The Relationship of Divine Forgiveness, Victim Forgiveness, and Physical Health Mediated by Stress

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Abstract: Though there is increasing evidence to support a relationship between forgiveness and physical health, certain subcategories of forgiveness, namely victim and divine forgiveness, are relatively understudied. This study seeks to add to the body of forgiveness literature by examining how divine and victim forgiveness relate to one's physical health, and whether that relationship is mediated by stress. Furthermore, a literature review is included to detail how stress, a potential mediating variable between forgiveness and physical health, affects physical health. The results of the study reveal that victim forgiveness positively predicts physical health, but is not mediated by stress. In contrast, divine forgiveness alone does not predict physical health, rather the effect of divine forgiveness on physical health was mediated by stress.

The Relationship of Divine Forgiveness, Victim Forgiveness, and Physical Health mediated by Stress

Studies related to forgiveness and its different aspects have been increasing in number throughout the years. However, despite a clear association between forgiveness and mental health (Akhtar & Barlow, 2018; Davis et al., 2015), research looking into the relationship between physical health and forgiveness is still in its early stages. Nevertheless, the research that has been conducted shows some promise that forgiveness may have a similar positive bearing on physical health as it does on mental health. For example, studies have been conducted involving patients who had coronary artery disease, in which they underwent forgiveness therapy; the results of the study showed a significant decrease in anger-induced myocardial perfusions in the therapy group compared to the controls (Waltman et al., 2009). Similarly, forgiveness intervention has also been observed to improve the overall fibromyalgia health of women who had been abused during childhood (Lee & Enright, 2014). These studies show the potential of forgiveness interventions in helping manage certain physical illnesses, and, as medicine shifts towards a more integrative approach, forgiveness and its different aspects may be worth giving some consideration in regard to promoting both mental and physical health.

Definition of Forgiveness

Forgiveness was originally a topic studied in academic disciplines such as theology, religion, and philosophy, but recently the study of forgiveness has found its way in the field of psychology as well, likely due in part to increasing research interest in positive psychology, which is psychology that deals with "optimal human functioning" (Linley, Joseph, Harrington, & Wood, 2006). The broadness of forgiveness as a topic means that there exist many definitions that attempt to conceptualize it. Even in the field of psychology, there are varying working

models of forgiveness, which can sometimes dictate how one operationalizes forgiveness as a variable. One of the earliest models of forgiveness was developed by Enright and the Human Development Study Group (1996), in which they refer to forgiveness as a triad consisting of forgiving others, self-forgiveness, and receiving forgiveness (Enright, 1996). This study will focus on the receiving forgiveness aspect of the triad, which is defined as "When one offends another, he or she receives forgiveness when the offended person willingly offers the cessation of negative attitudes, thoughts, and behaviors, and substitutes more positive feelings, thoughts, and behaviors toward the offender" (Enright, 1996, p.112).

Forgiveness and Stress

Interpersonal relationships are an important aspect of life, and preserving the harmony between these relationships can be necessary for us to function properly in our day-to-day lives. However, it is inevitable that within these relationships, transgressions, both committed against us and by us, will occur. Depending on the severity of the offense against us, it can be difficult to forgive; failing to do so is referred to in forgiveness literature as 'unforgiveness', or in more familiar terms, 'holding a grudge'. In forgiveness literature, unforgiveness is characterized by a prolonged experience of a combination of negative emotions such as "anger, resentment, hostility, fear, bitterness and hatred" as well as "motivations for revenge or avoidance" specifically towards the offender (Lee & Enright, 2019). In contrast to the health-promoting effects of forgiveness, as seen in the previously mentioned intervention studies, unforgiveness not surprisingly has negative effects on one's mental and physical health. The slew of negative emotions associated with unforgiveness paint a clear picture of how forgiveness/unforgiveness can directly affect one's mental health; however, it is unlikely that forgiveness directly influences health. Rather, a prominent theory in the literature proposes stress, particularly

psychological stress, as the physiological mechanism that mediates the relationship between the two (Green, DeCourville, & Sadava, 2012; Lee & Enright, 2019).

General Effect of Stress on Health

Stress, both psychological and physical, can be defined as an individual's response to demands that are placed upon them, otherwise known as stressors (Kogler et al., 2015). Stressors can also be thought of as things that threaten or unbalance our 'homeostasis'; the stress response is, therefore, our body's attempt at restoring the homeostatic imbalance. Psychological and physical stressors can differ in origin and certain characteristics, but both elicit real, physiological responses within the body (Kogler et al., 2015). Both types of stress, in limited amounts, can be beneficial, often initiating psychological and physical adaptation in order to cope with the present stressor (Dhabhar, 2018). Chronic exposure to stress, however, can have detrimental effects not only on one's mental health, but one's physical health as well (Yaribeygi, Panahi, Sahraei, Johnston, & Sahebkar, 2017). Long-term physical stressors, such as illness, injury, and infection, are clear and explicit threats to one's health, but it can be difficult to see that chronic psychological or emotional stressors also have a negative effect on one's physical health. Chronic psychological stress has been associated with the weakening of the immune system, cardiovascular disease, cognitive decline, and other systemic illnesses (McEwen & Sapolsky, 2006; Yaribeygi et al., 2017). In a sense, psychological stress can promote adverse physical stresses, like those previously mentioned, and can seriously harm an individual if left unaddressed.

Evidence of Unforgiveness as Stress

The psychological literature further adds that unforgiveness can be thought of as part of the stress response to a transgression or conflict – a type of social stressor and that the act of

forgiving is, therefore, a coping method used to deal with the stressor (Harris & Thoresen, 2005). Unforgiveness being stressful is supported by research that shows unforgiveness giving rise to the same physiological events that occur in the regular stress response. For example, PET scans have shown that individuals in a state of anger, a strong predictor of unforgiveness (Wu, Chi, Zeng, Lin, & Du, 2019), have decreased activity in the prefrontal cortex and an increase in activity of the limbic system, particularly the amygdala (Pietrini, Guazzelli, Basso, Jaffe, & Grafman, 2000); both effects are characteristic of the changes we see in brain activity during the stress response (Arnsten, Amy F. T., 2009). Research has shown that holding a grudge was associated with sympathetic nervous system (SNS) activation and muscle tensions (Lawler-Row, Karremans, Scott, Edlis-Matityahou, & Edwards, 2008), and that self-reported unforgiveness was linked with higher levels of salivary cortisol (Berry & Worthington Jr., 2001), both of which are physical markers of the stress response. (Worthington & Scherer, 2004). Of course, correlational studies were also in support of the relationship between forgiveness and stress (Green, DeCourville, & Sadava, 2012; Toussaint, Shields, & Slavich, 2016).

