Attachment Traumatology: Interpersonal neurosynchronistic phylogenesis

Orientation: Dyadic trauma is contagious. Converging neurosynchronistic constructs and the application of attachment focused-somatic experiencing (AF-SE) to traumatised dyads have revealed phenomena that required examination of the relationship between trauma, attachment and community psychopathology.

Research purpose: The phylogenetic impact of trauma on attachment is under-reported in attachment traumatology. The purpose of the study was to introduce the theory of dyadic trauma, and SPA and interpersonal neurosynchronistic phylogenesis (INP) as constructs to explain the relationship between trauma, attachment and community psychopathology.

Motivation for the study: Widespread loneliness and loss of social cohesion indicate significant, trauma-driven phylogenetic shifts in secure phylogenetic attachment (SPA). Interpersonal neurosynchronistic constructs emerged to elucidate the phenomena.

Research approach/design and method: Conceptualisation based on a synthesis of pertinent research provided for an analysis with theory adaptation as an approach. Secure phylogenetic attachment transposed interpersonally is compromised by maladaptive-interpersonal neurosynchronistic phylogenesis (M-INP). Attachment traumatology was chosen as the domain theory and INP as the method theory.

Main findings: Maladaptive-interpersonal neurosynchronistic phylogenesis is complicit in community psychopathology. It was found that INP served as a valuable method theory in generating new insights regarding dyadic trauma, attachment and psychopathology. Three unique categories of attachment, namely SPA, the antithesis of trauma, traumatic and monozygotic attachment were proposed.

Implications for practice: Attachment traumatologists are provided with a theoretical model, dyadic trauma and descriptive terminology to elucidate the phylogenetic impact of trauma on attachment.

Contribution/value add: Specific nomenclature described the interpersonal neuro-dynamics of INP and its functional role in traumatic attachment thereby indicating a paradigm shift in attachment traumatology.

Keywords: attachment focused-somatic experiencing; dyadic trauma; dyadic completion; interpersonal neurosynchronistic phylogenesis; loneliness; monozygotic attachment; monozygotic attunement; secure phylogenetic attachment and social isolation.

Introduction

Escalating community psychopathology is evident in widespread loneliness, social isolation and loss of social cohesion (Riordan et al., 2019). Loneliness and social isolation have been identified as risk factors in mortality (Holt-Lunstad et al., 2015).

There is a linear, interpersonal neurosynchronistic relationship between trauma, loneliness, loss of social cohesion and psychopathology. Stress induced perturbations in the brain of the traumatised person emphasise vigilance in the survival networks of the limbic brain, specifically, innervation of the amygdala (Riordan et al., 2019) generating maladaptive-interpersonal neurosynchronistic phylogenesis (M-INP) and contagious dyadic trauma resulting in the loss of secure phylogenetic attachment (SPA) initiating avoidance psychopathology. This in turn compromises social engagement with loss of trust in relationships due to increased fear-vigilance that then leads to interpersonal perturbance and loss of attunement in the dyad. The inevitable outcome after unresolved dyadic trauma is a tendency towards avoidant social isolation, loneliness, and loss of social cohesion across relationships, families and communities.
There may be many reasons for loss of social cohesion in our communities, such as popular immiseration, misconduct on social media, a dramatic reduction in the face-heart connection (Porges, 2011), which is a primal social survival imperative in human bonding, loss of trust in our traditional institutions, divergent social and political norms, and geopolitical tensions. However, loneliness and social isolation are initiated predominantly by the trauma-based emotions namely fear, anger, grief, guilt, powerlessness, abandonment, shame and disgust.

