Concept Paper

The "Double-Edge Sword" of Human Empathy: A Unifying Neurobehavioral Theory of Compassion Stress Injury

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Abstract: An integrative neurobehavioral model for "compassion stress injury" is offered to explain the "double-edge sword" of empathy and inherent vulnerability of helping professionals and care-givers. One of the most strikingly robust, yet largely invisible scientific findings to emerge over the past decade is identifying the neurophysiological mechanisms enabling human beings to understand and feel what another is feeling. The compelling convergence of evidence from multi-disciplinary lines of primary research and studies of paired-deficits has revealed that the phenomenon of human beings witnessing the pain and suffering of others is clearly associated with activation of neural structures used during first-hand experience. Moreover, it is now evident that a large part of the neural activation shared between self- and other-related experiences occurs automatically, outside the observer's conscious awareness or control. However, it is also well established that full blown human empathic capacity and altruistic behavior is regulated by neural pathways responsible for flexible consciously controlled actions of the observer. We review the history, prevalence, and etiological models of "compassion stress injury" such as burnout, secondary traumatic stress, vicarious traumatization, compassion fatigue, and empathic distress fatigue, along with implications of the neurobehavioral approach in future research.

Keywords: compassion fatigue; empathy; stress injury; burnout; vicarious trauma; neuroscience

1. Introduction

On a clear summer night in 2006, an ambulance is urgently dispatched to the family residence of doctor "R", a well-respected senior military psychologist and the only credentialed mental health provider for a U.S. Marine base of 6000. Initial reports by Dr. R's frantic spouse indicated sudden paralysis, inability to speak, and unresponsiveness, suggesting possible seizure or stroke. Emergency personnel are greeted by a panic-stricken wife and two crying children. They enter the home to find the high-ranking patient sitting upright on a bed, conscious and breathing on his own, but immobilized, non-communicative, and with fixed gaze.

As Dr. R.'s listless body is loaded onto a gurney, medical staff learns of his gradually worsening insomnia, night sweats, and weight loss in the context of 14-hour days, 6–7 days a week, and excessive work demands. A normally upbeat, fun-loving, and energetic personality has steadily given way to a serious, easily frustrated, fatigued, preoccupied, and socially withdrawn persona. Dr. R. frequently expressed consternation, guilt, and moral outrage over his and the military's inability to provide adequate treatment to the high volume of traumatized war veterans. Mrs. R exclaims, "My husband cares too much…it's eating him alive, and now maybe it's even killing him!" [1].

Within two-days, the naval officer was walking and talking, and back to full-duty. Extensive medical and neurological examination revealed no identifiable organic cause, resulting in a vague, nondescript diagnosis of "Idiopathic seizure", a medically unexplained physical condition often associated with a spectrum of stress-related injuries [2]. Absent secondary gain and prior psychiatric or epileptic history, Dr. R. concurred with his wife's initial assessment-but how exactly can "caring too much" or empathy be a double-edge sword for helpers and caregivers?

2. Purpose of the Study

On the face of it, contemporary euphemisms like "compassion fatigue" (CF) and kindred terminology (e.g., "vicarious traumatization") imply the causation of helper distress is the act of caring itself and exposure to another's pain; however, to date there is no mechanism explaining how. This study aims to: (1) introduce the concept of compassion stress injury (CSI) and explore its relationship with other stress-related conditions; (2) review the universality of compassion stress across history and disciplines; (3) examine current etiological models of CSI; (4) review developmental, social psychological; and neuroscientific studies related to empathy; and (5) propose a unifying neurobehavioral theory of CSI.

3. The Phenomenology of Compassion Stress Injury

Returning to Dr. R., it appears woefully understated to explain his collapse as mere compassion "fatigue". To be awake and alert, but unable to speak, respond, or move one's own body communicates more than just being tired and needing a respite. In every sense of the word, the paralysis was experienced as genuine and very "real". Try as he did, Dr. R. could not reply to his wife's frantic questioning, or the queries from the perplexed medical attendants who had invaded his bedroom. He could not even raise a finger, until whatever had caused this frozen immobilized state had eventually passed.

Why compassion stress injury? In the wake of the Persian Gulf War (1990–1991), the American government commissioned several national scientific investigations of the immediate and long-term health effects from stress. After an exhaustive review, the Institute of Medicine (IOM) [3] concluded:

> "In the brain, there is evidence of structural and functional changes resulting directly from chronic or severe stress. The changes are associated with alterations of the most profound functions of the brain: memory and decision-making" ([3], p. 60) and "profound effects on multiple organ systems…the continuation of altered physiologic states over months and years contribute to the accumulation of adverse long-term health consequences." ([3], p. 66).

Linkage between stress and health is nothing new. In fact, Walter B. Cannon, an American Army psychiatrist of the First World War (WWI: 1914–1918) who set out to explain the neurobiological effects of extreme stress, coined the phrase "fight or flight" [4]. One of Cannon's lines of inquiry was the phenomenon known as "voodoo death" found in "primitive" cultures [4,5] also known as "death by fright" in "modern" societies [6]. Similar post-Second World War (WWII: 1939–1945) research identified universal stages of General Adaptation Syndrome consisting of alarm, resistance, and eventual exhaustion leading to innumerable "diseases of adaptation" and possible death [7]. Taken together, if stress and emotions like fear cause permanent structural and functional changes to the mind-brain-body, including sudden death in otherwise medically fit men, women, and children [5,8], the explanatory concept of "stress injury" appears infinitely better-suited to explain Dr. R's predicament.

Overview of CSI. Etiological explanations for stress injuries whether caused by combat, non-war-related trauma, or taking care of traumatized human beings, consists of identifying a host of risk/vulnerability and resilience/protective factors. However, the common pathogenic thread for the universality of human vulnerability is the cumulative "dosage" effects of first-hand and secondary exposure to chronic inescapable stress and/or potentially traumatic stress. Documented occupational hazards for helpers bearing witness to intense human suffering has given birth to labels such as "professional burnout (PB)", "secondary traumatic stress (STS)", "vicarious traumatization (VT)" "CF" and "empathic distress fatigue (EDF)", reflecting a possible spectrum of CSI. Below we define pertinent terminology and examine historical roots.

Compassion satisfaction. Is described by Stamm as the capability of the helper to gain a sense of meaning and purpose from their work [9], similar to Figley's "sense of achievement" [10], which can help counteract the physiological, emotional, and philosophical effects of compassion stress [11]. These concepts are roughly equivalent to constructs of "resilience" and "post-traumatic growth" [12] applied to traumatic stress, and buttressed by preventative self-care.

Compassion stress. The term compassion is derived from the Latin *pati*, meaning "to suffer", and *cum*, meaning "with"—translated literally as "to suffer with", and fully-defined as "sympathetic consciousness of others' distress together with a desire to alleviate it". When combined with "stress"—"a physical, chemical, or emotional factor that causes bodily or mental tension and may be a factor in disease causation", and "injury"—"an act that damages or hurts" [13], we have the makings of CSI. Figley defines compassion stress as "the residue of emotional energy from the empathic response to the client and is the on-going demand for action to relieve the suffering of a client" ([10], p. 1437). Later, we expand this definition.

4. Scope and Universality of CSI

Research has shown that CSI is quite common, and has shown to affect 40% to 80% of resident physicians In addition, CSI is also seen across a broad swath of helping populations: self-identified trauma therapists [14,15]; police officers [16]; humanitarian aid workers [17]; audiologists [18]; early childhood special education teachers [19]; military psychologists [20]; military chaplains [21]; civilian clergy [22]; child welfare workers [23]; emergency department nurses [24]; pediatric intensive care providers [25]; Veterans Administration counselors [26]; hospice care professionals [27]; children of war veterans [28]; and family care-givers [29].

4.1. Historical Roots of Compassion Stress Injury

Early cautionary statements to those in the healing arts are evident in the ancient biblical proverb "*Physician Heal Thyself*" (Luke 4:23 King James Version), but by the turn of the 20th century, the potentially adverse effects of helping traumatized soldiers became evident.

