variables measured in the MIDUS study. Additional covariates of race, smoking status, BMI, and current emotional/mental health were also included in the analysis in which physical health variables were operationalized as the dependent measure. Each of these additional covariates have been previously associated with an increased risk of reporting a chronic physical health condition (Sin et al., 2021; Piazza, 2013; Charles et al., 2008).

Education was assessed through an ordinal scale item of less than a high school education, a high school diploma or a General Equivalency diploma, some college but no degree, a four-year degree, and finally, at least some graduate school. *Race* was operationalized as a dichotomous variable of white (0) or other (1) due to a lack of ethnic minorities included in the

sample. Parenthetically, it should be noted that the MIDUS researchers used the term 'race' rather than 'ethnicity', and so we followed their conceptualization in the current study. *History of smoking* was measured by asking the sample if they had "ever smoked cigarettes regularly", which was dummy coded on the basis of whether participants have ever smoked or not. *BMI* was calculated through the collection of participant's self-reported height and weight (i.e., self-reported weight divided by height in kilograms and meters squared, respectively). *Current mental/emotional health* was assessed by asking the following: "Would you say you mental or emotional health is excellent, very good, good, fair, or poor?". Employment status was assessed by asking if participants were currently working or not. Parental status was assessed by asking participants if they currently have no children or one or more children. Partner status was assessed by asking participants if they were currently married or living in a marital-like relationship or if they had neither relationship. Finally, T1 measures (MIDUS 2) of the respective dependent variable was operationalized as a covariate in each of the four main analyses described below.

Data Analysis

After collating the variables of interest from the MIDUS Colectica and exporting them into Excel, the data were subsequently imported into SPSS (version 28). Prior to conducting the main analyses, which involved examining whether group involvement moderates the relations between cumulative stress and the physical and mental health measures described above, the data were first examined to ascertain both the amount and patterns of missingness. Newman (2014) emphasized the importance of addressing missing data in research studies, noting that researchers in psychology often ignore recommended treatments for handling missing data, which can lead to bias and errors in their findings. As Newman noted, missing data can be

categorized into three types of missingness; that is data that are missing at the item-level, construct-level, and/or person-level. Item-level missingness refers to situations where respondents leave one or more items unanswered on a multi-item questionnaire instrument. This can happen for various reasons, including if or when individual items deal with sensitive information or when participants are confused about individual items. Construct-level missingness occurs when respondents do not provide responses in relation to an entire sub-scale scale or all questions that pertain to a specific construct. Person-level missingness refers to cases where an individual fails to respond to any part of the survey. These distinctions can help researchers better understand the nature of missing data and tailor their handling of it accordingly. Before reporting the levels of missingness in the current study, it is important to emphasize one major suggestion as reported by Newman (2014): that is, one should not use listwise deletion. Listwise deletion is a method of handling missing data that involves removing all cases or persons who have any missing data before proceeding with the analysis. Essentially, the analysis is conducted only with complete cases, and any incomplete cases are excluded from the analysis. Such an approach has the disadvantage of treating missing data at the item and/or construct levels as missing at the person level, which can be problematic for several reasons. Specifically, the use of listwise deletion often greatly reduces a study's sample size and statistical power (while also increasing the odds of Type II error) and only supports inferences to those individuals that filled out the survey completely (Newman, 2014).

When data are missing, Newman (2014) recommended the pursuit of some key steps. First, if data are missing at the item-level, the suggested practice is to use each person's mean across available items to replace the missing data and represent the focal construct. Once item-level missing data are dealt with, it is essential to ascertain the amount of missing data at the

construct-level and/or person-level and examine the patterns of missingness; that is, as per Rubin's (1976) typology, examine whether data are Missing Completely at Random (MCAR), Missing at Random (MAR), or Missing Not at Random (MNAR). Once these patterns are identified, the remaining missing data can be handled using techniques such as multiple imputation or maximum likelihood techniques (Newman, 2014).

With regard to the cumulative stress data, missing data were handled by the MIDUS researchers prior to utilization in the current study (as per Williams & Slopen, 2019). Specifically, when sub-facets of cumulative stress were found to be missing, but those missing data were found to be unrelated to participants' demographic information, missing data (accounting for 0% to 22% of cases) for those sub-facets were substituted with the lowest possible value (Haight et al., 2023). When missing data were found to be MNAR, sequential multiple imputation was used following procedures by Ranghunathan et al. (2001).

In addition to the nature, and handling, of missingness within the cumulative stress measures, the extent of missing data varied across the four main analyses described below, and thus, a separate description and explanation of the missing data patterns is provided. Item-level missing data was initially identified for three variables. Specifically, participants had missing data at the item-level in the general affective distress model for the covariate variable of T1 general affective distress (11.0% of participants), as well as for the dependent variable of T2 general affective distress (11.1% of participants). It is important to mention that some participants were missing either the T1 or T2 general affective distress measures, while others were missing both. Furthermore, item-level missing data was observed for two participants (0.0005% of participants) in the physical health model, specifically for the covariate of emotional health. To address these item-level missing values, the missing values were replaced

with the mean value across the available items for each respective participant, as suggested by Newman (2014).

With regard to ascertaining missing data at the construct-level, 15.8% of the participants had missing data for the moderator variable of group involvement. Additionally, in all four main analyses, a very small percentage of participants (only four individuals, amounting to less than 0.1% of the sample) had missing data at the construct level for the covariate of education. In the analysis focusing on self-reported affective disorder, it was observed that there was exclusively construct-level missingness across the variables in the model. This corresponded to T1 self-reported affective disorder for 10.7% of the participants, as well as T2 self-reported affective disorder, which was missing for 11.8% of participants. Lastly, in the analysis examining physical health, construct-level missing data was found for several covariates. Specifically, BMI had missing data for 14.3% of the participants, smoking status for 25.9% of the participants, and race for 0.4% of the participants.

With regard to Rubin's (1976) typology, data that are MCAR means that the probability of missing data is unrelated to both the observed data values and the missing data values. MAR means that the probability of missing data depends on other observed data in the dataset but not on the missing data values. Lastly, MNAR can occur when the likelihood of missingness is influenced not only by the observed data but also by the specific values that are missing. To assess whether the missing data in each analysis followed the Missing Completely at Random (MCAR) pattern, Little's (1988) chi-squared test was performed. The results indicated that the missing data for self-reported affective disorder ($\chi 2$ (2) = 0.21, p = 0.90), physical health ($\chi 2$ (5) = 6.8, p = 0.29), general affective distress ($\chi 2$ (27) = 40.1, p = 0.051), and affective diagnosis based on self-report ($\chi 2$ (2) = 0.19, p = 0.91) were consistent with the MCAR (with *p*s > .05). To

address the missing data, multiple imputation methods (Newman, 2014) were employed in the analyses involving physical health, general affective distress, and affective diagnosis based on self-report. However, in the modeling of self-reported affective disorder, expectation maximization (Newman, 2014) was used to handle missing data on the basis of the binary nature of the dependent measure (T2 self-reported affective disorder) and covariate (T1 self-reported affective disorder).

Following the completion of missing data imputation, moderation analyses were conducted for each of the four health outcomes. All covariates for these analyses were derived from T1 (i.e., MIDUS 2), which was the baseline measurement point for this study. The independent variable of cumulative stress was based on those measures derived at T1. Similarly, the moderating variable of group involvement was also based T1 measures. The dependent variables of mental and physical health outcomes were measured at T2 (i.e., MIDUS 3), which occurred approximately 10 years after T1.The moderation analysis for general affective distress was conducted using a general linear regression, while the moderation analyses for the dichotomous dependent variables of self-reported affective disorder and affective diagnosis based on self-report were undertaken using binary logistic regression. Finally, for physical health, due to the count data of the dependent variable and the high number of zeros in the dataset, a negative binomial Poisson regression was utilized. Not only did this dataset have a higher number of zeros but it also displayed overdispersion when analyses were conducted using a simple Poisson regression. Negative binomial Poisson regression is an extension of Poisson regression that allows for larger variances than means, which makes it suitable for count data that are overdispersed (Cameron & Trivedi, 2013).

Results

Descriptive Statistics

Bivariate correlations between study variables are shown in Table 2. Of particular relevance to the current research, there were significant correlations between T1 cumulative stress and T2 mental health outcomes for each of general affective distress ($r = 0.30$, $p < 0.01$), self-reported affective disorder ($r = 0.17$, $p < 0.01$), and affective diagnosis based on self-report ($r = 0.19$, $p < 0.01$). The bivariate relationship between T1 cumulative stress and T2 physical health outcomes was also significant although very small ($r = 0.07$, $p < 0.01$).

Mental Health Conditions

Moderation Analyses – General Affective Distress

For the moderation analyses, linear regression was used to test whether group involvement moderates the effects of T1 cumulative stress in relation to T2 general affective distress. Prior to conducting this analysis, tests for linearity were undertaken using the Box-Tidwell procedure (1962), scatter plot analysis, and the Durbin-Watson test. The Durbin-Watson test checks for autocorrelation in the residuals, which would violate linearity assumptions. A value between 1.5 and 2.5 indicates no significant autocorrelation. The Box-Tidwell procedure and scatter plot analysis confirmed that the predictor was linearly related to the outcome variable. Furthermore, the Durbin-Watson test produced a value of 2.010, indicating that the assumption of linearity was met, and the regression model was appropriate for analyzing the relationship between variables. As shown in Table 3, there was a main effect for cumulative stress on general affective distress ($\beta = 0.12$, $p < 0.001$) such that higher levels of cumulative stress were associated with higher general affective distress scores. There was no main effect for gender ($\beta = 0.01$, $p = 0.31$) or group involvement ($\beta = 0.005$, $p = 0.39$) on general affective

distress. Finally, the stress*group involvement interaction effect was non-significant ($\beta = 0.02$, $p = 0.18$) which suggests that group involvement did not moderate the relationship between Time 1 stress and Time 2 general affective distress.

Moderation Analyses – Self-Reported Affective Disorder

For the moderation analyses, a binary logistic regression was performed to ascertain the interaction effect between cumulative stress and group involvement in predicting self-reported affective disorder. Prior to running the analyses, assumption testing was undertaken to confirm there was a linear relationship between the continuous independent variables and the logit transformation of the dependent variable. This involved evaluating the linearity of the continuous predictor variables with respect to the logit of the dependent variable using the Box-Tidwell procedure (1962). The predictor variable was linearly related to the logit of the dependent variable. Furthermore, this model explained 30% (Nagelkerke R^2) of the variance in self-reported affective disorder. The binary logistic regression model was statistically significant, $\chi2(10) = 771.9$, $p < 0.001$. Of the 10 predictor variables, 5 were shown to be statistically significant: cumulative stress, gender, T1 self-reported affective disorder (M2), employment status, and parental status (as shown in Table 4). The stress*group involvement interaction was non-significant ($\beta = 0.005$, $p = 0.60$) which suggests that group involvement did not moderate the relationship between T1 cumulative life stress (M2) and T2 self-reported affective disorder (M3).

Moderation Analyses – Affective Diagnosis based on Self-Report

For the moderation analyses, a binary logistic regression was performed to ascertain the interaction between cumulative life stress and group involvement in predicting the likelihood that participants had an affective diagnosis based on self-report at T2. Prior to running the analyses, the Box-Tidwell (1962) procedure was utilized to check for linearity between the continuous

predictor variable and the logit transformation of the dependent variable. Furthermore, this model explained 19% (Nagelkerke R^2) of the variance in affective diagnosis based on self-report. The binary logistic regression model was statistically significant, $\chi 2(10) = 385.1$, $p < 0.001$. Of the 10 predictor variables, 6 were shown to be statistically significant: cumulative stress, gender, T1 affective diagnosis based on self-report, parental status, employment status, and age (as shown in Table 5). The stress*group involvement interaction effect was non-significant ($\beta = 0.009$, $p = 0.47$) suggesting group involvement did not moderate the relationship between Time 1 stress and Time 2 affective diagnosis based on self-report.