Cognitive behavioural therapy (CBT) and talk therapies primarily engage the social networks of the prefrontal cortex as the dominant change and recovery agency of treatment for trauma. However, the neural origins and survival imperatives of trauma exist in the limbic and primitive brain structures that initiate flight, fight, freeze neurosequences, (Payne et al., 2015) and the polyvagal network of dorsal vagal collapse (Porges, 2011), indicating that subcortical and somatic representations of trauma may be more accurate targets for psychotherapy. While uncompromised SPA promotes social cohesion, trauma impacts the interpersonal synergy of SPA, manifest in dyadic trauma which is then transposed through M-INP in communities leading to escalating psychopathology.

The purpose of this study is therefore to examine the role of INP in attachment traumatology to determine the functional dynamics involved in the inter-relatedness of attachment, trauma and psychopathology. The interpersonal neurosynchronistic mechanisms of trauma as a contagion and the maladaptive impact of somatic perturbations in attachment dyads after trauma can be explained with the theory of dyadic trauma and in particular the construct of INP. Attachment focused-somatic experiencing (AF-SE) is an integrated somatic, neurobiological intervention with demonstrated utility to resolve trauma as a contagion in attachment relationships (Riordan, 2022). Elucidation and reconceptualisation of the nature and phylogenesis of trauma in attachment dyads may explain emerging phenomena and therefore offer AF-SE as an alternative treatment modality to address dyadic trauma.

Research design

Research approach

The relationship between trauma and community psychopathology was considered through the lens of recent discoveries identifying dyadic trauma and completion as phylogenetic mechanism of trauma and recovery. The constructs of SPA as the antithesis of trauma, dyadic trauma, dyadic completion, and monozygotic attunement were assessed quantitatively and inferentially in previous studies, and these studies (Riordan, 2022; Riordan et al., 2017, 2019) are synthesised in this article to better understand the impact of trauma on attachment.

The author hereby introduces the construct of interpersonal neurosynchronistic phylogenesis as the interpersonal neurophysiological mechanism of change in dyads during dyadic trauma and dyadic completion as a plausible hypothesis to explain the evolution of SPA in dyads and why traumatically compromised INP leads to M-INP, dyadic trauma and the psychopathology of loneliness and loss of social cohesion.

Research method

The method starts with addressing attachment traumatology as a domain theory. Then highlights INP as a method theory, with special attention to the path leading to INP and new terminology used to describe this method theory. The method theory is used to evaluate attachment traumatology and the limitations thereof. The discussion concludes with recommendations on refining attachment traumatology, with a view to enhancing treatment of trauma with AF-SE.

Targeted body of literature

Trauma impacts the subcortical structures of the brain (Payne et al., 2015; Porges, 2011, 2021; Riordan et al., 2017, 2019; Schore 2019a, 2019b; Siegel, 2012; Van der Kolk, 2014) and is complicit in the interpersonal neurobiology of attachment dyads (Atzil & Gendron, 2017; Feldman, 2017) through interpersonal right brain to right brain synchronicity (Schore, 2019a). This research therefore targets the neuroscience of trauma and emerging attachment focused somatic therapies. Specific emphasis will be placed on a quantifiable monozygotic twin study (Riordan, 2022) that serves to illustrate the use of AF-SE.

Data gathering method

Cognitive-behavioural theories and their clinical interventions (behaviour-based talk therapies) were excluded because they primarily target the prefrontal cortex and cognition as the agency for change. Bottom-up neurobiological constructs of trauma and attachment were included to explain loneliness and loss of social cohesion. The keywords in this literature search were loneliness and social isolation, trauma, dyadic trauma and attachment, the neurobiology of trauma, phylogenetics and somatic experiencing.

Data analysis and presentation

Data gathered from historical attachment traumatology served as the domain theory and the synthesis of recent research established INP as the method theory. Theoretical and conceptual explanations with supporting evidence were developed to substantiate theoretical hypotheses, inferences and conclusions. From the method theory, the researcher considered statistical and inferential evidence of pre- and post-treatment changes after AF-SE interventions, presented in previous literature (Riordan, 2022; Riordan et al., 2017, 2019), to elucidate the neurosynchronistic nature of rupture and repair dynamics in dyadic trauma and dyadic completion. Outcomes were considered in the context of existing research in attachment traumatology (domain theory), and previous studies from the method theory, indicating that ‘trauma is...
contagious in dyads’ where INP is the interpersonal neurophysiological mechanism of change.

