De-pathologizing the Symptoms of Adverse Childhood Experiences

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A Paper

Presented to School of Health and Social Sciences, City University of Seattle

In Partial Fulfillment of the Requirements

For the Degree of Master of Arts

CPC680: Counselling Research Capstone

August 24, 2021

De-pathologizing the Symptoms of Adverse Childhood Experiences

APPROVED:

(Faculty Advisor)

(Principal of Canadian Programs)

Acknowledgments

To my partner, Allison, who is the true wizard behind the curtains of this project and my sons, Sawyer and Cohen, who have helped me to reclaim the joy and adventure of childhood. Thank you for your love, your support and your sacrifices to make this a reality.

To my classmates and teachers, who embraced, inspired, and challenged me. Thanks for the incredible learning experience no textbook could have provided. I look forward to working together in the future as colleagues.

To my supervisor, Jill Taggert, whose rapid response time always surprised me. Thank you for the practical and always encouraging feedback. It was a pleasure working with you.

To my friends, who have stuck with me through unprecedented times. Thank you for making me laugh, for challenging me to be better, and for checking in on me when you haven't heard from me in a while.

Abstract

Adverse Childhood Experiences (ACEs) directly affect more than half the world, with the most vulnerable populations experiencing ACEs at even higher rates. ACE research reveals a strong, graded relationship between childhood stress and trauma and an increased risk of poor mental health, adult diseases, and risky or harmful behaviours. The purpose of this paper is to consider how our body's neuro-biological survival systems can have unintended consequences when chronically activated or mistuned in our early years of development to help us move from symptom management to treating root causes. The mislocation of client's problem through labelling their ACE symptoms a disease (a problem with their body), a disorder (a problem with their mind/personality), or bad behaviour (a problem with their morality) can lead to ineffective treatment strategies and further reinforcement of their pathologization and stigmatization particularly for those most vulnerable to ACEs. Using examples from Somatic Experiencing, Internal Family Systems, and Eye-Movement Desensitization and Reprocessing, this paper proposes five assumptions for de-pathologizing ACE symptoms: 1.) ACE impacts are not the problem; they are a symptom of the problem, 2.) Focus treatment on the underlying problem (isolation, alienation, shame, rejection, fear, abuse, neglect, and nervous system dysregulation), not the symptoms, 3.) ACE symptoms result from what went right to help survive, and the motivating goal is still to protect, 4.) Integrative compassion for ourselves and others comes through a deep understanding of the symptoms' motivation (5.) The solution lies within our body and mind's innate wisdom and ability to heal.

Keywords: adiposity, adverse childhood experiences, bullying, peer victimization, child abuse, maltreatment, community violence, depathologize, deprivation, early attachments, psychological abuse, emotional abuse, isolation, peer rejection, maladaptive functioning,

neglect/lack of care, neurobiology, neuroplasticity, physical abuse, polyvagal theory, poverty, resiliency, sexual abuse, somatic, trauma, violence against mother or step-mother, intimate partner violence, domestic violence, socioeconomic status, substance abuse, child development, mental health, stress

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Chapter 1: Introduction

Adverse Childhood Experiences (ACEs) have become an important area of study in the last 25 years, revealing the lasting impacts of childhood stress and trauma on our minds, bodies, behaviours, and relationships (Finkelhor et al., 2015). This research brings a biological, psychological, social (BPS) perspective that challenges traditional notions locating the problem within the person by labelling it a disease (a problem with your body), a disorder (a problem with your mind/personality), or problematic behaviour (a problem with your morality). Instead, ACEs research invites us to consider how our protective biological survival responses can have devastating outcomes when they are chronically activated in our early years of development.

This chapter will discuss the significance of this capstone project on depathologizing ACE symptoms and its potential to benefit all of us, particularly vulnerable populations. I will reflect on my position within the research. I will then review some of the relevant literature. Following that, I will discuss the purpose of my capstone project—to depathologize the effects of ACEs. Finally, I will define key terms, propose subheadings for a more extensive literature review in chapter two, and provide a chapter summary.

Background to the Research Problem

ACEs Defined

Adverse childhood experiences (ACEs) are stressful or traumatic events experienced during childhood that have a strong, graded relationship with life-long negative health outcomes. Gershon et al. (2013) suggest that the definition of ACE should be ‘acute or chronic stressful events which may be biological or psychological in nature, occurring during childhood and resulting in a biological and/or psychological stress (as cited in Sachs-Ericsson et al., 2016). The

original ACE scale developed for the original Kaiser Permenente ACEs study (Felitti et al., 1998) is a prevalent tool among researchers and advocates concerned about the long-term effects of childhood trauma (Finkelhor et al., 2015). The scale uses a ten-item survey to assess early life experiences. Five items address aspects of child maltreatment: physical abuse, psychological abuse, sexual abuse, physical neglect and emotional neglect. The other five items address parental or family incapacities: parental loss through divorce, death or abandonment, parental imprisonment, parental mental illness, parental substance abuse, and violence against the mother. While the original ten-item ACE scale is the most commonly used, there is mounting evidence that other common childhood adversities such as childhood bullying and peer victimization, isolation and peer rejection, and poverty have negative long-term developmental effects (Finkelhor et al., 2015).

Overview of ACEs Symptoms

The higher one's ACE score (having multiple categories of ACE exposure) increases the likelihood of having multiple health risk factors later in life (Felitti et al., 1998). While trauma has historically been viewed from a mental health lens the research on Adverse Childhood Experiences (ACEs) reveals the negative outcomes associated with ACEs fall across a broad spectrum of the human experience including: poor mental health such as: panic reactions, depressed affect, anxiety, hallucinations, and sleep disturbances (Anda et al., 2006; Kessler et al., 2010); adult diseases such as: increased risk of diabetes, asthma, stroke, myocardial infarction, and all-cause mortality (Dong et al., 2004; Dube et al, 2003; Edwards et al., 2003; Geoffroy et al., 2016; Gilbert et al., 2015; Nikulina et al., 2012; Young & Widom, 2014 as cited in Koyashi et al, 2020), cancer, skeletal fractures, liver disease, sexually transmitted infections and adiposity; and increased risk of participating in risky behaviours such as: physical inactivity, substance use,

addictions, promiscuity, self-harm, suicide (Anda et al., 2006, Dube et al., 2003, Felitti et al., 1998), early sexual intercourse, poor anger control, and perpetuating intimate partner violence (Anda et al., 2006).

Relationship to Physical Health Risks

The original ACEs study of 9508 American adults found that 52.1% had at least 1 ACE factor and one-fourth reported ≥2 ACE factors (Felitti et al., 1998). Further, Felitti et al.'s study showed that persons with multiple categories of ACE exposure were likely to have multiple health risk factors later in life. For example, they found that four or more ACEs increased 4- to 12-fold the health risks for alcoholism, drug abuse, depression, and suicide attempt, and a 2- to 4-fold increase in smoking, poor self-rated health, ≥50 sexual intercourse partners, and sexually transmitted disease; and a 1.4- to 1.6-fold increase in physical inactivity and severe obesity. The number of ACEs also showed a graded relationship to the presence of adult diseases, including ischemic heart disease, cancer, chronic lung disease, skeletal fractures, and liver disease.

Relationship to Poor Mental Health

Kessler et al. 's (2010) cross-national study found that ACEs account for 29.8% of all Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) and Composite International Diagnostic Interview (CIDI) disorders. This study surveyed 51945 international participants and found that maladaptive family functioning, such as parental mental illness, child abuse, neglect were the strongest predictors of disorders. Researchers concluded that ACEs have strong associations with all classes of disorders, at all life-course stages, in all groups, in all countries surveyed.

Anda et al. (2006) found that ACEs scores had a strong, graded relationship to the prevalence and risk of affective disturbances (panic reactions, depressed affect, anxiety, and

hallucinations), somatic disturbances (sleep disturbance, severe obesity), substance use and abuse (smoking, alcoholism, illicit drug use, and injected drug use), sexuality measures (early intercourse, promiscuity, and sexual dissatisfaction), impaired memory, high perceived stress, difficulty controlling anger, and the risk of perpetrating intimate partner violence (IPV). Researchers concluded that the graded relationship of the ACE score to 18 different outcomes in multiple domains theoretically parallels the cumulative exposure of the developing brain to the stress response with resulting impairment in multiple brain structures and functions. These factors will be discussed in greater detail in Chapter 2.

Relationship to Vulnerable Populations

Soares et al. (2016) compared British adolescents with lower income Brazilian adolescents and found that approximately 85% experienced at least one ACE, and females reported a higher number of adversities. They were unable to determine if ACEs were associated with adolescent adiposity due to poor sample quality. They did conclude that ACEs are more likely to impact vulnerable populations, such as women and lower socioeconomic populations. This finding is consistent with other research (Bellis et al., 2014; Brown et al., 1998; Logan-Greene et al., 2014; Sidebotham et al., 2006; Ye & Reyes-Salvail, 2014 as cited in Soares et al., 2016).

Merrick et al. (2018) found that childhood adversity is common across sociodemographic characteristics, but some individuals are at higher risk of experiencing ACEs than others. This study looked at 214157 American respondents. It found that 61.55% had at least 1 ACE, and 24.64% reported ≥3 ACEs. Researchers identified racial minorities, high school dropouts, low income, unemployed, unable to work, and gay or lesbian populations as particularly vulnerable. Researchers concluded that although identifying and treating ACE exposure is essential,

prioritizing the prevention of ACEs is critical to improving health and life outcomes throughout the lifespan and across generations.

Capstone Significance

While prevalence rates of ACEs vary considerably depending on the definition, measurements, sample characteristics, and methodologies of the study (Soares et al., 2016), the research does show that ACEs affect a majority of people across countries, populations, and cultures (Kessler, 2010). ACEs are associated with poor physical health, mental health, as well as risky behaviours (Felitti et al., 1998). Given the abundance of research on showing that children's brains are exceedingly and malleable in the early years, raising awareness about ACEs' pervasiveness and devastating impacts is an important project (Shonkoff, 2000, as cited in Novoa & Morrissey, 2020).

Historically, the scientific method has sought to sort, categorize, and specialize in treating many of the challenges that we now understand to have a strong-graded relationship with ACEs. The creation of discrete siloed areas of expertise has created experts, yet it has also hindered the cross-pollination of ideas and possibly misdirected the location of the underlying problem. Physical illness gets labelled a disease to be treated by medical doctors. Mental illness gets labelled a disorder to be treated by a psychologist, problematic behaviours get labelled immoral, deviant, sinful, or anti-social by societies and treated accordingly. Yet scientific research is now has come full circle, reaffirming the interconnectedness of the human experience. ACE research is one of those areas pointing to our interconnectedness. Dr. Jennifer Mervyn, a local psychologist and ACE advocate, suggests that understanding, preventing, and treating *ACEs is not just mental healthcare; it is healthcare* (personal communication, 2018). Considering ACEs

as the most significant factor in screening for many medical conditions seems shocking, and yet here we find the science suggesting that may indeed be the case.

Understanding the etiology of diseases, disorders, and problematic behaviours help us to better understand ourselves, our world, and the solutions available to us. Many of the labels for problems locate the problem within a person rather than recognizing them as biologically protective responses to external danger. ACEs research brings a biological, psychological, social (BPS) perspective to the life-long effects of ACEs on our physiology, personality, behaviours, relationships. In doing so, it helps us to see that when our body's short-term solution to stress becomes a chronic state, the protective solution can itself can become a long-term problem. Is obesity a human flaw caused by a weak will and overeating, or is it caused by 25 years of continuously high cortisol levels produced by a chronically dysregulated nervous system resulting from childhood trauma?

Neuroscience has changed how many therapists view trauma—no longer is it seen as a disorder of events, but rather as a disorder of the body, brain, and nervous system (Fisher, 2017). This neurobiological perspective led to another significant paradigm shift which Fisher eloquently lays out in the introduction to her book:

> If the brain and body are inherently adaptive, then the legacy of trauma response must also reflect an attempt at adaptation, rather than evidence of pathology. Through that neurobiological lens, what appears clinically as stuckness and resistance, untreatable diagnoses, or character-disordered behavior simply represent how an individual's mind and body adapted to a dangerous world in which the only "protection" was the very same caretaker who endangered him or her. Each symptom was an ingenious solution by the body to create some semblance of safety for the developing child or endangered adult.

> The trauma-related issues with which the client present for help, I now believe, are in truth a "red badge of courage" that tells the story of what happened even more eloquently than the events each individual consciously remembers. (Fisher, 2017, p. 1-2)

It is with this two-fold lens: 1) that trauma is a disorder of the body, brain, and nervous system; and 2) that the symptoms of trauma are the body's attempts at adaptation, rather than evidence of pathology, that I will explore in-depth in our literature review.

Who would benefit from this research?

Based on the prevalence rates of ACEs research, more than half the world's population has at least one ACE. This suggests that almost everyone will benefit from this research, whether it's one's self personally or it's someone you know and love; ACEs impact us all in particular, vulnerable and marginalized populations that are the most impacted ACEs and will also benefit the most from addressing it. Researchers have identified some of these vulnerable ACE populations, such as lower socio-economic populations (Merrick et al., 2018, Soares et al., 2016), gays and lesbians, racial minorities (Merrick et al., 2018), and women (Soares et al., 2016). This list is by no means exhaustive. I expect to further identify other ACE vulnerable populations in our literature review, such as indigenous, sexual minority, and transgender populations. All of these populations experience current and historical systemic oppression, exploitation, and pathologization of their minds, bodies, relationships, cultures, and behaviours.

Purpose of the Capstone

Based on these research problems, the purpose of this capstone project is to depathologize ACEs by 1) bringing a holistic awareness to treating medical conditions that known to be the result, in part, of untreated somatic survival responses to trauma and stress; 2) reframing ACEs impacts on our mind, body, behaviour, and relationships through a lens that

views them as the natural defence responses of our mind and body attempting to survive and protect itself; and 3) to consider how I might use this information interrupt the cycle of oppression perpetuated on people vulnerable to ACEs.