Physical Health Conditions

Before performing the moderation analysis, the assumption of linearity was assessed by creating a residual scatterplot, which showed no violations. As for the moderation analyses, a negative binomial Poisson regression was conducted to analyze whether group involvement moderated the effects of cumulative life stress in relation to physical health outcomes. Due to the nature of the dependent variable being count data, Poisson regression was initially chosen. However, due to increased variation in the data that was greater than the mean (overdispersion), a negative binomial model was implemented (Cameron & Trivedi, 2013). Furthermore, as the negative binomial model allows for the variance to be greater than the mean, this model is applicable and encouraged when dealing with count data that has a high frequency of zero values, as occurred in the current dataset (Cox et al., 2019). The likelihood ratio chi-square test (Omnibus test) indicated that the full model was a significant improvement in fit over a no predictor model ($p < 0.001$). Within the model, 7 variables were significantly related to physical health at T2. These included cumulative stress, gender, T1 physical health, race, smoking status, BMI, and age (as shown in Table 6). Of direct relevance to this study, the stress*group

involvement interaction was non-significant ($\beta = -0.01$, $p = 0.17$) which indicates that group involvement did not moderate the relationship between T1 cumulative stress and T2 physical health outcomes.

Discussion

A growing body of evidence suggests that belonging to or being a member of social groups positively contributes towards individual heath and wellbeing (Lam et al., 2018; Haslam et al., 2018). Given this knowledge, the goal of this research was to investigate whether involvement in sport or social groups moderated the relationship between cumulative life stress and the presence of long term chronic mental and physical health conditions experienced within a large sample of adults from the United States.

Mental and Physical Health Trends within MIDUS

The results of the regression analyses suggest that specific demographic and lifestyle factors may contribute to downstream general affective distress, self-reported affective disorder, affective diagnosis based on self-report, and physical health illnesses. The regression coefficients for gender in three of the models, namely self-reported affective disorder, affective diagnosis based on self-report, and physical health illnesses, suggest that women report experiencing more health problems than men for each of the aforementioned illnesses. These findings align with prior research, which has consistently shown that women exhibit higher rates of depression and anxiety compared to men (Bromet et al., 2011).

However, research on the prevalence of chronic physical conditions between genders has yielded inconsistent results. While women generally have a longer lifespan than men (Crimmins et al., 2019; Matud, 2017), studies have found that men are more likely to have heart disease, stroke, and diabetes, whereas women are more likely to have arthritis and depression (Crimmins et al., 2019; Tam, 2020). In Canada's Aging and Chronic Diseases Report, Dr. Theresa Tam, the Chief Public Health Officer in Canada, highlighted that men aged 65+ exhibit a higher occurrence of specific chronic health diseases such as diabetes, cancer, heart failure, and gout compared to women aged 65+ (Public Health Agency of Canada, 2021). In contrast, women

aged 65+ present with higher levels of chronic health conditions such as osteoporosis, arthritis, dementia, and anxiety disorders compared to men. At a minimum, the results of the current study suggest that sex and gender need to be considered in any predictive longitudinal models, at least in controlling for their effects as substantive covariates, when examining the relations between stressors and putative physical and psychological outcomes.

In addition, employment status was found to be a significant predictor of general affective distress, self-reported affective disorder, and affective diagnosis based on self-report with unemployed individuals being at a higher risk of developing such a disorder. This finding is consistent with previous studies that have shown the impact of job loss in relation to the onset of poor mental health outcomes (Norstrom et al., 2019; Shimzu et al., 2015). The results also revealed a positive correlation between age and the number of physical health outcomes at T2, indicating that older age is associated with increased physical health conditions. This finding is consistent with previous research that has linked aging to increased risk of chronic diseases, decreased physical function, and overall decline in health (Crimmins & Beltrán-Sánchez, 2011; Lowsky et al., 2014). Taken together, these findings suggest that age may be an important factor, and should (as with gender), be considered as a control variable within any longitudinal study that is designed to ascertain the predictive utility of various candidate variables.

Does Group Involvement Protect against the Effects of Stress in Relation to Downstream Health?

The findings of the moderation analyses highlight that cumulative stress is related to the occurrence of downstream health problems; in particular, mental health problems. Indeed, this finding would appear to underscore the necessity of early interventions to alleviate the adverse effects of stress. The positive correlations observed between T1 cumulative stress and T2 mental

health outcomes, assessed approximately 10 years later, are consistent with previous studies linking stress to affective disorders (Stansfeld et al., 2011; Zisook et al., 2007). Given the increasing prevalence of mental health disorders worldwide, the identification of stress as a risk factor for these conditions several years later can have important implications for public health. By recognizing stress as a significant contributor to these disorders, public health initiatives can prioritize stress management and prevention strategies to reduce the overall burden of mental health conditions in communities. These prevention and management strategies can target populations in schools, workplaces, and communities, which have been shown to mitigate the impact of stress and lower the risk of mental health disorders like major depressive disorder (Munoz et al., 2015). Moreover, the weaker yet significant correlation between T1 cumulative stress and T2 physical health outcomes suggests that stress may also have detrimental effects on physical health. This finding is in line with previous research linking stress to various physical health problems, such as cardiovascular disease and immune system dysfunction (Cohen et al., 2007; McEwen & Stellar, 1993).

Beyond ascertaining the relations between stressors and various health outcomes, the main objective of conducting the moderation analyses was to explore whether group involvement acted as a protective factor against the adverse effects of cumulative stress on subsequent mental and physical health conditions, when assessed approximately 10 years later. The results indicated, contrary to the study's *a priori* hypotheses, that involvement in sport or social groups did not moderate the association between cumulative stress at T1 and the health outcomes at T2. This finding stands in contrast to prior studies that emphasized the beneficial effects of group involvement on stress management and well-being, which were attributed to the psychosocial resources acquired through social interaction (Sandstrom & Dunn, 2014; Holt-Lundstad et al.,

2010). As one example, Jetten et al. (2022) conducted a study that examined the effects of physical activity and social group memberships on mental health outcomes over time. When considering all the groups that individuals specified that they were members (i.e., social and sport group), belonging to multiple groups was related to lower levels of depression and anxiety over time, independent of initial levels of depression and anxiety. In a similar regard, Stevens et al. (2021) aimed to investigate the effects of group involvement, as well as the loss of access to groups, on mental health and well-being. This research was conducted specifically in the context of group physical activity across two separate studies. In one study, data from a population study of older adults in England (N = 4,549) revealed that belonging to sport or exercise groups predicted fewer symptoms of depression four years later than those who did not belong to such groups (Stevens et al, 2019, study 1). This relationship was mediated by two factors: those who participated in groups engaged in physical activity more frequently than those who did not and experienced lower levels of loneliness. Non-group participants had almost twice the rate of clinical depression compared to group participants. A separate study focused on Australian adults (N = 635) who were members of sport and exercise groups (Stevens et al, 2019, study 2). Specifically, during the COVID-19 pandemic, when participants experienced greater loss of access to group-based activities, they subsequently experienced more severe depression symptoms than those with fewer losses of group-based activities. Clinical depression rates were over twice as high among those who had lost access to more than two groups compared to those who had lost access to fewer than two groups. The relationship between the number of groups lost and depression symptom severity was mediated by increased feelings of loneliness, but not by overall physical activity levels.

The lack of a buffering effect of group involvement on the relationship between stress and health outcomes suggests that group involvement is not a substantive third variable that protects against cumulative life stress. On the other hand, it is conceivable that the operationalization of group life that was used in this study was not a sensitive measure of group involvement. Group life was assessed by asking participants how many times in a typical month did participants take part in social or sport groups. Such a measure does not provide any indication of the *quality of group interactions*, or the extent to which participants felt connected to others within those groups, and so may well have been an imprecise measure of their group involvement as part of the MIDUS study. This issue is discussed in greater detail below. It is also conceivable that participants' involvement in group activities may have substantially changed during the 10-year period between data collection during MIDUS 2 and MIDUS 3. Of course, other factors (beyond group involvement) might be more salient moderate of the relations between cumulative stress and health outcomes, such as coping strategies and personality traits (Cohen & Janicki-Deverts, 2009; Korten et al., 2017). As such, it is important for future research to investigate the complex interplay between stress, individual differences, other candidate moderators and salient health outcomes.

Study Strengths

The utilization of the MIDUS dataset offers several notable strengths for this study. First, the dataset encompasses a comprehensive range of questions and variables that were collected by the MIDUS researchers. This extensive coverage allows for effective statistical control of various demographic factors and health behaviors, as highlighted by Kahan et al. (2014). By including relevant covariates, we enhance the accuracy of our modeling while reducing potential errors caused by confounding variables. Second, longitudinal studies, as emphasized by Caruana

and colleagues (2015), allow researchers to assess relationships between risk factors and disease development over different timeframes. By employing a research design utilizing the longitudinal nature of the MIDUS dataset, we can explore the dynamic nature of these relationships and gain a deeper understanding of the factors that might contribute to disease development. Third, as noted by Button et al. (2013) and Kline (2016), a larger sample enables us to detect smaller effect sizes and obtain more precise estimates. An effect size refers to the magnitude of the relationship or difference between variables. A larger sample size provides more statistical power, which increases the likelihood of detecting smaller effect sizes that may exist in the population, helps reduce uncertainty, and increase the precision of estimates (Cohen, 1992; Kline, 2016). Lastly, the public availability of the MIDUS dataset contributes to the overall strength of this research. The accessibility of the dataset enables researchers from various disciplines and institutions to access the data and conduct independent analyses. This open access framework facilitates cross-disciplinary collaborations, fostering a collaborative environment for researchers to exchange insights and findings and supporting the progress of scientific research throughout numerous fields.

Study Limitations

While this study has some notable strengths, there are also some limitations that need to be addressed. For one, the sample of participants included in the analyses was predominantly Caucasian, with only about 17% of participants coming from racial and ethnic minority groups. Although the MIDUS researchers attempted to address this issue by recruiting a sample of African American participants from Milwaukee, Wisconsin, the underrepresentation of minority groups in this study remains a concern. This is particularly important in the context of chronic health conditions, where racial and ethnic minorities have been shown to be at a higher risk than

their European American counterparts of multiple health disparities (Williams and Cooper, 2019). Research has consistently demonstrated that Black, Hispanic, and Latinx populations are more likely to develop chronic diseases and experience mental health disorders. For example, non-Hispanic Black participants have been found to have a higher level of chronic disease burden than non-Hispanic white participants, while Hispanic individuals are more than twice as likely to report diabetes or CVD when compared to non-Hispanic white individuals (Bobo et al., 2016; Davis et al., 2017; Quinones et al., 2019). The underrepresentation of these groups in the sample presents a challenge for generalizing the study findings to the wider population. Drawing inferences from particular observations to the population as a whole relies on having a sample that is representative of that population. Census data shows that white individuals make up around 60-65% of the US population, but in this study, they made up 83% of the sample (Gobo, 2004; US Census Bureau, 2010, 2020). Therefore, caution must be exercised when generalizing the study results. Given the higher prevalence of chronic health conditions in minority populations, it is increasingly important to conduct research that focuses on reducing these conditions in these groups. In future studies, efforts should be made to recruit a more representative sample of participants from diverse racial and ethnic backgrounds to improve the generalizability of the findings.