**Attachment traumatology**

**The neurosynchronistic impact of trauma on attachment**

Twenty-five per cent of Australian households are single-person homes (Australian Bureau of Statistics, 2022). The Queensland government ordered a parliamentary enquiry to address loneliness (abc.net.au, November 2020).

These data are unprecedented in human history because human bonding and social cohesion as mechanisms for survival, have been inherently self-evident components of social organisation.

Attachment traumatology is poorly informed of the phylogenetic impact of trauma on relationships. The phenomenon of widespread psychopathology, loneliness, social isolation and loss of social cohesion is not yet fully understood. However, the destructive impact of social isolation is known to increase stress, impair health and hasten death (Cacioppo et al., 2014). What has been established is that trauma in dyads is complicit in the social dynamics of attachment (Schore, 2019a, 2019b; Seigel, 2012; Van der Kolk, 2014). What is not clearly established are the neurological mechanisms of phylogenetic rupture and repair when trauma is introduced into the dyad.

Van der Kolk (2014) predicted a dramatic surge in developmental disorders due to the changes in attachment styles in child–parent relationships. Emphasising attachment that is phylogenetically enhancing as in SPA, may be a counterpoint to van der Kolk’s prediction. Primary attachment relationships form the foundations of all further attachment relationships by sharing an underlying neurobiology (Feldman, 2017). This underlying neurobiology was further elucidated by Schore (2019a) in his observations regarding ‘Right Brain to Right Brain’ right-lateralised interbrain synchronisation in non-verbal communication (p. 178) that foreshadowed the constructs of dyadic trauma and SPA by noting that, ‘an expansion of the child’s right brain regulatory coping capacities… underlie the developmental principle that secure attachment is the primary defense against trauma-induced psychopathology’ (p. 42).

Previously, Schore (2003) noted that trauma impacts attachment most significantly during life-cycle events. ‘Early failures in dyadic regulation ...skew the developmental trajectory of the corticolimbic systems that mediate the social and emotional functioning for the individual for the rest of the lifespan’ (p. 33) signalling the emergence of dyadic trauma as complicit in community psychopathology.

**Neurosynchronistic research foretelling secure phylogenetic attachment**

Feldman (2017) contends that, ‘humans can repair, via top-down, processing, commitment, and discipline, the effects of early maladaptive relationships by later benevolent ones’ (p. 81) foreshadowing SPA as a valid construct for psychotherapists to pursue in treatment.

Psychopathology generates social avoidance through the top-down, bottom-up, self-defence sequences of flight, fight, fear-immobilisation in the dorsal vagal complex prominent in trauma presentations (Payne et al., 2015; Porges 2011; Porges & Dana, 2018). However, loving, romantic and close friendship bonds (SPA) can heal trauma in the context of completing the traumatised individual’s survival imperative with ‘the capacity to feel safe in the arms of another’ (Porges & Dana, 2018, p. ix; in Badenoch, 2018b).

Neurosynchronicity has been the focus of neuroscientific research to determine the underlying neuro-mechanisms of attunement and connectedness for romantic couples (Kinreich et al., 2017), parent–adolescent relationships (Deng et al., 2022) and in theories of human consciousness, ‘that have been associated with subjective reports of social connectedness engagement and cooperativeness as well as experiences of social cohesion’ (Valencia & Froese, 2020, p. 1). Secure phylogenetic attachment incorporates historical neurosynchronistic research into a viable construct supporting the theory of dyadic trauma.