Victim Forgiveness

The majority of forgiveness research, including that on stress and physical health, has focused primarily on forgiveness from the victim's perspective, and many of the health benefits of forgiveness intervention studies come from the act of forgiving. But since virtually everybody will at some point in time be both victims and transgressors within their relationships, there is good reason to further examine the effects of unforgiveness and its related concepts from the side of the offender. Since transgressors are the individuals who committed the offense, they do not have access to the active forgiving aspect, rather they must wait to receive forgiveness. The receiving of forgiveness is one of the parts of Enright's forgiveness triad and is defined as

"When one offends another, he or she receives forgiveness when the offended person willingly offers the cessation of negative attitudes, thoughts, and behaviors, and substitutes more positive feelings, thoughts, and behaviors toward the offender" (Enright, 1996), p.112). From this we derive our definition of victim forgiveness, which is "the offenders' perception of having received forgiveness from the victim" (Kim et al, 2020). Victim forgiveness involves the transgressor feeling as if the person he offended no longer harbors any negative emotions towards the transgressor, and that there is a sense of peace in the relationship. It is hypothesized that there is a fine distinction between receiving forgiveness and victim forgiveness; while a transgressor may have been offered genuine forgiveness from his victim, certain psychological factors, such as severe shame, can prevent the transgressor from genuinely perceiving that he is forgiven. Victim forgiveness is a novel concept, so research that directly examines its difference from receiving forgiveness is difficult to find; however, we can use other measures of feeling forgiven, such as repentance and prosocial behavior to infer the relationship between the victim forgiveness and receiving forgiveness. For example, one study showed that whether the victim offers forgiveness explicitly or implicitly affects the feelings of repentance and prosocial behavior measured in transgressors (Struthers, Eaton, Shirvani, Georghiou, & Edell, 2008). These findings lend support to the hypothesis that being offered forgiveness and actually feeling forgiven are not one and the same, but that there can be a gradation in how much a transgressor feels forgiven.

Divine Forgiveness

Divine forgiveness is another subcategory of forgiveness and is similar to victim forgiveness in that it deals with the transgressor's perception of being forgiven. The distinction is in the party offering forgiveness; where victim forgiveness deals with the transgressor's

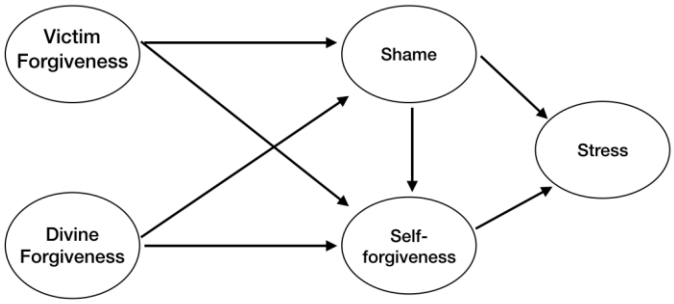
perception of being forgiven by the individual they offended, divine forgiveness deals with the transgressor's perception that they have been forgiven by God regarding a certain offense. Formally it is defined as "when offenders perceive that God has forgiven them and that there is a sense of peace in the relationship between God and themselves" (Kim et al, 2020).

Victim Forgiveness, Divine Forgiveness, and Stress

The feeling of not being forgiven, either by the person that the transgressor has offended or by God, can be a stressful experience. One way by which divine and victim forgiveness can lead to stress is through their effect on the transgressor's self-forgiveness. Self-forgiveness is the third part of the forgiveness triad and is defined as " a willingness to abandon self-resentment in the face of one's own acknowledged objective wrong, while fostering compassion, generosity, and love toward oneself " (Enright, 1996). Self-forgiveness involves the transgressor acknowledging that he has committed an "objective wrong", or taking moral responsibility for his offense (Worthington & Wade, 2019). Failing to achieve self-forgiveness implies that the transgressor continues to hold negative attitudes toward themselves such as resentment, anger, or hatred. The transgressor then is essentially experiencing unforgiveness toward himself, which we've previously mentioned as being a stress response. Victim and divine forgiveness can then cause stress by preventing the transgressor from achieving self-forgiveness. Studies have shown that receiving forgiveness from others contributes to one's self-forgiveness (Hall & Fincham, 2008; Ingersoll-Dayton & Krause, 2005). Feeling that one has not been forgiven by God has also been shown to negatively correlate with self-forgiveness (Ingersoll-Dayton, Torges, & Krause, 2010; Long, Chen, Potts, Hanson, & VanderWeele, 2020). These studies evidence that feeling unforgiven by either God or the person you hurt could make it more difficult for you to forgive yourself.

Achieving self-forgiveness not only requires the transgressor to acknowledge his behavior, but also to acknowledge his self-worth and recognize that he is capable of change (Enright, 1996). Victim and divine forgiveness could also inhibit self-forgiveness by causing feelings of shame. Shame is defined as a negative emotion that is brought about from feeling that one is being judged by others, and that "one's self is perceived to be inadequate, inappropriate or immoral" (Dolezal & Lyons, 2017). Conceptually, shame is tied to one's identity, and therefore directly opposes the acknowledgement of self-worth that is necessary for the transgressor to be able to self-forgive. This is further evidenced by studies that consistently find shame to be negatively linked to self-forgiveness (Carpenter, Isenberg, & McDonald, 2019; Hall & Fincham, 2005; McGaffin, Lyons, & Deane, 2013). Feelings of not being forgiven can produce deep feelings of shame (Riek, Luna, & Schnabelrauch, 2014; Struthers et al., 2008) that can make it more difficult for one to forgive themself. Shame itself is maladaptive and destructive compared to its guilt counterpart and is more likely to produce chronic psychological stress (Dolezal & Lyons, 2017). The conceptual model for these relationships is similar to the model proposed by Ingersoll-Dayton, Torges, & Krause (2010): 1) divine unforgiveness and victim unforgiveness relate to self-unforgiveness, 2) all three unforgiveness are related to shame 3) self-unforgiveness directly relates to psychological stress 4) shame directly relates to psychological stress.