The First World War (WWI: 1914–1918)—Shell shock by proxy. The term "shell shocked by proxy" was introduced in Pat Barker's acclaimed novel *Regeneration* depicting true-life exploits of the legendary British WWI Army social anthropologist, neurologist, and psychiatrist William H. R. Rivers at Craiglockhart War Hospital [30]. According to Nickerson and Shea [31]:

> We learn from Rivers the capacity of patients to teach us, and when need be, to heal us. The act of listening to individuals is fundamental, and it is not passive. It implies a willingness to be changed by what we hear. Otherwise one no longer listened attentively enough to the individual voice. There was a real danger, he thought, that in the end the stories would become one story, the voices blend into a single cry of pain. ([31], p. 4).

> While at Craiglockhart, Dr. Rivers began to mimic the symptoms of his shell shocked patients such as severe speech impediment, disrupted sleep, excessive fatigue, irregular heartbeat, tremors, and disturbed gait leading to the diagnosis of a CSI and mandatory respite [30].

Second World War (WWII: 1939–1945)—Old sergeant's syndrome and burnt out. Traces of CSI during WWII is evident in the terms "old sergeant's syndrome" and "burnt out" coined by U.S. Army psychiatrist Raymond Sobel, referring to large numbers of battle hardened, highly decorated proven enlisted leaders considered the backbone of the infantry who succumbed to diverse constellations of psychiatric, somatic, and behavioral symptoms such as: abnormal tremulousness, profuse sweating, excessive fear, speech deficit, severe fatigue, sleep disturbance, guilt, depression, and an uncharacteristic tendency to be the first to get in and the last to leave a foxhole ([32], p. 1238).

These "old sergeants" were deeply concerned and committed to the welfare of their men, often serving as surrogate caregivers, while at the same time having to make impossible decisions leading to the deaths of those they cared for. In a similar vein, concerns of "old psychiatrist syndrome" appear evident as U.S. Army psychiatrist Albert J. Glass warned [33]: "the psychiatrist himself is likely to become weary and emotionally exhausted…each case a decision that may mean life or death for the soldier must be reached…he may identify himself with his patients and see them as all equally deserving of evacuation; or...adopt a harsh policy, assume a severe and caustic manner ([33], p. 61)…When the psychiatrist

observes himself becoming angry with his patients or figuratively crying with them, it is a good rule to stop work and see no patients for an hour or two" ([33], p. 62).

Vietnam War (1965–1973): Secondary Traumatic Stress (STS). In 1978, American Vietnam veteran, psychologist Charles R. Figley expressed concern for family members and helping professionals of returning war veterans coining the term "secondary catastrophic stress reactions" [34], which he later reformulated as "secondary traumatic stress" (STS), "The natural consequent behaviors and emotions resulting from knowing about a traumatizing event experienced by a significant other—the stress resulting from helping or wanting to help a traumatized or suffering person" ([35], p. 7). Figley's STS concept is distinguished from other types of CSI by suggesting helpers and family members can actually acquire symptoms of Post-Traumatic Stress Disorder (PTSD) from another person; however, he carefully noted secondary exposure which always mimics symptoms of the distressed other [35].

Figley cited cultural precedents like fathers exhibiting pregnancy symptoms and folie a deux or "shared psychotic disorder" [36], whereby a spouse acquires the psychiatric symptoms of the other, and anecdotal descriptors of STS like "co-victimization" [37], "secondary survivor" [38], "rape-related family crisis" [39], and "proximity effects" of female partners of veterans [36,40]. Of interest, however, is the absence of a clear explanatory mechanism for these diverse phenomena.

Inter-generational transmission. Figley's STS concept has been extended specifically to previously non-traumatized children acquiring characteristic trauma-like responses from a parent or caregiver [28,35].

Trans-generational trauma. Danieli's (1985) research on "*generational trauma*" within families of Jewish holocaust survivors reveals the potential for unresolved STS to cross-generational boundaries [41].

4.2. Non War-Related Conceptualizations of Compassion Stress Injury

Professional Burnout (PB). There is no consistent definition for the term *burnout* first introduced by Sobel [32], and later expanded by Freudenberger who is credited for writing the first published paper on generalized job "burnout" not specific to helpers [42]. Maslach provided a contemporary definition of PB when she noted an insidious syndrome "of emotional exhaustion, depersonalization, and reduced accomplishment" ([43], p. 3). PB is associated with conflict between work and family domains, role ambiguity, excessive workload, perception of low rewards, and lack of administrative support [44]. Within helping professions, PB is considered to be prevalent due to the deeply personal nature of the work and the types of clients served [45].

Traumatic Countertransference. Freud described countertransference as arising "in the physician as a result of the patient's influence on his unconscious feelings, and have nearly come to the point of requiring the physician to recognize and overcome this countertransference in himself" ([46], p. 19). Whereas Freudian countertransference applied across the board, Herman coined the phrase "traumatic counter-transference" to describe the inevitable risk of trauma-focused therapists listening to narratives that are "bound to revive any personal traumatic experiences" of the therapist ([47], p. 140). As a result, the therapist struggles with similar difficulties as the client, be that a crisis of faith, a sense of personal vulnerability, a fear of intimacy, or feelings of helplessness. The empathic involvement of the therapist with the client leaves the therapist vulnerable, and "trauma is contagious" ([47], p. 140).

Vicarious Traumatization (VT). McCann and Pearlman defined VT as "the transformation in the inner experience of the therapist that comes about as a result of empathetic engagement with clients' trauma

material" ([48], p. 145). As Harrison and Westwood noted "Clinicians must listen to graphically detailed descriptions of horrific events and bear witness to the psychological and physical aftermath of intense cruelty or violence" ([49], p. 203). Consequently, helpers may be disturbed by persistent imagery or intrusive thoughts of their clients' involvement in traumatic events. Such imagery can remain long after client contact [50].

Compassion Fatigue (*CF*). In Carla Joinson's 1992 study on burnout in emergency department nurses, she coined the term CF defined as a form of burnout that "affects people in caregiving professions" ([51], p. 116). Figley adopted the term in his seminal text *Compassion Fatigue: Coping with Secondary Traumatic Stress Disorder in Those Who Treat the Traumatized*, and elaborated CF as "a state of tension and preoccupation with the traumatized patients by re-experiencing the traumatic events, avoidance/numbing of reminders persistent arousal (e.g., anxiety) associated with the patient. It is a function of bearing witness to the suffering of others" ([36], p. 1435).

Why fatigue? According to Joinson, the underlying reason for introducing the phrase "compassion fatigue" to her nursing colleagues was to garner general acceptance and constructive solutions to a serious and widespread problem [51]. It was Joinson's belief that existing terminology like VT or STS would solicit automatic rejection due to stigma and fears that one's peers would prejudge them as weak and unable to cope with professional demands [51]. Her decision appears justified given pervasive mental health stigma in the 21st century. Moreover, there is a national precedent for using "fatigue" to describe stress casualties.

In 1943, the U.S. Army adopted the terms "battle fatigue" and "combat exhaustion" in place of psychiatric nomenclature in order to de-pathologize universal human responses to extreme stress, as well as (erroneously) convey the transient nature of nervous breakdowns after brief respite [52]. The military's use of "fatigue/exhaustion" had inadvertent effects. It minimized individual suffering, and generated a sense of shame and guilt in the afflicted who didn't recover quickly after respite-in that failure to recover shortly after breakdown was perceived as evidence of predisposed vulnerability, personality weakness, or malingering [52].

5. Differentiation between Compassion Stress Injuries

Harrison and Westwood contrasted countertransference with VT in that the former is often experienced as short-term within-session intrusions of the clinician's own concerns triggered by a particular client's material, whereas VT occurs cumulatively via repeated sharing of traumatic experiences and persists outside of the helping setting [49]. Additionally, STS involves the direct mimicking of another's symptoms, which can emerge and resolve quickly, whereas the onset of diffuse symptoms and behavioral indicators of PB, CF, and VT tend to be insidious and chronic [50]. In PB, the source of distress is primarily dissatisfaction in the work environment; in contrast to dosage effects from exposure to traumatic stimuli (e.g., STS, VT, CF; [49]).