This capstone will seek to answer the research questions: 1) what are the underlying roots of ACEs impacts? 2) what are the impacts *resulting* from ACEs on our physiology, behaviours, and relationships? 3) What are the protective factors that make people more *resilient* towards their ACEs? 4) How can I best help *restore* those impacted by ACEs? 5) How can I depathologize the survival *responses* people use to cope with their ACEs?

Definition of Terms.

- **adiposity:** fat around the belly and organs
- **adverse childhood experiences:** a list of difficult and traumatic events and circumstances that people experience before the age of eighteen. The original ACEs questionnaire is made up of 10 items: physical abuse, psychological abuse, sexual abuse, physical neglect, emotional neglect, parental loss through divorce, death or abandonment, parental imprisonment, parental mental illness, parental substance abuse, and violence against the mother/step-mother. At least four additional items have been proposed: childhood bullying and peer victimization, isolation and peer rejection, poverty and deprivation, exposure to community violence, and low socioeconomic status (Finkelhor et al., 2015).
- **bullying/peer victimization:** the routine use or threat of violence, coercion, or social pressure (teasing, shunning, ignoring, and spreading rumours) to abuse, dominate or intimidate others. There is often a perceived imbalance of physical or social power by the bully or victim (Bullying, 2020; Youngblade et al., 2009).

- **child abuse/maltreatment:** cruel or unusual behaviour towards another child that carries a substantial risk of causing physical or emotional harm. This includes any form of physical, emotional, or sexual mistreatment or lack of care (Perkarsky, 2018; Red Cross, n.d.).
- **community violence:** harm committed in public by individuals who are not related to the victim or witness (TeachTrauma, n.d.)
- **depathologize:** refusing to label something as a disease or illness (Boundless, n.d.).
- **deprivation:** the consequences of a lack of income and other resources, which cumulatively can be seen as living in poverty. Deprivation happens when resources are so seriously below the average individual or family that one is, in effect, excluded from ordinary patterns, customs and activities (Townsend, 1979, as cited in Mack, n.d.).
- **early attachments:** the emotional connection that a child forms with their primary caregiver, often the mother. It binds them together, endures over time, and helps the child experience pleasure, joy, safety, and comfort. It is highly influential in the development of adult personality, relationship styles, and ability to cope. (Encyclopedia, n.d.).
- **psychological/emotional abuse:** the chronic attack on a child or youth's self-esteem by a person in a position of trust or authority. Rejecting, degrading, isolating, terrorizing, corrupting, ignoring, and exploiting are all forms of emotional abuse (Red Cross, n.d.).
- **isolation/peer rejection:** exclusion from a social group
- **maladaptive functioning:** reactions and behaviours that are inappropriate, unhelpful, or unhealthy.
- **neglect/lack of care:** a lasting carelessness towards the necessities in life such as clothing, shelter, healthy diet, education, hygiene, supervision, medical and dental care, adequate rest, safe environment, moral guidance and discipline, exercise, and fresh air (Red Cross, n.d.).

- **neurobiology:** the study of the nervous system and how the brain works.
- **neuroplasticity:** the brain's ability to reorganize, heal, and learn by forming new connections throughout life.
- **physical abuse:** when a person purposefully injures or threatens to injure another person (Red Cross, n.d.)**.**
- **polyvagal theory:** a theory by Stephen Porges that identifies three types of responses based on the level of threat one perceives: the fight or flight response, the freeze response, and the social engagement response (Wagner, 2016).
- **poverty:** the lack of resources needed for the food, activities, and living conditions needed in the society to which one belongs.
- **resiliency:** a person's ability to recover from or adapt to adversity or change (Merriam-Webster, n.d.-b).
- **sexual abuse:** when a younger or less powerful person is used by an older or more powerful child, youth or adult for sexual gratification (Red Cross, n.d.).
- **somatic:** a person's body, body parts, or body experiences such as physical sensations. Used to distinguish from emotions, thoughts, and behaviours
- **trauma:** a person's response to a deeply distressing or disturbing event that overwhelms their ability to cope, causes feelings of helplessness, diminishes their sense of self and their ability to feel the full range of emotions and experiences. (Onderko, n.d.)
- **violence against mother or step-mother/intimate partner violence/domestic violence:** any form of physical, emotional, or sexual abuse, or neglect done against spouses and dating partners, either in current or former relationships. Approximately 80% of victims are female (Sinha, n.d.). ACEs questionnaires typically specify mothers and/or step-mothers.

- **socioeconomic status:** the social standing of an individual or group, often measured as a combination of education, income and occupation.
- **substance abuse:** the unhealthy use of drugs or alcohol despite the negative consequences
- **child development:** the physical, language, thought and emotional changes that occur in a child from birth to adulthood
- **mental health:** the well-being of a person's thoughts, emotions and behaviours
- **stress:** a pressure or strain placed on someone

Reflectivity and Positionality Statement

I recognize that almost every location I currently inhabit is one of privilege, power, and majority. I am an educated, white, middle-class, able-bodied, married, heterosexual, cis-gender Canadian male. I am a land-owner on the traditional, unceded lands of the Kwantlen, W̱SÁNEĆ, Stó:lō, Tsawwassen, Semiahmoo, and Coast Salish nations. My ancestors were settlers who benefited from the colonization of Canada. At the end of the day, it is my hope that the privileges in my life are used in the service of lifting others. Despite my privileged locations, my life has been touched by ACEs, both personally and professionally.

This capstone was seeded by those experiences before I even applied to the City University's counselling program. In many ways, this project is a product of my own journey to understand how profoundly my childhood experiences have shaped my adult life. It is also a culmination of my life's work with vulnerable youth populations. I have seen ACEs profoundly impact the youth with whom I work. I knew their ACEs were impacting their relationships, their decision-making and their mental health. I have even suspected, at times, that their ACEs were playing a part in their physical health problems. Time and time again, I saw the adults and systems that were supposed to help youth make things worse with band-aid solutions due to lack

of funding and misinterpreting survival behaviours as the source of the problem. I now know that what I had anecdotally observed was just the tip of the ACE iceberg.

Chapter Summary

ACEs are as stressful or traumatic events experienced during childhood with a strong, graded relationship with life-long negative outcomes. These outcomes fall across a broad spectrum of the human experience, including poor mental health (panic reactions, depressed affect, anxiety, hallucinations, and sleep disturbances), adult diseases (diabetes, asthma, stroke, myocardial infarction, cancer, skeletal fractures, liver disease, sexually transmitted infections, obesity, adiposity, all-cause mortality) and risky or harmful behaviours (physical inactivity, substance use, addictions, promiscuity, self-harm, suicide, early sexual intercourse, poor anger control, and perpetuating intimate partner violence). ACEs are more likely to impact vulnerable populations (women, racial minorities, high school dropouts, low income, unemployed, unable to work, and gay or lesbian populations); however, the research shows that ACEs affect a majority of people across countries, populations, and cultures. This means almost everyone stands to benefit from this capstone project.

The purpose of this capstone is to raise awareness of the consequences of ACEs, to depathologize these impacts by revealing them as unintended results of our innate protective survival responses, and to contribute to the interruption of oppression perpetuated on people vulnerable to ACEs. I hope to learn how ACEs impact our physiology, behaviours, and relationships? Are there any resiliency factors to ACEs? How can I restore those impacted by ACEs? How can I depathologize the survival responses people use to cope with their ACEs? In my literature review, I propose to examine the neurobiological roots of ACEs, the results of

ACEs, and some of the ways I can restore those suffering from their ACE experiences including this author.

Chapter 2: Literature Review

In the previous chapter, I defined Adverse Childhood Experiences (ACEs). I then overviewed the wide-ranging impacts ACEs have on physical health, mental health, and vulnerable populations. I discussed the significance of my capstone project and how almost everyone stands to benefit from understanding ACEs better, and I located myself within the topic. I then defined many of the key terms and finally proposed headings (roots, results, restoration of ACEs) for my literature review in order to help us imagine a better response

In this chapter, I will review some of the relevant research literature on ACEs. I will start in the Roots of ACE Impacts with how ACEs shape our brain and body function, our brain and personality, our brain and relationships, our nervous system, the hypothalamic-pituitary-adrenal axis' regulation of the stress hormone cortisol, the immune system, the endogenous opioid system, and cell telomeres. In Results of ACEs, I will look at how ACEs influence anxiety and depression, shame, impaired cognitive function, sleep disturbance, obesity, cancer, and substance use. Finally, in Restoring Interventions For ACE Clients, I will explore how Somatic Experiencing (SE),Eye-Movement Desensitization and Reprocessing (EMDR), and Internal Family Systems (IFS) can help us restore clients suffering from ACE impacts.

Roots of ACE Symptoms

This section will examine some of the underlying and interconnected biological systems that help us adapt and survive in our environments. Because many of these protective systems are immature at birth, their function and development are heavily influenced by the environment with which they interact. When these systems function appropriately, they are highly adaptive to protect us. However, given their malleable nature, particularly in childhood, their protective

function can become disorganized, impaired, or over-activated resulting in a lifetime of potentially problematic outcomes.

The Brain and Body Functions

Survival is the brain's number one job, and everything else comes after that (Van der Kolk, 2014). According to Van der Kolk, the brain accomplishes this in five ways: 1) signalling bodily needs (food, rest, protection, sex, and shelter); 2) mapping the environment to locate where to satisfy these needs; 3) produce the energy and actions required for these needs; 4) warning of dangers and opportunities; 5) adapting actions to the requirements of the situation. As mammals, humans best accomplish these task in groups, thus

> Psychological problems occur when our internal signal don't work, when our maps don't lead us where we need to go, when we are too paralyzed to move, when our actions do not correspond to our needs, or when our relationships break down (Van Der Kolk, 2014, p. 55).

Perry's Neurosequential Model of Therapeutics (NMT) helps us to understand how the timing of ACEs can impact specific brain structures and functions. NMT is based on three assumptions (Perry, 2009). First, the human brain develops from the bottom up. Second, trauma inhibits the part of the brain (brainstem, limbic, neocortex) that is developing at the time of the trauma. Third, that treatment should start from where the individual is currently functioning and work up the hierarchy as function improves. For example, if someone has an underdeveloped or hyperactive brain stem, they likely operate in survival mode, with quick transitions into flight, flight, or freeze; therapy needs to start there. One cannot form proper relationships, learn to control their impulses, emotions and behaviour if they primarily function in a primitive brain state. NMT then helps match the nature and timing of specific therapeutic techniques to the

developmental stage, brain regions, and neural networks mediating the neuropsychiatric problems (Perry, 2009).

Our experiences shape the brain in combination with our genetics and our temperament (Van der Kolk, 2014). In explaining how our experiences shape the brain, Perry (2009) states that the brain organizes itself in a use-dependent fashion. According to Perry, in the developing brain, undifferentiated neural systems are determined by signals (neurotransmitters, neurohormones, cellular adhesion molecules, amino acids, ions) to organize them from their undifferentiated, immature forms. The quantity, pattern of activity, and nature of the activation of these molecular cues are dependent, in part, upon the experiences of the developing child. Thus when the child has adverse experiences—loss, threat, neglect, and injury—there can be disruptions of neurodevelopment leading to compromised functioning.

Our environment impacts the development of our brain and affects which pathways are the most used. Because neurons that fire together, wire together, repeated experiences often become the default setting (Van der Kolk, 2014). A brain that consistently feels safe and loved can default to states of play, cooperation and exploration; whereas, a brain that consistently perceives threats and being unwanted defaults to managing feelings of fear and abandonment (Van der Kolk, 2014). Young developing brain stems and limbic brains are particularly shaped by early emotional and relational experiences, although they can be modified by later experiences (Van der Kolk, 2014).

First to develop is the brain stem, sometimes referred to as the reptilian brain. It is responsible for basic functions like eating, breathing, sleeping and waking, and getting rid of toxins. The brainstem and the hypothalamus are responsible for the body's energy levels by

maintaining the basic life-sustaining systems of our heart, lungs, immune, and endocrine systems within a balance called homeostasis (Van der Kolk, 2014).

Above the brain stem and second to develop is the limbic system, also called the mammalian brain. The development of the limbic system rapidly expands after a child is born. The limbic system "is the seat of emotions, the monitor of danger, the judge of what is pleasurable or scary, the arbiter of what is or is not important for survival purposes. It is also the central command post for coping with the challenges of living within our complex social networks" (Van der Kolk, 2014). Van der Kolk (2014) refers to the brainstem and limbic system together as the emotional brain—its' primary job is to detect danger or opportunities and motivate us towards or away from them by releasing hormones through the hypothalamic-pituitary-adrenal axis (HPA axis). The HPA axis will be discussed in greater detail later. The emotional brain structures and biochemistry are simpler than the neocortex in that it assesses information globally (Van der Kolk, 2014). This results in rapid decisions based on rough similarities. If it detects a threat, the emotional brain will put into action a preplanned automatic escape plan called fight or flight response before our brain's neocortex has a chance to assess the situation.

The top layer of the brain is the neocortex or the rational brain. This is the part of the brain responsible for language, abstract thought, creativity, empathy, planning for the future, and absorbing and ascribing meaning to large amounts of information (Van der Kolk, 2014). The frontal lobes, which make up the largest portion of the neocortex, begin to rapidly develop around the age of two. While the triune brain structures are each responsible for discreet body functions, the two hemispheres of the brain have distinctive functions that can become

disconnected by stress and trauma. I will be exploring how this affects personality in the next section.

The Brain and Personality

Structural Dissociation Model

The brain develops vertically from the bottom up with different functions in the brain stem, limbic and cortex structures. The brain is also organized horizontally into two separately functioning hemispheres that communicate through the corpus callosum. Van der Hart, Nijenhuis, & Steele (2006) propose the structural dissociation model (SDM) for understanding how the brain hemispheres act protectively for our mind and personality when it experiences trauma. SDM describes how the brain's left and right hemispheres have separate, specialized functions that disconnect or 'dissociate' under stress and threat (Fisher, 2017). SDM uses parts language to describe different functions. The left brain personality is referred to as the apparently normal part (ANP) in SDM (Van der Hart et al., 2006) functions under stress by remaining positive, task-orientated, and logical—to keep going on with the tasks of daily life (Fisher, 2017). The right brain personality or emotional part's (EP) function is to cope with the emotions, sensations, and survival mode (hypervigilance, fight, flight, freeze, submission, cry for help) that was activated at the time of the trauma (Van der Hart et al., 2006; Fisher, 2017). Van der Hart et al. (2006) suggest that structural dissociation is not necessarily a complete split but instead lacks cohesion and coordination among the parts.