Figure 1. Conceptual model of the relationship between victim forgiveness, divine forgiveness, self-forgiveness, shame, and stress



Review of Stress Pathophysiology

Cardiovascular Stress Response

There is increasing evidence that chronic psychological stress and cardiovascular diseases are positively linked. Stressors such as work stress, low socioeconomic status, and negative emotions have been associated with coronary heart disease (CHD), and the relationship is prevalent even when controlling for the typical predictors of heart disease such as obesity, smoking, and alcohol consumption (Rozanski, Blumenthal, Davidson, Saab, & Kubzansky, 2005; Vitaliano et al., 2002; Vujcic et al., 2016). Chronic psychological stress has also been linked to myocardial infarctions, hypertension, and endothelial dysfunction (Golbidi, Frisbee, & Laher, 2015; Spruill Tanya et al., 2019; Vujcic et al., 2016). These studies are examples of the maladaptation of the stress response within the cardiovascular system; however, in proper conditions, the stress response is built for our survival and intended to prepare our body to address the present threat or stressor. The adaptive stress response begins with activation of the hypothalamic pituitary adrenal axis (HPA) and the sympathetic-adreno-medullar (SAM) axis. Activation of the HPA axis ultimately results in the release of glucocorticoids, such as cortisol,

which bring about a variety of adaptive results, such as increased blood glucose, and mediation of the effects of catecholamines, such as epinephrine and norepinephrine, on the blood vessel (Yang, S. & Zhang, 2004). The SAM axis is the sympathetic activation side of the acute stress response and generally results in immediate changes mediated by catecholamines. Catecholamine release can bring about increased heart rate by acting on pacemaker cells, increased force of myocardial contraction by increasing cardiac cell contractility, and increased blood pressure through vasoconstriction as needed throughout the body (William Tank & Lee Wong, 2014).

The body's prolonged attempts to address the stressors and restore homeostasis can result in a dysregulation of the adaptive stress response and cause stress disorders. This dysregulation can lead to the variety of cardiovascular problems that were previously mentioned. Since the stress response has a system-wide effect, there are multiple mechanisms that can bring about damage to the cardiovascular system. Prolonged exposure to cortisol has been observed to impair endothelial health through the reduction in membrane adenylyl cyclase activity and cyclic adenosine monophosphate (cAMP) levels (Borski, Hyde, & Fruchtman, 2002). cAMP has a role in endothelium repair and has been suggested to prevent atherosclerosis by removing excess cholesterol in macrophages and reducing the amount of free circulating cholesterol (Fantidis, 2010). Nitric oxide (NO) is produced by the endothelium and functions to regulate vascular resistance and dilator tone, prevent platelets and other cells from aggregating, and regulate cell growth (Tousoulis, Kampoli, Tentolouris, Papageorgiou, & Stefanadis, 2012). Cortisol potentially inhibits endothelial NO synthase, the NO synthesizing enzyme for the endothelium. Cortisol could also reduce NO availability through the production of reactive oxygen species that cause NO uncoupling (Golbidi et al., 2015; Iuchi et al., 2003). Cortisol's disruption of NO

synthesis and availability over time likely leads to some of the cardiovascular diseases that we see associated with stress. This is supported by the fact that diabetes mellitus, hypercholesterolemia, hypertension, and even heart failure are associated with reduced NO function due to inhibition or excessive oxidative degradation (Tousoulis et al., 2012). A third mechanism of cortisol-related dysfunction is its role in increasing blood glucose. Chronic stress, both physical and psychological, can cause stress-related hyperglycemia, which has downstream effects of reducing NO activity and increasing the number of inflammatory cytokines in the blood (Golbidi et al., 2015). Both of these effects may promote cardiovascular risks through increasing oxidative stress (Esposito, Marfella, & Giugliano, 2003).

Chronic SNS and renin-angiotensin-aldosterone system (RAAS) activation may also provide another pathophysiological link between stress and cardiovascular diseases. Episodes of chronic psychological stress may lead to continuous activation of the SNS (Won & Kim, 2016). This SNS hyperactivity results in increased levels of catecholamines, particularly norepinephrine, in circulation (Ayada, Toru, & Korkut, 2015; Won & Kim, 2016). Normally catecholamines would bind to beta-adrenergic receptors in cardiac muscle, activating cAMP, resulting in the opening of calcium channels, and the subsequent influx of calcium in the sarcolemma (Pinnell, Turner, & Howell, 2007), but an excess of catecholamines can endanger the cardiac muscle cell. High concentrations of catecholamines are oxidized into aminochromes, which can alter gene activity in cardiomyocytes, cause intracellular calcium overload, and cause oxidative stress to the cardiomyocyte, ultimately resulting in apoptosis of the cell.

The RAAS has recently been observed to be an important hormone system in the stress response. Angiotensin II is believed to have a wide contribution to the stress response, as it interacts with many of the stress response intermediates such as potentiation of

adrenocorticotropic hormone (ACTH) and stimulation of vasopressin release; angiotensin II may also strengthen the SNS and HPA response overall (Yang, G., Wan, & Zhu, 1996). However, similar to the effects of excess catecholamines, excess angiotensin may lead to increased oxidative stress through the production of superoxide anions and increase oxidative toxicity (Ayada et al., 2015). Continued damage to cardiac muscle cells due to SNS and RAAS hyperactivation would likely lead to a variety of cardiovascular complications and perhaps ultimately heart failure (Ayada et al., 2015).

Immune System and Stress

The systematic study of mental health and the immune system is called psychoneuroimmunology, and many developments have been made in that field of study since the term was first coined in 1975. One of the major topics of interest within psychoneuroimmunology is the relationship between stress and immune function. The deleterious effects of chronic stress and the immune system are prevalent in the literature. Viral challenges, where healthy participants are given a pathogen or virus, showed prolonged durations of stress predicted susceptibility to infection (Cohen, Tyrrell, & Smith, 1991), and further research showed that in the case of infections such as the flu, increased psychological stress is associated with more severe symptoms (Cohen, 2005). Stress and the immune cancer response have also been examined, and while evidence that chronic stress plays a role in causing cancer is relatively weak, some findings suggest that chronic stress may be linked to cancer progression, tumor growth, and metastasis (Kemeny & Schedlowski, 2007). A study on human papillomavirus (HPV)-associated cervical intraepithelial neoplasia (CIN), a condition that usually precedes the development of breast cancer, found that higher levels of stress correlated with greater severity of CIN symptoms (Antoni, Schneiderman, & Penedo, 2007). Also, studies

using rat models found that social stress could reduce resistance to tumor metastasis (Reiche, Nunes, & Morimoto, 2004). In contrast, acute stress has been found to enhance the properties of the immune system. For example, acute stress during the time of infection can enhance the amount of memory T cells, thereby increasing the duration of immune memory (Dhabhar & Viswanathan, 2005). Acute stress also increases the number of neutrophils, lymphocytes, helper T cells (Th), cytolytic T cells (CTL), and B cells, and mobilizes them to areas like the skin (Dhabhar, Malarkey, Neri, & McEwen, 2012). The differences in the effects of acute and chronic stress illustrate how the stress's overall effect on immune function is more complex than simply suppression or enhancement.

First, there are physiological mechanisms that allow the stress response to interacting with the immune system. The stress-mediating molecules, such as epinephrine, cytokines, corticotropin-releasing hormone (CRH), ACTH, and cortisol, have all been found to affect humoral and cellular immune responses (Kemeny & Schedlowski, 2007). Sympathetic adrenergic nerves, which are active as part of the SAM axis of the stress response, have also been found to terminate at immune-related organs such the thymus, spleen, bone marrow, and lymph nodes (Felten et al., 1987). Furthermore, all immune cells have beta-adrenergic receptors that can bind to the catecholamines that are released during the stress response (Sanders & Kavelaars, 2007).