**Attachment traumatology historical constructs in interpersonal neurosynchronicity**

In the origins of attachment theory, Bowlby (1969) hypothesised that the individual’s capacity to cope with stress is correlated with certain maternal behaviours and that developing emotional and limbic systems are impacted by attachment transactions. This observation emphasised by Schore (2003) highlights attachment outcomes and environments of adaptiveness to have consequences that are ‘vital to the survival of the species’ (p. 178).

In neuroscience, trauma has been largely conceptualised as existing in the neurophysiology of the individual experiencing the trauma (Porges, 2011; Scaer, 2014), ‘producing enduring neurobiological alterations that underlie right brain affective instability, inefficient stress tolerance, memory impairment, and dissociative disturbances’ (Schore, 2019a, p. 51).

Similarly, post-traumatic (PTSD) generates increased tendency towards social isolation and loss of social cohesion (Wilson et al., 2018) because sympathetic arousal devolves in a cascade of trauma responses defined as the Defence Cascade (Payne et al., 2015), a fight, flight, freeze sequence in the limbic and primitive regions of the brain.

Schore (2019a) also noted that chronic relational trauma in infancy, ‘leads to the dysregulation of emotions’ (p. 240).

Modern attachment theorists agree that attachment is an interpersonal, neurobiological process (Schore, 2019a) involving bonding (Scaer, 2014), manifest in attuned...
connectedness (Porges & Dana, 2018), for individual homeostatic neurodevelopment (Schore, 2019a, 2019b) and mutual regulation (Tronic, 2007). Feldman (2017) elucidated the neurosynchronistic nature of healthy INP:

Humans’ representation-based attachments are characterized by biobehavioral synchrony and integrate subcortical with cortical networks implicated in reward/motivation, embodied simulation and mentalization. (p. 80)

The neurobiology of attachment rides on systems that maintain brain plasticity through time-sensitive increased flow of dopamine and oxytocin (Feldman, 2017), and allostasis is regulated with ‘bio-behavioural synchrony’ (Atzil & Gendron, 2017), an ancient phylogenetic dance in human attachment that can be defined in the construct of SPA. In contradiction, discordant bio-behavioural attachment counteracts phylogenetic allostatic/homeostasis which, fuelled by stress hormones and trauma-based affective states, is the embodiment of the theory of dyadic trauma as a contagion generating psychopathology in our communities over time.

**Interpersonal neurosynchronistic phylogenesis**

The theoretical journey from trauma to dyadic trauma, secure phylogenetic attachment and interpersonal neurosynchronistic phylogenesis

Riordan and his colleagues, while observing trauma in attachment relationships realised there were no theoretical terms to describe their discoveries; therefore, descriptive terms were coined that reflected the phenomenon observed over three progressive publications (Riordan, 2022; Riordan et al., 2017, 2019). Researchers examined the interpersonal neurosynchronistic nature of trauma to derive descriptive terms and constructs to elucidate the nature of trauma as a contagion in attachment dyads, intergenerationally and phylogenetically.

Riordan et al. (2017) in their observations of a single subject treatment protocol for toddler trauma using somatic experiencing coined several new terms to describe the phenomenon observed: attachment perturbation, attachment neuroception, comfort-seeking, somatic-attachment-soothing, quiescent attunement, dyadic completion, traumatised attachment dyad and traumatic attachment.

These terms evolved sequentially from observing the interpersonal processes of trauma in an otherwise secure attachment dyad and the behavioural changes of participants in the dyad during and after treatment with somatic experiencing therapy.

Following their 2017 discoveries, Riordan et al. (2019) introduced several new terms to describe the application of SE in attachment trauma across the life cycle. A ‘flow of life’ trajectory of trauma and recovery was developed (see Figure 3). Two major constructs emerged, namely dyadic trauma and SPA that led to a new treatment modality to address trauma in relationships, namely AF-SE.