According to Van der Hart et al. (2006), structure dissociation has three levels of severity: 1) Primary structural dissociation—the most basic trauma-related division of the personality having a single ANP and a single EP. In this case, the ANP is the majority stakeholder of the personality, while the EP is often restricted in scope, function, and sense of self. Primary

structural dissociation is typically reflected in a post-traumatic stress diagnosis (PTSD). 2) Secondary structural dissociation—occurs when traumatizing events are increasingly overwhelming or prolonged. In this case, a single ANP remains intact, but there is an additional division of EP. The additional EPs may have different and conflicting defence strategies resulting in further lack of integration. Secondary structural dissociation is typically reflected in complex PTSD and Borderline Personality Disorder (BPD). 3) Tertiary structural dissociation—occurs when the unavoidable aspects of daily life have become associated with past trauma or when the functioning of ANP very poor. As a result, there is a further division of the ANP and possibly EP. In tertiary and severe secondary dissociation, multiple parts may have great elaboration (names, ages, genders, preferences) and real or perceived autonomy from the influence of other parts. Tertiary structural dissociation is reflected in dissociative identity disorder (DID).

There is a growing body of supporting empirical evidence for dissociation and splitting (Brand et al., 2016; Dorahy et al., 2014). SDM is rooted in neuroscience and is a well-accepted theory in Europe (Fisher, 2017). The biological basis for this is found in the regions and structures of the brain itself (Van der Hart et al., 2004). For example, the right hemisphere dominance in childhood with the left hemisphere dominance slowly developing throughout childhood and adolescence (Shore, 2001). Split-brain research has shown that the two hemispheres of the brain can communicate with each other but operate independently and differently (Gazzaniga, 2015). The left hemisphere uses language, while the right is more visual and lacks language. The right recalls memories episodically and implicitly, while the left recalls autobiographical memory and acquired knowledge. The left brain remembers the gist of a situation deleting details that do not fit the general schema of the event and making inferences to fill any missing details (Gazzaniga, 2015). In contrast, the right brain does not forget or interpret

the non-verbal aspects of the situation, “It is totally truthful and only identifies the original pictures” (Gazzaniga, 2015, p. 152).

The needs of the right brain EP (to manage the memories, emotions, sensations of the trauma) and the needs of the left brain ANP (to go on with normal life) maybe appear contradictory or fraudulent to the person experiencing them, yet both are real and necessary from an evolutionary perspective (Fisher, 2017). Splitting or fragmenting of personality is an adaptive survival strategy providing psychological distance from overwhelming or intolerable experiences. It is also adaptive in resolving contradictory needs and drives. For example, a child’s attachment needs to seek proximity and comfort from their caregiver may contradict their animal defence mechanisms to fight, flight, or flee (Liotti, 1999).

Whereas the left brain ANP is focused on everyday life, it also avoids traumatic memories (Van der Hart et al., 2006) or judging them as bad qualities to be fixed (Fisher, 2017). The right brain EP are likewise alienated from the other half, perceiving the ANP as having abandoned them, absent, or weak (Fisher, 2017). This results in a personality that is frantically trying to be normal but ends up feeling alienated from or invaded by the intrusive communication of the parts. For example, the going on with everyday life part might want to lose some weight by exercising and eating healthy; however, if the EP is triggered, it may not care about the weight loss goal and instead soothe the difficult emotions and sensations of traumatic memory by eating some chocolate cake. This results in one part feeling angry and judgemental at themselves for self-sabotaging their plan, and the other part feeling rejected and judged for trying to manage their pain. Fisher calls this experience self-alienation.

Self-alienation reflects the fact that victims of trauma often survive by denying part of their personality (Fisher, 2017). The price of this defence mechanism is maintaining the denial or

self-hatred for the disconnection long after the danger has passed. Trauma survivors often report better functioning as a result of their compartmentalization but also feel like frauds or that they are pretending.

> Without an explanatory paradigm that makes sense of these contradictions, there is no way for individuals to know that their intense feelings and distorted perceptions are evidence of fragmentation, not proof of internal defectiveness or fraudulence masked by the ability to function. (Fisher, 2017, p. 5)

According to Fisher (2017), continued self-alienation results in growing self-loathing, detachment from emotion, addictive or self-destructive behaviour, and internal battles between vulnerability and control, love and hate, closeness and distance, shame and pride. I will examine shame in more depth later. Fisher suggests the mindfulness-based approach to integrating parts developed by Richard Schwartz called Internal Family Systems (IFS). I will be examining this modality further in the restoration section.

Our Brain and Relationships

Attachment Theory (AT) states that one's childhood pattern of relating to others, learned from their primary caregiver, continue to be enacted in their relationships throughout their lifespan. John Bowlby (1969) defined attachment as the "lasting psychological connectedness between human beings" (p. 194). According to AT, infants need a consistent nurturing relationship with one or more sensitive caregiver to develop into healthy individuals (van Rosmelan et al., 2016). This style is called a secure attachment. On the other hand, parental unavailability or unresponsiveness may create insecure attachments and contribute to maladaptive behaviour or even psychopathology. Thus making AT an important line of research for ACEs. Attachment models have been conceptualized with two dimensions that underlie

insecure attachment styles: avoidant attachment and anxious attachment. Individuals with a highly avoidant attachment style tend to struggle for independence and maintain an emotional distance from significant others. Individuals with a highly anxious attachment style are excessively dependent on significant others and worry that persons in close relationships with them will not be available or supportive in stressful times. Disorganized attachments will fluctuate between anxious and avoidant and is thought to be the result of unpredictable parenting (Cherry, 2019; Shorey, 2020). While attachment styles exhibited in adulthood are not automatically the same as those seen in infancy, early attachments can have a significant influence on adult relationships (Cherry, 2019).

History

John Bowlby is credited with laying the foundations of AT. Bowlby countered the behaviourist theory that attachment was a classically conditioned behaviour resulting from feedings (Cherry, 2019). Instead, Bowlby saw that feedings did not reduce the anxiety experienced by children when they were separated from their primary caregivers. Thus he concluded that attachment also had an emotional motivation—when children are scared, they seek closeness to their primary caregiver to sense comfort and care. Bowlby argued that nurturance and responsiveness were the principal factors of attachment (Cherry, 2019).

Mary Ainsworth, both collaborated with and elaborated on Bowlby's work and has come to be recognized as a co-founder of AT in her own right (Royden, 2019; van Rosmelan et al., 2016). Ainsworth is famous for her ground-breaking research in the 1970s on mother-child relationships known as the 'Strange Situation' (Cherry, 2019). In this study, researchers observed young children between twelve and eighteen months old as they reacted to a situation in which they were momentarily left alone and then reunited with their mothers. Based on her

observations, Ainsworth identified three different styles of attachment: secure attachment, anxious/ambivalent attachment, and avoidant attachment (Cherry, 2019; Royden, 2019).

In 1986, researchers Mary Main and Judith Solomon added a fourth attachment style they called disorganized attachment (Cherry, 2019). Main and her colleagues also developed the Adult Attachment Interview (AAI). The AAI has provided an empirically validated way of following the transmission of attachment patterns from generation to generation (Wylie & Turner, n.d.). Another contribution Main brought to AT was the concept of an Earned Secure attachment (ESA). An ESA is when someone initially develops an insecure attachment style but later can gain or 'earn' a secure attachment style.

Another contributor to AT is Sue Johnson and her incorporation of AT with couples and families in her Emotionally-Focused Therapy (EFT). EFT is systems approach that considers how AT affects our relationships later in life. In EFT perceived distance or separation from close relationships gets interpreted by the brain as danger because losing the connection to a loved one jeopardizes one's sense of security. This primal fear emotion sets off the fight-or-flight response in the amygdala. This is what is thought to occur in relationships where people are either angry with one another or are withdrawing in response to a perceived challenge to their sense of attachment (Jones, 2009).

Peter Fonagy and Anthony Bateman's concept of mentalization continues to add to AT (MentalHelp.net, n.d.). Mentalization is the ability to reflect upon one's state of mind and have insight into one's feeling and why. The ability to mentalize is considered highly correlated to attachment style as caregivers' perceptive understanding of a child's experience and ability to give feedback to the child about their experience provides a valuable model. This modelling helps children learn how to pay attention to and to understand what they are experiencing.

Secure Attachment

In Ainsworth's strange situation, securely attached children became distressed when their mother left the room but quickly adjusted to being positive and happy when reunited with their mom (Royden, 2019). They were calmly interacting with strangers only when their mom was nearby and seemed to use their mother as a secure base from which to explore their situation. Whenever mom was nearby, they could relax and play, becoming accustomed to their new environment. Ainsworth hypothesized that securely attached children having been parented with consistency and reliable responses were able to relax because they knew their needs would be met.

Children who are securely attached as infants tend to develop stronger self-esteem and better self-reliance as they grow older (Cherry, 2019). They also tend to be more independent, perform better in school, have successful social relationships, and experience less depression and anxiety (Cherry, 2019). Those adults who are securely attached in childhood tend to have good self-esteem, healthy romantic relationships, and the ability to self-disclose to others. An adult with a secure attachment style is likely to miss their romantic partner but not become anxious or insecure about being briefly separated from them (Royden, 2019).

Anxious Attachment

Children with an anxious attachment, sometimes called anxious-ambivalent, presented increased levels of distress when their mothers left and angry upon her return (Royden 2019). They might avoid contact or push their mothers away. Still, others would desperately cling on to their moms. These children were very anxious around strangers, with and without their mother's presence. They also cried more and explored less than both the securely and avoidantly attached children. Ainsworth hypothesized the anxious attached child was likely parented by someone

inconsistent in responding to their needs. An adult with an anxious attachment style might continuously worry about their partner's faithfulness or safety. They might know it is irrational, yet still struggle not to continually send messages checking in on them. They may become angry or hurt if they do not receive a prompt response.

Avoidant Attachment

Children with an avoidant attachment, sometimes called dismissing in adults (Shorey, 2020), display no visible signs of distress when their mothers left the room, and little interest in her when she returned (Royden, 2019). They appeared to be just as happy with a stranger. Incredibly, later research revealed that despite their untroubled outward appearance, their heart rates and other physiological signs of stress were dramatically increased when their mothers left. Ainsworth hypothesized that children with avoidant attachment were likely to have been parented by someone unavailable or rejecting of their needs. Shorey (2020) states that an avoidant child will learn to deny their own negative emotions and needs for close relationships and augment this need by winning at things like academics and sports and acting self-assured and confident. Avoidant adults may thoroughly enjoy their freedom from their partner and be unlikely to give their partner a second thought during a night out.

Disorganized Attachment

Children with a disorganized attachment, sometimes called 'unresolved' attachment or 'fearful' in adults (Shorey, 2020), often present with an unclear assortment of behaviours. They might look disoriented, dazed, or confused. They may also avoid or resist the parent. Main and Solomon (1986) hypothesize that this lack of a clear attachment pattern is likely linked to inconsistent caregiver behaviour (cited in Cherry, 2019). Thus parents may serve as both a source of comfort and fear, leading to disorganized behaviour. This often is seen when parents are

frightened or frightening; thus, children will not be able to develop organized ways of coping or adapting because their environment is too unpredictable (Shorey, 2020).

Neurobiological Safety

The attachment relationship is so important our brain encodes it emotionally not as love, but as safety (Jones, 2009). Gordon Neufeld suggests this is why we ignore our own safety to run into a burning building to save loved ones (Cram & Salmond, 2020). The loss, perceived loss, or even the potential loss of any attachment relationship can activate the fight, flight, or freeze threat responses of our nervous system (Jones, 2009). Thus here we find AT intersecting with my next topic Polyvagal Theory.

The Nervous System

This section will explore the nervous system's role in safety, socializing, and survival responses and how ACEs can lead to maladaptive functioning in these systems. Polyvagal theory (PT) explains how our physiological and behavioural states are influenced by the nervous system's continuous monitoring for cues of risk and safety. According to Porges (2015)

> The theory explains why feeling safe requires a unique set of cues to the nervous system that are not equivalent to physical safety or the removal of threat. The theory emphasizes the importance of safety cues emanating through reciprocal social interactions that dampen defense and how these cues can be distorted or optimized by environmental or bodily cues. (p. 114)

PT proposes that as a result of evolutionary processes, social connectedness emerged as the chief biological impetus for mammals in their pursuit of survival. Social connectedness' function is to support proximity and co-regulation of one's physiological state (Porges, 2015). This begins

with the mother-infant attachment relationship and then expands to other significant partners throughout our lives.

The autonomic nervous system (ANS) responds to sensations in the body and signals from the environment to ensure our survival using three hierarchal pathways, each with different bodily activations (Dana, 2018). Stephen Porges calls this detection and subsequent response neuroception because it occurs subcortically beneath our conscious thoughts (Porges, n.d., as cited in Dana, 2018). Thus neuroception is different from perception in that it occurs without awareness. One experiences wellness when all three pathways work together in cooperation (Dana, 2018), but because this system is hierarchal in nature, one cannot access the higher pathways when the lower pathways are activated (Dana, 2018; Ogden & Fisher, 2018).

The highest and newest pathway is the ventral vagal nerve (VVN) of the PNS. The neuroception of safety activates the VVN pathway producing patterns of social engagement and connection (Dana 2018; Ogden & Fisher, 2018). For this reason, the VVN is sometimes referred to as the social engagement system (Ogden & Fisher, 2018). The VVN suppresses our heart rate to approximately 72 beats per minute by influencing the pacemaking sino-atrial node (Dana, 2018). This concept is called the vagal brake and helps maintain arousal within a window of tolerance (Siegel, 1999, as cited in Ogden & Fisher, 2018). The VVN also controls areas of the body used in social and environmental interaction—eyelid opening (promoting observation), facial muscles (enhancing emotional expression), middle ear muscles (distinguishing the human voice from background noise), muscle of mastication (stimulating eating), laryngeal and pharyngeal muscles (fostering the communication of emotional states through prosody), and head tilting and turning muscles (facilitating social gesture and orientation; Porges, 2005, as cited in Ogden & Fisher, 2018).