In order to understand the bi-directional influence of stress on the immune system, it is helpful to classify the physiological stress response in waves. The early wave of the stress response is characterized by the initial activation of the HPA axis, releasing CRH and ACTH, and the activation of the SAM axis, where the SNS is activated and catecholamines are quickly released (Sapolsky, Romero, & Munck, 2000). The second, slower wave, involves the increasing

amounts of steroid such as glucocorticoids (GC) and their subsequent effects (Sapolsky et al., 2000). SNS activation and catecholamines are dominant in the early wave response, and this can help partially explain the immune-enhancing effects of acute stress. For example, one study tested the changes in skin delayed type hypersensitivity, which is a type of immune response, in response to different amounts of corticosterone and epinephrine in the context of the adrenalectomy (Dhabhar & McEwen, 1999). In the study, the purpose of the adrenalectomy was to control for the immune-enhancing effects of acute stress (Dhabhar & McEwen, 1999). The researchers found that administering epinephrine along with low doses of corticosterone produced an immune-enhancing response (Dhabhar & McEwen, 1999). These findings suggest that catecholamine's primary effect on the immune system is enhancement (Dhabhar & McEwen, 1999). What was interesting was the role of the low dose of corticosterone, which is the primary corticosteroid released by the adrenal glands of laboratory mice, which showed that glucocorticoids, despite their low concentrations and delayed effect, also have immune-enhancing properties. Rather, it is because the glucocorticoid was in a low dose that it had an immune-enhancing response (Cain & Cidlowski, 2017; Sapolsky et al., 2000). Low concentrations of glucocorticoids can help sensitize or prepare the immune response to fight infection by upregulating pattern recognition receptors, which are necessary for recognizing pathogens, cytokine receptors, and complement factors. The upregulation of these factors can allow the immune system to respond much more quickly to signals of infection (Cain & Cidlowski, 2017). GC's effects are referred to as permissive rather than directly stimulating, serving to prime and ready the immune system for an effective and efficient response (Sapolsky et al., 2000). The concentration of GCs needed to produce the permissive response are similar to the levels of GCs produced during the peaks of the circadian rhythm; this can explain why some

experiments on animal models, which have controls with no GC, find an immune-enhancing effect when low-GC is administered (Sapolsky et al., 2000). It also shows why it is necessary that an increase in GC levels and GC genomic effects have to be delayed in the stress response; it is to allow the permissive effects of GC to play a role in moderating the rest of the immune-enhancing effect of the acute stress response (Sapolsky et al., 2000). Once GC reaches levels characteristic of stress, it no longer produces a permissive effect; rather, it exhibits a suppressing effect on the immune system (Sapolsky et al., 2000).

The biphasic model of the effect of glucocorticoids on the immune response paints a good picture of how the stress response was intended to work in tandem with the immune system and how chronic stress can cause things to go wrong. Similar to how the effects of the regular stress response on the cardiovascular system were intended to help in survival, the effects of acute stress on the immune system are beneficial as well. Since stressful situations are situations where injury, and therefore infection, may occur, it would make sense to have a boost in the immune system during this time (Dhabhar, 2009). This adaptation would have been incredibly helpful in the past, when humans, during hunts or other endeavors, would have frequently encountered physical dangers. Though we don't necessarily face many physical threats now, this immune response can still be beneficial; for example, activation of the acute stress response during surgery would boost the immune system, thereby decreasing the risk of infection (Dhabhar, 2009). Immune suppression caused by stress levels of GC is also an adaptive response. Stress-induced immune enhancement is a case where too much of a good thing becomes a bad thing. An overactive immune system can result in autoimmune disorders. Therefore, the glucocorticoid suppression of the immune system is a mechanism to keep the increased immune activity in check. The maladaptation occurs when stress becomes chronic.

Feeling unforgiven can cause feelings of shame and therefore cause psychological stress; unresolved stressors can then become chronic. Chronic stress means that one would continuously raise their GC levels beyond baseline, essentially keeping it in the range where it is immunosuppressive. This can account for why being chronically stressed can lead to the deleterious effects that were mentioned earlier, such as increased infection risk, lower tumor metastasis resistance, and reduction in immune cells.

The Brain and Stress

Looking at the brain, we see that the effects of stress follow a similar pattern, as seen in the immune and cardiovascular systems, where acute stress produces effects originally intended to be an adaptive response and chronic stress produces effects that are generally maladaptive. Two specific parts of the brain, the prefrontal cortex (PFC) and the amygdala, are most relevant when it comes to stress. The prefrontal cortex has a variety of functions that are generally associated with higher-level thinking such as abstract thought, planning, regulation of behavior, etc. The amygdala, which is part of the limbic system, is often thought of as the fear and aggression center of the brain and also plays roles in memory consolidation, primitive decision making, and emotional responses (Amunts et al., 2005). Another important aspect of the amygdala is that it has projections in the hypothalamus and brainstem allowing it to stimulate the HPA and SAM axes. The amygdala can also influence the activation of the locus coeruleus (LC) which is responsible for the synthesis and release of NE in the brain (Arnsten, Amy F. T., Raskind, Taylor, & Connor, 2015).

Generally, acute stress inhibits functioning of the PFC and enhances functioning of the amygdala. Under stress conditions, the amygdala activates the HPA axis and SAM axis (Arnsten, 2009). The amygdala also has projections in the neural network such as the LC that results in the

release of norepinephrine (Arnsten et al., 2015). Both norepinephrine and dopamine, though norepinephrine, in particular, are the major effectors of stress in this PFC and the amygdala. Glucocorticoids are also involved; however, studies have shown that the effects of GCs on the brain may be moderated by NE and may simply serve to accentuate the effects of NE (Arnsten et al., 2015; Wirth, 2015). The acute stress response elevates the levels of NE, which bind to α-1 adrenergic receptors on the neurons of the prefrontal cortex (Arnsten, 2009). Upon binding to these receptors, the phosphokinase C (PKC) pathway is activated, resulting in the release of intracellular calcium and the opening of calcium-activated potassium (K+) channels (Arnsten, 2009). The opening of the K+ channels hyperpolarizes the cell, thereby inhibiting or suppressing neuronal activity (Arnsten, 2009). Dopamine also binds to receptors on PFC neurons and then activates a PKA pathway resulting in increased intracellular levels of cAMP (Arnsten, 2009). This is important when we consider the presence of hyperpolarization-activated cyclic nucleotide–gated (HCN) channels at the dendrites of the PFC neurons. HCN channels are commonly known as the pacemaker channels of the heart, which help to regulate the rhythmic activity of cardiac cells (Benarroch, 2013). The HCN channels have a similar function in the brain where they regulate the patterned firing of neurons that is characteristic of brain activity (Benarroch, 2013). For the HCN channels to perform their regulatory function, they can be both excitatory or inhibitory, depending on cell conditions (Arnsten, 2009). In the PFC neurons, high levels of cAMP seem to open HCN channels, resulting in the prevention of depolarization at the dendrite, likely through the opening of K+ channels (Arnsten, 2009). This results in a decrease in the efficiency of the synaptic input at the dendrite (Arnsten, 2009). Lack of excitatory input contributes to the hyperpolarization of the neuron and further contributes to the "shut-down" or the repression of the PFC (Arnsten, 2009). Under normal non-stressed conditions, NE will much