Empathic Distress Fatigue (EDF). Klimecki and Singer described CF as a form of "pathological altruism" defined as "the willingness of an individual to place the needs of others above him-or herself to the point of causing harm" ([53], p. 369). The authors argued that EDF be used instead of CF because empathic distress due to perceiving others' suffering *versus* compassion per se, causes CF and possible burnout [53]. According to Klimecki and Singer, empathy results in two different empathic reactions:

(1) compassion, empathic concern, and sympathy (e.g., other-related emotion, loving kindness, good health, and positive motivation) [53]; or (2) empathic/personal distress (e.g., self-related emotion, negative feelings, poor health, burnout, and withdrawal)-each reaction corresponding to separate, but related neural pathways [53].

5.1. Limitations of Current Classifications of Compassion Stress Injury

There is considerable overlap of symptoms and behavioral indicators between the various CSI constructs (Appendix A), with each emphasizing the central role of empathy and second-hand exposure to another's suffering. However, notable limitations include: (a) confusion of overlapping constructs; (b) minimizing the extent of suffering with euphemisms of "fatigue" for injuries resulting in structural changes in the brain and multiple organ systems that can lead to significant health impairment, premature death, and even suicide; and (c) absence of a coherent empirically-driven explanation of CSI.

5.2. Linking Compassion Stress with Empirically-Based Stress-Related Conditions

The diverse range of symptoms and signs associated with compassion stress (Appendix A) are nearly identical to those in Table 1 below attributed to established constructs of "acute stress reactions" (ASR) and "combat/operational stress reactions" (COSR) [54] reflecting sympathetic stress activation. Moreover, descriptions of cumulative "dosage" effects buffered by "compassion satisfaction" and lists of risk/vulnerability and protective/resilient factors in CSI (Appendix B), mirror empirically-grounded findings in etiology and resilience of post-traumatic stress injuries [54].

Table 1. Human stress response to acute stress.

Physical	Cognitive/Mental	Emotional	Behavioral
• Chills	• Blaming someone	• Agitation	• Increased alcohol consumption
• Difficulty breathing	• Change in alertness	• Anxiety	
• Dizziness	• Confusion	• Apprehension	• Antisocial acts
• Elevated blood pressure	• Hyper-vigilance	• Denial	• Change in activity
• Fainting	• Increased or decreased awareness of surroundings	• Depression	• Change in communication
• Fatigue		• Emotional shock	• Change in sexual functioning
• Grinding teeth	• Intrusive images	• Fear	
• Headaches	• Memory problems	• Feeling overwhelmed	• Change in speech pattern
• Muscle tremors	• Nightmares	• Grief	• Emotional outbursts
• Nausea	• Poor abstract thinking	• Guilt	• Inability to rest
• Pain	• Poor attention	• Inappropriate emotional response	• Change in appetite
• Profuse sweating	• Poor concentration		• Pacing
• Rapid heart rate	• Poor decision-making	• Irritability	• Startle reflex intensified
• Twitches	• Poor problem solving	• Loss of emotional control	• Suspiciousness
• Weakness			• Social withdrawal

Source: Adapted from ([54], p. 25). Spiritual or moral symptoms are also often present including feelings of despair, excessive guilt, questioning religious or spiritual beliefs, existential crisis, rejection of spiritual practice and community, and a sense of nihilism [2].

5.3. Comparing CSI and ASR/COSR and Traumatic Stress Injuries

A critical difference between CSI and traumatic stress injury concepts is the second-hand exposure in CSI to threat stimuli from bearing witness to another's emotional and physical suffering, combined with other internal and external demands the helper encounters. For example, helpers may experience empathic stress from their desire to help alleviate client suffering, internalized threats triggered by the helper's own past trauma history, and external stressors from workload demands-either pathway activates sympathetic stress response system-but how is empathy itself a potential threat?

5.4. Etiological Considerations of Compassion Stress Injuries

The superordinate etiologic factor posited across all CSI types is empathic attunement to the suffering of others. However, only a few cogent theories have been articulated to explain the actual mechanism(s) by which empathic resonance to another's pain can be risky, and even injurious. We identified four models: (a) Figley's Ten Factor Causal Model; (b) McCann and Pearlman's Cognitive Schema Model; (c) Neurobiological Model of Burnout; and (d) Klimecki and Singer's Empathic Distress Model.

Figley's Ten Factor Causal Model. Figley outlined ten factors that formed a casual model proposed as predictive of compassion fatigue [10]: *empathetic ability* (e.g., therapist aptitude for noticing the pain of others); *empathetic concern* (e.g., therapist drive to respond to those in need); *exposure to the client* (e.g., face-to-face exposure to clients and their material); *empathetic response* (e.g., therapist effort to alleviate pain of others); *compassion stress* (e.g., "the residue of emotional energy from the empathic response to the client and is the on-going demand for action to relieve the suffering of a client", [10], p. 1437); *sense of achievement* (e.g., therapist satisfaction with their work); *disengagement* (e.g., therapist ability to distance her or himself from client suffering); *prolonged exposure* (e.g., therapist's continued sense of responsibility for client well-being); *traumatic recollections* (e.g., intrusive memories that cause an emotional reaction); *life disruption* (e.g., unanticipated changes in daily living). For Figley: "An emotional arousal appears to be associated with an empathic and sympathetic reaction. Sometimes…we become emotionally drained by [caring so much] we are adversely affected by our effort. Indeed, simply being a member of a family and caring deeply about its members makes us emotionally vulnerable to the catastrophes that impact them" ([35], p. 12).

McCann and Pearlman's Cognitive Schema Model. McCann and Pearlman identified seven major cognitive schema alterations in helping professionals exposed to VT which are believed to underlie compassion stress injury [55]: (1) *frame of reference* (e.g., clinicians may question their personal or professional identity); (2) *safety* (heightened sense of fear); (3) *trust/dependency* (viewing significant others with suspicion); (4) *esteem* (criticizing and devaluing the self); (5) *independence* (loss of freedom as a result of feeling vulnerable); (6) *power* (e.g., clinicians may feel overly responsible for their clients' recovery); and (7) *intimacy* (e.g., loss of faith in humanity may lead clinicians to block against feelings of intimacy). Finally, VT may contribute to behaviors that promote emotional numbing including alcohol consumption, overeating, overspending, and overworking [56]. Phelps, Lloyd, Creamer, and Forbes noted the personal vulnerability of the individual therapist's cognitive schemas, as well as the therapist's ability to process the client's traumatic material both serve to determine the likelihood the therapist will experience VT [57].

Empirical support for ten factor and cognitive schema models. We were unable to locate any research that specifically tested either model.

Neurobiological Model of Burnout. Eriksson and Wallin proposed a neurobiological explanation of job burnout as primarily overlapping with mood and somatic symptoms associated with depression [58]. According to Eriksson and Wallin, failure of adult hippocampal neurogenesis provides the biological and cellular basis of altered brain plasticity in burnout, and that burnout is the result of inadequate brain plasticity caused by stress-induced lowering of neurogenesis [58]. According to their theory, the effect of stress is mediated by Hypothalamic-Pituitary-Adrenal (HPA)-axis dysfunction.

Empirical support for neurobiological model of burnout. Overall empirical support is mixed. Recent studies found normal functioning of HPA axis in burnout suggesting possible adaptation process [59]. Supporting this finding, researchers found that chronic stress does not always present with hyperactivity of the HPA axis [60]. van Luijtelaar, Verbraak, van den Bunt, Keijsers, and Arns conducted a study utilizing EEG to determine possible neurobiological markers for burnout in the general population, not specific to helping professions or caregivers [61]. Burnout patients showed reduced P300 amplitude, a lower alpha peak frequency and reduced beta power, which is different than the literature in depression and chronic fatigue patients. Van Luijtelaar *et al.*, concluded that burnout be considered a separate clinical syndrome [61]. However, other researchers reported no distinction between burnout and depression [62]. In addition, Gleichgerrcht and Decety [63] reported that burnout among physicians may be related to dispositional empathy and personal empathic distress associated with perceiving the pain of others, particularly female patients.