Attachment focused-somatic experiencing was then applied in a quantifiable monozygotic twin study (Riordan, 2022) to test the veracity of the observable concepts and the theoretical constructs that had emerged. The contagious nature of trauma in relationships led to several new terms describing trauma and attachment in monozygotic twins including monozygotic attunement, monozygotic attachment and consequently the realisation of INP emerged as a plausible theoretical explanation for the mechanisms by which trauma as a contagion is transposed interpersonally in dyads, intergenerationally and phylogenetically in communities.

It can be argued that these research studies (Riordan, 2022; Riordan et al., 2017, 2019) target the interpersonal neurosynchronistic mechanisms of trauma in dyads and devise therapeutic protocols to resolve it with AF-SE. To consider the contagious nature of trauma in attachment dyads and its role phylogenetically in human survival, specific emphasis will be placed on dyadic trauma, SPA and interpersonal neurosynchronicity in a quantifiable monozygotic twin study (Riordan, 2022), which serves as an illustration of the interpersonal neurosynchronistic mechanisms of trauma in dyads.

While not all neurosynchronistic phenomenon and terminology mentioned will be defined in this paper, as they have been defined in previous papers (Riordan, 2022; Riordan et al., 2017, 2019), the most important terminology relevant to this paper is briefly explained.

**Trauma**, according to Levine (2015), is an incomplete survival response after fear and/or terror immobilisation after which, ‘persistent maladaptive procedural and emotional memories form the core mechanism that underlies all traumas, as well as many problematic social and relationship issues’ (p. 38). Riordan et al. (2019, Riordan, 2022) deepen this definition of trauma, arguing that trauma compromises whole brain function by innervating phylogenetic survival imperatives of flight, fight, freeze sequences in the limbic and primitive brain that override social and cognitive function. Social engagement systems are downregulated, and neuroception and secure attachment dynamics are compromised by traumatic perturbations in the attachment dyad.

**Dyadic trauma** is socially destructive (Riordan, 2022) and occurs:

When resonance and attuned connectedness in secure attachment alters for both participants in the dyad. Social engagement, governed by mutually integrated whole-brain connection, shifts to disorganised social avoidance. Fear and anger drive survival imperatives dominated by limbic and subcortical brain structures. Dyadic trauma is neurologically dis-integrating and contagious in attachment dyads and may promote psychopathology throughout the life cycle. (p. 3)

**Traumatic attachment** is the interpersonal neurobiological relationship between individuals in the traumatised dyad characterised by flight, fight, freeze or shutdown trajectories.
in a devolving interpersonal neurobiological synchronicity. Traumatic attachment occurs when one or both participants experience trauma leading to dyadic trauma (Riordan et al., 2017, 2019).

Secure phylogenetic attachment ‘promotes dyadic synergy that is resonant, attuned, engaged, and mutually regulating. Secure phylogenetic attachment invites social dynamics that involve nurturing, support, acceptance, regard, tolerance, love, and respect’ (Riordan et al., 2019, p. 1). Secure phylogenetic attachment is embodied in the innate capacity to form secure attachments in adaptive-INP incorporating neurobiological, neurochemical (Feldman, 2017), behavioural and affective interpersonal dynamics for survival throughout the life cycle.

Dyadic completion is the neural synchronisation between participants in the traumatised dyad to reinstate allostatic/homeostasis during trauma by restoring the attuned concordance of SPA (Riordan et al., 2017, 2019).

Interpersonal neurosynchronistic phylogenesis is the interpersonal neurophysiological mechanism of change between members of a dyad during the face–heart connection that is transposed from one member to another and one dyad to another intergenerationally and phylogenetically as either SPA or dyadic trauma. Interpersonal neurosynchronistic phylogenesis is separated into contagious adaptive or maladaptive modalities.

Somatic experiencing

Somatic experiencing is a comprehensive trauma treatment to target the nervous system as the agency of change after trauma and is now established as a clinically effective treatment for the neurobiological resolution of trauma within the individual nervous system (Brom et al., 2017; Leitch, 2007; Leitch et al., 2007; Levine, 2010; Parker et al., 2008; Riordan et al., 2017, 2019).