The middle pathway is the sympathetic nervous system (SNS), facilitating mobilization when neurocepting danger (Dana 2018; Ogden & Fisher, 2018). The SNS activates our primal hyper-arousal defence systems of fight, flight, cry for help, and alert freeze (Ogden & Fisher, 2018). It mobilizes us to action through two systems, the sympathetic-adrenal-medullary (SAM) and the HPA axis (Dana, 2018). The SAM is quick-acting, providing a short burst of adrenaline. This system is designed for rapid response and return to regulation. The HPA axis activates when the rapid response does not resolve the danger. The HPA axis releases the stress hormone cortisol. While the SAM response takes seconds, the HPA response takes minutes. This prepares us for action by increasing our heart rate, shortening our breath, dilating our pupils, activating sweating, increasing blood sugar, activating our muscles, and shutting down unnecessary systems such as digestion, the immune system, and growth processes (Dana, 2018; Ogden & Fisher, 2018). Our ability to hear the middle frequencies associated with human voices diminish and shift to focus on the lower frequencies of predators and the high frequencies of distress (Dana, 2018). Our ability to read facial expressions diminishes, resulting in neutral faces appearing angry or dangerous (Dana, 2018).

The oldest and most primitive pathway is the dorsal vagal nerve (DVN), part of the parasympathetic nervous system (PNS). The neuroception of life threat or inescapable danger activates the DVN system facilitating survival-related immobilization: hypo-arousal, feigning death, behavioural shutdown, and syncope (Dana, 2018; Ogden & Fisher, 2018). The neuroception of life threat is different from danger, in that, with danger, survival still appears possible (Ogden & Fisher, 2018). This system seeks to conserve resources by increasing fuel storage and insulin activity, providing endorphins to numb and raise pain threshold (Walker, 2017). Simultaneously, it attempts to reduce metabolic demands by decreasing muscle tone, heart

rate, digestion via emptying the bowel and bladder (Sullivan et al., 2018), temperature, sexual responses, and immune activity (Walker, 2017). As a result, one might experience disassociation and changes in cognitive function (Dana, 2018). This might feel like a sense of numbness, shutting down within the mind, or separation from one's sense of self (Siegel, 1999, p. 254, as cited in Ogden & Fisher, 2018). When action impossible, extreme dorsal vagal arousal may produce fainting, vomiting, or loss of control of the rectal sphincter (Frijda, 1986, as cited in Ogden & Fisher, 2018). This last resort is often called "total submission" (van der Hart et al., 2006, as cited in Ogden & Fisher, 2018).

The hierarchical relationships between these three systems are shaped early in life, resulting in enduring arousal tendencies, reactions under stress, and even vulnerability to psychiatric disorders (Cozolino, 2002; Lyons-Ruth & Jacobvitz, 1999; Porges, 2011; Schore, 2001, p. 209; Sroufe, 1997; Van Ijzendoorn, Schuengel, & Bakermans-Kranenburg, 1999, as cited in Ogden & Fisher, 2018). Early and repeated ACEs train our neuroception to be hypersensitive to risk detection even when there may be no risk (Porges, 2011, p. 253, as cited in Odgen & Fisher, 2018). Thus those with ACEs often have a reduced ability to correctly judge environmental and relational safety. As a result, they developed what Stephen Porges calls "faulty" neuroception (2011, as cited in Odgen & Fisher, 2018). As a result, engagement with the people and environments in our daily life, which requires an accurate neuroception of safety, is impeded.

The inability to manage danger cues can trigger the SNS and DVN to become chronically active (Dana, 2018). With ACEs, there is a long-term calibration of the ANS during early life events, resulting in consequential patterns of autonomic and HPA responsivity that are sustained long after the events that precipitated them and possibly long after they are adaptive (Del

Giudice et al., 2011; Pluess, 2015, as cited in Price et al., 2018). When one is unable to move into a pro-social ventral vagal state, their nervous system is chronically hyper- or hypo-aroused, and this leads to physical illnesses, distressed relationships, altered cognitive capacities, and incessant drive to find safety and relief from an out of balance nervous system (Dana, 2018). Chronic DVN activation is associated with health problems such as impaired immune function, chronic fatigue, and digestive issues, and psychological problems such as disassociation, depression, with withdrawal from relationships (Dana, 2018).

HPA Axis and Cortisol

In this section, I will explore cortisol's potential relationship to ACE outcomes. The stress response system (SRS) involves several subsystems, including the parasympathetic nervous system (PNS), the sympathetic nervous system (SNS), hypothalamic-pituitary-adrenal (HPA) axis (Price et al., 2018). When the amygdala perceives a threat, it activates the HPA axis, the locus coeruleus, and the noradrenergic system (Shorter et al., 2015). This results in increased cortisol and adrenaline levels to help arouse the body to fight or flee the threat. This occurs before the medial prefrontal cortex (mPFC) has had a chance to assess the situation. As long as you are not too upset, the mPFC can help assess for false alarms and stand down the body's stress response if needed. This balance between the amygdala and mPFC is vital in being able to respond appropriately to our environment. However, difficulty arises when a response that is adaptive to difficult environments remains set even when the environment is changed. As discussed previously in describing the SNS pathway of polyvagal theory, with ACEs, there can be a long-term calibration of the SRS during early life events, resulting in consequential patterns of autonomic and HPA responsivity that are sustained long after the events that precipitated them

and possibly long after they are adaptive (Del Giudice et al., 2011; Pluess, 2015, as cited in Price et al., 2018).

Homeostasis, a complex and dynamic biological equilibrium, is continuously unbalanced by internal or external forces called stressors (Chrousos, 2009). Stress occurs when homeostasis is threatened or perceived to be threatened. The body maintains homeostasis through numerous physiological and behavioural responses. The steroid hormone cortisol, a type of glucocorticoid produced by the adrenal cortex, helps maintain homeostasis allowing the body and its many systems to respond to life‘s many challenges in an adaptive manner (Dedovic & Duchesne, 2012). For this reason, cortisol is both essential for survival and commonly used as a biological measure of stress.

Basal levels of cortisol prime numerous physiological systems (cardiovascular, immune, metabolic and cognitive) for action prior to and at the onset of stress (Sapolsky et al., 2000). For example, cortisol facilitates the metabolism of fat and increases blood sugar availability. Cortisol also signals the body to end its’ stress response (Praag, 2004). This suppressive function prevents the stress response from overrunning and becoming harmful. Finally, cortisol prepares the body for future stressors by influencing metabolic processes, appetite, reproductive behaviour, physiology, and cognitive processes (Sapolsky et al., 2000).

While is the cortisol stress response is designed to be an adaptive response to threat, inappropriate responses such as too little, too much, or for too long can be harmful (Dedovic & Duchesne, 2012). Excess cortisol, for example, can contribute to many health problems such as the increased risk for cardiovascular, metabolic, immune, cognitive, and emotional disorders. Because cortisol influences so many body systems, it may contribute to a wide array of problems from allergies like asthma or eczema; to gastrointestinal pain, diarrhea or constipation; to

anxiety, depression, and cognitive dysfunction; as well as hypertension, metabolic syndrome, degenerative neurovascular disease and sleep disorders (Chrousos, 2009; Dedovic & Duchesne, 2012; Tsigos & Chrousos, 2002). While cortisol initially reduces appetite and may lead to weight loss, long-term exposure can have the opposite effect, stimulating over-eating and weight gain (Sapolsky et al., 2000). Chronic exposure to elevated cortisol levels is associated with adiposity and long-term obesity (Jackson et al., 2017).

The Immune System

In this section, I will explore the immune system and how its protective functioning can become impaired or maladaptive due to ACE exposure. The immune system is another crucial means of bodily surveillance and defence (Danese & Lewis, 2017). Like our brains, our immune system is not fully developed when we are born. Instead, through experience, the immune system increases its abilities over time, maturing in adolescence. There are a number of potentially protective aspects to a child's immature immune system: 1) it permits maximum flexibility to adapt to their unique environment (Danese & Lewis, 2017). 2) It could prevent a harmful or persistent immune response as they move from a sterile womb to their colonized external world.

The immune system has two arms—the innate immune system (IIS) and the adaptive immune system (AIS). The IIS is the evolutionary inheritance that gives infants the innate ability to recognize pathogens common to their ancestor's environments (Janeway and Medzhitov, 2002, as cited in Danese & Lewis, 2017). At birth, an infant's IIS can recognize common pathogens, but their ability to process antigens and respond with the correct defence mechanism is underdeveloped (Dowling & Levy, 2014). According to Danese and Lewis (2017), newborns have fewer and less functional antigen-presenting cells. Their white blood cells, both monocytes and neutrophils, have diminished function. They have reduced cytotoxicity and reduced ability to

destroy virus-infected cells. As well, they have very low levels of complementary factors that back chemotaxis (cell movement in response to chemicals) and phagocytosis (the ingestion of cells or particles by other cells).

The AIS provides two key survival advantages (Delves and Roitt, 2000a, b, as cited in Danese & Lewis, 2017). 1) It produces antigen–receptor diversity enabling more specific and effective responses to pathogens. 2) It creates long-lived antigen-specific cells that provide immune memory of previously encountered pathogens. The cellular arm of the AIS undergoes significant development after birth (Dowling & Levy, 2014). According to Danese and Lewis (2017), in the first weeks post-birth, there is a rapid increase of immature lymphocytes (a type of white blood cell) as they colonize the skin, lungs, and digestive tract. Neo-natal memory T cells and B cells levels are very low, achieving adult levels in adolescence. In contrast to the cellular arm, the humoral arm of the AIS is less developed in infants. Infants have high levels of immunoglobulin M but low levels of immunoglobulin G and immunoglobulin A. Infants typically receive secretory immunoglobulin A from their mother through breastfeeding during this phase of humoral deficiency.

The prolonged exposure to environmental chemicals that may be carcinogenic, often referred to as ‘toxic load,’ is a well-known stressor of our immune system; however, chronic exposure to psychologically toxic environments and their cumulative effects are not as well-known (Porges, 2015). Despite the mounting evidence of ACE research, even less is understood about the mechanisms or processes through which danger and risk express their harmful effects (Porges, 2015). Childhood exposure to severe psychosocial stress may affect the development of the immune system (Danese & Lewis, 2017). Evidence for this theory can be found in experiments with animals that have shown that early-life stress can alter immune function

(Danese & Lewis, 2017). Ajdacic-Grosset et al. (2019) found ACEs were associated with chronic inflammatory diseases and neurodevelopmental/mental disorders. The cumulative exposure to childhood maltreatment was found to be associated with a significant graded increase in inflammation levels 20 years later (Danese et al., 2007). A systemic review of 20 studies found childhood maltreatment is associated with a chronic inflammatory state independent of clinical comorbidities (Coelho et al., 2014), and a meta-analysis of 25 studies found that childhood trauma was associated with a pro-inflammatory state in adulthood and had a specific inflammatory profile depending on the type of trauma—physical, emotional, or sexual (Baumeister, 2015). Childhood adversity may also be associated with impaired AIS (Shirtcliff et al., 2009).

The Endogenous Opioid System

In this section, I will be exploring the how Endogenous Opioid System's (EOS) role in our moods and stress responses. I will also examine how it can become impaired or dysregulated by ACE exposure and how many behaviours associated with ACEs may be unconscious attempts to self-regulate the EOS. The EOS function is to help regulate moods and respond to stressful events (Lovallo, 2018; Van der Kolk, 2014). It rewards certain behaviours through feelings of euphoria and regulates pain through analgesics. The EOS comprises three types of opioids: β-endorphins, enkephalins, and dynorphins. These three opioids activate three types of receptors: the μ-opioid, δ-opioid, and κ-opioid receptor. These EO influence social and affective regulation and have a crucial mediating role in separation distress, relief/pleasure on reunion, self-soothing, and pain of social exclusion and rejection (Stanley & Siever, 2010, as cited Tadros et al., 2019). The addictive and rewarding properties of endorphins are comparable to those exhibited by morphine (van Ree, 1979, as cited in Bandelow et al., 2010).

β-endorphins are an essential EO released during stress (Roth-Deri et al., 2008, as cited in Bandelow et al., 2010). It has a particular affinity for the μ-opioid receptors (Bandelow et al., 2010); these receptors are distributed in regions of the brain linked to emotional processing, decision-making, and pain regulation (Moghaddas et al., 2017). This release induced through pain or exercise produces euphoria and analgesic effects. This is why severely injured people may not feel pain immediately or why long-distance runners experience a runner's high. They are released during childbirth and play a role in positive activities like love, kissing and sex. On the other hand, Dynorphins stimulating the κ-opioid receptor can induce dysphoria, depersonalization, derealization, and perceptual alterations (Kumor et al., 1986; Walsh, Strain, Abreu, & Bigelow, 2001, as cited in Bandelow et al., 2010).

Trauma can result in the dysregulation of the endogenous opioid system (EOS) (Bandelow et al., 2010; Savulich et al., 2017). Early life adversity was associated with diminished EOS activity in adult women (Lovallo et al., 2018). It is theorized that the high comorbidity rates of trauma, addiction, and self-harm reveal unconscious attempts by trauma survivors to regulate their EOS, as well as the APA axis (Bandelow et al., 2010; Lovallo et al., 2018; Moghaddas et al., 2017; Tadros et al., 2019). For example, Bandelow et al. (2010) suggest the pathology of Borderline Personality Disorder (BPD) is partially the result of dysregulation of the EOS. They posited that the self-destructive behaviours and symptoms of BPD (self-injury, food restriction, aggressiveness, sensation seeking, substance abuse, fear of abandonment, frequent/risky sexual contacts, attention-seeking behaviour, anhedonia, and feelings of emptiness and depersonalization) were explained by uncontrollable and unconscious attempts to stimulate their endogenous opioid system (EOS) and the dopaminergic reward system. Further evidence of this can be found in the off-label use of opiate antagonist drug Naltrexone (NTX) in the

treatment of BPD (Aboujaoude & Salame, 2016; Moghaddas et al., 2017). In fact, Timäus et al. (2019) found that 35.6% of BPD patients are being treated with NTX, although the efficacy of such off-label use is unclear at this time, requiring further research.