more readily bind to alpha-2 adrenergic receptors, since NE has a higher affinity to alpha-2 receptors compared to alpha-1 (Ramos & Arnsten, 2007). This explains why the alpha-1 receptors are only activated in response to higher amounts of NE (Arnsten, Amy FT, Wang, & Paspalas, 2012). The literature shows that the activation of the alpha 2a receptors generally enhances the activity of the PFC (Arnsten, A. F., 2000). One mechanism by which it achieves this is through the inhibition of intracellular cAMP, which allows HCN channels to do their proper regulative functioning of stimulating appropriate networks while inhibiting inappropriate networks (Arnsten, 2009). One of the PFC's main functions is the regulation of emotional or habitual impulses derived from the amygdala; therefore, reducing PFC activity also results in the enhancement of amygdala function (Arnsten, 2009). Furthermore, NE and dopamine themselves will bind to the amygdala and enhance its functions, which can often result in a cycle of amygdala activation and stress hormone release (Arnsten, 2009).

While it might seem that the shutting down of the PFC during acute stress is disadvantageous, there is some survival value to it. In premodern times, life or death situations were likely to be better handled by the habitual, instinctive, and primitive actions that are generated by the subcortical structures such as the amygdala (Arnsten, 2000). The higher cognitive functions of the PFC such as abstract thinking and judgment may even be counterproductive to producing the necessary fast and rapid actions or decision making that is needed to deal with the situation. Furthermore, enhancement of amygdala function aids in that quick impulse and emotion-driven decision-making. Also due to the amygdala's projection into the hippocampus, its enhancement can better consolidate the memory of the events surrounding the stressor, which may aid in an individual's future survival (Arnsten, 2009).

Chronic stress has been shown to produce chemical and structural changes within the PFC that seem to exaggerate the prioritization of amygdala activity over the PFC. Chronic stress seems to prime the brain to more readily produce NE through the increased firing of the LC. Animal studies have also shown that chronic stress causes dendrite and dendritic spine loss in the PFC, perhaps due to the lack of patterned and simultaneous firing that usually strengthens synapses (Arnsten, 2009). In contrast, chronic stress increases the number of dendrites in the amygdala. It has also been shown that chronic stress results in the reduction of gray matter in humans (Ansell, Rando, Tuit, Guarnaccia, & Sinha, 2012) as well as decreased regulation of the amygdala by the PFC (Won & Kim, 2016). These changes in the brain manifest in impairment of behavior and cognitive function. Dendritic loss has been shown to reduce working memory and the ability to shift attention (Arnsten et al., 2015). The reduction in PFC activity can result in impaired impulse control and increased emotional reactivity. The persistent suppression of the PFC and activation of the amygdala can help explain a more indirect method by which chronic stress can deteriorate overall physical health. Chronic stress is linked with unhealthy behaviors such as smoking, excess alcohol consumption, excess food consumption, and reduced physical activity (Rodriquez, Gregorich, Livaudais-Toman, & Pérez-Stable, 2017; Vgontzas & Bixler, 2008). The lack of impulse control and increased emotional decision-making resulting from stress's effects on the brain could further explain why chronically stressed individuals are associated with more frequent instances of unhealthy behaviors. The previous sections of this literature review showed how chronic stress can harm physical health through the maladaptation of the body's innate acute stress mechanism. Stress may also be able to indirectly impair physical health through the engagement of unhealthy behaviors by individuals, which is likely to be exacerbated by stress's effects on the brain.

Current Study

Research into divine and victim forgiveness has only recently begun to take shape. Therefore, there are few studies that directly examine the effects of feeling unforgiven and physical health. A study was conducted that measured the physiological responses of transgressors (individuals who had offended another person) when they were asked to imagine situations either where they had been forgiven by the person that they had offended, or situations where they had not been forgiven (vanOyen-Witvliet, Ludwig, & Bauer, 2002). The results were contrary to what was expected; there were very few changes in physiological markers such as heart rate, blood pressure, skin conductance, and corrugation (21). Similarly, Chen et al. (2019) conducted a meta-analysis that examined the correlations between types of forgiveness, i.e. self-forgiveness, forgiveness of others, and divine forgiveness, and physical health outcomes (Chen, Harris, Worthington, & VanderWeele, 2019). Once again, the researchers reported that there was no strong correlation found between divine forgiveness and physical health, though the researchers attribute this null finding to the method by which they measured physical health – it was measured through outcomes of diseases, which may take longer to fully see effects (Chen et al., 2019).

Looking at both the theoretical and empirical basis for the effect of divine and victim forgiveness on physical health, it's difficult to predict what the outcome of the proposed study might be. In theory, it would make sense that feeling that one has received forgiveness from God and the person you have harmed should ease feelings of shame, which in turn can lower stress, thereby improving overall physical health. However, the few studies that have attempted to observe these variables seem to contradict the proposed theory. It may be that the effects of perceived interpersonal and divine forgiveness on physical health may be too gradual or subtle to

be noticed in disease outcome and cross-sectional biological measures of physical health. Perhaps a broader measure of physical health would be more able to detect the effects. If so, then this study could bear positive results.

Research Questions:

1) Does victim forgiveness predict physical health?
2) Does divine forgiveness predict physical health?
3) Does stress play a mediating role in the relationship between perceived forgiveness and physical health?

The hypothesis for the current study is that both victim and divine forgiveness will have a positive correlation with physical health (H1, H2), and that stress will mediate the relationship between the two forgiveness and physical health (H3a and H3b).

Methods

Participants and Procedure

A sample was taken from undergraduate Liberty University students, who were 18 or older, currently taking a Liberty University psychology course, and believe in God or a higher power. Institutional Review Board (IRB) approval was obtained before recruitment began. Participants were recruited by sending out an email with a link to the online survey and through the announcement of the study within psychology classes. Upon clicking the link for the survey, the participants were taken to an online page containing the details of the study and information regarding consent. Those who consented to participate in the study were then routed to another page that contained the screening survey. Participants who were younger than 18, not Liberty students currently taking a psychology course, or did not believe in God or some form of a higher power were redirected to the online page signifying the end of the online survey. Upon

completing the screening portion of the survey, the participants were asked to recall a time when they had wronged somebody. The participants were then asked to answer survey questions regarding the measure of victim forgiveness, divine forgiveness, perceived stress, and physical health. The measures were randomly presented, and the survey was conducted anonymously. Participants were then able to receive credit for one Psychology Activity for participating in the survey.