Klimecki and Singer's Empathic Distress Model. Klimecki and Singer are the first to begin shedding light on possible neural substrates of CF and PB, by drawing upon the compelling convergence of developmental, social psychological and neuroscientific research on empathy [53]. In a nutshell, the authors stressed the importance of maintaining self-other distinction in order to regulate our empathic reactions, whereby overuse of self-oriented responses result in activation of brain areas responsible for processing threat or pain (e.g., amygdala), thereby causing the helper to experience "empathic distress" from vicariously induced negative emotions and stress which can lead to CF and PB [53]. Conversely, one can prevent CF by cognitively controlling our empathic reactions thru activating neural pathways associated with maintaining a compassionate "other-perspective" that stimulates empathic concern, positive feelings, and altruistic behavior [53].

5.5. Limitations of Etiological Research on Compassion Stress Injury

Prior to Klimecki and Singer's neuroscience-based formulation for CF and PB, the actual mechanisms for explaining the double-edge sword of human empathy have been elusive [53]. However, major limitations exist in Klimecki and Singer's model [53]. Most importantly, the cause of CSI almost exclusively is attributed to poor cognitive (other-oriented) regulation of empathic reactions, thus not accounting for the combined dosage effects from other sources of helper sympathetic stress (e.g., countertransference), the interactive role of known risk/protective factors (e.g., sleep), and the cumulative impact of automatic, uncontrolled components of empathic responses.

6. A Neurobehavioral Model of Compassion Stress Injury

We came to our neurobehavioral perspective by reviewing six diverse, yet converging lines of inquiry: (1) developmental research on imitation, mirror neurons, and emotional contagion; (2) social psychology of mimicry; (3) neuroscience of pain perception and empathy; (4) neurobiology of the human stress response; (5) military research on ASR/COSR; and (6) CSI research. Together, these provide a compelling integrative explanation for the universal vulnerability of humans helping humans.

6.1. Defining Empathy and Compassion

As social animals, human beings possess a remarkable innate capacity to perceive, imagine, feel, understand, and respond to another's emotional state-particularly distress and suffering-often referred to as "empathy". According to Singer and Lamm, the word "empathy" is derived from Greek—from *empatheia* (passion), which is composed of "en" (in) and "pathos" (feeling), that was later introduced in German as *Einfuhlung* ("feeling into" something) [64]. Compassion on the other hand, is often distinguished from empathy, as both a feeling of concern and the motivation to alleviate the suffering of another (e.g., [65]).

6.2. Counseling and Psychotherapy Models of Empathy

The innate human capacity to communicate shared understanding and feelings that lead to prosocial behavior has obvious and profound adaptive benefits for individuals and society. Winnicott observed, "a sign of health in the mind is the ability of one individual to enter imaginatively and accurately into the thoughts and feelings and hopes and fears of another person'' ([66], p. 117), and Rogers explained: "Gradually my understanding of empathy extended to an intuitive capacity…where I would find something rising in myself…I don't really understand the workings of intuition. Do I simply pick up nonverbal cues? I don't think that's sufficient to explain it. Somehow there is a way in which the inner core of me relates to the inner core of the other person, and I understand better than my mind understands…somehow, my non-conscious mind understands more than my conscious mind understands, so I'm able to respond to something in this other person that I didn't know I was responding to" ([67], p. 285).

6.3. Neuroscientific Research on Empathy

Perhaps the most compelling finding over the past decade is identifying the neurophysiological mechanisms that enable human beings to understand, imagine, and feel what another is experiencing. Converging evidence from multi-disciplinary lines of primary research in such diverse fields as social neuroscience, human development, social psychological and pain physiology reveals that observing or imagining the actions, sensations, and emotions of others results in the dual activation of neural pathways involving both automatic (bottom-up) and executive (top-down) control during first-hand witnessing another [63,65,68–70]. Literally, we empathize by actually recreating in ourselves the physiological emotional experience of another [53].

Contemporary neuroscientific models view full-blown empathy as generated by two parallel processes: (a) automatic, non-conscious or "bottom up" matching between perception and action; and (b) conscious, cognitive controlled or "top down" processes that regulate empathic responses and altruistic (helping) behavior [68].

6.4. Evidence of Dual-Processing of Shared Neural Representations

In short, the immediate human empathic response is the result of neurophysiological simulation (reproduction) of another's experience. This initial empathic response occurs automatically by activating mirroring neural circuits involved in imitation, mimicry, and emotional contagion, all outside the observer's conscious awareness or control [68,69,71,72]. However, it is also well-established that full human empathic capacity and prosocial behavior is regulated by "top-down" neural pathways responsible for flexible deliberately controlled actions including self/other awareness, perspective-taking, and emotional reappraisal [53,71,72].

6.5. Automatic Processes: Mirror Neurons, Imitation, Mimicry, and Emotional Contagion

The concept of unconscious automatic simulation was introduced as far back as 1903 by Lipps, positing that, "involuntary, instinctual, kinesthetic imitation of the observed vital activity of another occurs in empathy [73]. When empathy produces this physical mimicry in the spectator, the intentional focus does not remain on the spectator's body but is projected into the other" ([74], p. 93). In 1964, Ax observed that empathy might be thought of, "as an autonomic nervous system state, which tends to simulate that of another person" ([74], p. 93).

Understanding these automatic "bottom-up" empathic and sympathetic responses is crucial for explaining the neurophysiological mechanisms responsible for inherent human vulnerability toward CSI. The relationship between the observer and the distressed other can dictate the reaction and perhaps commitment to help. For example, behavioral neuroimaging studies found judgments of infant pain differed systematically across parents, nurses, and pediatricians [75].

The mirror neuron system. How does our brain link observation of another's emotion, facial expression, or behavior with the automatic action of copying it? In 1996, Giacomo Rizzolatti and colleagues discovered specialized mirror neurons in the monkey prefrontal cortex responded not only when they grasped food, but also when observing another reaching for food [76]. In comparison, the human mirror neuron system (MNS) is viewed by some researchers as uniquely extensive and widely involved [77].

In humans, the MNS has been found to correspond to movements all over the body, and is activated in the actual or imagined presence of others irrespective of goal, whereas the monkey's MNS appears to fire when witnessing a goal-directed action (e.g., another reaching for food) [78]. This may explain the paucity of imitation behaviors reported throughout the animal kingdom, but more importantly, why human beings may be unique in the extensiveness of our empathic capacity and altruistic behaviors, as well as our inherent susceptibility to CSI. Furthermore, human research has shown that observing emotions and bodily sensations appears to active brain regions largely overlapping those occurring during first-hand experience, indicating that empathic responses may involve mirror-like systems [79,80].

That said, the empirically based conclusions of MNS-related research is not without dissenters. For instance, Carmazza and colleagues [81] believe claims that conceptual knowledge can be mediated by sensori-motor reproduction is largely unsubstantiated because it fails to discriminate between existing cognitive theories, as well as the lack of clarity over whether the human MNS also involves abstract, non-motor brain activation. In addition, there is considerable debate over the specific brain regions

involved in MNS activation, with some studies implicating prefrontal regions, whereas others pointing at the lateral occipto-temporal region [82]. Clearly further research is needed to address these questions. However, despite such limitations, there appears to be overwhelming evidence for an inherent, automatic neurobiological vulnerability of empathic attunement arising from diverse lines of scientific inquiry-as briefly outlined below.

Innate imitation. Meltzoff and Moore's seminal report of neonatal imitation has been substantially replicated, showing human beings are innately hard-wired with the capacity for recognizing and duplicating the actions of others (e.g., tongue protrusion)-critical building blocks for attachment and empathy [83]. A review on innate imitation by Meltzoff and Decety indicated that infants as young as forty-two minutes to seventy-two hours can accurately imitate facial expressions [84]; human actions, but not objects, and by three months of age this type of imitation fades. A robust literature reveals the innate ability of human newborns to imitate from memory and individual differences in the proclivity and extent that one demonstrates such early imitation [84]. Children (and adults) retain their uncanny natural ability to imitate others as a potent source for social learning and communication.