Cell Telomere

There is mounting research showing that accelerated biological ageing, measured by telomere length, may contribute to associations between ACEs and negative long-term health outcomes (Bürgin et al., 2019). Telomeres are repeated, non-coding deoxyribonucleic acid (DNA) sequences at the end of chromosomes which protect them from damage (Dagan et al., 2018). Telomeres shorten during cell division as a result of incomplete replication of the chromosome ends (Blackburn, 2000, 2001, as cited in Bürgin et al., 2019). When telomeres reach a critically short length, the cells become unstable and can malfunction. (Blackburn, 2000, as cited in Bürgin et al., 2019). Telomere shortening is a natural process that occurs over one's lifespan, making measurement of telomere length a viable marker of biological ageing (Cawthon et al., 2003; Blackburn, 2005; Aubert and Lansdorp, 2008; Takubo et al., 2010 as cited in Bürgin et al., 2019). Shorter telomeres are associated with numerous age-related diseases, including cardiovascular diseases and metabolic disorders, cancer, dementia, type 2 diabetes, earlier mortality (Dagan et al., 2018), and several psychiatric disorders (Bürgin et al., 2019). It is theorized telomere shortening is the result of the accumulated physiological wear and tear from chronic stress exposure, commonly referred to as allostatic load (Dagan et al., 2018).

Despite exposure to ACEs, some individuals show remarkable resilience (Dagan et al., 2018). Bügin et al.'s (2019) review of 38 studies suggests that there is a negative association between ACEs and telomere length, but the diversity in sample and stressor characteristics made firm conclusions difficult. In fact, a number of studies showed no association, and two studies

showed association to longer telomeres. A number of studies suggest that parent sensitivity may buffer children from the accumulated physiological wear and tear resulting from ACEs (McEwen, Gray, & Nasca, 2015, as cited in Dagan et al., 2018). Dagan et al. (2018) found a significant interaction between attachment style and ACE in predicting telomere length in young adulthood. Shorten telomere length was associated with insecure-dismissing but not with secure-autonomous or insecure-preoccupied attachment styles. Their study supports the growing evidence that parenting can shape pathways toward physical well-being despite ACEs (Cicchetti & Blender, 2006; Gunnar & Quevedo, 2007, as cited in Dagan et al., 2018).

Results of ACEs

ACEs affect our minds, our bodies, our behaviours, and our relationships. In this section, I will examine some of the known impacts that are associated with ACEs. This section is not meant to be exhaustive but rather to provide a survey of the wide-ranging consequences of ACES.

Anxiety and Depression

The robust and graded relationship between ACEs and anxiety and depression is supported in numerous studies (Afifi et al., 2008; Anda et al., 2006; Edwards et al., 2003; Felitti et al., 1998; Kessler et al., 2010; Radford et al., 2017; Selah et al., 2017; Takizawa et al., 2014). Felitti et al. (1998) found 4.6 times increase in the risk of depressed mood in ACE scores of 4 or more. Anda et al. (2006) also found a strong, graded relationship with four or more ACEs increasing the risk of depression 3.6 times and anxiety 2.4 times. Depression and anxiety are considered mediating factors in numerous other ACE impacts, such as cognitive impairment (Selah et al., 2017), suicide (Sachs-Ericsson et al., 2016), and sleep disturbances (Park et al., 2021). Kessler et al.'s (2010) study of 51945 adults in 21 countries estimate the eradication of

ACEs would result in a 22.9% reduction in mood disorders and 31.0% in anxiety disorders. Merrick et al. (2019) found four or more ACEs increase the risk of depression 5.3 times.

Cognitive Function

Anda et al. (2006) found ACEs have graded association with impaired childhood memory with a 4.4-fold increase in participants with four or more ACEs. Gould et al. (2012) found ACEs associated with altered neurocognitive functioning. Abuse had a slightly more substantial effect than neglect on visual memory, executive functioning, and spatial working memory, while neglect was strongly associated with emotional processing/inhibition. Selah et al. (2017) found ACEs associated with poorer cognitive performance and alterations in brain morphology and that this effect differed between depressed and non-depressed ACE groups with smaller hippocampal regions in the depressed ACE group. They also noted that many ACEs independently predict adult major depression (Selah et al., 2017), which is also associated with impaired cognitive function, including attention, memory, and decision making (Rubin & Zorumski, 2016; Keller et al., 2017). Treat et al. (2019) found parental ACE scores were negatively correlated with children's working memory scores. They also found a negative correlation between harsh parenting and a child's inhibitory control and a moderate negative correlation between harsh parenting and a child's cognitive flexibility scores.

Although ACE exposure is associated with impaired cognitive development during childhood and adolescence, it is not as clear whether these relationships persist with respect to cognitive health outcomes later in life (Gold et al., 2021; Kobayashi et al., 2020). Similar to many ACE-associated effects, when examining their impacts later in life, there are many different moderators and mediators that may affect the extent and nature of the relationship (Sachs-Ericsson et al., 2016). Geoffroy et al.'s (2016) longitudinal study of a 1958 British Birth

Cohort showed that childhood exposure to neglect or abuse was negatively associated with domain-specific cognitive functions through adolescence and at age 50. Barnes et al. (2012) found protective associations between childhood adversity and cognitive decline in older African American adults, but no associations in older White adults (Barnes et al., 2012). Burri et al.'s (2013) study of Swiss former child labourers over 60 years old found associations between childhood trauma exposure, PTSD symptoms and cognitive performance. Donley et al. (2018) found that childhood stress was associated with an increased risk of dementia and marginally significant association with Alzheimer's disease among Eastern Finnish men. Kobayashi et al.'s (2020) South African study found that ACEs were not associated with cognition, except that having a parent who drank excessively, used drugs, or had mental health problems was associated with lower memory scores. Kobayashi et al. (2020) conclude that later-life memory is negatively impacted in populations with a historical context of pervasive trauma.

Interestingly ACEs may be associated with some cognitive benefit, mainly when dealing with uncertain situations. Mittal et al.'s (2015) study found people who experienced unpredictable childhoods performed worse at inhibition (overriding dominant responses) but performed better at shifting (efficiently switching between different tasks). This finding was consistent with the notion that shifting, but not inhibition, is adaptive in unpredictable environments.

Shame

Shame is a universal emotion of feeling flawed, deficient, or defective commonly experienced by ACE survivors. This feeling of "flawedness" causes one to hide when their defects are exposed or at-risk of being exposed. Shame is about a fear of disconnection. A shamed person feels there is something unacceptable about themselves, therefore deserving of

abandonment (Park, 2016). One can both feel shame and be shamed (Mercer, 2018), and as a result, one feels alone, rejected, or scorned by others (Volk et al., 2016). Over time this feeling can become internalized. Shame then becomes dispositional—shaping one's personality, identity and interpersonal relationships. This type of dispositional shame is referred to as "chronic shame" or "toxic shame" (Mercer, 2018; Bradshaw 1988 & Pattison, 2000 as cited in Park, 2016; Szentágotai-Tătar & Miu, 2016).

The difference between guilt and shame is a helpful distinction in understanding shame. Shame focuses on the self as the problem, whereas guilt sees the behaviour as the problem. In other words, guilt says, "I have done something bad," while shame says, "I am a bad" (Dearing et al., 2005 as cited in Chisholm & Gall, 2015). Guilt is adaptive for changing behaviour, while shame is maladaptive for changing behaviour (Chisholm & Gall, 2015; Park, 2016; Szentágotai-Tătar & Miu, 2016). One online study found that shame was the strongest predictor of hypersexuality, reduced motivation to change problematic behaviours, and fewer attempts to change. At the same time feelings of guilt predicted lower levels of hypersexuality and greater motivation to change problematic behaviours (Gilliland et al., 2011, as cited in Chisholm & Gall, 2015). Guilt has also been found to be unrelated to psychopathology, whereas dispositional shame has been linked to many psychological problems (Park, 2016).

Suicide

The greater the number of ACEs, the more significant increased suicidal ideation across the lifetime (Afifi et al., 2008; Bellis et al., 2014; Dube et al., 2001; Dube et al., 2003; Felitti et al., 1998, Radford et al., 2017; Sachs-Ericsson et al., 2016; Takizawa et al., 2014). Dube et al. (2001) found the population-attributable risk fractions for one or more ACE were 67% for lifetime suicide attempts, 64% for adult, and 80% for childhood/adolescent. Sachs-Ericsson et

al.'s (2016) review of ACE studies concluded that ACEs have persistent and multifaceted effects on suicidality in late life. This review cited 24 key articles between 2001 and 2015 linking ACEs to suicidal behaviour and another 36 articles that identified associations between ACE and other negative health and psychological outcomes. While not all studies differentiate effects of specific ACEs, some studies showed sexual and physical abuse having the strongest association with suicide (Bruffaerts et al., 2010; Hardt et al., 2008, as cited in Sachs-Ericsson et al., 2016), while others show sexual abuse having the strongest association (Molnar et al., 2001, as cited in Sachs-Ericsson et al., 2016).

Sachs-Ericsson (2016) proposes a heuristic framework through which to understand how ACEs may lead to suicide in later life. First, ACEs may affect biological processes such as neurological development, making people more sensitive to subsequent stressors and epigenetic gene expression, leading to increased risk of mood dysregulation, psychiatric disorders and suicidal ideation and behaviour. Secondly, ACEs are associated with psychiatric disorders and poor health functioning comorbidities, which increased the risk of suicide. Third, ACEs influence psycho-social development by shaping cognitive biases, problem-solving, coping skills, affective states, and interpersonal deficits. Finally, Sachs-Ericsson et al. propose that it is through the interaction of the above domains (biological, psychiatric and health, and psycho-social development) with the unique late-life stressors that suicidality in older adulthood emerges.

Sleep

ACEs may affect sleep over a lifetime. ACEs show a negative association with sleep disturbances such as trouble falling asleep, staying asleep, feeling tired post-sleep (Anda et al., 2006), insufficient sleep duration (Chapman et al., 2013, Sullivan et al., 2019), and nightmares (Agargun et al., 2003; Cuddy et al., 1992; Nielsen et al., 2019; Punamäki et al., 2005). Anda et

al. (2006) found four or more ACEs increases the risk of sleep disturbance 2.1 times. Chapman et al. (2013) found that 21.3% of no ACEs participants had frequent insufficient sleep as opposed to 47.0% of those with five or more. Sullivan et al. (2019) found ACEs increased the odds of chronic short sleep duration in adulthood and showed both a time-dependent and dose-response nature. Talvitie et al.'s (2019) study is one of the few that found no ACE effect on sleep in adulthood; however, their study had very broad definitions of ACEs such as parental smoking and changing schools. Talvitie et al. (2019) suggest, "the accumulation of more typical childhood adversities might not persistently affect self-reported sleep duration, sleep deficiency or sleep problems in adulthood."

Nightmares are significant among the sleep disorders associated with early adversity (Nielsen, 2017). Agargun et al. (2004) found nightmare sufferers were more likely to report incidents of childhood trauma. Cuddy et al. (1992) found sexual or physical abuse predicted frequent nightmares, frequent recurrent nightmare themes, and more difficulties returning to sleep after nightmares. No differences were found between participants abused before or after age 14. Nielsen et al. (2019) found self-reported adversity occurring as young as 0-6 years of age associated with nightmare severity and sleep spindle anomalies. Punamäki et al. (2005) found that adverse experiences before age 14 resulted in a more frequent recall of nightmares, more distress from nightmares, and a greater waking impact of nightmares compared to the no adversity group.

Sleep is interrelated to a number of other ACE impacts, such as mood and anxiety disorders (Park et al., 2021), impaired executive function and cognitive control (Meldrum et al., 2015), and delinquent behaviours (Hambrick et al., 2018). For example, sleep problems are both a symptom of mood and anxiety disorder and a probable path that links ACE-related mental and

physical health problems (Park et al., 2021). Park et al. (2021) found that depression and anxiety partially mediated the relationship between ACEs and sleep problems. Further impaired sleep is associated with poor executive function and cognitive control (Meldrum et al., 2015). Hambrick et al. (2018) found sleep partially mediated the association between ACEs and delinquency in adolescents.

Obesity

The ACE score has a graded relationship to obesity, with four or more ACEs increasing the risk of severe obesity by 1.9 times (Anda et al., 2006). In a meta-analysis of 41 studies, Danese & Tam (2014) found those with a history of childhood maltreatment, the maltreated group, were 1.36 times more likely to be obese than those without. Heerman et al. (2016) found children with two or more ACEs were 1.8 times more likely to develop adolescent obesity. Merrick et al. (2019) found four or more ACEs increase the risk of obesity 1.2 times. Further supporting these findings, Danese et al. (2014) found maltreated children had blunted elevation in leptin levels, relative to increasing levels of physiological stimuli, adiposity and inflammation, compared with a group of non-maltreated children matched for gender, zygosity and socioeconomic status. Leptin is an essential molecule in the regulation of both energy balance and immunity that promotes lipolysis, the breakdown of fat into energy.

Baldwin & Danese (2019) outline a plausible but mostly untested explanation for the underlying mechanisms involved in obesity's association with ACEs. ACEs could increase obesity risk by giving rise to a "thrifty" phenotype, typified by increased energy intake and storage and/or decreased energy expenditure (Danese & Tan, 2014). Maltreated children may eat more food due to alterations in reward processing, the HPA axis, and executive function (Baldwin & Danese, 2019). Maltreated children show reduced sensitivity to reward (Guyer et al.,

2006; Mehta et al., 2010). This may support overeating as a means of overcoming the reward deficit associated with ACEs (Baldwin & Danese, 2019). Consuming food high in calories has shown a reduction in HPA axis activation (Pecoraro et al., 2004). As a result, it is possible that food consumption is an unconscious attempt to self-medicate (Baldwin & Danese, 2019). As discussed previously, ACEs are associated with poor inhibitory control (Gould et al., 2012; Keller et al., 2017; Rubin & Zorumski, 2016; Selah et al., 2017; Treat et al., 2019), which may result in excessive food intake (Wonderlich et al., 2001).