Limitations related to the use of self-report measures should also be recognized. Due to the low cost of implementation, relative ease of use, and flexibility, self-reports are often a preferred method of data collection for large scale epidemiological studies (Hidalgo & Goodman, 2012; Kormos & Gifford, 2014). Within health research, self-reported survey data can provide information on particular experiences and perspectives that may not be captured by medical records or structured interviews (Hidalgo, 2012). With that said, the use of self-report

data also brings into play the risk of recall bias, in which there is an error in the recollection of information or events (Brusco & Watts, 2015). This can include an incorrect timeline of events, forgetting that an event occurred, or recalling an event that did not actually occur (Brusco, 2015). Due to the nature of the questions in this study, in particular, the physical health questions that asked whether or not a chronic condition had been experienced in the past 12 months, recall bias might have contributed to participant responses. Within health research, specifically in situations in which one is being asked about the presence or occurrence of chronic diseases/illnesses, one way to address the validity of self-reported data is to cross-check participant responses with official health records (Hidalgo & Goodman, 2012). This was not done in the MIDUS study, and it is conceivable that respondents may not have answered a question(s) accurately, particularly in the case of sensitive questions regarding their health (Demetriou et al., 2015). This limitation is tied to the phenomenon of social desirability bias, in which respondents answer questions in a seemingly socially acceptable way (Vesely & Klockner, 2020).

A further potential limitation of this study corresponds to the validity of the measure that was used to assess group involvement. Previous research has identified that group involvement can contribute to the development of social identity and enhance individual health and wellbeing by providing psychosocial resources to group members (Haslam et al., 2018; Taylor, 2011; Wiley et al., 2018). With that said, the number of groups with which a person engages over a one-month period sheds limited light on the quality of interactions that one experiences within those groups, the meaningfulness of any group ties, and/or the extent to which a person's social identities are intertwined with other group members. Indeed, it is very plausible that the measure of group involvement was not sufficiently sensitive to tap into the quality of participants' group lives. As a result, we were unable to focus on the quality of the groups while solely focusing on

the quantity of groups with which participants were involved. To address this limitation, researchers could, in future, use measures of group involvement that reflect the quality of interactions among group members. This could include carefully considering the goals, values, and culture of each group to which a person belongs, as well as their level of engagement and commitment towards those groups (Uchino, Cacioppo, & Kiecolt-Glaser, 1996). By using such methods, researchers can obtain a more nuanced understanding of the psychological benefits of group involvement and potentially develop more effective interventions to promote health and wellbeing.

Furthermore, this study was limited by the way in which participants' physical health conditions were measured by the MIDUS researchers. A focal question asked whether participants had been treated or experienced symptoms of specific conditions in the previous 12 months before answering the questionnaire. It is important to acknowledge the limitations of using a passive prospective design with only two timepoints, as this may not capture changes in health status that occur outside of the data collection periods. In this study, if participants had an illness between the first timepoint (MIDUS 2) and 12 months before the second timepoint (MIDUS 3) but did not recall its presence within the last 12 months, they would have answered negatively. This may have resulted in an underestimation of the true prevalence of illness and influenced how participants answered. Despite this limitation, the 10-year span of the MIDUS study is a strength and provides valuable insights into the long-term health outcomes of individuals.

A final limitation of the current study lies in the adopted longitudinal design, which involved pre and post-tests conducted over a span of 10 years. The lengthy intervals between measurements may have resulted in the loss of valuable information and may not capture the

dynamic nature of the variables under investigation (Dimitrov & Rumrill, 2003). Additionally, relying on a passive prospective observational design with only two assessments over 10 years will likely have resulted in important short-term fluctuations and intra-individual variations that were not captured (Dimitrov, 2003). To address these limitations, future epidemiological research involving more frequent assessments of stressors, group involvement, and health outcomes should be considered. This approach offers several advantages, including a more detailed understanding of within-person dynamics and the ability to investigate dynamic processes that unfold over shorter timeframes.

Conclusion

In conclusion, the research reported in my Master's thesis sought to investigate the potential moderating effects of social and sport group involvement on the relationship between cumulative stress and long-term physical and mental health outcomes. The findings revealed several important insights. Contrary to my *a priori* expectations, the findings did not support the hypothesis that group involvement would moderate the relationship between stress and health outcomes. Specifically, greater involvement in group life, as observed within MIDUS 2, did not appear to mitigate the effects of cumulative stress on either physical or mental health outcomes approximately 10 years later, as assessed in MIDUS 3. These results suggest that the protective effects of group membership on health outcomes, as reported in previous research, may not be applicable in the context of the MIDUS dataset. It is possible that other individual or contextual factors not examined in this study may interact with various stressors to protect (to some extent) against depleted health outcomes. It is also conceivable that the measure of group involvement that was used in the current study lacked sufficient sensitivity and validity with respect to reflecting the quality of ‘group life’. As such, future research would appear warranted to

ascertain whether high quality relationships embedded within group life protect against stressor-strain effects, or whether such an analysis would yield comparable null findings to those reported in this study.

Tables

Table 1

Descriptive Statistics for the Full Sample (N = 3682)

Characteristic	T1	
	N or Mean	% or SD
Gender (%)		
Female	2064	56.1
Male	1618	43.9
T1 Age, *M* (SD)	54.05	11.4
T2 Age, *M* (SD)	63.37	11.3
Marital status (%)		
Single	1006	27.3
Married/partnered	2676	72.7
Children (%)	3207	87.1
Highest educational level (%)		
Did not graduate high school	257	7
High school/some college	1715	46.6
University or postgraduate degree	1706	46.3
Employment (%)		
Unemployed	1026	27.9
Employed	2656	72.1
Race[a] (%)		
White	3038	82.8
Non-white	631	17.1
Smoking[b] (%)		
Smoker	1725	46.8
Non-Smoker	1002	27.2
BMI, *M* (SD)	28.4	7.5
Emotional Health *M* (SD)	2.14	.92

[a]. 0.1% of respondents did not respond.

[b]. 26% of respondents did not respond.

Table 2

Correlations for all continuous study variables

Variable	Mean	SD	Range	1	2	3	4	5	6	7	8	9	10	11	12
1. Cumulative Stress	.02	.99	-2.16-5.26	—											
2. Group Inv.	1.95	4.44	0-55	-.07**	—										
3. GAD T2	1.53	.60	1-5	.30**	-.04*	—									
4. Age	54.05	11.37	30-84	-.39**	.00	-.11**	—								
5. GAD T1	1.53	.56	1-5	.37**	-.04*	.59**	-.16**	—							
6. SR-Aff T2	.40	.20	0-1	.16**	-.03	.46**	-.03	.34**	—						
7. SR-Aff T1	.37	.19	0-1	.20**	-.03*	.34**	-.06**	.47**	.47**	—					
8. AD-SR T2	.12	.33	0-1	.19**	-.02	.40**	-.11**	.32**	.33**	.26**	—				
9. AD-SR T1	.12	.32	0-1	.20**	-.004	.30**	-.12**	.46**	.25**	.38**	.32**	—			
10. PH T1	1.68	1.73	0-15	.09**	-.08**	.20**	.24**	.23**	.24**	.27**	.11**	.15**	—		
11. PH T2	2.13	1.98	0-15	.07**	-.07**	.26**	.25**	.22**	.30**	.20**	.15**	.15**	.56**	—	
12. BMI	28.33	7.08	14-82	.12**	-.05**	.06**	-.04**	.08**	.05**	.06**	.04*	.03	.18**	.18**	—

Note: **. Correlation is significant at the 0.01 level (2-tailed); *. Correlation is significant at the 0.05 level (2-tailed); Group Inv. = Group Involvement; GAD = General affective distress; SR-Aff = Self-reported affective disorder; AD-SR = Affective diagnosis based on self-report; PH = Physical Health

Table 3

Linear Regression Predicting General Affective Distress

	B	*SE*	β	t	*p*	95% CI	
						Lower	*Upper*
Cumulative Stress	.07	.01	.12	7.11	<.001	.05	.09
Gender	.02	.02	.01	1.02	.31	-.02	.05
Group Inv.	.001	.002	.005	.39	.70	-.003	.004
T1 Gen. Aff. Distress	.56	.02	.53	35.6	<.001	.53	.59
Education	-.01	.003	-.05	-3.56	<0.001	-.02	-.005
Age	-0.001	.001	-.01	-.69	.49	-.002	.001
Employment Status	-.13	.02	-.10	-6.22	<.001	-.17	-0.10
Partner Status	-0.04	.02	-.03	-1.94	.05	-0.7	.00
Parental Status	-0.06	.03	-.03	-2.30	.02	-.11	.008
Group Inv.* Cumulative Stress	.002	.001	.02	1.33	.18	-.001	.005

Note: Gen. Aff. Distress = General Affective Distress; Group Inv. = Group Involvement

Table 4

Logistic Regression Predicting Likelihood of Self-Reported Affective Disorder

	B	*SE*	Wald	*df*	*p*	Odds Ratio	95% CI	
							Lower	*Upper*
Cumulative Stress	.29	.05	27.91	1	<.001	1.33	1.20	1.48
Gender	-.49	.10	23.44	1	<.001	.61	.50	.75
Group Inv.	.002	.01	.02	1	.89	1.0	.98	1.03
SR Disorder T1	2.39	.11	490.66	1	<.001	10.78	8.74	13.31
Education	-.02	.02	.97	1	.33	.98	.94	1.02
Age	.003	.005	.27	1	.61	1.0	.99	1.01
Employment Status	-.54	.12	22.23	1	<.001	.58	.46	.73
Partner Status	.14	.11	1.54	1	.21	1.15	.93	1.42
Parental Status	-.49	.14	11.73	1	<.001	.62	.47	.81
Group Inv* Cumulative Stress	.005	.009	.27	1	.60	1.01	.99	1.02

Note: SR Disorder = Self-reported affective disorder; Group Inv. = Group Involvement

Table 5

Logistic Regression Predicting Likelihood of Affective Diagnosis based on Self- Report

	B	*SE*	Wald	*df*	*p*	Odds Ratio	95% CI	
							Lower	*Upper*
Cumulative Stress	.42	.06	49.40	1	<.001	1.52	1.35	1.71
Gender	-.33	.12	8.13	1	.004	.72	.57	.90
Group Inv.	-.01	.02	.34	1	.56	.99	.96	1.03
T1 AD-SR	1.59	.13	162.05	1	<.001	4.90	3.84	6.27
Education	-.04	.02	2.51	1	.11	.96	.92	1.01
Age	-.02	.01	11.03	1	<.001	.98	.97	.99
Employment Status	-.66	.13	24.71	1	<.001	.52	.40	.67
Partner Status	.06	.12	.23	1	.63	1.10	.83	1.35
Parental Status	-.33	.16	4.18	1	.04	.72	.53	.99
Group Inv* Cumulative Stress	.009	.01	.53	1	.47	1.01	.99	1.03

Note: AD-SR = Affective diagnosis based on self report; Group Inv. = Group Involvement

Table 6

Negative Binomial Poisson Regression for Physical Health Outcomes

	B	*SE*	Wald	*df*	*p*	Odds Ratio	95% CI	
							Lower	*Upper*
Cumulative Stress	.09	.03	11.04	1	<.001	1.10	1.04	1.16
Gender	-.13	-.04	8.88	1	.003	.88	.81	.96
Group Inv.	-.01	-.01	.73	1	.39	.99	.98	1.01
T1 PH	.20	.01	233.44	1	.00	1.22	1.19	1.26
Education	-.01	.01	.93	1	.33	.99	.97	1.01
Race	-.16	.06	7.96	1	.005	.85	.76	.95
Smoking Status	.14	.04	7.23	1	.007	1.14	1.04	1.26
BMI	.01	.003	12.92	1	<.001	1.01	1.01	1.02
Emotional Health	0.10	.02	6.65	1	.01	1.10	1.02	1.11
Age	.02	.002	59.54	1	<.001	1.02	1.01	1.02
Employment Status	-.09	.05	2.93	1	.09	.92	.83	1.01
Partner Status	-.02	.05	.17	1	.68	.98	.89	1.08
Parental Status	-.05	.07	.55	1	.46	.95	.84	1.08
Group Inv* Cumulative Stress	-.01	.01	1.90	1	.17	.99	.97	1.01