Measures

Divine Forgiveness

The Divine Forgiveness Scale (DFS) was recently developed as part of a previously conducted undergraduate study by Dr. Jichan Kim and his research team. The DFS scale was developed to measure perceived divine forgiveness in the context of the specific transgression committed by the participants. Many of the studies concerning divine forgiveness had used single-item measures (Ingersoll-Dayton et al., 2010), and given the subtle differences between explicitly receiving forgiveness and perceiving that one has been forgiven, the team felt it necessary to develop a measure that might be more specific for divine forgiveness. The DFS contains 7 items; participants were asked to respond to each item in the specific context of their wrongdoing. The seven items were as follows: “1. I feel that I am forgiven by God for what I have done,” “2. I have a sense of peace in the relationship between me and God,” “3. I believe God is compassionate even though I have done wrong,” “4. I do not feel that God still loves me after what I have done,” “5. I feel abandoned by God for what I have done,” “6. God is merciful to me despite my wrongdoing,” and “7. I feel reconciled to God even after what I have done.” Participants indicated their level of agreement with each item on a scale of 1 (strongly disagree) to 7 (strongly agree) where there were two reverse items (Item #s 4 and 5). The total possible

scores (after reversing two items) ranged from 7 to 49 where higher scores indicated higher perceived victim forgiveness. In the same study that the scale was developed for, the internal consistency of the scale was analyzed, and results showed a Cronbach's alpha of .92, which indicated a high internal consistency. Furthermore, the results of the graduate study showed that divine forgiveness's "relationship with self-forgiveness, victim forgiveness, anxiety, and depression presents evidence for its construct validity (criterion validity)" (Kim et al, 2020).

Victim Forgiveness

The Victim Forgiveness Scale (VFS) was developed to measure perceived victim forgiveness in the context of the specific transgression committed by the participants. The scale was developed following the same procedure and rationale of creating the 7-item DFS. The VFS scale contains 7 items. Participants indicated their level of agreement with each item on a scale of 1 (strongly disagree) to 7 (strongly agree) where there were three reverse items. The seven items were as follows: "1. I feel that I am forgiven by the person I have wronged," "2. I have a sense of peace in the relationship between me and the person I have wronged, "3. The person I have wronged has shown compassion towards me," "4. The person I have wronged harbors bitterness towards me because of what I have done," "5. The person I have wronged recognizes my inherent worth despite what I have done, "6. The person I have wronged is unkind to me because of what I have done," and "7. I feel I have not been granted forgiveness by the person I have wronged." The total possible scores (after reverse scoring reverse items: #4, #6, and #7) ranged from 7 to 49 where higher scores indicate higher perceived victim forgiveness. Cronbach's alpha for the VFS scale was also high at an alpha of .92., suggesting high internal consistency. Similar to the DFS scale, the measure's "relationship with self-forgiveness, divine

forgiveness, and depression presents evidence for its construct validity (criterion validity)" (Kim et al., 2020).

Stress

Stress was operationalized by the Perceived Stress Scale. This measure consists of 14 items designed to "measure the degree to which situations in one's life are appraised as stressful" (Cohen, Kamarck, & Mermelstein, 1983). Participants indicated "how often they have found their lives unpredictable, uncontrollable, and overloaded in the last month" from a scale of 0 (never) to 4 (very often) (Cohen et al., 1983). Some of the items included were: "In the last month, how often have you been upset because of something that happened unexpectedly?" and "In the last month, how often have you felt nervous and "stressed"?" (Cohen et al., 1983). The scale was reported to have a Cronbach's alpha of .78, indicative of a good internal consistency (Cohen et al., 1983).

Physical Health

Physical Health was operationalized by the Cohen-Hoberman Inventory of Physical Symptoms (CHIPS). This measure consists of 33 items regarding physical symptoms. Participants indicated how much the listed symptoms had bothered them in the past two weeks from a scale of 0 (they have not been bothered by the problem) to 4 (the problem has been an extreme bother) (Cohen & Hoberman, 1983). Some of the symptoms listed were "constant fatigue", "headache", "cold or cough" etc. The scale was designed in the context of recent stressful life events and how these symptoms may relate to psychological effects (Cohen & Hoberman, 1983). The scale was reported to have a Cronbach's alpha of .88, which is indicative of a good internal consistency (Cohen & Hoberman, 1983).

Results

Eighty responses were received; however, 18 were removed for incompletion and 3 more were excluded from analysis due to missing answers, leaving a total n = 59 participants (Mean age = 20.00, SD = 1.63). Analysis of the demographic survey revealed that 83.1% of participants identified as female and 84.7% identified as white/Caucasian, implying a level of bias in who this data may represent. Furthermore, all of the participants identified as Christians of some denomination. The internal consistency of each measure was also analyzed. DFS (M = 44.86, SD = 4.99) had a Cronbach's alpha of .79; VFS (M = 35.53, SD = 2.56) had a Cronbach's of .94; PSS (M = 22.51, SD = 5.91) had a Cronbach's alpha of .83; CHIPS (M = 19.98, SD = 23.11) had a Cronbach's alpha of .96. The results of the reliability analysis provide evidence that the measures used all had acceptable to high degrees of internal consistency, that is that items used for each scale all measured the same construct. Even DFS, which had a lower alpha than what was seen in previous uses of the scale, still had an alpha value greater than .7, which is a traditionally used standard for Cronbach's alpha (Taber, 2018).

Correlation analyses were conducted to examine the relationships between each variable (Table 1). Only 3 correlations were statistically significant at a .05 level, and the effect sizes of the correlations were generally small to moderate. Results showed a significant negative correlation between VFS and CHIPS ($r(57) = -.35$, $p = .007$), which had moderate effect size. DFS and PSS showed a significant negative correlation as well ($r(57) = -.28$, $p = .029$) which indicates a smaller effect size. Results also showed a significant positive correlation between PSS and CHIPS ($r = .47$, $p < .001$), which had a moderate to large effect size.