ACEs may result in decreased energy expenditure due to alterations in metabolic hormones, mental health problems and abnormal immune functioning (Baldwin & Danese, 2019). ACEs are associated with low stimulated and basal levels of leptin (Danese et al., 2014). Lower leptin levels may reduce metabolic energy level availability from lipolysis (Baldwin & Danese, 2019). Mental health problems may contribute to a lack of exercise. For example, ACEs is associated with depression (Afifi et al., 2008; Anda et al., 2006; Edwards et al., 2003; Felitti et al., 1998; Radford et al., 2017; Selah et al., 2017; Takizawa et al., 2014) which is also associated with physical inactivity (Baldwin & Danese, 2019). Finally, ACEs are associated with higher inflammation levels (Danese et al., 2007) which can increase fatigue (Dantzer et al., 2008).

Cancer

Numerous studies have shown ACEs associated with cancer (Brown et al., 2010; Brown et al., 2013; Kelly-Irving et al., 2013; Merrick et al., 2019), cancer risk behaviours such as smoking, alcoholism, and decreased screening (Mouton et al., 2016), and cancer-associated health problems such as obesity (Mouton et al., 2016) and asthma (Santillan et al., 2003). This complex interplay of associations makes research challenging, but overall the research agrees that ACEs increase the risk of cancer.

Santillan et al.'s (2003) meta-analysis showed nearly two times the risk of lung cancer among persons with asthma independent of smoking history (Brown et al., 2006). Brown et al. (2010) found a positive association between ACEs and the risk of lung cancer. This association was particularly strong for those who died from lung cancer at younger ages. Further, they found that the increase in the risk of lung cancer was only partly due to relationships between ACEs and smoking. Brown et al. (2013) found approximately 10% of individuals exposed to ACEs were diagnosed with cancer and sexual abuse had the strongest association with an odds ratio (OR) of 1.21. Kelly-Irving et al. (2013) found that cancer risk in women before the age of 50 increased 2.1 times for those who had two or more ACEs. Merrick et al. (2019) found four or more ACEs increased the risk of cancer 1.4 times. Mouton et al. (2016) found ACEs associated with some cancer risk behaviours, including smoking, decreased mammography, and cervical cancer screening. This trend was not seen in prostate or colorectal cancer screening, suggesting possible gender differences. Alcalá et al. (2018) also found ACEs associated with lower cancer screening rates.

Substance Use

One of the most remarkable findings of the original ACE study (Felitti et al., 1998) was that individuals who experienced four or more types of ACEs were 4-12 times more likely to develop a substance use problem (LeTendre & Reed, 2017). Dube et al. (2003) found that each additional ACE increased the risk of smoking by 20–30 and self-reported alcoholism by 40-50%. Anda et al. (2006) found substance use increases as the ACE score increased with four or more ACEs increasing the risk of smoking 1.8 times, alcoholism 7.2 times, illicit drug use 4.5 times, and injected drug use 11.1 times. Kessler et al.'s (2010) study of 51945 adults in 21 countries estimate the eradication of ACEs would result in a 27.5% decrease in substance use disorders

(SUD). LeTendre & Reed (2017) found that each additional ACE was associated with at least a 34% increase in the odds of developing a clinically significant SUD in adulthood. Bryant et al. (2020) found having one or more ACE predicted having a SUD but did not find the dose relationship between ACEs and SUD that other studies have found.

Restoring Interventions for ACE Symptoms

In this section, I will examine a number of counselling modalities and interventions for restoring clients who are experiencing the negative neurological effects of ACEs on adult functioning. This is not intended to be an exhaustive section but rather to highlight some best counselling practices that one might consider in parallel to medical treatments for ACE outcomes. The criteria I used for selecting modalities were five-fold: 1) evidence-based—the modality has a body of supporting research, 2) trauma-informed—the modality understands the prevalence of trauma, recognizes its impacts, and that knowledge informs the work, 3) attachment-informed—it recognizes the lasting impact of early attachment relationships on how people relate to each other, how this impacts therapy and is intentional in using corrective emotional experiences to help clients earn secure attachment styles, 4) a depathologizing lens—it locates the solution within the client and the problem outside of the client, and 5) integrative—the modalities are complementary to each other in treating different aspects of ACEs. In particular, I will focus on SE for ANS regulation, EMDR for processing traumatic memories, and IFS for resolving internal self-alienation.

Somatic Experiencing

While talk therapies have been the traditional treatment for trauma, the work and research of Peter Levine, Allan Schore, Stephen Porges, Bessel Van Der Kolk, Janina Fisher, and Pat Ogden, among others, have brought new understanding to the importance of a bottom-up

approach that starts with utilizing the body's innate ability to regulate the ANS. While ACEs encompass more than just trauma, they both share ANS dysregulation as a significant source of pathology, making its' regulation an important goal of treatment. For purposes of this project, I will provide a brief overview of Peter Levine's SE and its' efficacy.

Overview

Levine developed his body-oriented approach based on his observations of how wild animals respond to a life-threatening event with no signs of trauma after the fact. According to Levine, trauma resides in the nervous system, not in the traumatic event itself (Heller & Heller, 2004). Levine says that trauma has to do with the ANS freeze response to perceived life threat (Somatic Experiencing International, n.d.-b). When the fight or flight response cannot overcome the threat, we freeze, also called tonic immobility or "playing dead." This makes us less threatening in the hopes the danger will leave us alone. Once the life threat is removed, the energy that our body had built up for fight or flight must still be discharged. Our body does this through shaking and trembling. If this process is unable to complete, then that energetic charge remains trapped. From our body's perception, then, it is still under threat. SE then works to release this stored energy and switch off the body's threat alarm that produces dysregulation and dissociation.

SE is a resiliency-based treatment for ANS dysregulation disorders, such as posttraumatic stress disorder, anxiety, depression, and physical pain such as chronic pain, migraines, and fibromyalgia (Briggs et al., 2018). Its' ideas and principles can be integrated into many other modalities (Heller & Heller, 2004). For example, attachment work is understood in how attachment wounds impact the nervous system (Somatic Experiencing International, n.d.-b, October & Carleton, 2009). SE is different from cognitive-behavioural and exposure

interventions in its bodily focus on interoception and musculoskeletal sensations rather than cognitions and emotional experiences (Payne et al., 2005). It does not require extensive nor complete retelling of the traumatic events. It does require the client to engage with traumatic memories that cause high arousal (Brom et al., 2017). Instead, the client learns to monitor the arousal and downregulate it in an early phase by using body awareness and applying self-regulation tools like engagement in pleasant sensations, positive memories, or other experiences that help regulate arousal. According to Payne et al. (2015), inner attention, in addition to the use of body awareness and interoceptive imagery, can lead to the resolution of symptoms resulting from chronic and traumatic stress. This is accomplished through the completion of thwarted, biologically based, self-protective and defensive responses and the discharge and regulation of excess autonomic arousal.

Principles of Treatment

According to October and Carleton (2009), SE has ten principles: 1.) Resources: SE helps the client identify resources in their life. A resource is anything that is positive in their life or imagination—sensations, images, behaviours, affects or meanings. Resources are considered reparative as they aid in titration. 2.) Narrative: SE uses narrative for tracking ANS activation, not as a search for memories. 3.) Resilience: SE therapists must work within a range of resilience, neither pressing through resistance nor providing catharsis. 4.) Pendulation and Titration: optimally, the ANS is wired to pendulate between SNS and PNS modes and traumatic material should be titrated or introduced in small amounts to maintain the client's ANS activation within their window of tolerance. 5.) Sensation, Image, Behaviour, Affect, Meaning (SIBAM): an SE therapist works with as many channels of the client's experience as possible in order to deepen and widen their experience as discharge can come through awareness of any

channel. 6.) Time: you must allow plenty of time for the nervous system to reorganize itself. 7.) Discharge: recognize signs of ANS discharge (Exhaling, yawning, burping, tingling, numbing, sense of flow, warmth/heat, sweating, crying, shaking, trembling, coughing). 8.) Dosing Arousal and Stabilization: The right amount of arousal allows a client to expand their tolerance; excess arousal results in reduced functioning. Stabilization is needed to allow the client's time to get used to increased energy in their system without reverting to old patterns of dysregulation. 9.) Continuity through Language: SE therapists use connecting words and phrases such as: "and then?" or "as you notice the resource in your lower body what is happening in your upper body?" to facilitate successful movement through the experience when the client is stuck. Because trauma interrupts the experience, the goal is for clients to move toward integration of the experience. 10.) Maintaining Stable Arousal: it's the therapist's job to manage client arousal, highly activated clients lose their capacity for creative self-regulation.

Efficacy

Brom et al.'s (2017) randomized control trial (RCT) showed SE had a large effect size (Cohen's $d > 0.8$) both on PTSD symptoms and depression symptoms. Andersen et al.'s (2017) RCT of PTSD sufferers with comorbid lower back pain showed that combining SE with physical therapy had a large effect size in reducing the number of PTSD symptoms and a moderate effect size in reducing fear of movement compared to physical therapy alone. Andersen et al.'s (2020) follow up RCT of PTSD sufferers with comorbid lower back pain compared physical therapy and SE to physical therapy alone and found no difference in outcomes between groups. Briggs et al.'s (2018) small study (n=8) SE showed moderate effect size on psychological quality of life and possible improvement in symptoms of depression, somatization for gender non-conforming individuals. Winblad et al.'s (2018) study showed that professionals who attend the 3-year SE

training showed a significant improvement in self-reported measures associated with resiliency, including physical and interpersonal quality of life and psychological symptoms (anxiety and somatization). According to Bission et al.'s (2020) review and meta-analysis of non-pharmacological and non-psychological interventions for PTSD, SE meets the 2018 International Society of Traumatic Stress Studies Prevention and Treatment Guidelines.

Eye Movement Desensitization and Reprocessing

Francine Shapiro developed EMDR to help trauma survivors access and process traumatic memories so they can be brought to an adaptive resolution (Shapiro, 2001, as cited by Cronk, 2018). Underlying this modality is the understanding that disturbing memories are typically processed in the brain by thinking, talking, and sometimes dreaming about the event (Shapiro & Maxfield, 2002); however, this process can be interrupted by highly charged memories. This interruption is considered the underlying cause of PSTD symptoms (Cronk, 2018). Solomon et al. (2009) explains,

> As the brain slowly processes the memory, it is transferred to the left cerebral cortex where it is filed away along with other memories and becomes part of one's life story. As with neutral memories, the stored information can be retrieved when needed to understand new experiences. Traumatic experiences are highly charged emotionally and overwhelm the brain's capacity to process information. The brain has difficulty integrating traumatic memories, connecting them with similar memories, and storing them. Instead, the episodic memory of the experience may be stored in the limbic system indefinitely. Dysfunctionally stored traumatic memories can lead to maladaptive coping strategies. (p. 393)

History

In 1987, Francine Shapiro was walking in the park when she realized that her eye movements seemed to decrease the emotion of a distressing memory (Prochaska & Norcoss, 2018, p. 187). Assuming that eye movements had a desensitizing effect, she experimented with this and found that others also had similar responses to eye movements. In 1989, the first controlled studies investigating what Shapiro called Eye Movement Desensitization for treating PTSD were published. Shapiro then began training clinicians and optimizing her procedures. It became apparent through this process that eye movements alone did not create comprehensive therapeutic effects, and so she added other treatment elements, including a cognitive restructuring component—resulting in the renaming it Eye Movement Desensitization and Reprocessing. In 1995, independent control studies were published, and the label "experimental" and the training restrictions were removed; as well, a textbook of procedures was published.

Overview

This section will briefly overview the eight-step process of EMDR treatment as described in Shapiro & Forrest (2016). 1.) History Taking: the client is encouraged to focus on their emotions and physical sensations surrounding the event rather than on recalling the details. 2.) Preparation: involves building client trust, assessing readiness to move forward, teaching grounding and stress reduction skills, as well as helping them create a safe-place image and psychoeducation on bilateral stimulation (BS). 3.) Assessment: after determining what event to target and the client is asked to provide three things: an image or scene that best represents the target event, a negative cognition/belief about yourself associated with that event, and a positive cognition/belief you would rather believe about yourself and the event. The client is then asked to rate how true the positive cognition feels on a scale of 1-7 and how disturbing the target is on

a scale of 0-10 and to describe what they experience in their body. 4.) Desensitization: Without talking to the therapist, the client focuses on the image representing the target event while following the BS (typically the therapist's fingers or a light bar.) Client pays mindful attention to any thoughts, beliefs, insights, emotions or sensations that arise. This process is repeated until the client rates their distress level at a zero. In between sets of BS, the therapist may also throw some things in for the client to think about; these are called *cognitive interweaves*. 5.) Installation: Uses that same BS process, but client now focuses on the positive cognition/belief that they chose. In between BS sets therapist has the client assess how true that positive cognition feels to them, using the same 1-7 scale from the assessment phase. When the client gets to a 7, meaning completely true, this phase is complete. 6.) Body Scan: the client is asked to mentally scan their body to find any sensations or tensions that signal that something is still stuck regarding the target. If there are any lingering sensations, the therapist will continue with the BS. 7.) Therapist closes down the material and does a final check using various methods, including imagery, to contain any unprocessed material. This may include returning the client to a safe place or creating a container to hold anything that may become too distressing in between sessions. 8.) Reevaluation: the beginning of the next session begins with reevaluating the client's disturbance level regarding the previous target. This helps to determine if client disturbance is still neutral and if the client's positive cognition still rings true. If the previous session was incomplete, the cycle begins again at phase four.