Note: PH = Physical health conditions; Group Inv. = Group Involvement

References

Afrisham, R., Paknejad, M., Soliemanifar, O., Sadegh-Nejadi, S., Meshkani, R., & Ashtary-Larky, D. (2019). The Influence of Psychological Stress on the Initiation and Progression of Diabetes and Cancer. *International Journal of Endocrinology and Metabolism*, *17*(2), e67400. https://doi.org/10.5812/ijem.67400

Belkic, K., Landsbergis, P. A., Schnall, P. L., & Baker, D. (2004). Is job strain a major source of cardiovascular disease risk? *Scandinavian Journal of Work, Environment & Health*, *30*(2), 85–128. https://doi.org/10.5271/sjweh.769

Bernabé, D. G., Tamae, A. C., Biasoli, É. R., & Oliveira, S. H. P. (2011). Stress hormones increase cell proliferation and regulates interleukin-6 secretion in human oral squamous cell carcinoma cells. *Brain, Behavior, and Immunity*, 25(3), 574–583. https://doi.org/10.1016/j.bbi.2010.12.012

Black, D. S., & Slavich, G. M. (2016). Mindfulness meditation and the immune system: A systematic review of randomized controlled trials. *Annals of the New York Academy of Sciences*, *1373*(1), 13–24. https://doi.org/10.1111/nyas.12998

Block, J. P., He, Y., Zaslavsky, A. M., Ding, L., & Ayanian, J. Z. (2009). Psychosocial stress and change in weight among us adults. *American Journal of Epidemiology*, *170*(2), 181–192. https://doi.org/10.1093/aje/kwp104

Bobo, W. V., Yawn, B. P., St. Sauver, J. L., Grossardt, B. R., Boyd, C. M., & Rocca, W. A. (2016). Prevalence of combined somatic and mental health multimorbidity: Patterns by age, sex, and Race/Ethnicity. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *71*(11), 1483–1491. https://doi.org/10.1093/gerona/glw032

Boomershine, C. S., Wang, T., & Zwilling, B. S. (2001). Neuroendocrine regulation of macrophage and neutrophil function. In R. Ader, D. L. Felten, & N. Cohen (Eds.), *Psychoneuroimmunology* (3rd ed., pp. 289– 300). New York: Academic Press.

Bromet, E., Andrade, L. H., Hwang, I., Sampson, N. A., Alonso, J., de Girolamo, G., de Graaf, R., Demyttenaere, K., Hu, C., Iwata, N., Karam, A. N., Kaur, J., Kostyuchenko, S., Lépine, J.-P., Levinson, D., Matschinger, H., Mora, M. E., Browne, M. O., Posada-Villa, J., … Kessler, R. C. (2011). Cross-national epidemiology of DSM-IV major depressive episode. *BMC Medicine*, *9*(1). https://doi.org/10.1186/1741-7015-9-90

Brusco, N. K., & Watts, J. J. (2015). Empirical evidence of recall bias for primary health care visits. *BMC Health Services Research*, *15*(1). https://doi.org/10.1186/s12913-015-1039-1

Bucholz, E. M., Strait, K. M., Dreyer, R. P., Geda, M., Spatz, E. S., Bueno, H., Lichtman, J. H., D'Onofrio, G., Spertus, J. A., & Krumholz, H. M. (2014). Effect of low perceived social support on health outcomes in young patients with acute myocardial infarction: results from the VIRGO (Variation in Recovery: Role of Gender on Outcomes of Young AMI Patients) study. *Journal of the American Heart Association*, *3*(5), e001252–e001252. https://doi.org/10.1161/JAHA.114.001252

Bureau, U. S. C. (2022, August 18). *Race and ethnicity in the United States: 2010 census and 2020 census*. Census.gov. Retrieved March 20, 2023, from https://www.census.gov/library/visualizations/interactive/race-and-ethnicity-in-the-united-state-2010-and-2020-census.html

Button, K. S., Ioannidis, J. P., Mokrysz, C., Nosek, B. A., Flint, J., Robinson, E. S., & Munafò, M. R. (2013). Power failure: Why small sample size undermines the reliability of neuroscience. *Nature Reviews Neuroscience*, *14*(5), 365–376. https://doi.org/10.1038/nrn3475

Cacioppo, J. T., Hawkley, L. C., & Thisted, R. A. (2010). Perceived social isolation makes me sad: 5-year cross-lagged analyses of loneliness and depressive symptomatology in the Chicago Health, Aging, and Social Relations Study. *Psychology and Aging*, *25*(2), 453–463. https://doi.org/10.1037/a0017216

Cameron, A. C., & Trivedi, P. K. (2013). *Regression analysis of Count Data* (2nd ed.). Cambridge University Press.

Campos, C. (2012). Chronic hyperglycemia and glucose toxicity: Pathology and clinical sequelae. *Postgraduate Medicine, 124*(6), 90–97. https://doi.org/10.3810/pgm.2012.11.2615

Cargile, A. C. (2017). Self-categorization theory. *The International Encyclopedia of Intercultural Communication*, 1–5. https://doi.org/10.1002/9781118783665.ieicc0173

Carreras, G., Miccinesi, G., Wilcock, A., Preston, N., Nieboer, D., Deliens, L., Groenvold, M., Lunder, U., van der Heide, A., & Baccini, M. (2021). Missing not at random in end of life care studies: Multiple imputation and sensitivity analysis on data from the Action Study. *BMC Medical Research Methodology*, *21*(1). https://doi.org/10.1186/s12874-020-01180-y

Caruana, E. J., Roman, M., Hernández-Sánchez, J., & Solli, P. (2015). Longitudinal studies. *Journal of Thoracic Disease*, 7(11), E537-E540. https://doi.org/10.3978/j.issn.2072-1439.2015.10.63

Charles, S. T., Gatz, M., Kato, K., & Pedersen, N. L. (2008). Physical health 25 years later: the predictive ability of neuroticism. *Health Psychology : Official Journal of the Division of Health Psychology, American Psychological Association*, *27*(3), 369–378. https://doi.org/10.1037/0278-6133.27.3.369

Charles, S. T., Piazza, J. R., Mogle, J., Sliwinski, M. J., & Almeida, D. M. (2013). The wear and tear of daily stressors on mental health. *Psychological Science*, *24*(5), 733–741. https://doi.org/10.1177/0956797612462222

Chopik, W. J., O'Brien, E., & Konrath, S. H. (2016). Differences in empathic concern and perspective taking across 63 countries. *Journal of Cross-Cultural Psychology*, *48*(1), 23–38. https://doi.org/10.1177/0022022116673910

Chu, B., Marwaha, K., Sanvictores, T., & Ayers, D. (2021). Physiology, Stress Reaction. In *StatPearls*. StatPearls Publishing.

Cohen, J. (1992). Statistical Power Analysis. *Current Directions in Psychological Science*, *1*(3), 98–101. http://www.jstor.org/stable/20182143

Cohen, S., Janicki-Deverts, D., & Miller, G. E. (2007). Psychological stress and disease. *JAMA*, *298*(14), 1685–1687. https://doi.org/10.1001/jama.298.14.1685

Cohen, S., & Janicki-Deverts, D. (2009). Can we improve our physical health by altering our social networks? *Perspectives on Psychological Science*, *4*(4), 375–378. https://doi.org/10.1111/j.1745-6924.2009.01141.x

Creaven, A.-M., Higgins, N. M., Ginty, A. T., & Gallagher, S. (2020). Social Support, social participation, and cardiovascular reactivity to stress in the midlife in the United States (MIDUS) study. *Biological Psychology*, 155, 107921. https://doi.org/10.1016/j.biopsycho.2020.107921

Creswell, J. D., Taren, A. A., Lindsay, E. K., Greco, C. M., Gianaros, P. J., Fairgrieve, A., Marsland, A. L., Brown, K. W., Way, B. M., Rosen, R. K., & Ferris, J. L. (2016). Alterations in resting-state functional connectivity link mindfulness meditation with reduced interleukin-6: A randomized controlled trial. *Biological Psychiatry*, *80*(1), 53–61. https://doi.org/10.1016/j.biopsych.2016.01.008

Crimmins, E. M., & Beltran-Sanchez, H. (2010). Mortality and morbidity trends: Is there compression of morbidity? *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, *66B*(1), 75–86. https://doi.org/10.1093/geronb/gbq088

Crimmins, E. M., Shim, H., Zhang, Y. S., & Kim, J. K. (2019). Differences between men and women in mortality and the health dimensions of the morbidity process. *Clinical Chemistry*, *65*(1), 135–145. https://doi.org/10.1373/clinchem.2018.288332

Cruwys, T., Dingle, G. A., Haslam, C., Haslam, S. A., Jetten, J., & Morton, T. A. (2013). Social group memberships protect against future depression, alleviate depression symptoms and prevent

depression relapse. *Social Science & Medicine*, *98*, 179–186. https://doi.org/10.1016/j.socscimed.2013.09.013

Cruwys, T., Alexander Haslam, S., Dingle, G. A., Jetten, J., Hornsey, M. J., Desdemona Chong, E. M., & Oei, T. P. S. (2014). Feeling connected again: Interventions that increase social identification reduce depression symptoms in community and clinical settings. *Journal of Affective Disorders*, *159*, 139–146. https://doi.org/10.1016/j.jad.2014.02.019

Cruwys, T., Haslam, S. A., Dingle, G. A., Haslam, C., & Jetten, J. (2014). Depression and social identity. *Personality and Social Psychology Review*, 18(3), 215–238. https://doi.org/10.1177/1088868314523839

Demetriou, C., Ozer, B. U., & Essau, C. A. (2015). Self-report questionnaires. *The Encyclopedia of Clinical Psychology*, 1–6. https://doi.org/10.1002/9781118625392.wbecp507

Devinsky, O., Morrell, M. J., & Vogt, B. A. (1995). Contributions of anterior cingulate cortex to behaviour. *Brain*, 118(1), 279–306. https://doi.org/10.1093/brain/118.1.279

Dhabhar, F. S. (2018). The short-term stress response - Mother nature's mechanism for enhancing protection and performance under conditions of threat, challenge, and opportunity. *Frontiers in Neuroendocrinology*, 49, 175–192. https://doi.org/10.1016/j.yfrne.2018.03.004

DiMatteo, M. R., Lepper, H. S., & Croghan, T. W. (2000). Depression is a risk factor for noncompliance with medical treatment. *Archives of Internal Medicine*, *160*(14), 2101. https://doi.org/10.1001/archinte.160.14.2101

Dimitrov, D. M., & Rumrill, P. D., Jr (2003). Pretest-posttest designs and measurement of change. *Work*, *20*(2), 159–165.