Table 1

Descriptive statistics and correlations of all study variables

Variables	1	2	3
1. SF	1	-	-
2. DF	.33**	1	-
3. VF	.36**	.34**	1
Range	64-175	7-49	10-49
Mean	132.21	43.40	38.06
SD	21.71	7.92	9.66
Alpha	.94	.92	.92

Note. SF, DF, and VF connote self-forgiveness, divine forgiveness, and victim forgiveness.
*p<.05 **p<.01

Regression

Regression analyses were conducted to test H1 and H2 – whether VFS and DFS predict CHIPS to some degree (Table 2). First, two simple linear regression analyses (Model 1 and Model 2 in Table 2) were run to establish predictive power between the variables. The results of these two simple regressions reflected the results of the correlational analyses, indicating that VFS significantly predicted CHIPS (R^2 = .12, F (1,57) = 7.93, p < .01), whereas DFS did not significantly predict CHIPS (R^2 = .02, F (1,57) = 1.03, *ns*) A multiple regression analysis was then conducted with both DFS and VFS as predictors (Model 3); the results showed that DFS and VFS together predicted a small, but significant amount of the variance in the CHIPS scores (R^2 $_{\text{Adjusted}}$ = .09, F (2,56) = 4.02, p = .023), and that of the two forgiveness variables, VFS alone significantly predicted CHIPS (β = -.34, p = .011), whereas DFS did not (β = -.06, p = .645). To test the predictive power of all three variables as well as the relationship between PSS and

CHIPS, a second multiple regression analysis was conducted that included PSS as an independent variable (Model 4). The analysis showed PSS had a significant contribution in being able to predict the variances of CHIPS ($\beta = .48$, $p < .001$). In this analysis, VFS also had a significant effect, while DFS did not. Ultimately, this model which considers DFS, VFS, and PSS together as predictors, had a significant effect on CHIPS ($R^2 = .33$, F (3,55) = 8.85, $p < .001$). Further hierarchical regression analysis showed that when DFS was added to a regression model consisting of PSS and VFS, DFS did not contribute any significant change in the R^2 (R^2 change = .005, Sig. F change = .537), further corroborating the previous regression analyses in which DFS did not significantly predict CHIPS.

Table 2

Summary of Regression Analyses for Variables Predicting CHIPS as the Criterion Variable

	Model 1[a]			Model 2[b]			Model 3[c]			Model 4[d]		
Variable	B	SE	*β*	B	SE	*β*	B	SE	*β*	B	SE	B
VFS	-.64	.23	-.35**	–	–	–	-.62	.24	-.34*	-.62	.21	-.34**
DFS				-.62	.61	-.133	-.28	.59	-.06	.34	.55	.07
PSS										1.83	.45	.48**
R^2		.12**			.02			.13			.33**	
F		7.93			1.03			4.02			8.85	

a. Predictors: VFS
b. Predictors: DFS
c. Predictors: VFS, DFS
d. Dependent VFS, DFS, PSS
*p < .05. **p < .01.

Mediation

Mediation analysis was conducted using the Hayes PROCESS macro tool, a computational sequence used in SPSS, to test H3: stress mediates the relationship of either of the forgiveness variables and CHIPS (Table 3). Looking first at DFS, the total effect, which considers both the direct and indirect (mediated) effect, was found to be nonsignificant (total effect: -.27, 95% bootstrap CI [-1.46 to .91]). The direct effect, which is the effect of DFS on CHIPS controlling for PSS, was also shown to be nonsignificant (direct effect: .34, 95% bootstrap CI [-.76 to 1.44]. The indirect effect, which is the effect of DFS on CHIPS via PSS, was found to be significant (indirect effect: -.27, 95% bootstrap CI [-1.46 to .91]), indicating that mediation has occurred. In contrast, the mediation analysis for VFS revealed the exact opposite results and that PSS did not mediate the relationship between VFS and CHIPS. Both the total effect (total effect: -.62, 95% bootstrap CI [-1.09 to -.15]) and the direct effect (direct effect: -.27, 95% bootstrap CI [-1.04 to .20]) were significant, but the indirect effect between DFS and CHIPS was nonsignificant (indirect effect: -.27, 95% bootstrap CI [-1.46 to .91]. A summary of the mediation analyses are illustrated in Figure 2 and Figure 3.

In summary, the results of analyses seem to indicate the following: 1) VFS has a significant negative relationship with CHIPS, supporting our hypothesis that victim forgiveness positively predicts physical health, as CHIPS is a negative measure of physical health. 2) DFS does not have a significant relationship with CHIPS, and that our analyses did not support our second hypothesis that divine forgiveness positively predicts physical health. 3) There is a significant indirect effect between DFS and CHIPS, providing evidence that PSS does act as a mediator. Furthermore, there was no significant total nor direct effect between DFS and CHIPS, implying that divine forgiveness primarily affects physical health through mediators such as

stress. 4) Regarding VFS and CHIPS, there was no significant indirect effect, implying that PSS did not mediate the relationship between the two. However, since a mechanism is required to explain how victim forgiveness could affect one's health, it can be inferred that other factors that were not accounted for in the model also mediate the relationship between victim forgiveness and physical health.

Table 3

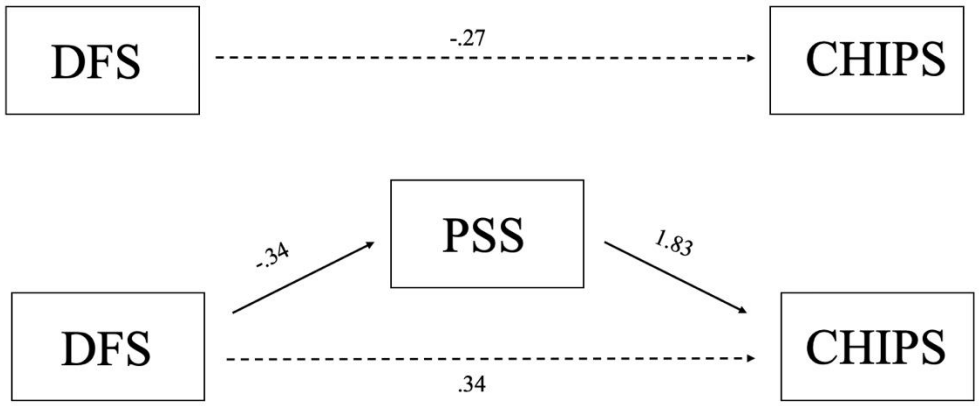
Total, Direct, and Indirect Effects of the two Different Mediation Models

Hayes PROCESS Mediation (n = 59)				
	Mediation Models			
	VFS → PSS → CHIPS		DFS → PSS → CHIPS	
		95% CI [lower, upper]		95% CI [lower-upper]
Total Effect	-.62*	-1.09, -.15	-.27	-1.46 .91
Direct Effect	-.62*	-1.04, -.20	.34	-.76, 1.44
Indirect Effect	.00	-.19, .23	-.61*	-1.24, -.16

Note. A confidence interval range that does not include zero implies significance at a .05 level.
*p < .05

Figure 2.