The core tenet of SE in the resolution of trauma is ‘the completion of thwarted, biologically based, self-protective and defensive responses, and the discharge and regulation of excess autonomic arousal’ (Payne et al., 2015, p. 1). Engaging interoceptive, kinaesthetic and proprioceptive neurophysiology during dynamic SE, the client experiences somatic shifts as they internally regulate and reorganise neural synchrony between the cortex and subcortical survival structures. This allows for a whole brain, flexible state of relaxed readiness, renegotiating trauma triggers to effectively respond to current events (see Figure 1 and Figure 2).

The processes of interoception and the impact of somatic experiencing are symbolised in Figure 1 showing the major structures of the brain and the general action of SE and AF-SE pendulation during and after interoception, a shift from sympathetic arousal (flight, fight, freeze responses) to parasympathetic (rest and digest) state. Attachment relationships are positively impacted through the processes of INP attunement to generate dyadic completion and the reinstallation of SPA.

In sessions of AF-SE, the SE therapist invites the client to interocept while targeting primarily traumatic sensation that can also lead to somatic-affective experiences and new understanding of traumatic memory that transposes from the prefrontal cortex to the hippocampus during neurogenic discharge to create new meaning around the trauma, and resolution in the traumatised dyad reinstating SPA through INP.

Attachment Focused-Somatic Experiencing offers neurobiological coordinates and a therapeutic process to address dyadic trauma (see Figure 3). Attachment focused-somatic experiencing utilises the interpersonal neurosynchronistic construct of SPA to identify and understand threat-recovery and its role in resolving dyadic trauma with dyadic completion (Riordan et al., 2017, 2019).

The process and events of trauma and recovery with AF-SE are identified and explained. These neurobiological and therapeutic coordinates of threat response and recovery provide a map of the neurophysiological sequences of re-traumatisation and recovery through adaptive-INP to achieve SPA. For a comprehensive explanation of the individual stages of re-traumatisation and recovery with AF-SE, see Riordan et al. (2019).

Interpersonal neurosynchronistic phylogenesis:
An illustration and confirmation using a monozygotic twin study

Monozygotic attachment impacted by trauma is suboptimal and antagonistic to the dynamics of SPA and compromises life cycle procreative pair bonding (Riordan, 2022).

Riordan’s (2022) monozygotic twin study revealed quantifiable autonomic measures of change after treatments with AF-SE, providing insight into the dynamics of how ‘trauma as a contagion’ is transposed in the interpersonal neurobiological dynamics of suboptimal sibling attachment.

The term INP was coined here to identify the phenomena. Riordan demonstrated that autonomic physiological measures of recovery from PTSD after treatment with AF-SE in the traumatised twin were transposed neurosynchronistically to the non-traumatised twin via their monozygotic, interpersonal neurosynchronistic attunement.

Monozygotic attunement

Monozygotic twins, in traumatic circumstances, share a level of somatic congruence in their attachment style that goes beyond typical sibling attachment dynamics to a platform of somatic attunement where, one twin may experience the sensations, symptoms and experience of traumatic overwhelm in a state of sympathetic arousal with the other...
twin, who remains in the state of dorsal vagal numbness (Riordan, 2022).

This phenomenon offers insights into the dynamics of attachment including the phenomena of dyadic trauma, dyadic completion (Riordan et al., 2017), attachment-neuroception (Porges, 2011; Riordan et al., 2017) and INP. Objective autonomic measures of pre- and post-treatment evidence of neurogenic concordance in this twin pair have validated the constructs of dyadic trauma and completion, monozygotic attachment, monozygotic attunement, SPA and INP.