Duijts, S. F. A., Zeegers, M. P. A., & Borne, B. V. (2003). The association between stressful life events and breast cancer risk: A meta-analysis. *International Journal of Cancer*, *107*(6), 1023–1029. https://doi.org/10.1002/ijc.11504

Du, H., King, R. B., & Chi, P. (2017). Self-esteem and subjective well-being revisited: The roles of personal, relational, and collective self-esteem. *PLOS ONE*, 12(8). https://doi.org/10.1371/journal.pone.0183958

Engel, G. L. (1977). The need for a new medical model: A challenge for biomedicine. *Science, 196*(4286), 129–136. https://doi.org/10.1126/science.847460

Epel, E. S., Crosswell, A. D., Mayer, S. E., Prather, A. A., Slavich, G. M., Puterman, E., & Mendes, W. B. (2018). More than a feeling: A unified view of stress measurement for population science. *Frontiers in Neuroendocrinology*, *49*, 146–169. https://doi.org/10.1016/j.yfrne.2018.03.001

Evans, G. W., Li, D., & Sepanski Whipple, S. (2013). Cumulative risk and child development. *Psychological Bulletin*, 139(6), 1342–1396. doi:10.1037/a0031808

Fitzgerald, M., & Berthiaume, K. (2021). Does spirituality moderate the relationship between child maltreatment and adult men and women's social anxiety, depression and loneliness. *Journal of Aggression, Maltreatment & Trauma*, *31*(2), 235–253. https://doi.org/10.1080/10926771.2021.1912875

Folkman, S., Lazarus, R. S., Gruen, R. J., & DeLongis, A. (1986). Appraisal, coping, health status, and psychological symptoms. *Journal of Personality and Social Psychology*, *50*(3), 571–579. https://doi.org/10.1037/0022-3514.50.3.571

Gayman, M. D., Cislo, A. M., Goidel, A. R., & Ueno, K. (2013). SES and race-ethnic differences in the stress-buffering effects of coping resources among young adults. *Ethnicity & Health*, *19*(2), 198–216. https://doi.org/10.1080/13557858.2013.828827

Geyer, S. (1991). Life events prior to manifestation of breast cancer: A limited prospective study covering eight years before diagnosis. *Journal of Psychosomatic Research*, 35(2-3), 355–363. https://doi.org/10.1016/0022-3999(91)90090-b

Gobo, G. (2004). Sampling, representativeness and generalizability. *Qualitative Research Practice*, 405–426. https://doi.org/10.4135/9781848608191.d34

Greenaway, K. H., Cruwys, T., Haslam, S. A., & Jetten, J. (2015). Social identities promote well-being because they satisfy global psychological needs. *European Journal of Social Psychology*, *46*(3), 294–307. https://doi.org/10.1002/ejsp.2169

Grzywacz, J. G. (2000). Work-family spillover and health during midlife: is managing conflict everything? *American Journal of Health Promotion : AJHP*, 14(4), 236–243. https://doi.org/10.4278/0890-1171-14.4.236

Haight, B. L., Peddie, L., Crosswell, A. D., Hives, B. A., Almeida, D. M., & Puterman, E. (2023). Combined effects of cumulative stress and daily stressors on daily health. *Health Psychology*, *42*(5), 325.

Hannibal, K. E., & Bishop, M. D. (2014). Chronic stress, cortisol dysfunction, and pain: A psychoneuroendocrine rationale for stress management in pain rehabilitation. *Physical Therapy*, *94*(12), 1816–1825. https://doi.org/10.2522/ptj.20130597

Harris, M. L., Oldmeadow, C., Hure, A., Luu, J., Loxton, D., & Attia, J. (2017). Stress increases the risk of type 2 diabetes onset in women: A 12-year longitudinal study using causal modelling. *PLOS ONE*, 12(2). https://doi.org/10.1371/journal.pone.0172126

Harris, M. A., & Orth, U. (2019). The Link Between Self-Esteem and Social Relationships: A Meta-Analysis of Longitudinal Studies. *Journal of Personality and Social Psychology*. http://dx.doi.org/10.1037/pspp0000265

Haslam, C., Holme, A., Haslam, S. A., Iyer, A., Jetten, J., & Williams, W. H. (2008). Maintaining group memberships: Social Identity Continuity predicts well-being after stroke. *Neuropsychological Rehabilitation*, *18*(5-6), 671–691. https://doi.org/10.1080/09602010701643449

Haslam, C., Cruwys, T., & Haslam, S. A. (2014). “the WE's have it”: Evidence for the distinctive benefits of group engagement in enhancing Cognitive Health in aging. *Social Science & Medicine*, *120*, 57–66. https://doi.org/10.1016/j.socscimed.2014.08.037

Haslam, C., Jetten, J., Cruwys, T., Dingle, G. A., & Haslam, S. A. (2018). The social identity approach to health. In The New Psychology of Health: Unlocking the Social Cure (1st ed., pp. 13–35). *Routledge*. https://doi.org/10.4324/9781315648569

Haslam, S. A., McMahon, C., Cruwys, T., Haslam, C., Jetten, J., & Steffens, N. K. (2018). Social Cure, what social cure? the propensity to underestimate the importance of social factors for health. *Social Science & Medicine*, *198*, 14–21. https://doi.org/10.1016/j.socscimed.2017.12.020

Haslam, S. A., Haslam, C., Jetten, J., Cruwys, T., & Bentley, S. (2019). Group life shapes the psychology and biology of Health: The Case for a sociopsychobio model. *Social and Personality Psychology Compass*, 13(8). https://doi.org/10.1111/spc3.12490

Hemingway, H., & Marmot, M. (1999). Evidence based cardiology: Psychosocial factors in the aetiology and prognosis of coronary heart disease: Systematic review of Prospective Cohort Studies. *BMJ*, 318(7196), 1460–1467. https://doi.org/10.1136/bmj.318.7196.1460

Heshizer, B., & Knapp, D. E. (2016). Revisiting the buffering hypothesis: Social support, work stressors, stress related symptoms, and negative affectivity in a sample of public school teachers. *OALib*, *03*(10), 1–11. https://doi.org/10.4236/oalib.1103057

Hidalgo, B. (2012). Validation of self-reported measures in Health Disparities Research. *Journal of Biometrics & Biostatistics*, *03*(07). https://doi.org/10.4172/2155-6180.1000e114

Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine*, 7(7), e1000316. https://doi.org/10.1371/journal.pmed.1000316

Hornsey, M. J. (2008). Social identity theory and self-categorization theory: A historical review. *Social and Personality Psychology Compass*, *2*(1), 204–222. https://doi.org/10.1111/j.1751-9004.2007.00066.x

House, J., Landis, K., & Umberson, D. (1988). Social relationships and health. *Science,* 241, 540–545. doi: 10.1126/science.3399889

Jamieson, J. P., Hangen, E. J., Lee, H. Y., & Yeager, D. S. (2018). Capitalizing on Appraisal Processes to Improve Affective Responses to Social Stress. *Emotion Review : Journal of the International Society for Research on Emotion*, *10*(1), 30–39.

Jetten, J., Haslam, C., & Haslam, S. A. (Eds.). (2012). *The social cure: Identity, health and well-being.* Psychology Press.

Jetten, J., Branscombe, N. R., Haslam, S. A., Haslam, C., Cruwys, T., Jones, J. M., ... & Zhang, A. (2015). Having a lot of a good thing: Multiple important group memberships as a source of self-esteem. *PloS one*, *10*(5), e0124609.

Jetten, J., Haslam, S. A., Cruwys, T., Greenaway, K. H., Haslam, C., & Steffens, N. K. (2017). Advancing the social identity approach to health and well-being: Progressing the social cure research agenda. *European Journal of Social Psychology*, 47(7), 789–802. https://doi.org/10.1002/ejsp.2333

Jetten, J., Haslam, C., von Hippel, C., Bentley, S. V., Cruwys, T., Steffens, N. K., & Haslam, S. A. (2022). "Let's get physical"—or social: the role of physical activity versus social group memberships in predicting depression and anxiety over time. *Journal of affective disorders*, *306*, 55-61.

Jiang, T., Yakin, S., Crocker, J., & Way, B. M. (2022). Perceived social support-giving moderates the association between social relationships and interleukin-6 levels in blood. *Brain, Behavior, and Immunity*, *100*, 25–28. https://doi.org/10.1016/j.bbi.2021.11.002

Kahan, B. C., Jairath, V., Doré, C. J., & Morris, T. P. (2014). The risks and rewards of covariate adjustment in randomized trials: An assessment of 12 outcomes from 8 studies. *Trials*, *15*(1). https://doi.org/10.1186/1745-6215-15-139

Karasek, R. (1985) *Job Content Questionnaire and User's Guide.* University of Massachusetts; Lowell.

Kawachi, I., & Berkman, L. F. (2001). Social ties and mental health. *Journal of Urban Health : Bulletin of the New York Academy of Medicine*, *78*(3), 458–467. https://doi.org/10.1093/jurban/78.3.458

Kemeny, M. E. (2003). The psychobiology of stress. Current Directions in Psychological *Science*, 12(4), 124–129. https://doi.org/10.1111/1467-8721.01246

Kessler, R. C., Andrews, G., Mroczek, D., Ustun, B., & Wittchen, H.-U. (1998). The World Health Organization Composite International Diagnostic Interview Short-form (CIDI-SF). *International Journal of Methods in Psychiatric Research*, *7*(4), 171–185. https://doi.org/10.1002/mpr.47

Kessler, R. C., Mickelson, K. D., & Williams, D. R. (1999). The Prevalence, Distribution, and Mental Health Correlates of Perceived Discrimination in the United States. *Journal of Health and Social Behavior*, *40*(3), 208–230. https://doi.org/10.2307/2676349

Kessler, R. C., Andrews, G., Colpe, L. J., Hiripi, E., Mroczek, D. K., Normand, S. L., Walters, E. E., & Zaslavsky, A. M. (2002). Short screening scales to monitor population prevalences and trends in non-specific psychological distress. *Psychological Medicine*, 32(6), 959–976. https://doi.org/10.1017/s0033291702006074

Kessler, R. C., Birnbaum, H., Bromet, E., Hwang, I., Sampson, N., & Shahly, V. (2010). Age differences in major depression: results from the National Comorbidity Survey Replication (NCS-R). *Psychological medicine*, *40*(2), 225–237. https://doi.org/10.1017/S0033291709990213

Keyes, C. L. (1998). Social well-being. *Social Psychology Quarterly*, *61*(2), 121–140. https://doi.org/10.2307/2787065

Kivimäki, M., Virtanen, M., Elovainio, M., Kouvonen, A., Väänänen, A., & Vahtera, J. (2006). Work stress in the etiology of coronary heart disease—a meta-analysis. *Scandinavian Journal of Work, Environment & Health, 32*(6), 431–442. https://doi.org/10.5271/sjweh.1049

Kline, R. B. (2016). *Principles and practice of structural equation modeling* (4th ed.). The Guilford Press.

Kormos, C., & Gifford, R. (2014). The validity of self-report measures of Proenvironmental Behavior: A meta-analytic review. *Journal of Environmental Psychology*, *40*, 359–371. https://doi.org/10.1016/j.jenvp.2014.09.003

Korten, N. C., Comijs, H. C., Penninx, B. W., & Deeg, D. J. (2017). Perceived stress and cognitive function in older adults: which aspect of perceived stress is important?. *International journal of geriatric psychiatry*, *32*(4), 439–445. https://doi.org/10.1002/gps.4486

Krause N. (2004). Stressors arising in highly valued roles, meaning in life, and the physical health status of older adults. *The journals of gerontology. Series B, Psychological sciences and social sciences*, *59*(5), S287–S297. https://doi.org/10.1093/geronb/59.5.s287

Kumari, N., Dwarakanath, B. S., Das, A., & Bhatt, A. N. (2016). Role of interleukin-6 in cancer progression and therapeutic resistance. *Tumor Biology*, *37*(9), 11553–11572. https://doi.org/10.1007/s13277-016-5098-7

Kyprianides, A., Easterbrook, M. J., & Brown, R. (2019). Group identities benefit well-being by satisfying needs. *Journal of Experimental Social Psychology*, *84*, 103836. https://doi.org/10.1016/j.jesp.2019.103836

Lachman ME, Weaver SL. The Midlife Development Inventory (MIDI) Personality Scales: Scale construction and scoring. *Technical report.* 1997

Lam, B. C. P., Haslam, C., Haslam, S. A., Steffens, N. K., Cruwys, T., Jetten, J., & Yang, J. (2018). Multiple Social Groups support adjustment to retirement across cultures. *Social Science & Medicine*, *208*, 200–208. https://doi.org/10.1016/j.socscimed.2018.05.049

Lantz, P. M., House, J. S., Mero, R. P., & Williams, D. R. (2005). Stress, life events, and socioeconomic disparities in health: results from the Americans' Changing Lives Study. *Journal of Health and Social Behavior*, *46*(3), 274–288. https://doi.org/10.1177/002214650504600305

Lazarus, R. S., & Folkman, S. (1984). Stress, Appraisal, and Coping. New York: *Springer Publishing*.