Mimicry. Researchers distinguish between active or conscious imitation and passive, involuntary physical simulation of behavior known as "mimicry" [64]. A robust line of social psychology queries indicates that people unconsciously and automatically mimic the physiological bases of emotional expressions, mannerisms, facial expressions, postures, body movements, vocal intonations, accents, and speech patterns of others [85]. For instance, using facial electromyography, studies have revealed that when an observer perceives another person's emotional facial expression such as a smile or a frown, corresponding expressions occur in the observer [64]. Investigators have repeatedly confirmed the social function of involuntary mimicry in terms of increasing rapport and fondness between self and other, finding mimicked subjects were significantly more helpful and generous toward others than non-mimicked subjects [86].

Emotional contagion. Understanding another's emotional signals has clear adaptive advantages especially in the formation and maintenance of social relationships. In this regard, developmental research has identified "emotional contagion" or the ability to detect or "catch" the immediate emotional state of another person, as the most rudimentary form of empathy [64]. For example, studies have repeatedly shown neonatal crying produced significantly more reactive crying in the newborn than did either white noise, a cry from a 5-month old, or a synthetic cry [74]. Studies with newborns led developmental researchers to conclude that the understanding of another is primarily a form of "embodied practice" [87]. Subsequent replications provide strong evidence that human infants are capable of early self/other distinction and emotional resonance-important innate precursors of empathy.

Developmental research on infant-mother emotional contagion. Research on emotional or mood contagion during infant-mother interaction reveals emotional synchrony in mother-infant play is an important foundation for the development of empathy evident by 2–3 months of age. Malatesta and Haviland found mothers were highly likely to imitate infant expressions of enjoyment and interest (which occurred most frequently), as well as expressions of surprise, sadness, and anger when they occurred [88]. However, non-depressed mothers rarely displayed negative emotions to their baby. Thus, optimal infant-mother dyads exhibit notable positive synchrony partly due to maternal matching of her infant's positive affective expressions [88]. However, repeated studies have shown that when depressed mothers interact with their infants they often display flattened affect, provide less stimulation, and

respond less appropriately to their infant's actions-thereby stimulating sympathetic stress in infants (e.g., elevated salivary cortisol levels), as well as infant simulation of maternal depression by becoming less attentive, having fewer positive emotional expressions, and becoming morefussythanbabieswith non-depressed mothers [89].

Neural mechanisms for emotional contagion. Functional neuroimaging studies have shown that recognizing the facial expressions of disgust in someone else involves the automatic activation of the same part of the brain, the left anterior insula and right anterior cingulate cortex, normally activated during the actual first-hand experience of disgust. Research on shared representations for both negative and positive emotions have similarly identified shared representations for first-hand and vicarious experiences [69,90].

Physiological simulation of emotion. When another feels a negative emotion (e.g., fear, pain, grief) the observer automatically simulates a physiological response that matches the other [80], just as babies experience a stress response to hearing other newborn crying, or when interacting with a depressed mother. Levenson and Rauf demonstrated automatic simulation of negative emotions in couples finding that when one member's sympathetic arousal more closely mirrored the observed other's, they interpreted the others' negative emotions more accurately [91]. This was not true for positive emotions. Results suggest a relation between physiological linkage (how closely one simulates the physiological response of others) and accuracy in interpreting another's negative emotions. Another study found that the larger the right insula, the more accurate the person was at detecting their own and others' internal physiological states [92].

Effects of witnessing another' pain. Research on the perception of other's pain is particularly salient in regards to understanding the potential iatrogenic impact of empathy. Jackson and colleagues reviewed the evolutionary implications for human empathic ability to quickly and accurately identify, communicate, and interpret expressions of human idioms of distress/suffering (*i.e.*, paralinguistic vocalizations, facial expression, body posture, avoidance behavior, *etc.*), thus allowing for recognition of potential danger as well as understanding another's condition, all of which may lead to life-saving care [68].

Helpers inherently experience an immediate, automatic, and profound neurobiological impact from the act of witnessing another's pain, revealing that perception of another's suffering, automatically activates the observer's own representation of that emotion and behavior [93]. This hard-wired human capacity to synchronize with the pain experiences of others has been established by meta-analysis [68], confirmatory factor analysis [94], and systematic reviews [64]. Researchers have identified the underlying neural circuit or "pain matrix" responsible for our innate empathic abilities including the dorsal anterior cingulate cortex (ACC), the anterior midcingulate cortex (aMCC) and anterior insula [68,69]. These shared neural pathways between self and other, prompt observers to "resonate" with the emotional state of others in order to understand another's internal experience [74]. Therefore, the more empathically attuned we are, the greater level of neural activation and shared pain we experience—a.k.a. empathy's double-edge sword [69,80].

6.6. Paired Deficits Research

Further substantiation of shared representation neural mechanisms underlying empathy is evident in research on paired-deficits between emotion production and emotion recognition. For example,

Gazzaniga reviewed studies involving patients with Huntington disease whose damaged insula and putamen limited their ability to recognize disgust in self and others [95]. Similar paired deficits have been demonstrated across a wide-range of shared sensations, emotions, and behaviors [64,74].

It is striking that most pain studies induced empathic-related neurophysiological changes in observers despite artificial laboratory settings and inconsequential nature of the relationship (e.g., two emotionally unattached study participants). Researchers predict more meaningful scenarios will solicit greater empathic and sympathetic responses in helpers [64]. In fact, Lamm *et al.* suggested empathic responses of caregivers is likely responsible for the high frequency of pain symptoms reported by spouses of chronic pain patients [68,96,97].

6.7. Relevance of Automatic Empathic Responses and Compassion Stress

Credible evidence now exists to explain the inherent universal risks/vulnerability associated with helping professions and caregiving- our automatic empathic responses via the innate MNS. In other words, the "double-edge" sword of the prosocial benefits from human empathy is the potentially deleterious, cumulative effects from under-regulated chronic physiological simulation of clients' or loved ones' emotional and physical pain. The common refrain of feeling emotionally and/or physically drained after a particularly intense session in the presence of distressed others, now has a neurophysiological reality.

Redefining compassion stress. It bears mentioning that humans are predisposed to find emotional suffering and distress in others as aversive-stimulating a sympathetic stress response and desire to avoid prolonged exposure [68]. Fortunately, nature has endowed humans with the capacity for intentionally regulating our empathic responses-allowing for potential altruistic behaviors to override our sympathetic arousal and avoidance. Nevertheless, definitions of compassion stress must account for both the automatic and cognitively controlled empathic responses of helpers in addition to inherent sympathetic stress responses related to engaging in helping behaviors.

6.8. "Top-Down" Control: Self-Awareness, Re-Appraisal, and Perspective-Taking

Contemporary neuroscientific models of empathy posit that full-blown human empathy and altruistic behavior requires conscious, cognitive processing, rather than mere automatic empathic reactions [53,68]. Hein and Singer reviewed of the role of regulatory top-down processes such as self/other awareness, emotion reappraisal, perspective-taking and the helper-client relationship by allowing observers to modulate their empathic reactions [98]. Consistent evidence from diverse lines ofprimaryand paired-deficit research has substantiated the role of executive neural networks in regulating empathic responses, as well as providing key insight into the mechanisms whereby helping professionals and caregivers may exhibit either resilience or compassion empathic distress [69].

Self/Other awareness. In human development, the emergence of a self-representation is vital for the empathic process. Conversely, sharing emotion without top-down processes like self-awareness corresponds to the phenomenon of emotional contagion, which takes the form of "total identification without discrimination between one's feelings and those of the other"—an evolutionary threat and clear pathway toward a CSI [74]. Therefore, self-oriented neural networks provide the critical capacity to differentiate self and other, in order to detach and regulate bottom-up empathic responses.