Efficacy

EMDR has a large body of research supporting it. A number of meta-analyses have shown that EMDR is an effective treatment of PTSD with large effect sizes compared to control conditions and similar effects to CBT (Boccia et al., 2015; Bisson & Andrew, 2007; Chen et al.,

2014; Cusack et al., 2016; Ehring et al., 2014; Hoogsteder et al., 2021; Khan et al., 2018; Moreno-Alcázar et al., 2017). Another measure of effectiveness is the numbers needed to treat (NNT). EMDR, Prolonged Exposure Therapy, Cognitive Process Therapy, Cognitive Therapy, CBT-mixed Therapies showed <4 NNT to achieve a loss of PTSD diagnosis (Cusack et al., 2016). It has been shown effective with children (Khan et al., 2018; Moreno-Alcázar et al., 2017; Hoogsteder, 2021) and across cultures (Cronk, 2018). Although there remains a number of questions about how and if the eye movements contribute to the therapeutic process, Lee & Cuijpers (2013) meta-analysis of dismantling studies, in which the process was compared with and without eye movements, suggests they do contribute to the effectiveness of treatment. EMDR is best known as a treatment for PTSD; however, it has since been used in treating many other mental health problems (Cuijpers et al., 2020) EMDR is used in other disorders such as bipolar disorder, conduct disorder, and eating disorders because traumatic memories, current stresses, and future challenges have a significant role in these disorders (Cuijpers et al., 2020).

Internal Family Systems

Richard C. Schwartz developed IFS in the 1980s while working with eating disordered (ED) adolescents who regularly talked about their internal conversations with “different parts.” (Anderson et al., 2017). Using his clients’ own words, he dubbed their subpersonalities “parts.” Schwartz discovered in borrowing from his family systems experience that he and his client could encourage a difficult part to allow the client some mental separation from its own distorted perspective. When a part is able to step back, the client would suddenly become nonjudgmental and curious toward their part. This new and compassionate relational posture between the client and their parts has proved to be the essential mechanism of change in IFS therapy.

Overview

The IFS is a mindfulness-based modality that holds a plural model of the mind, stating that everyone has an "internal system of countless parts who interact internally with each other and externally with other people" (Anderson et al., 2017; p. 3). As well, the mind holds a core resource called "the Self" that is not a part, which can be recognized by qualities such as curiosity, calm, clarity, connectedness, confidence, courage, creativity and compassion. IFS divides parts into three categories based on their roles: injured parts called "exiles," proactive protective parts called "managers," and reactive protective parts called "firefighters." Exiles are parts that have been shamed, dismissed, abused, or neglected in childhood. They have been exiled by protective parts either for their own safety or to prevent their emotional pain from overwhelming the system. Managers concentrate on learning, functioning, preparing, stability, and preventing exiles from flooding the system with their emotion. While they are hard-working, managers can be relentless in their criticism and shaming. Firefighters activate when an exile's memories and emotions break through the manager's wall. When this happens, firefighters react by attempting to extinguish the pain in ways that managers despise, such as alcohol, drugs, overeating, shopping, sex, self-harm, suicide, and homicide.

Goals

The first goal of IFS is called unblending (Anderson et al., 2017). The therapist supports the client to help their protective parts differentiate or unblend. The second goal is to support the client in befriending their firefighters/managers and get permission to help exiles. The third goal is to support the client to form a constructive relationship with their exiles, listen to their experiences, and help them heal by letting go of extreme or damaging feeling states and beliefs.

This then releases protectors and allows the healed parts to reintegrate, and restores the Self as leader of the inner system.

Underlying Assumptions

According to Anderson et al. (2017), IFS has six major underlying assumptions: 1.) All parts have good intentions, even those who misbehave. Therefore therapy begins with the invitation that all parts are welcome. 2.) The psychic response to injury is predictable: when parts are wounded, other parts step into protective roles. 3.) Protective parts behave predictably and can appear pathological. 4.) A destabilized, disrupted inner system is able to reintegrate and balance itself when it is in relationship with the Self. 5.) The Self is intrinsic and present from birth. It is neither created nor cultivated and cannot be destroyed. 6.) Everyone has a Self; it can be accessed for healing in every person.

Efficacy

The IFS model is acknowledged by the National Registry of Evidence-based Programs and Practices (NREPP) as an evidence-based treatment for effectively improving phobia, panic and generalized anxiety disorder, physical health conditions, and depressive symptoms (Matheson, 2015). IFS has been shown to be beneficial in a number of qualitative studies, including a teenager dealing with shame (Sweezy, 2011), blended families (Carter, 1999), an African American mother supporting their sexually abused daughter (Wilkins, 2007) and increasing self-awareness in new therapists (Mojita et al., 2014). Shaddick et al.'s (2013) RCT of rheumatoid arthritis sufferers showed IFS improved overall pain and physical function that was sustained at the one-year follow-up with participants self-reporting improved joint pain, self-compassion, and reduced depressive symptoms. Haddock et al.'s (2016) RCT of depression among female college students found IFS had similar effectiveness at CBT or Interpersonal

Psychotherapy (IP). DiGloria's (2020) pilot study found that an integrated IFS and Mindful Self-Compassion (MSC) program supported improving same-sex couples relationship satisfaction, depressive symptoms, mindful attention and awareness, and self-compassion.

Schwartz and many other trauma experts have endorsed IFS as a beneficial approach to trauma (Twornbly and Schwartz 2008; Schwartz et al. 2009; Schwartz and Sparks 2014, as cited by Lucero et al., 2018). However, there is no direct research on IFS effectiveness for trauma. There is some secondary supporting evidence for IFS found in methylenedioxymethamphetamine (MDMA) or "ecstasy" research for the treatment of PTSD, which has shown great promise and is currently in phase three of clinical trials. In the first two clinical trials, MDMA-assisted psychotherapy was shown to be efficacious and well-tolerated in a large sample of adults with PTSD (Mithoefer et al., 2019). Interestingly, these studies showed a large number of participants experiencing parts or multiplicity during their treatment.

> In one MDMA study of veterans, firefighters, and police officers who had been diagnosed with PTSD, Mithoefer (2013) added an additional internal pilot study for which he developed a "parts-work" measure to discover the rate at which study participants spoke of parts while in session. This measure revealed that awareness of parts came up in 78% of the active-dose MDMA-assisted sessions. Additionally, therapists in 92% of active-dose sessions, compared to 29% of low-dose sessions, observed marked increases in qualities that signify Self-energy, along with "greater understanding and acceptance of these parts" (p. 13) (p.673; Schwartz & Sweezy, 2019).

In fact, although the therapists in these studies are encouraged to be non-directive, the manual for treatment in these studies states that,

Therapists may benefit from training and experience in Internal Family Systems Therapy (IFS) or some other model (e.g. Voice Dialogue or Psychosynthesis) that recognizes and addresses the multiplicity of the psyche [3, 23-25]. Training in these methods provides an understanding that multiplicity in the absence of high degree dissociation is a normal phenomenon. This approach is applicable because multiplicity often becomes more apparent during MDMA-assisted psychotherapy. During and after MDMA-assisted sessions, the participant may have a heightened awareness of different parts of their psyche and it is essential that such experiences not be pathologized by the therapists (Mithoefer et al., 2013).

Chapter Summary

In chapter two I reviewed some of the relevant literature on ACEs roots, results, and restoration. First I reviewed some of the many interconnected neurobiological origins of ACE associations on the development and function of our brain and body, our personality, our nervous system, our cortisol levels, our immune system, our endogenous opioid system, and our cell telomeres. The NMT reveals how ACEs inhibit development of the specific brain structures and functions that are developing at the time of the experience and that ACE treatment modalities must be matched to clients current functioning. The SDM demonstrates how right and left brain splitting or fragmenting of personality is an adaptive survival strategy providing psychological distance from ACEs and resolving the survivors contradictory needs and drives but if unresolved can lead to self-alienation. AT explains how our early experiences with our primary caregiver are important our brain encodes it emotionally not as love, but as safety thus shaping how we related to others throughout our lives. PT explains the nervous system's role in safety, socializing, and survival responses and how ACEs can result in maladaptive functioning. The cortisol stress

response is designed to be an adaptive response to threat, but ACEs can lead to inappropriate responses such as too little, too much, or for too long can be harmful increasing risk for cardiovascular, metabolic, immune, cognitive, and emotional disorders. ACE exposure can negatively affect immune system development leading to compromised or over-functioning. Many behaviours associated with ACEs, such as self-harm and addiction may be unconscious attempts to self-regulate an EOS is was impaired or dysregulated by ACEs. Accelerated biological ageing, measured by telomere length is associated with ACE exposure. Second, I looked at how ACEs negatively affect our minds, our bodies, our behaviours, and our relationships through increasing our risk of anxiety and depression, shame, impaired cognitive function, impaired sleep, obesity, cancer, and substance use. Finally, in Restoring Interventions For ACE Clients, I reviewed three treatment modalities for ACEs (SE for ANS regulation, EMDR for processing traumatic memories, and IFS for resolving internal self-alienation) based on five criteria: evidence-based, trauma-informed, attachment-informed, a depathologizing lens, and integrative.

Chapter 3: Analysis and Conclusion

In Chapter 1 I defined Adverse Childhood Experiences (ACEs) and overviewed the wide-ranging impacts ACEs have on physical health, mental health, and vulnerable populations. I discussed how almost everyone stands to benefit from understanding ACEs better and located ourselves within the topic. I then defined many key terms and finally proposed headings (roots, results, restoration of ACEs) for my literature review to help us imagine a better response. In chapter two, I reviewed the relevant research literature on ACEs: 1.) How ACEs shape our brain and body function, our brain and personality, our brain and relationships, our nervous system, the hypothalamic-pituitary-adrenal axis' regulation of the stress hormone cortisol, the immune system, the endogenous opioid system, and cell telomeres. 2.) How ACEs increase the risk for anxiety and depression, impaired cognitive function, sleep disturbance, obesity, cancer, and substance use. 3.) How Somatic Experiencing (SE), Eye-Movement Desensitization and Reprocessing (EMDR), and Internal Family Systems (IFS) can help us restore clients suffering from ACE impacts.

In this chapter I will review the purpose of my capstone project on ACEs and what I diversed in the research literature. I will highlight what I appreciated from the research and discuss the limitations I found. Finally, I will propose a depathologizing response to ACEs.

Capstone Purpose

In this project I sought to depathologize ACEs by increasing understanding of the causes of ACE associations for example, how this results in maladaptation in our physiology, behaviour, and relationships to best help respond to, restore, and foster resiliency in those impacted by ACEs. I intend to bring a holistic awareness to treating medical conditions known to often be the result, in part, of untreated somatic survival responses to trauma and stress; to reframe ACEs

impacts through a lens that views them as the natural defense responses of our mind and body attempting to survive and protect itself, and to consider how one might use this information to interrupt the cycle of oppression perpetuated on people vulnerable to ACEs.

Research Literature Summary

ACEs are stressful or traumatic events experienced during childhood with a strong, graded relationship with life-long negative outcomes. These outcomes include poor mental health, poor physical health, and risky or harmful behaviours. ACEs are more likely to impact vulnerable populations (women, racial minorities, high school dropouts, low income, unemployed, unable to work, and gay or lesbian populations); however, the research shows that ACEs affect most people across countries, populations, and cultures.

ACEs affect the development and function of our brain and body, our personality, our nervous system, our cortisol levels, our immune system, our endogenous opioid system, and our cell telomeres. The Neurosequential Model of Therapeutics (NMT) reveals how ACEs inhibit the development of the specific brain structures and functions that are developing at the time of the experience and that ACE treatment modalities must be matched to the client's current functioning. The Structural Dissociation Model (SDM) demonstrates how right and left brain splitting or fragmenting of personality is an adaptive survival strategy providing psychological distance from ACEs and resolving the survivor's contradictory needs and drives, but if unresolved, can lead to self-alienation. Attachment Theory (AT) explains how our early experiences with our primary caregiver are so important our brain encodes it emotionally, not as love, but as safety, thus shaping how we related to others throughout our lives. Polyvagal Theory (PT) explains the nervous system's role in safety, socializing, and survival responses and how ACEs can result in maladaptive functioning. The cortisol stress response is designed to be an

adaptive response to threat, but ACEs can lead to inappropriate responses such as too little, too much, or for too long can be harmful, increasing the risk for cardiovascular, metabolic, immune, cognitive, and emotional disorders. ACE exposure can negatively affect immune system development leading to compromised or over-functioning. Many behaviours associated with ACEs, such as self-harm and addiction, may be unconscious attempts to self-regulate an EOS impaired or dysregulated by ACEs. Accelerated biological aging, measured by telomere length, is associated with ACE exposure.

These interconnected neurobiological systems at the root of ACEs negatively affect our minds, bodies, behaviours, and relationships. ACE research demonstrates a dose-response in increasing the risk of anxiety and depression, impaired cognitive function, impaired sleep, obesity, cancer, and substance use. Three evidence-based, trauma-informed, attachment-informed, depathologizing, and integrative modalities for ACEs are SE for Autonomic Nervous System (ANS) regulation, EMDR for processing traumatic memories, and IFS for resolving internal self-alienation.

Research Limitations

This capstone was limited in several ways: First, given the complex interconnectedness of the underlying neurobiology involved in ACEs, causality and directionality are very difficult, if not impossible, to determine with any confidence. Second, in identifying ACE associations, particularly later in life, there many different moderators and mediators that can confound the extent and nature of the relationship (Sachs-Ericsson et al., 2016). For example, anxiety and depression are associated with ACEs, but they are also associated with many other ACE impacts such as cognitive function, sleep, suicide, obesity, and substance use. Another example is that attachment style was shown to moderate effects on cell telomere length (Dagan et al., 2018).

This supported other research showing parenting can shape pathways toward physical well-being despite ACEs (Cicchetti & Blender, 2006; Gunnar & Quevedo, 2007, as cited in Dagan et al., 2018).