Lazarus, R. S. (1991a). Emotion and Adaptation. New York: Oxford University Press.

Levine, M., & Thompson, K. (2004). Identity, place, and bystander intervention: Social categories and helping after natural disasters. *The Journal of Social Psychology*, *144*(3), 229–245. https://doi.org/10.3200/socp.144.3.229-245

Levine, M., Prosser, A., Evans, D., & Reicher, S. (2005). Identity and emergency intervention: How Social Group Membership and inclusiveness of group boundaries shape helping behavior. *Personality and Social Psychology Bulletin*, *31*(4), 443–453. https://doi.org/10.1177/0146167204271651

Little, R. J. (1988). A test of missing completely at random for multivariate data with missing values. *Journal of the American Statistical Association*, *83*(404), 1198–1202. https://doi.org/10.1080/01621459.1988.10478722

Liu, W., Ge, T., Leng, Y., Pan, Z., Fan, J., Yang, W., Cui, R., Li, A., & Li, A. (2017). The Role of Neural Plasticity in Depression: From Hippocampus to Prefrontal Cortex. *Neural Plasticity*, *2017*, 6871089–11. https://doi.org/10.1155/2017/6871089

Lockwood, K. G., Peddie, L., Crosswell, A. D., Hives, B. A., Slopen, N., Almeida, D. M., & Puterman, E. (2022). Effects of Chronic Burden Across Multiple Domains and Experiences of Daily

Stressors on Negative Affect. *Annals of behavioral medicine : a publication of the Society of Behavioral Medicine*, *56*(10), 1056–1067. https://doi.org/10.1093/abm/kaac001

Lowsky, D. J., Olshansky, S. J., Bhattacharya, J., & Goldman, D. P. (2014). Heterogeneity in healthy aging. *The journals of gerontology. Series A, Biological sciences and medical sciences*, *69*(6), 640–649. https://doi.org/10.1093/gerona/glt162

Lucassen, P. J., Pruessner, J., Sousa, N., Almeida, O. F., Van Dam, A. M., Rajkowska, G., Swaab, D. F., & Czéh, B. (2013). Neuropathology of stress. *Acta Neuropathologica*, *127*(1), 109–135. https://doi.org/10.1007/s00401-013-1223-5

Marik, P. E., & Bellomo, R. (2013). Stress hyperglycemia: An essential survival response! *Critical Care*, *17*(2), 305. https://doi.org/10.1186/cc12514

Martiny S.E., Rubin M. (2016) Towards a Clearer Understanding of Social Identity Theory's Self-Esteem Hypothesis. In: McKeown S., Haji R., Ferguson N. (eds) *Understanding Peace and Conflict Through Social Identity Theory.* Peace Psychology Book Series. Springer, Cham. https://doi.org/10.1007/978-3-319-29869-6_2

Mariotti, A. (2015). The effects of chronic stress on Health: New Insights Into the molecular mechanisms of brain–body communication. *Future Science OA*, *1*(3). https://doi.org/10.4155/fso.15.21

Masjedi, A., Hashemi, V., Hojjat-Farsangi, M., Ghalamfarsa, G., Azizi, G., Yousefi, M., & Jadidi-Niaragh, F. (2018). The significant role of interleukin-6 and its signaling pathway in the immunopathogenesis and treatment of breast cancer. *Biomedicine & Pharmacotherapy*, *108*, 1415–1424. https://doi.org/10.1016/j.biopha.2018.09.177

Mattacks, C. A., & Pond, C. M. (1999). Interactions of noradrenalin and tumour necrosis factor alpha, interleukin 4 and interleukin 6 in the control of lipolysis from adipocytes around lymph nodes. *Cytokine*, *11*(5), 334–346. https://doi.org/10.1006/cyto.1998.0442

Matud, M. P. (2017). Gender and health. *Gender Differences in Different Contexts*. https://doi.org/10.5772/65410

Mazure, C. M. (1998). Life stressors as risk factors in depression. *Clinical Psychology: Science and Practice*, *5*(3), 291–313. https://doi.org/10.1111/j.1468-2850.1998.tb00151.x

McEwen, B. S., & Stellar, E. (1993). Stress and the individual. Mechanisms leading to disease. *Archives of internal medicine*, *153*(18), 2093–2101.

McEwen, B. S., & Akil, H. (2020). Revisiting the stress concept: Implications for affective disorders. *The Journal of Neuroscience*, *40*(1), 12–21. https://doi.org/10.1523/jneurosci.0733-19.2019

Mcloughlin, E., Fletcher, D., Slavich, G. M., Arnold, R., & Moore, L. J. (2021). Cumulative lifetime stress exposure, depression, anxiety, and well-being in elite athletes: A mixed-method study. *Psychology of Sport and Exercise*, *52*. https://doi.org/10.1016/j.psychsport.2020.101823

Melamed, S., Shirom, A., Toker, S., & Shapira, I. (2006). Burnout and risk of type 2 diabetes: A prospective study of apparently healthy employed persons. *Psychosomatic Medicine*, *68*(6), 863–869. https://doi.org/10.1097/01.psy.0000242860.24009.f0

Montoya, A. K. (2018). Moderation analysis in two-instance repeated measures designs: Probing methods and multiple moderator models. *Behavior Research Methods*, *51*(1), 61–82. https://doi.org/10.3758/s13428-018-1088-6

Moreno-Smith, M., Lutgendorf, S. K., & Sood, A. K. (2010). Impact of stress on cancer metastasis. *Future Oncology (London, England)*, *6*(12), 1863–1881. https://doi.org/10.2217/fon.10.142

Morton, P. M., Schafer, M. H., & Ferraro, K. F. (2012). Does childhood misfortune increase cancer risk in adulthood? *Journal of Aging and Health*, *24*(6), 948–984. https://doi.org/10.1177/0898264312449184

Multhoff, G., Molls, M., & Radons, J. (2011). Chronic inflammation in cancer development. *Frontiers in Immunology*, *2*, 98. https://doi.org/10.3389/fimmu.2011.00098

Muñoz, R. F., Beardslee, W. R., & Leykin, Y. (2012). Major depression can be prevented. *The American psychologist*, *67*(4), 285–295. https://doi.org/10.1037/a0027666

Murea, M., Ma, L., & Freedman, B. I. (2012). Genetic and environmental factors associated with type 2 diabetes and diabetic vascular complications. *The Review of Diabetic Studies*, *9*(1), 6–22. https://doi.org/10.1900/rds.2012.9.6

Newman, D. A. (2014). Missing Data: Five Practical Guidelines. *Organizational Research Methods*, *17*(4), 372–411. https://doi.org/10.1177/1094428114548590

Norström, F., Waenerlund, A.-K., Lindholm, L., Nygren, R., Sahlén, K.-G., & Brydsten, A. (2019). Does unemployment contribute to poorer health-related quality of life among Swedish adults? *BMC Public Health*, *19*(1). https://doi.org/10.1186/s12889-019-6825-y

Oakes, P. J. (1987). The salience of social categories. In J. C. Turner, M. A. Hogg, P. J. Oakes, S. D. Reicher & M. S. Wetherell (Eds.), *Rediscovering the social group: A self-categorization theory* (pp. 117–141). Oxford, UK: Blackwell.

Ong, A. D., Bergeman, C. S., & Boker, S. M. (2009). Resilience comes of age: Defining features in later adulthood. *Journal of Personality*, *77*(6), 1777–1804. https://doi.org/10.1111/j.1467-6494.2009.00600.x

Orth, U., & Robins, R. W. (2014). The development of self-esteem. *Current Directions in Psychological Science, 23*, 381-387. http://dx.doi.org/10.1177/0963721414547414

Ozbay, F., Johnson, D. C., Dimoulas, E., Morgan, C. A., Charney, D., & Southwick, S. (2007). Social support and resilience to stress: from neurobiology to clinical practice. *Psychiatry (Edgmont (Pa. : Township))*, *4*(5), 35–40.

Piazza, J. R., Charles, S. T., Sliwinski, M. J., Mogle, J., & Almeida, D. M. (2013). Affective reactivity to daily stressors and long-term risk of reporting a chronic physical health condition. *Annals of Behavioral Medicine : a Publication of the Society of Behavioral Medicine*, *45*(1), 110–120. https://doi.org/10.1007/s12160-012-9423-0

Polit, D. F., & Beck, C. T. (2010). Generalization in quantitative and qualitative research: Myths and strategies. *International Journal of Nursing Studies*, *47*(11), 1451–1458. https://doi.org/10.1016/j.ijnurstu.2010.06.004

Pottier, P., Hardouin, J.-B., Dejoie, T., Castillo, J.-M., Le Loupp, A.-G., Planchon, B., Bonnaud, A., & LeBlanc, V. (2015). Effect of extrinsic and intrinsic stressors on clinical skills performance in third-year medical students. *Journal of General Internal Medicine*, *30*(9), 1259–1269. https://doi.org/10.1007/s11606-015-3314-6

Public Health Agency of Canada. (2021, July 20). *Government of Canada*. Canada.ca. Retrieved March 20, 2023, from https://www.canada.ca/en/public-health/services/publications/diseases-conditions/aging-chronic-diseases-profile-canadian-seniors-report.html

Quiñones, A. R., Botoseneanu, A., Markwardt, S., Nagel, C. L., Newsom, J. T., Dorr, D. A., & Allore, H. G. (2019). Racial/ethnic differences in multimorbidity development and chronic disease accumulation for middle-aged adults. *PLOS ONE*, *14*(6). https://doi.org/10.1371/journal.pone.0218462

Raghunathan, T. E., Lepkowski, J., Van Hoewyk, J., & Solenberger, P. (2001). A multivariate technique for multiply imputing missing values using a sequence of regression models. *Survey Methodology*, *27*(1), 85-95.

Reading, S. R., Karlamangla, A. S., Swendeman, D. T., Ritz, B. R., Gruenewald, T. L., Slopen, N., . . . Seeman, T. E. (2016). Relationship between psychosocial stress and allostatic load: Findings from the MIDUS study. Annals of Behavioral Medicine, 50(Suppl. 1), S272.

Rector, N. A., & Roger, D. (1997). The stress buffering effects of self-esteem. *Personality and Individual Differences, 23*(5), 799–808. https://doi.org/10.1016/s0191-8869(97)00095-0

Reimer, N. K., Schmid, K., Hewstone, M., & Al Ramiah, A. (2020). Self-categorization and social identification: Making sense of us and them. In D. Chadee (Ed.), *Theories in social psychology (2nd ed.)*. Wiley-Blackwell.

Rosengren, A., Orth-Gomer, K., Wedel, H., & Wilhelmsen, L. (1993). Stressful life events, social support, and mortality in men born in 1933. *BMJ, 307*(6912), 1102–1105. https://doi.org/10.1136/bmj.307.6912.1102

Rosengren, A., Hawken, S., Ôunpuu, S., Sliwa, K., Zubaid, M., Almahmeed, W. A., Blackett, K. N., Sitthi-amorn, C., Sato, H., & Yusuf, S. (2004). Association of Psychosocial Risk factors with risk of acute myocardial infarction in 11 119 cases and 13 648 controls from 52 countries (the Interheart Study): Case-control study. *The Lancet, 364*(9438), 953–962. https://doi.org/10.1016/s0140-6736(04)17019-0

Rotenberg, S., & McGrath, J. J. (2016). Inter-relation between autonomic and Hpa Axis activity in children and adolescents. *Biological Psychology, 117*, 16–25. https://doi.org/10.1016/j.biopsycho.2016.01.015

Rubin, D. B. (1976). Inference and missing data. *Biometrika, 63*(3), 581-592.