Emotion reappraisal. The ability to deliberately cognitively reappraise ambiguous or negative emotions (e.g., expressing sadness) of another in a more positive manner (e.g., healthy ventilation) has been shown in neuroimaging studies to help observer's modulate automatic emotional synchrony with distressed others by decreasing activity in the brain's emotional processing regions while increasing activation in regions essential for memory, cognitive control, and self-monitoring [99]. Moreover, left hemisphere speech centers were more active during reappraisal as observers reportedly "talked to themselves" and accessed positive emotions [96], thus regulating emotional contagion.

Perspective-taking. Developmental research indicates that the ability to consider the perspective of another person gradually emerges after 18 months, representing another prominent top-down process to shape empathic responses and social understanding [61]. Top-down processes like perspective-taking activates neural pathways such as the frontopolar cortex that critically serves to both inhibit automatic neurophysiological simulation of another's behavior, as well as amplify cognitive appraisal by means of imagination or anticipation used to help recognize and interpret another's internal state [100]. Paired-deficits research of individuals experiencing frontal cortex damage reveals significant impairment in cognitive flexibility and perspective-taking ability [101,102].

6.9. Mental Flexibility and Self-Regulation

Mature empathy is described as the ability to recognize the other person is like the self, while maintaining an appropriate self-other boundary [74]. Mental flexibility and self-regulation represent two cognitive components deemed essential in maintaining self-other distinction during an emphatic reaction [74]. For example, the role of mental flexibility in regulating our empathic responses has been examined by Li and Han using event-related brain potential (ERP) to disentangle the bottom-up automatic empathic response from top-down control via perspective-taking during perception of others' pain [103]. The researchers concluded that the ability to shift between self-and other-perspectives regulates the controlled aspects of empathy, but not the automatic neurophysiological simulation [103]. Additionally, Decety and Jackson reported subjects experiencing intense emotions intensely, especially negative emotions, are prone to an aversive emotional reaction such as anxiety from recognizing another's emotional pain [74]. These studies demonstrate the importance of top-down processes like mental flexibility in CSI, as well as limitations of models that over-emphasize cognitive factors [53,55].

Inhibiting the default egocentric bias. Decety and Jackson argued that empathy involves the active inhibitory mechanism via the prefrontal cortex, in order to deliberately suppress our inherent egocentric bias about others, whereby we naturally infer other's experience based upon our own embodied experience, beliefs, attitudes, and biases including racial bias [74,104]. Interestingly, suppressing emotional expression, including positive emotions, increases blood pressure for suppressor and observer, as well as decreasing other-focused attention [105].

6.10. Research on Individual Differences in Empathic Ability

Individual differences and interactional effects mediating an observer's empathic responses have been studied including gender [74,106], culture [107], perceived emotional conflict [72], attachment styles [108], perspective-taking ability [100], empathy enhancement training [109] and level of trait or dispositional anxiety [110]. For instance, Loggia and colleagues measured state empathy (described as empathic

concern evoked in the here and now) with a five-item questionnaire shown to be a reliable measure of state empathic concern [111], and trait empathy (defined as dispositional tendency to respond empathically to everyday life situations) via the Interpersonal Reactivity Index and the Balanced Emotional Empathy Scale [111]. Findings indicated that observers with high levels of state empathy experienced more pain from observing others' pain, than low state empathy [111].

Implications for CSI. Research on individual differences and CSI are invaluable in developing more reliable and effective prevention, screening, and intervention options. Data provides tentative support for Figley's [10] factors of helper's empathic ability, empathic concern, life disruptions, and McCann and Pearlman's cognitive schema of therapist "safety", especially if the latter is associated with high trait levels of anxiety that may predispose helpers to more intense unregulated empathic responses [55]. A neuro-behavioral explanation for mimicking another's PTSD symptoms (e.g., STS, VT), implicates the combined effect of heightened activation of sympathetic stress responses due to helper/other distress and intense, automatic empathic responses that interfere with effective cognitive regulation resulting in "emotional contagion". Children without fully developed cognitive skills needed for adequate empathic regulation, would be particularly at-risk for emotional contagion (e.g., STS) in the presence of an overly distressed caregiver [28].

7. The Linnerooth Neurobehavioral Model of Compassion Stress Injury

This neurobehavioral model is dedicated in honor of U.S. Army psychologist Peter Linnerooth, a staunch advocate for increased CSI awareness [112].

7.1. Empathic Response

The universal neurophysiological alterations experienced by helpers during the process of shared understanding, imagining, and feeling of another's inner experience caused by the dual activation of automatic, non-conscious simulation via the mirror neuron system (e.g., mimicry, emotional contagion), and "top-down" regulation by deliberate controlled cognitive processes (e.g., self/other awareness, emotional reappraisal, perspective-taking), that can lead to prosocial behavior.

7.2. Compassion Stress

The combined neurophysiological effect experienced by helpers exposed to another's pain and suffering arising from two separate, but interrelated pathways: (1) shared emotional experience via empathic responses (automatic and regulated); and (2) sympathetic stress activation from: (a) *primary compassion stressors* inherent or directly related to the act of helping or caregiving including environmental, situational, or internalized care-giving demands as well as triggering of un-integrated, distressful or painful experiences in the helper's associative (memory) neural network (e.g., countertransference; see Appendix C); and (b) *secondary compassion stressors* associated with the helper's maladaptive reactions or attempts to cope with primary compassion stress (e.g., isolation, excessive work, *etc.*, see Appendix C).

7.3. Compassion Stress Reactions (CSR)

The adverse cumulative effects of severe or protracted compassion stress resulting in normally transient functional neurobehavioral changes that temporarily impact a helper's mental/physical health, sense of well-being, and may or may not interfere with the ability to fulfill some major professional or caregiving responsibilities. A CSR is caused by exposure to intense or prolonged pain and suffering of others and the combined cumulative effect of: (1) excessive under-regulated negative empathic responses (e.g., emotional contagion) and; (2) over-activation of sympathetic stress response to primary and secondary compassion stressors resulting in heightened risk/vulnerability factors and behavioral indicators (Appendix B); and (3) reduced effectiveness of self-care evidenced by decreased protective/resilience behaviors (Appendices B and D).

Onset of some signs, symptoms, or behavioral indicators may be simultaneous from witnessing another's intense emotional or physical suffering or within minutes of the other's vivid verbal disclosure or behavioral enactments after an interval of hours or days. In most cases, symptoms will disappear within minutes to hours, or in more intense exposure, reliving and/or avoidance behaviors may persist in fleeting, mild, and non-disruptive forms, but gradually fade within 2–5 days.

Examples of potential primary and secondary compassion stressors are reflected in Appendix C, including the myriad of risk/vulnerability factors that may exceed the potentially mediating effects of self-care via protective/resilient factors (Appendix B and D). Excessive sympathetic activation and ineffective self-care will eventually interfere with top-down cognitive regulation of empathic responses leading to emotional contagion (e.g., STS). Helpers may endorse behavioral indicators associated with CSI (Appendix B), or on standardized CSI measures [113]. In CSR, the helper's self-care activities are temporarily overwhelmed or only partly effective.

Severity of CSR. Severity can range from "mild to severe" as determined by the number, type, and frequency of reported signs, symptoms, or behavioral indicators (Appendix B) and measured on standardized assessments [113], as well as neurophysiological indices of brain function and stress response [69]. Normally CSR are short-lived, and do not cause significant impairment. The CSR construct is modeled after empirically established ASR/COSR constructs whereby the presence of CSR is universal to the human stress response and not a sign of psychopathology [54].

7.4. Compassion Stress Injury (CSI)

A severe maladaptive or prolonged CSR lasting greater than five-days and causes significant functional and/or structural neurophysiological changes as evidenced by noticeable negative impact to a helper's mental/physical health, sense of well-being, and/or ability to fulfill many major professional or caregiving responsibilities. A CSI is caused by the cumulative effects of severe or prolonged CSR due to excessive, unregulated empathic responses (e.g., emotional contagion) combined with chronic activation of the helper's sympathetic stress response to primary and secondary compassion stressors that overwhelms the helper's self-care and resilience capacity. CSI will typically manifest in a diverse constellation of neuropsychiatric and somatic symptoms corresponding to a broad array of psychiatric, medical, and/or medically unexplained physical diagnoses.