Finally, many leading trauma therapists recommend somatic modalities, IFS, and EMDR; however, there is very little high-level empirical evidence for somatic modalities upon reviewing the literature. SE only has three randomized control trials (RCTs) published: one on the treatment of post-traumatic stress disorder (PTSD; Brom et al., 2017) and the other two on chronic lower back pain co-morbid with PTSD (Andersen et al., 2017; Andersen et al., 2020). Brom et al. (2017) supported the efficacy of the therapy, even though a reversal of clinical symptoms as measured by the Clinician-Administered PTSD Scale was moderate at 44.1 percent. However, the second RTC did not demonstrate a similar level of effect as all outcomes were not clinically meaningful (Andersen et al., 2017). The third study showed no difference between physical therapy and physical therapy with SE (Andersen et al., 2020). Other studies performed were mostly exploratory studies that did not have controls, randomization or replication. Similar critiques could be brought to IFS, which only has two supporting RCTs, one on pain reduction and physical function in Rheumatoid Arthritis (RA) sufferers (Shaddick et al., 2013) and one on depressed female college students (Haddock et al., 2016). Other studies available are primarily qualitative case studies. There are currently no direct studies supporting IFS for treating trauma with only secondary evidence in psychotherapy-assisted 3,4-Methylenedioxymethamphetamine (MDMA) clinical trials. EMDR has a large body of empirical evidence; however, there is still debate about the purpose and necessity of bilateral eye movements compared to prolonged exposure.

A Depathologizing Response to ACEs

Based on the research, I will propose a depathogizing lens for responding to ACEs. Traditional notions pathologize people by locating the problem within the person by labelling it a disease (a problem with your body), a disorder (a problem with your mind/personality), or problematic behaviour (a problem with your morality). When one locates the problem within ourselves, we experience the self-alienating and the maladaptive emotion of shame. In this section, I will propose five assumptions to help us as therapists and our clients depathologize our ACEs.

ACE Impacts Are Symptoms

ACE impacts are not the problem, they are a symptom of the problem. For example, addiction is not the problem. Addiction is problematic because it is often harmful, but it's not the problem. Instead, ACE research invites us to investigate and treat the underlying problem that have created the symptoms such as: disconnection, isolation, alienation, shame, rejection, fear, abuse, neglect, and nervous system dysregulation to name a few. Too often, oppressed and vulnerable groups are dehumanized by the coping mechanisms they have adopted to survive the trauma of their experiences. Depathologizing seeks to understand coping strategies such as addiction as resiliency and resistance in the face of systemic harm.

Using addiction as an example, SE understands addiction as a symptom of unconscious attempts to regulate the users' dysregulated nervous system. The specific substance of choice provides clues for the therapist about what state of dysregulation the client is in—stimulants usage indicating the client's is in a chronic dorsal vagal (freeze) state and the client is needing support upregulating themselves, depressant usage indicating a client is in a chronic sympathetic nervous (fight/flight) state and the client is needing support downregulating themselves.

EMDR understands the symptom of addiction as a maladaptive coping strategy developed to deal with the underlying problem of dysfunctionally stored traumatic memories. Thus EMDR focuses treatment on desensitizing and reprocessing these memories in order to remove the client's need to actively suppress them through their maladaptive coping strategy.

IFS provides a helpful framework understanding addiction and the shame cycle common to addiction as a symptom of maladaptive self-protection by parts. This cycle starts when the exile is triggered and floods the system. The reactive firefighter part's then attempts to distract or numb the sensation and emotional pain of the triggered exile through use of their preferred substance. The self-hatred and shame felt post-use is understood as the manager part's criticism of the firefighter. This internal shaming dynamic typically result in the firefighter's attempts once again to numb the pain.

Treat The Problem, Not The Symptoms

If the underlying problems behind ACE symptoms are disconnection, isolation, alienation, shame, rejection, fear, abuse and dysregulation then focusing treatment on the symptoms is a temporary solution. Like swatting flies without cleaning up the stuff attracting them, they almost always return, and sometimes they bring their bigger and scarier friends. Our clients typically come to therapy seeking help for their symptoms, but those are typically just the external manifestations of something deeper. Treating symptoms looks like taking anti-depressants without understanding and changing how you got depressed in the first place, or only taking diuretics for weight loss, or expecting willpower alone will help you quit an addiction. Taking this even further ACEs research invites us to ask if medical conditions such as heart disease and cancer are ACE symptoms that could or should be treated at least partially through counselling. More often than not when we as counsellors concentrate solely on treating ACE

symptoms, we become focused behaviour and thought management. This risks the unintentional pathologizing of our clients because we subtly reinforced the message that the problem is located in their body, thoughts, behaviours, or morality rather than their ACEs.

Treating the root problem behind ACE symptoms is found at the core of the modalities we examined in Chapter 2. For example, SE states that it is an event's effect on our nervous system that is problematic more than the event itself. Thus trauma resides in our body and nervous system, not in the event. SE then focuses on treating the root ACE problem of nervous system dysregulation using the client's own body and resources. IFS focuses on treating the root ACE problem of disconnection from Self and the self-alienation created by the contradictive protective responses of parts. EMDR treats root ACE problems such as: rejection, fear and abuse by helping the brain desensitize and process traumatic memories into long-term storage which it has been unable to because the intense emotional content of the memory interrupted this process. As a result, we reexperience the trauma as a present event when the memory is accessed rather than just remembering as an event in the past. Attachment Theory, which informs SE, IFS, EMDR and many other modalities helps treat the root ACE problem of disconnection and isolation from others. By helping clients heal attachment wounds and earning more secure attachment styles they are able to participate more fully in trusting and secure relationships.

Our Symptoms Are Still Trying To Protect Us

The third assumption is that symptoms result from what went right in a client's life to survive their ACEs. Short-term survival responses are rarely adaptive long-term, and thus we see the devastating cost of surviving ACEs in our physical health, mental health and relationships. Yet, when we retroactively view the things our mind and body did to survive as maladaptive because they no longer help us, we become further alienated from ourselves. Instead, suppose we

can approach ourselves with the compassion that comes through a deep conviction that our current symptoms were initially and have always will be trying to protect us.

In SE the short-term protective fight/flight/freeze response activated by our nervous system to survive our ACEs are interrupted and result in a chronic state of dysregulation. The alarm bells continue to ring long after the robbers have left because the biological signal for safety has failed to arrive. Thus our body is still trying to protect us from a danger long past.

In EMDR, many ACE symptoms such as flashbacks and nightmares are understood as the interrupted attempts by our brain to process and store our ACE experiences in long-term memories due the disturbing emotional and sensate content. Like a skipping record, the brain unsuccessfully continues its' attempt to process this content resulting in symptoms such as chronic nightmares and flashbacks.

In IFS a core assumption is that all parts have good intentions, even those who misbehave (Anderson, 2017). Thus IFS therapy begins with the invitation that all parts are welcome. This requires us as therapists to interpret all client behaviour as protective in some way even when on the surface it appears to produce the opposite results. This reorientation allows us to move towards self-compassion, integration, and invitation to learn new and adaptive ways to protect ourselves.

Integrative Compassion

In truly understanding the protective motivation behind the symptoms (our parts, our behaviours, our energetic state, our physical and mental limitations, and our memories) that frustrate and alienate us from ourselves and others, then we can begin to step into true compassion for ourselves and compassion for others. Orson Scott Card's character Ender expresses this concept beautifully when he says,

> In the moment when I truly understand my enemy, understand him well enough to defeat him, then in that very moment I also love him. I think it's impossible to really understand somebody, what they want, what they believe, and not love them the way they love themselves. And then, in that very moment when I love them... I destroy them. (Card, p. 238)

When we know our enemy completely, we can love them, and at that moment, we destroy them by making them our friends. True self-compassion comes through understanding the aspects of ourselves we most want to reject, and true compassion for others comes through truly understanding their story.

Not only does SE help us to understand the protective nature our nervous system states but SE seeks to help the clients return to the safety of and maintain a ventral vagal state. In this state, we are not only able to feel safe but also able to access the resources of the prefrontal cortex such as empathy, impulse control, planning for the future, and coordinating complex behaviours. These resources are essential in being able to find compassion for ourselves and others and are only available to us when we are in the calm, grounded ventral vagal state.

In EMDR because traumatic memories are unable to be processed and stored as complete past events, the constant replay of these events such as leave us feeling unsafe, regretful, self-blaming, and second guessing. Introducing an interweave in EMDR can be a helpful way to diffuse and reframe these painful emotions and interpretations that come from the block processing of our experience. By reprocessing the memory with a positive interweave such as allowing the body to move a desired way, or reminding the client that it's over they are often able to move from being stuck to being able to adaptively process the memory.

IFS is particularly suited to this assumption as it is design is to help the parts step back from their internal conflicts to begin to understand how the other parts they are in conflict are also acting from a motivation of protection. To help all the parts learn to trust the Self with their burdens and become integrated with the whole as they allow the Self to lead. This becomes most apparent when the protective parts are able to step back and allow the Self to work with the exile parts. The exile, as the name implies, is often the aspects of us that our parts most resent and avoid and often have the hardest time showing compassion towards. As the exile is allowed space to be understood and unburdened the protective parts are able to view them from the compassionate perspective of the Self. As the exile finds healing and safety in the Self the protective parts no longer have to work to avoid the exiles pain because it no longer is overwhelming the system. The end results is increased compassion and parts that can appreciate each other more and work together as a whole led by the Self.

The Solution Lies Within

The final assumption is that the solution is located within the person. In SE, it is guiding the body's own innate abilities, resources, and resiliency to complete biological processes that were interrupted. In EMDR, it can be seen in the client's positive replacement cognition and using the eye movement activate the brain's ability to process and consolidate disturbing memories. In IFS, this is called the Self, which is accessible to everyone for healing. This is not advocating for self-help or that there's no need for help from others, or that external resources such as spirituality are not part of the solution, but rather it is a strength-based conviction that believes that people are innately resilient and have access to everything they need to move towards healing and that we as therapists should help clients access and leverage this innate wisdom and ability located within ourselves.

In SE a traumatic event is defined as an experience that causes a long-term dysregulation in the autonomic and core extrapyramidal nervous system (Levine, 1997). According to Levine, the commonly used term post-traumatic stress disorder (PTSD), suggests pathology; however SE, (developed several years before PTSD's definition and inclusion in the DSM III) views the trauma response as part of a natural, non-pathological process that has been interrupted. SE, therefore finds the solution to trauma in completing these interrupted processes by using our body's own innate ability and resources to regulate itself. SE works to help the client build their innate body sensations associated with safety and comfort before approaching any sensations associated with trauma. Once established these safety sensations become the reservoir of innate, embodied resource for the client to return repeatedly as they encounter titrated trauma sensations. This titrated pendulation between stress and safety allows the biological completion and autonomic discharge to occur in measured and manageable steps. Thus the solution in SE is found within the clients own biological resources.

In EMDR the bilateral stimulation is thought to replicate the eye movements of rapid eye movement (REM) sleep in which it is theorized that the brain is able to process our experiences and store them in long-term memory. This process is thought to be interrupted when we are woken up by the emotional content of traumatic memories. By simulating REM sleep while a client is awake a client is able to complete the processing of memories without being interrupted by sleep disturbances and as a result the traumatic memories are able to resolve into long-term memory as completed events rather than present experiences. Thus too in EMDR the therapist is strengthen and utilizing the client own innate ability to process memory in order to create change and healing for the client.

In IFS a destabilized, disrupted inner system is able to reintegrate and balance itself when it is in relationship with the Self. The Self is intrinsic and present from birth. It is neither created nor cultivated and cannot be destroyed. It is characterized by the traits of curiosity, calm, clarity, connectedness, confidence, courage, creativity and compassion. This is the essential mechanism of change/healing in IFS. Thus a primary goal is to help clients increasingly become more Self led rather than parts led. One of the first steps in IFS is to help a client unblend from their parts. Often clients are unaware of their parts and often over-identify themselves with certain parts, typically, the manager/self-critical parts. This internal triangulation contributes to the internal conflict rather than helping resolve it. Instead unblending is helping the client to recognize their Self as separate from their parts and helping the Self to befriend their parts. To have the parts unburden themselves upon the Self, to begin to trust the Self as an attachment figure, providing the loving protective role, the parts, themselves once step into the absence to provide.

Summary

The purpose of this capstone was to explore the neurobiological roots of and ACEs symptoms and reframe them as the natural defence responses of our mind and body attempting to survive and protect itself, and to consider how we might use this information to interrupt the cycle of oppression perpetuated on people vulnerable to ACEs by proposing a depathologizing lens.

ACEs are stressful or traumatic events experienced during childhood with a strong, graded relationship with life-long negative outcomes, including poor mental health, poor physical health, and risky or harmful behaviours. ACEs are more likely to impact vulnerable populations (women, racial minorities, high school dropouts, low income, unemployed, unable to

work, and gay or lesbian populations); however, the research shows that ACEs affect most people across countries, populations, and cultures.

ACEs affect the development and function of our brain and body, our personality, our nervous system, our cortisol levels, our immune system, our endogenous opioid system, and our cell telomeres. These interconnected neurobiological systems at the root of ACEs affect our minds, bodies, behaviours, and relationships. ACE research demonstrates a dose-response in increasing the risk of anxiety and depression, shame, impaired cognitive function, impaired sleep, obesity, cancer, and substance use. Three evidence-based, trauma-informed, attachment-informed, depathologizing, and integrative modalities for ACEs are SE for ANS regulation, EMDR for processing traumatic memories, and IFS for resolving internal self-alienation. The research for this project was limited by the complex interconnectedness of the neurobiological systems, the numerous moderators and mediators confounding the relationship between ACEs and adulthood, and the lack of high-quality research into SE and IFS treatment modalities and EMDR mechanisms of change.

I proposed a depathogizing lens for responding to ACEs characterized by five assumptions: 1.) ACE impacts are symptoms of the problem, 2.) Treat the problem (isolation, alienation, shame, rejection, fear, abuse, neglect and nervous system dysregulation), not the symptoms, 3.) Our symptoms are still trying to protect us, 4.) Integrative compassion for ourselves and others comes through a deep understanding of the symptoms' motivation (5.) The solution lies within our own innate wisdom and ability to heal.

Conclusion

In conclusion, ACEs are an important area of research in the last 25 years, revealing the lasting impacts of childhood stress and trauma on our minds, bodies, behaviours, and

relationships. This research challenges traditional notions that locate ACE sufferers' mental and physical problems and problematic behaviours within the person—in their mind, body, and morality. Instead, ACEs research invites us to consider how our protective biological survival responses can have devastating outcomes when they are chronically activated in our early years of development. As a result, it is vitally important that we bring a depathologizing lens to the people affected by ACEs to bring integration, compassion, and healing.

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