Ryff, C. D., Magee, W. J., Kling, K. C., & Wing, E. H. (1999). Forging macro-micro linkages in the study of psychological well-being. In C. D. Ryff & V. W. Marshall (Eds.), *The self and society in aging processes* (pp. 247-278). New York: Springer.

Ryff, C., Almeida, D., Ayanian, J., Binkley, N., Carr, D. S., Coe, C., Davidson, R., . . . Williams, D. (2017). Midlife in the United States (MIDUS Refresher): Milwaukee African American Sample, 2012-2013 - Version 1. [Data set]. ICPSR36722-v1. doi:10.3886/ICPSR36722.v1

Salleh M. R. (2008). Life event, stress and illness. *The Malaysian Journal of Medical Sciences : MJMS*, *15*(4), 9–18.

Sandstrom, G. M., & Dunn, E. W. (2014). Social Interactions and well-being. *Personality and Social Psychology Bulletin*, *40*(7), 910–922. https://doi.org/10.1177/0146167214529799

Schneiderman, N., Ironson, G., & Siegel, S. D. (2005). Stress and health: psychological, behavioral, and biological determinants. *Annual Review of Clinical Psychology*, *1*, 607–628. https://doi.org/10.1146/annurev.clinpsy.1.102803.144141

Sheng, J. A., Bales, N. J., Myers, S. A., Bautista, A. I., Roueinfar, M., Hale, T. M., & Handa, R. J. (2021). The hypothalamic-pituitary-adrenal axis: Development, programming actions of hormones, and maternal-fetal interactions. *Frontiers in Behavioral Neuroscience*, *14*. https://doi.org/10.3389/fnbeh.2020.601939

Shimazu, A., Schaufeli, W. B., Kamiyama, K., & Kawakami, N. (2015). Workaholism vs. work engagement: the two different predictors of future well-being and performance. *International journal of behavioral medicine*, *22*(1), 18–23. https://doi.org/10.1007/s12529-014-9410-x

Sin, N. L., Rush, J., Buxton, O. M., & Almeida, D. M. (2021). Emotional vulnerability to short sleep predicts increases in chronic health conditions across 8 years. *Annals of Behavioral Medicine*, *55*(12), 1231-1240.

Singh, N., Baby, D., Rajguru, J. P., Patil, P. B., Thakkannavar, S. S., & Pujari, V. B. (2019). Inflammation and cancer. *Annals of African Medicine*, *18*(3), 121–126. https://doi.org/10.4103/aam.aam_56_18

Slopen, N., Dutra, L. M., Williams, D. R., Mujahid, M. S., Lewis, T. T., Bennett, G. G., . . . Albert, M. A. (2012). Psychosocial stressors and cigarette smoking among African American adults in midlife. *Nicotine & Tobacco Research, 14*(10), 1161–1169. doi: 10.1093/ntr/nts011

Slopen, N., Kontos, E. Z., Ryff, C. D., Ayanian, J. Z., Albert, M. A., & Williams, D. R. (2013). Psychosocial stress and cigarette smoking persistence, cessation, and relapse over 9–10 years: a prospective study of middle-aged adults in the United States. *Cancer Causes & Control, 24*(10), 1849–1863. https://doi.org/10.1007/s10552-013-0262-5

Slopen, N., Non, A., L., Williams, D. R., Roberts, A. L., & Albert, M. A. (2014). Childhood adversity, adult neighborhood context, and cumulative biological risk for chronic diseases in adulthood. *Psychosomatic Medicine, 76*(7), 481–489.

Slopen, N., Meyer, C., & Williams, D. R. (2018). Cumulative stress and health. *The Oxford Handbook of Integrative Health Science*, 74–86. https://doi.org/10.1093/oxfordhb/9780190676384.013.5

Smith, S. M., & Vale, W. W. (2006). The role of the hypothalamic-pituitary-adrenal axis in neuroendocrine responses to stress. *Dialogues in Clinical Neuroscience, 8*(4), 383–395. https://doi.org/10.31887/DCNS.2006.8.4/ssmith

Stansfeld, S. A., Clark, C., Smuk, M., Power, C., Davidson, T., & Rodgers, B. (2017). Childhood adversity and midlife suicidal ideation. *Psychological medicine, 47*(2), 327–340. https://doi.org/10.1017/S0033291716002336

Stephens, M. A., & Wand, G. (2012). Stress and the HPA axis: role of glucocorticoids in alcohol dependence. *Alcohol Research : Current Reviews, 34*(4), 468–483.

Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. *Brain, Behavior, and Immunity, 21*(7), 901–912. https://doi.org/10.1016/j.bbi.2007.03.011

Sternthal, M. J., Slopen, N., & Williams, D. R. (2011). Racial disparities in health: How much does stress really matter? *Du Bois Review: Social Science Research on Race, 8*(01), 95–113. doi:10.1017/S1742058X11000087

Steptoe, A., Deaton, A., & Stone, A. A. (2015). Subjective wellbeing, health, and ageing. *Lancet (London, England), 385*(9968), 640–648. https://doi.org/10.1016/S0140-6736(13)61489-0

Stevens, M., Lieschke, J., Cruwys, T., Cárdenas, D., Platow, M. J., & Reynolds, K. J. (2021). Better together: How group-based physical activity protects against depression. *Social science & medicine (1982), 286*, 114337. https://doi.org/10.1016/j.socscimed.2021.114337

Stevens, M., Rees, T., Coffee, P., Steffens, N. K., Haslam, S. A., & Polman, R. (2017). A Social Identity Approach to Understanding and Promoting Physical Activity. *Sports Medicine (Auckland, N.Z.), 47*(10), 1911–1918. https://doi.org/10.1007/s40279-017-0720-4

Tajfel, H. (1972). La catégorisation sociale (English Trans.). In S. Moscovici (Ed.), *Introduction à la psychologie sociale* (Vol. **1**) (pp. 272– 302). Paris: Larousse.

Tajfel, H., & Turner, J. (1979). An integrative theory of intergroup conflict. In W. Austin, & S. Worchel (Eds.), *The social psychology of intergroup relations*. Brooks: California.

Thau, L., Gandhi, J., & Sharma, S. (2021). Physiology, Cortisol. In *StatPearls*. StatPearls Publishing.

Thoits, P. A. (1995). Stress, coping, and Social Support Processes: Where are we? what next? *Journal of Health and Social Behavior, 35*, 53–79. https://doi.org/10.2307/2626957

Townsend, N., Wilson, L., Bhatnagar, P., Wickramasinghe, K., Rayner, M., & Nichols, M. (2016). Cardiovascular disease in Europe 2016: An epidemiological update. *European Heart Journal, 37*(42), 3182–3183. https://doi.org/10.1093/eurheartj/ehw468

Tsenkova, V. K., Dienberg Love, G., Singer, B. H., & Ryff, C. D. (2008). Coping and positive affect predict longitudinal change in glycosylated hemoglobin. *Health Psychology*, *27*(2, Suppl). https://doi.org/10.1037/0278-6133.27.2(suppl.).s163

Turner, J. C. (1982). Towards a redefinition of the social group. In H. Tajfel (Ed.), *Social identity and intergroup relations* (pp. 15– 40). Cambridge: Cambridge University Press.

Turner, R. J., & Lloyd, D. A. (1995). Lifetime Traumas and Mental Health: The significance of cumulative adversity. *Journal of Health and Social Behavior*, *36*(4), 360. https://doi.org/10.2307/2137325

Turner, J. C. (1999). Some current issues in research on social identity and self-categorization theories. In N. Ellemers, R. Spears & B. Doosje (Eds.*)*, *Social identity: Context, commitment, content* (pp. 6–34). Oxford, UK: Blackwell.

Turner, R. J., & Lloyd, D. A. (2004). Stress burden and the lifetime incidence of psychiatric disorder In young adults. *Archives of General Psychiatry*, *61*(5), 481. https://doi.org/10.1001/archpsyc.61.5.481

Turner, J. C., & Reynolds, K. J. (2012). Self-categorization theory. In P. A. M. Van Lange, A. W. Kruglanski, & E. T. Higgins (Eds.), Handbook of Theories of Social Psychology (pp. 399–417). SAGE. https://doi.org/10.4135/9781446249222.n46

Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin*, *119*(3), 488–531. https://doi.org/10.1037/0033-2909.119.3.488

Vesely, S., & Klöckner, C. A. (2020). Social Desirability in environmental psychology research: Three meta-analyses. *Frontiers in Psychology*, *11*. https://doi.org/10.3389/fpsyg.2020.01395

von Hippel, P. T. (2007). Regression with missing Ys: An improved strategy for analyzing multiply imputed data. *Sociological Methodology*, *37*(1), 83-117.

Walen, H. R., & Lachman, M. E. (2000). Social Support and Strain from Partner, Family, and Friends: Costs and Benefits for Men and Women in Adulthood. *Journal of Social and Personal Relationships, 17*(1), 5–30. https://doi.org/10.1177/0265407500171001

Whitesell, N. R., Beals, J., Mitchell, C. M., Keane, E. M., Spicer, P., & Turner, R. J. (2007). The relationship of cumulative and proximal adversity to onset of substance dependence symptoms in two American Indian communities. *Drug and Alcohol Dependence, 91*(2-3), 279–288. https://doi.org/10.1016/j.drugalcdep.2007.06.008

Wiley, J. F., Gruenewald, T. L., & Seeman, T. E. (2018). Psychosocial Resources and physiological dysregulation. *The Oxford Handbook of Integrative Health Science*, 202–220. https://doi.org/10.1093/oxfordhb/9780190676384.013.13

Williams, D., & Cooper, L. (2019). Reducing racial inequities in health: Using what we already know to take action. *International Journal of Environmental Research and Public Health, 16*(4), 606. https://doi.org/10.3390/ijerph16040606

Williams, D. R., & Slopen, N. (2019). Documentation of Stress Summary Measures for MIDUS 2 and Milwaukee Surveys.

Won, E., & Kim, Y.-K. (2016). Stress, the autonomic nervous system, and the immune-kynurenine pathway in the etiology of depression. *Current Neuropharmacology, 14*(7), 665–673. https://doi.org/10.2174/1570159x14666151208113006

Yusuf, S., Hawken, S., & Ounpuu, S. (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the Interheart Study): Case-control study. *ACC Current Journal Review, 13*(12), 15–16. https://doi.org/10.1016/j.accreview.2004.11.072

Ziegler, M. G. (2004). Psychological stress and the autonomic nervous system. *Primer on the Autonomic Nervous System*, 189–190. https://doi.org/10.1016/b978-012589762-4/50051-7

Zimmerman, S. L. (1999). *Self-esteem, personal control, optimism, extraversion, and the subjective well-being of midwestern university faculty* (thesis). Retrieved January 22, 2022, from https://go.exlibris.link/ffKqXjPy.