7.5. Interaction of Empathic Responses, Risk/Protective Factors, and Self-Care

We hypothesize that there is a dosage effect of severe stress which is overwhelming. In CSI, there is a strong negative association between the combined cumulative effect of unregulated empathic and sympathetic stress responses from primary and secondary compassion stressors (Appendix C), and its nullifying impact of protective/resilience self-care factors (Appendix B). In short, combined unmitigated stress from work, personal, and/or professional risk factors may overload the helper's adaptive resources (resilience factors) serving to initiate and/or exacerbate the healer's incapacity to properly regulate their empathic and sympathetic responses, that in turn leads to emotional contagion and erosion of self-care. Conversely, routine use of protective/resilient factors associated with self-care (e.g., exercise, relaxation, recreation, sleep, social activity, balanced diet, *etc.*) is related to adequate regulation of empathic and sympathetic responses, however, if the dosage of compassion stress remains severe and protracted, eventually, like "old psychiatrist syndrome"—we all break. In essence, individual differences in terms of vulnerability of CSI is akin to individual vulnerability for developing or protecting against trauma-stress related injuries such as PTSD.

7.6. Slippery Slope of Dysregulated Sleep and Compassion Stress Injury

Meta-analyses and systematic reviews on the temporal relationship of sleep disturbance and stress-related injuries like PTSD concluded that it can be both a primary symptom and primary cause, a common finding in the CSI literature as well [10,114,115]. In CSI, we expect dysregulated sleep to be a key early marker and catalyst due to combined sympathetic activation from unregulated empathic responses and compassion stressors, as well as erosion of sleep hygiene. Thus monitoring sleep is essential.

7.7. Limitations of Neurobehavioral Research and Future Direction

General methodological and conceptual limitations of the neuroscience on empathy has been reviewed [116], but not in terms of its applicability toward CSI. First and foremost, few of the existing neurobehavioral research has involved helping professionals or caregivers, or explicitly examined compassion stress/injury as a dependent variable [116]. Secondly, research has emphasized the artificial unidirectional experience of observers, and not the interactional effects of automatic and controlled empathic processes of each participant. Fields of counseling espouse constructs depicting the bidirectional influence between client and healer (e.g., transference/counter-transference, modeling, pacing, mirroring, joining, *etc.*). There is now compelling evidence that the majority of human beings without congenital or acquired empathy-deficits constantly utilize their innate MNS during social exchange.

Therefore, future research on human change processes must take into account the reciprocal regulation of empathic responses. For example, we would reasonably expect to see some measure of client change due to the client's neurophysiological simulation of the helper's prosocial emotion and behaviors, which may account for psychotherapy outcome findings that 80% of positive early responders improved within the first three sessions irrespective of theoretical orientation [117]. Conversely, future investigations into the cause, prevention, and intervention of CSI should include the trajectory, underlying mechanisms, and diagnostic features of the model, interactional effects of empathic capacity and neurophysiological responses in helpers, especially in combination with dosage effect of compassion

stressors, and established risk/protective variables. For instance, we hypothesize that CSI is highly correlated to the healer's inability to adequately regulate joint empathic and sympathetic responses, leading to a form of emotional contagion, and erosion of self-care.

8. Conclusions

The understanding of neural mechanisms underpinning the empathic experience has increased significantly the past decade due to the application of social neuroscience. Our main goal was to introduce a unifying neurobehavioral formulation of CSI as well as demonstrate its shared heritage with traumatic and war stress injuries. Implications of this model extend beyond CSI, offering potential insight into the effect of simulating graphic violence, domestic conflict, and traumatic grief, as well as potential curative impact of empathic responses. It is our hope that an integrated mind-body paradigm may finally replace antiquated dualistic beliefs responsible for harmful stigma and disparities between mental and medical aspects of health. Research is needed to refine this model with the superordinate goal of vastly improving our understanding, prevention, screening, and intervention of stress-injuries like CSI. A science of self-care is needed to eliminate unnecessary suffering and loss of those who nobly extend the self, in service of others.

Author Contributions

Mark Russell conceived and designed the experiments; Matt Brickell performed the literature review; both authors analyzed the data and wrote the paper.

Conflicts of Interest

The authors declare no conflict of interest.

Abbreviations

CF: Compassion fatigue;
CSI: Compassion stress injury;
IOM: Institute of Medicine;
WWI: First World War;
WWII: Second World War;
PB: Professional burnout;
STS: Secondary traumatic stress;
VT: Vicarious traumatization;
EDF: Empathic distress fatigue;
PTSD: Post-Traumatic Stress Disorder;
ASR: Acute stress reactions;
COSR: Combat/operational stress reactions;

HPA: Hypothalamic-Pituitary-Adrenal;

MNS: Mirror neuron system;

ACC: Anterior cingulate cortex;

aMCC: Anterior midcingulate cortex;

ERP: Event-related brain potential.

Appendix A

Table A1. Comparison of Symptoms, Signs, and Behavioral Indicators for CSI.

Compassion Stress Injury	Symptoms, Signs, and Behavioral Indicators
Burnout	
Freudenberger [42]; Freudenberger and Robbins [118]	Sleeplessness, irritability, chronic fatigue, depression, anxiety, anger, cynicism, suspicion, physiological symptoms, boredom, loss of compassion, discouragement
Pines and Maslach [119]; Maslach [43]	Sleep disturbance, irritability, chronic fatigue, depression, anxiety, anger, pessimism, physical exhaustion, emotional exhaustion, poor work performance, reduced personal achievement, professional detachment, conflict between work and family, mental exhaustion, social isolation
Figley [10,36]	Gradual onset; Sleeping difficulties, chronic fatigue, irritability, somatic problems, anxiety, depression, guilt, helplessness, cynicism, poor work performance, professional withdrawal
Jenkins and Baird [120]	Sleep disturbance, irritability, aggression, poor work performance, professional isolation, physiological symptoms, emotional exhaustion
Eriksson and Wallin [58]	Gradual onset; insidious, dissatisfaction with career, reject career
Secondary Traumatic Stress	
Figley [10,35,36,121]	Abrupt onset; Sleep disturbance, irritability, chronic fatigue, erosion of idealism, anxiety, depression, poor work performance, professional withdrawal, helplessness, ASD/PTSD symptoms matching sufferer
Gentry, Baranowsky, and Dunning [122]	Abrupt onset; irritability, depression, outbursts of anger or rage, poor work performance, hopelessness, intrusive thoughts/images of another's experiences, hypervigiliance, lowered frustration tolerance, social isolation, inability to separate personal and professional life
Vicarious Traumatization	
McCann and Pearlman [48], McCann and Pearlman [55]	Sleep disturbance, irritability, chronic fatigue, anxiety, depression, professional withdrawal, intrusive thoughts/images of another's experiences, hypervigiliance, social isolation, helplessness, suspicion, cynicism, distrust
Herman [47]	Sleep disturbance, irritability, chronic fatigue, anxiety, depression, anger, professional withdrawal, social isolation, intrusive thoughts/images of another's experiences, hopelessness
Pearlman and Saakvitne [123]	Disturbances in self-identity, spirituality, world view, and cognitive frame of reference
Compassion Fatigue	
Joinson [51]	Sleep disturbance, irritability, chronic fatigue, depression, anger, inability to separate personal and professional life, professional withdrawal, reduced memory capacity, physiological disturbances
Figley [10,36,121,124]	Abrupt onset; sleep disturbance, irritability, chronic fatigue, depression, anxiety, anger, helplessness, guilt, intrusive thoughts/images of another's experiences, hypervigiliance, poor work performance, professional withdrawal, confusion, hopelessness, disorientation, social isolation, physiological symptoms, poor communication, inability to separate personal and professional life, irresponsibility

Appendix B

Table A2. List of therapist risk/vulnerability and protective/resilience factors.