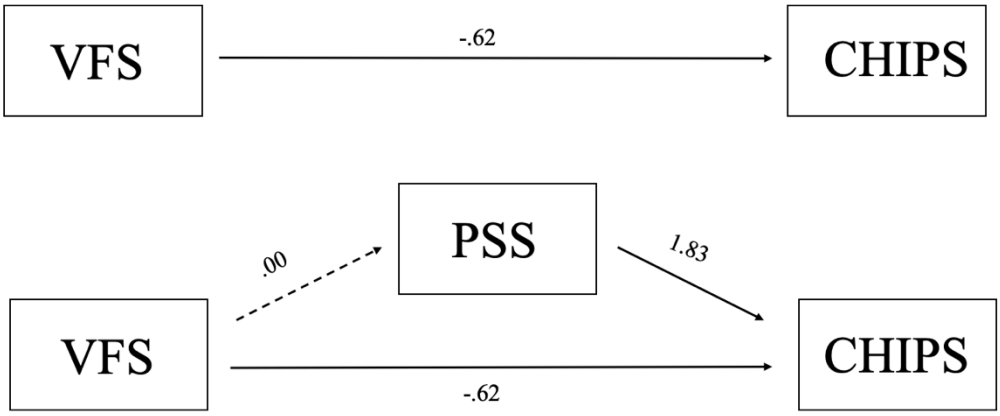
Standardized Regression Coefficients for the Relationship between Divine Forgiveness and Physical Health as Mediated by Perceived Stress



Note. dashed lines indicate nonsignificance

Figure 3.

Standardized Regression Coefficients for the Relationship between Victim Forgiveness and Physical Health as Mediated by Perceived Stress



Note. dashed lines indicate nonsignificance

Discussion

In this study, one of our aims was to answer whether either victim or divine forgiveness predicted physical health, hypothesizing that both forgiveness variables would have a positive correlation with physical health (H1 and H2). We further hypothesized that stress would mediate the relationship between victim forgiveness and physical health (H3a) and divine forgiveness and health (H3b). Both correlational and regression analyses revealed stress to have a negative relationship with physical health and that stress can predict physical health with moderate strength. These results were corroborated by the extensive literature regarding stress and health.

Both the correlational and regression analyses indicate that VFS had a significant positive correlation with physical health, thus supporting H1. Specifically, the correlation results for VFS showed a moderate negative relationship with CHIPS, while the linear regression with VFS as the sole predictor indicated a small negative correlation; both effects were statistically significant. Currently, no studies have directly examined the transgressor's perception of receiving forgiveness and its relationship with said transgressor's overall physical health, therefore this study may be the first of its kind to do so. The studies that have looked at victim forgiveness have examined its relationship with negative emotions and stress, upon which we based our third hypothesis (H3a) that stress mediates the relationship between victim forgiveness and physical health (da Silva, vanOyen Witvliet, & Riek, 2017; Dolezal & Lyons, 2017). However, the mediation analyses showed no significant indirect effect between VFS and CHIPS via PSS, therefore failing to support H3a. In fact, the correlational analysis revealed that VFS and PSS were not significantly correlated. This result was somewhat expected, considering other studies that have looked at receiving forgiveness and physiological measures of stress also

showed no significant effect (vanOyen-Witvliet et al., 2002). The mediation analysis did indicate a significant total and direct effect between victim forgiveness and physical health, and if stress does not mediate that relationship, then there must be another mediation variable that can help explain the mechanism by which victim forgiveness affects physical health. One alternate explanation could be a behavioral mechanism. Some studies have shown that negative emotional states, such as shame and un-self-forgiveness, which are inversely correlated with victim forgiveness, may be associated with unhealthy behaviors such as overeating (Swan & Andrews, 2003), addiction, and alcoholism. These behaviors may help to reduce feelings of stress, but will eventually begin to harm one's health.

The results of the analyses regarding DFS were opposite that of VFS. Both correlational and regression analysis showed no significant relationship between divine forgiveness and health. However, consistent with divine forgiveness literature (Ingersoll-Dayton et al., 2010), there was a significant negative correlation between DFS and PSS, implying that as divine forgiveness increases, perceived stress decreases. Nevertheless, H2 was not supported. The number of studies that examine divine forgiveness and physical health are still relatively scarce, though the general results of these studies seem to find a small correlation between divine forgiveness and physical health (Chen et al., 2019; Long et al., 2020). The small sample size is a limitation that may reconcile the results of this study with the results in previous studies. The mediation analysis did reveal a significant indirect effect in which DFS affects CHIPS via PSS, therefore supporting H3b. Furthermore, the mediation analysis showed no significant total or direct effect, which could imply that the effect of divine forgiveness on physical health is primarily mediated through variables such as stress. Another explanation for the lack of total effect is the presence of a mediating variable that negatively affects physical health. For

example, one study found that self-forgiveness can actually promote chronic unhealthy behavior by removing the negative emotions that are associated with that behavior (Wohl & Thompson, 2011). Granted, some would argue that a lack of taking responsibility would connotate pseudo-self-forgiveness rather than genuine self-forgiveness (Hall & Fincham, 2005). Nevertheless, if such an effect was found for divine forgiveness, then it could counteract the positive effect that divine forgiveness would have on physical health via stress, thus rendering the total effect null. Such an effect for divine forgiveness has yet to be analyzed; and therefore this alternative explanation is mostly conjecture, though it does highlight a greater need to more thoroughly research divine forgiveness as a whole.

Limitations and Future Directions

One of the key limitations to this study was the small sample size (n = 59) which would have surely affected the correlation and regression results. Previous studies have suggested that the effect size between the variables would be small; therefore, a more substantial sample size would be required to more accurately detect it. The fact that convenience sampling was used is also a limitation that may hinder the generalizability of the results, especially in light of the homogeneity of the participant demographics. Further studies should strive to diversify the sample according to age, ethnicity, and religious beliefs as well. It may also be beneficial to purposefully select a sample for those who have committed transgressions they deem as more serious; doing so might amplify the effect size that would normally be small in a regular random sample. The use of self-report measures is another likely limitation. Future studies should use other methods of operationalizing the variables, for example, using physiological measures such as cortisol serum levels as an indicator of stress, or physical check-ups to determine physical

health. While using these measures would certainly be more resource-intensive, it may produce a more accurate picture of the effects that we're attempting to discern.

Conclusion

The findings in this study, while mixed, lay the groundwork for future research that could potentially lead to the development of clinical applications. Forgiveness intervention studies conducted with patients who were victims of some transgressions have shown promising results in being able to reduce the severity of certain symptoms (Lee & Enright, 2014; Tibbits, Ellis, Piramelli, Luskin, & Lukman, 2006; Waltman et al., 2009). Therefore, the findings of this study may encourage the development of interventions that focus on healing for the transgressor. Of course, more research is required before the clinical use phase can be reached, and this study serves to further reveal the gap in the current literature regarding divine and victim forgiveness as a whole.

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