Zisook, S., Lesser, I., Stewart, J. W., Wisniewski, S. R., Balasubramani, G. K., Fava, M., Gilmer, W. S., Dresselhaus, T. R., Thase, M. E., Nierenberg, A. A., Trivedi, M. H., & Rush, A. J. (2007). Effect of age at onset on the course of major depressive disorder. *The American journal of psychiatry*, *164*(10), 1539–1546. https://doi.org/10.1176/appi.ajp.2007.06101757

Appendix

Group Involvement Questionnaire

In a typical month, about how many times do you attend the following? (If none, enter "0".) –

1. MEETINGS OF SPORTS OR SOCIAL GROUPS?

Cumulative stress Measures (From Slopen et al., 2013)

Work Stress: 20 Item Scale (All responses range from 1-5 from "all of the time" to never, unless otherwise noted)

i. Skill discretion

a. How often do you learn new things at work?

b. How often does your work demand a high level of skill or expertise?

c. How often does your job provide you with a variety of things that interest you?

ii. Decision authority

a. In you job, how often do you have to initiate things – such as coming up with you own ideas, or figuring out on your own what needs to be done?

b. How often do you have a choice in deciding how you do your tasks at work?

c. How often do you have a choice in deciding what tasks you do at work?

d. How often do you have a say in decisions about your work?

e. How often do you have a say in planning your work environment – that is, how your workplace is arranged or how things are organized?

f. How often do you control the amount of time you spend on task?

iii. Demands scale

a. How often do you have to work very intensively – that is, you are very busy trying to get things done?

b. How often do different people or groups at work demand things from you that you think are hard to combine?

c. How often do you have too many demands made on you?

d. How often do you have a lot of interruptions?

iv. Coworker support

a. How often do you get help and support from your coworkers?

b. How often are you coworkers willing to listen to your work-related problems?

v. *Supervisor support*

a. How often do you get the information you need from your supervisor or superiors?

b. How often do you get help and support from your immediate supervisor?

c. How often is your immediate supervisor willing to listen to your work-related problems?

vi. *Risk of injury on the job*

a. To what extent, over the past 10 years, have you been exposed to the risk of accidents or injuries on your job?

vii. *Job insecurity (response ranges from 1-5, from "all of the time" to "never")*

a. If you wanted to stay in your present job, what are the chances that you could keep it for the next 2 years?

Relationship Stress: 19 Item Scale

i. *Family strain (responses range 1-4, from "often" to "never")*

a. Not including your spouse or partner, how often do members of your family make too many demands on you?

b. How often do they criticize you?

c. How often do they let you down when you are counting on them?

d. How often do they get on your nerves?

ii. *Friend strain (response range 1-4, from "often" to "never")*

a. How often do your friends make too many demands on you?

b. How often do they criticize you?

c. How often do they let you down when you are counting on them?

d. How often do they get on your nerves?

iii. *Marital risk scale*

a. During the past year, how often have you thought your relationship might be in trouble? (Responses range 1-5, from "never" to "all the time")

b. Realistically, what do you think the chances are that you and your partner will eventually separate? (Responses range 1-4, from "Very Likely" to "not likely at all")

c. How much do you and your spouse or partner disagree on the following issues?

1. Money matters, such as how much to spend, save or invest?

2. Household tasks, such as what needs doing and who does it?

3. Leisure time activities, such as what to do and with whom? (Responses range 1-4, from "not at all" to "a lot")

iv. Spouse/partner strain scale (responses range 1-4, from "a lot" to "not at all)

How much

a. Does you spouse/partner really care about you?

b. Does he/she understand the way you feel about things?

c. Does he/she appreciate you?

d. Do you rely on him/her if you have a serious problem?

e. Can you open up to him/her if you need to talk about your worries?

f. Can you relax and be yourself around him/her?

Financial Stress: 2 Item Scale

a. In general, would you say you (and your family living with you) have more money than you need, just enough money for your needs, or not enough money to meet your needs? (Responses range 1-3, from "more money than you need" to "not enough money")

b. How difficult is it for you (and your family) to pay your monthly bills? (Responses range 1-4, from "very difficult" to "not at all difficult")

Work-family Spillover: 8 Item Scale (responses range 1-5, from "all of the time" to "never")

i. Negative work-to-family spillover

a. Your job reduces the effort you can give to activities at home.

b. Stress at work makes you irritable at home.

c. Your job makes you feel too tired to do the things that need attention at home.

d. Job worries or problems distract you when you are at home.

ii. Negative family-to-work spillover

a. Responsibilities at home reduce the effort you can devote to your job.

b. Personal or family worries and problems distract you when you are at work.

c. Activities and chores at home prevent you from getting the amount of sleep you need to do your job well.

d. Stress at home makes you irritable at work.

Perceived Deprivation in Family, Home/Neighbourhood, and Work: 18 Item Scale (responses range 1-4, from "a lot" to "not at all")

i. Perceived deprivation in family

a. I feel good about the opportunities I have been able to provide for my children.

b. It seems to me that family life with my children has been more negative than most peoples.

c. Problems with my children have caused me shame and embarrassment at times.

d. As a family, we have not had the resources to do many fun things together with the children.

e. I believe that I have been able to do as much for my children as most other people.

f. I feel a lot of pride about what I have been able to do for my children.

ii. Perceived deprivation in home

a. I live in as nice a home as most people.

b. I am proud of my home.

c. Most people live in a better neighbourhood than I do.

d. I don't like to invite people to my home because I do not live in a very nice place.

e. I feel very good about my home and neighbourhood.

f. It feels hopeless to try to improve my home and neighbourhood situation.

iii. Perceived deprivation in work

a. I feel cheated about the chances I have had to work at good jobs.

b. When I think about the work I do on my job, I feel a good deal of pride.

c. I feel that others respect the work I do on my job.

d. Most people have more rewarding jobs than I do.

e. When it comes to my work life, I've had opportunities that are as good as most peoples.

f. It makes me feel discouraged that other people have much better jobs than I do.

Neighbourhood Stress: 4 Item Scale (responses range 1-4, from "a lot" to "not at all")

a. I feel safe being out alone in my neighbourhood during the daytime.

b. I feel safe being out alone in my neighbourhood at night.

c. I could call on a neighbour for help if I needed it.

d. People in my neighbourhood trust each other.

Discrimination: 19 Item Scale

You were:

a. Discouraged by a teacher or advisor from seeking higher education.

b. Denied a scholarship.

c. Not hired for a job.

d. Not given a promotion.

e. Fired.

f. Prevented from renting or buying a home in the neighbourhood you wanted.

g. Prevented from remaining in a neighbourhood because neighbours made life so uncomfortable.

h. Hassled by the police.

i. Denied a bank loan.

j. Denied or provided inferior service by a plumber, car mechanic, or other service provider.

i. Everyday discrimination (responses range 1-4, from "often" to "never")

a. You are treated with less courtesy than other people.

b. You are treated with less respect than other people.

c. You receive poorer service than other people at restaurants or stores.

d. People act as if they think you are not smart.

e. People act as if they are afraid of you.

f. People act as if they think you are dishonest.

g. People act as if they think you are not as good as they are.

h. You are called names or insulted.

i. You are threatened or harassed.

Past-year problems in immediate family: 10 Item Scale (asked separately for (i) spouse/partner, (ii) parents, (iii) children; yes/no responses)

a. Chronic disease or disability?

b. Frequent minor illness?

c. Emotional problems (such as sadness, anxiety)?

d. Alcohol or substance problems?

e. Financial problems, such as low income or heavy debts?

f. Problems at school or at work (such as failing grades, poor job performance)?

g. Difficulty finding or keeping a job?

h. Marital or partner relationship problems?

i. Legal problems (such as involved in lawsuits, police charges, traffic violations)?

j. Difficulty getting along with people?

Mental Health Measures (From Charles et al., 2013)

General Affective Distress: (Scaled from 1-5, being "None of the time" to "All of the time" in the past 30 days how often do they experience the following emotions and emotional states).

i. *Emotions*

a. Worthless

b. Hopeless

c. Nervous

d. Restless/Fidgety

ii. *Emotional States*

a. How much of the time everything was an effort.

b. How often they felt so sad nothing could cheer them up.

Self-Reported Affective Disorder: In the past 12 Months, participants indicated either "Yes" or "No" if they had experienced or been treated for:

1. Anxiety, depression, or some other emotional disorder

CIDI-SF Affective Diagnosis based on self-report: Participants were diagnosed with an affective disorder within the past twelve months if they met the criteria for ONE of the following disorders

i. *Major Depressive Disorder:* Participants must meet the conditions in Part 1(a) and indicate that they feel these feelings "most of the day" or "all day long" along with "every day" or "almost every day" from Part 1(b). Further, participants must endorse feeling at least 4 of the symptoms from Part 2 during the two-week period. Both criteria (from part 1 and part 2) must be met for a positive classification.

Part 1:

a. Participants were asked if they felt sad, blue or depressed for two weeks or more in a row in the past twelve months

b. During these two weeks, participants were asked whether these feelings lasted all or most of the day (options range from "Less than half the day" to "All day long") along

with everyday or almost everyday (options range from “Everyday” to “Less often than that”).

Part 2:

a. Lost interest in most things.

b. Felt more tired or low energy.

c. Lost appetite.

d. Increased appetite.

e. More trouble falling asleep.

f. More trouble concentrating.

g. Felt down, no good or worthless.

h. Thought a lot about death.

ii. *Dysthymia:* Participants must meet the conditions in Part 1(a) via a (yes) and indicate that they feel this way “all day long” or “most of the day” along with “everyday” or “almost everyday” from Part 1(b). Further, participants must endorse feeling at least 4 of the symptoms from Part 2 during the two-week period. Both criteria (from part 1 and part 2) must be met for a positive classification.

Part 1:

a. Indicate with a yes or no if participants have lost interest in most things during a two-week period in the past 12 months.

b. During these two weeks, participants were asked whether these feelings lasted all or most of the day (options range from “Less than half the day” to “All day long”) along with everyday or almost everyday (options range from “Everyday” to “Less often than that”).

Part 2:

a. Feeling more tired or having less energy than usual.

b. Lost appetite

c. Trouble falling asleep.

d. More trouble concentrating.

e. Felt down, no good or worthless.

f. Thought a lot about death.

iii. *Generalized Anxiety Disorder:* Participants must indicate that they felt "a lot more" worried than most people (Part 1(a)) along with feeling worried "every day", "just about everyday", or "most days" in Part 1(b). Participants must also experience at least 3 of the symptoms from part 2. Both criteria (from part 1 and part 2) must be met for a positive classification.

Part 1:

c. Participants had to indicate they felt worried "a lot more" than most people.

a. Participants were asked whether these feelings occurred everyday or just about everyday or most days in the past 12 months (options range from "Everyday" to "Less than half the days").

Part 2:

a. Restless because of worry.

b. Keyed up, on edge or nervous energy.

c. Irritable because of worry.

d. Trouble falling asleep because of worry.

e. Trouble staying asleep because of worry.

f. Trouble concentrating because of worry.

g. Trouble remembering because of worry.

h. Low on energy because of worry.

i. Tire easily because of worry.

j. Sore or aching muscles because of worry.

Physical Health Measures (From Piazza et al., 2013)

For each of the following Chronic Health Conditions, participants were asked whether or not they had experienced or been treated for the following conditions (Answered either Yes or No):

1. Autoimmune Disorder or Lupus
2. Cardiovascular Conditions
    a. Stroke
    b. High Blood Pressure or Hypertension
3. Diabetes or High Blood Sugar
4. Digestive Conditions

a. Recurring stomach trouble, Indigestion or Diarrhea
b. Constipated all/most of the time
c. Ulcer

5. Foot Trouble
6. Hay Fever
7. Gall Bladder Trouble
8. Lung Conditions
    a. Asthma, Bronchitis or Emphysema
    b. Tuberculosis
    c. Other Lung Problems
9. Neurological Conditions
10. Pain-Related Conditions
    a. Arthritis, Rheumatism or other bone/joint diseases
    b. Sciatica, Lumbago or Recurring Backache
    c. Migraine Headaches
11. Skin Trouble
12. Thyroid Disease
13. Trouble with Gums, Mouth or Teeth
    a. Persistent trouble with gums or mouth
    b. Persistent trouble with teeth
14. Urinary or Bladder Problems

For the following, participants noted whether or not they have EVER experienced each Chronic Health Condition as they typically require ongoing care (Answered either Yes or No):

1. Cancer