Contributing Factors	Author(s)
Primary/Secondary Compassion Stressors: Risk/Vulnerability	
Exposure to client trauma	Pines and Maslach [119]; Maslach [43]; McCann and Pearlman [48]; Herman [47]; Joinson [51]; Figley [10,36,121,124]; Pearlman and Saakvitne [123]; Hesse [50]; Wee and Myers [15]; McLean, Wade, and Encel [125]; Imai *et al.* [45]; Creamer and Liddle [126]; Flannelly *et al.* [22]; Dekel *et al.* [127]; Moulden and Firestone [128]; Shah, Garland, and Katz [17]; Tyson [26]; Killian [129]; Bush [130]; Harrison and Westwood [49]; Robins, Meltzer, and Zelikovsky [131]; Ghahramanlou and Brodbeck [132]; Jenkins and Baird [120]; Meadors, Lamson, Swanson, White, and Sira [25]
Helping professional's empathetic engagement	McCann and Pearlman [48]; Herman [47]; Figley [10,36,121,124]; Pearlman and Saakvitne [123]; Jenkins and Baird [120]; Flannelly *et al.* [22]; Moulden and Firestone [128]; Tyson [26]; Killian [129]; Harrison and Westwood [49]; Robins *et al.* [131]; Meadors, Lamson, Swanson, White, and Sira [25]
Professional isolation	Figley [121]; Flannelly *et al.* [22]; Killian [129]; Bush [130]; Harrison and Westwood [49]; Phelps *et al.* [57]
Relationship problems	Flannelly *et al.* [22]
Therapist's trauma history	Herman [47]; Pearlman and Saakvitne [123]; McCann and Pearlman [55]; Ghahramanlou and Brodbeck [132]; Hesse [50]; Jenkins and Baird [120]; McLean *et al.* [125]; Creamer and Liddle [126]; Flannelly *et al.* [22]; Killian [129]; Bush [130]; Robins *et al.* [131]
Professional disappointment	Maslach [43]; Flannelly *et al.* [22]; Dekel *et al.* [127]; Killian [129]; Vallerand *et al.* [44]
Professional inexperience	McLean *et al.* [125]; Creamer and Liddle [126]; Dekel *et al.* [127]; Bush [130]; Phelps *et al.* [57]
Personal life stressors	Figley [10,121]; Bush [130]; Robins *et al.* [131]
Youth of therapist	Ghahramanlou and Brodbeck [132]; Creamer and Liddle [126]; Dekel *et al.* [127]; Bush [130]
Job Stress	Pines and Maslach [119]; Maslach [43]; Joinson [51]; Figley [36,121]; Maytum *et al.* [133]; Creamer and Liddle [126]; Dekel *et al.* [127]; Tyson [26]; Killian [129]; Bush [130]; Phelps *et al.* [57]; Robins *et al.* [131]; Meadors, Lamson, Swanson, White, and Sira [25]; Vallerand *et al.* [44]
Low Socioeconomic Status	Shah, Garland, and Katz [17]
Self-Care: Protective/Resilience	
Regular exercise	Hesse [50]; Maytum *et al.* [133]; Flannelly *et al.* [22]; Shah, Garland, and Katz [17]; Harrison and Westwood [49]; Linnerooth, Moore, and Mrdjenovich [20]
Maintains boundaries home/work	Herman [47]; Joinson [51]; Pearlman and Saakvitne [123]; Figley [10,121]; Hesse [50]; Harrison and Westwood [49]; Phelps *et al.* [57]
Social Support Network	Herman [47]; Figley [121]; Hesse [50]; Maytum *et al.* [133]; Killian [129]; Harrison and Westwood [49]; Phelps *et al.* [57]; Robins *et al.* [131]

Table A2. *Cont.*

Contributing Factors	Author(s)
Professional Support Network	Figley [121]; Hesse [50]; McLean *et al.* [125]; Maytum *et al.* [133]; Killian [129]; Harrison and Westwood [49]; Phelps *et al.* [57]; Linnerooth, Moore, and Mrdjenovich [20]
Humor	Joinson [51]; Maytum *et al.* [133]
Professional Satisfaction	Pearlman and Saakvitne [123]; Figley [10,121]; Dekel *et al.* [127]; Tyson [26]; Harrison and Westwood [49]; Phelps *et al.* [57]; Robins *et al.* [131]
Effective Training and Supervision	McCann and Pearlman [48]; Pearlman and Saakvitne [123]; Figley [121]; Hesse [50]; Dekel *et al.* [127]; Killian [129]; Harrison and Westwood [49]

Appendix C

Example of Primary and Secondary Compassion Stressors

Primary Compassion Stressors: Stressors inherent or directly related to the act of helping or caregiving include, but not limited to:

- Exposure to intense emotional or physical pain and suffering of others
- Felt demand to help or care for others
- Helping setting involves exposure to traumatic events (e.g., disaster, war, *etc.*)
- Frequently facing moral, ethical, or legal dilemmas
- Managing parasuicidal, suicidal behaviors, or completed suicide
- Exposure to unusual physical or emotional demands to meet others' needs
- Caregiving associated with severe illness, disease, or disability
- Working with death and dying of others and their family members
- Frustration with bureaucratic systems and policies
- Heightened sense of lack of control or feelings of helplessness
- Exposure to anger and/or lack of gratitude
- Dealing with non-compliance
- Being reminded of one's own mortality or traumatic experiences
- Difficulty paying attention
- Being reminded of one's own traumatic experiences
- Hyper vigilant response to certain cases
- Negative client behaviors and responses
- Increase in caseload sizes/decrease in staff
- Decreased individual control over work
- Increased number of work hours
- Increased time engaged in paperwork or administrative duties
- Dealing with inequities, disparities, and prejudice

Secondary Compassion Stressors: Stressors caused by the helper's maladaptive reactions or attempts to cope with primary compassion stress include:

- Withdrawal from colleagues, friends, or loved ones
- Impatience/intolerance

- Decrease in quality of professional and personal relationships
- Decreased interest in intimacy or sex
- Self-isolation and loneliness
- Reduced joy and sense of purpose with career
- Increased interpersonal conflicts
- Trouble separating work from personal life
- Diminished functioning in non-professional circumstances
- Increases in ineffective or self-destructive tension-reduction behaviors
- Work Performance decrease in quality, quantity
- Accident proneness
- Losing things
- Ineffective or harmful self-care practices (e.g., excessive drinking)
- Absenteeism/tardiness
- Overwork
- Frequent job changes
- Blaming clients for their experiences
- Setting perfectionist standards
- Personal illness or injury
- Changes in family relationships
- Changes in financial status
- Death of family member or close friend
- Personal exposure to trauma outside of the help setting
- Isolation from peers; limited access to supervision or consultation

Appendix D

Self-Care: Protective/Resiliency Factors
Adapted from: Figley, C.R. [121]. *Treating Compassion Fatigue.* New York: Brunner-Rutledge.

- Setting and maintain boundaries.
- Establish clear boundaries between your personal and professional life.
- Don't take on more than you can realistically do!
- Work only your assigned hours.
- When you go home at the end of a hard day, try your best to leave your work at work.
- Remember that it's alright to say "No"!
- Counter isolation in professional, personal and spiritual domains of life.
- Attend Trainings, professional development, and organizational support classes.
- Diversify professional roles.
- Establish a personal support community.
- Developing mindful awareness.
- Consciously expand perspective to embrace complexity.
- Active optimism and exquisite empathy.
- Holistic self-care.

- Maintain clear boundaries and honor limits.
- Professional satisfaction.
- Create meaning in your personal and professional life.

Prevention of Compassion Fatigue

- Must be well rested
- Utilize your positive supportive connections to process your feelings
- Take negatives and turn them into positives
- Research shows those who can "turn off thoughts about work" are more resilient during their careers
- Instead of waiting for the clinicians to become symptomatic use inventory tools to look for the presence of challenges to their fundamental assumptions, values and beliefs
- With early assessment clinicians have the opportunity to transform their discomfort into personal growth and development

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