**Interpersonal neurosynchronistic phylogenesis**

Adaptive INP originates in the face-heart connection (Porges, 2011) postpartum that initiates interpersonal neurosynergy and attuned reciprocity in the attachment bond between mother and child (Schore, 2019a). Unresolved traumatic ruptures of the attachment bond emerge as perturbations in the dyad that then compromise somatic attunement and reciprocity, creating loss of concordance and reduced interpersonal synergy which generates fear-vigilance sequences in the subcortical brain. Functional connectivity changes in the individual’s brain during social exclusion (Schmalzle et al., 2017) shifting from social engagement to avoidance in the brains of both participants in the dyad. The resulting M-INP further confounds SPA and erodes neuroception creating dyadic trauma.

This process is reversed during AF-SE to restore SPA (Riordan, 2022) during somatic and affective attunement via interpersonal neurobehavioural synergy with the participants in the dyad and the SE therapist. An attuned, allostatic/ homeostatic nervous system will engage the dysregulated nervous system in somatic-attachment-soothing (Riordan...
Hyper arousal (hypervigilance)
- Flight
- Somatic overwhelm
- Hyper arousal (agression avoidance)
- Relationship perturbation
- Loss of secure phylogenetic attachment
- Social isolation
- Dyadic trauma

Homeostasis (whole brain integration and recovery)
- AF-SE
- Neurogenic discharge
- Quiescent immobilization
- Quiescent attunement
- Dyadic completion
- Heart-face connectedness
- Social engagement
- Reinstatement of secure phylogenetic attachment

Hypoarousal (dorsal vagal shutdown)
- Freeze
- Shutdown
- Tonic immobilization (visceral vagal syncopa)
- Loss of secure phylogenetic attachment
- Social isolation (loneliness, depression)
- Dyadic trauma

Flow of life


FIGURE 3: Flow of life.
et al., 2017) as with a mother soothing her infant or pair-bonded partners comforting each other in adversity. This process also occurs in the therapeutic crucible where the therapist as the neuroregulated, empathic witness, promotes synergistic, whole brain function and dyadic repair where the dysregulated nervous system (the client) attunes to and synchronises with the empathic regulated nervous system (the AF-SE therapist) in the interpersonal neurosynchronistic dynamics of AF-SE. These are the key principles of adaptive-INP allowing dyadic completion to restore SPA. Riordan and colleagues (2019, 2022) demonstrated AF-SE as a top-down and bottom-up interpersonal, neurobiological treatment for dyadic trauma to reinstate SPA.

Interpersonal neurosynchronistic phylogenesis offers a platform for conceptualisation of ‘trauma as a contagion’ in communities because the evidence implies that SPA and dyadic trauma are conflicting phenomenon in a neurosynchronistic dance of adaptive and maladaptive INP where the impact of trauma on dyads can be examined.

Discussion

Historical research in trauma and attachment has indicated that neurosynchronicity is an important construct. Dyadic trauma offers an inclusive theory with constructs that explain the impact of trauma on attachment.

The application of AF-SE to trauma in attachment dyads reveals several phenomena hereto unexplored by traumatologists and attachment theorists. Previous experimental and quantitative research has identified several new terms in the study of trauma and attachment to describe emerging phenomena associated with the application of AF-SE to traumatised dyads:

- attachment focused-somatic experiencing (Riordan et al., 2019)
- dyadic trauma (Riordan et al., 2019)
- dyadic completion (Riordan et al., 2017)
- secure phylogenetic attachment (Riordan et al., 2019)
- monozygotic attachment (Riordan, 2022)
- interpersonal neurosynchronistic phylogenesis.

Relational trauma (Schore, 2019a) may evolve into dyadic trauma (Riordan et al., 2017, 2019) due to loss of cohesion and trust in bonding due to interpersonal emotional triggers in relationships. Trauma triggers, in turn, add increasingly traumatising perturbations to the dyad that escalate onto loss of synchronistic attunement thereby compromising SPA. Avoidance, a key fear-vigilance sequence of flight-trauma, generates loss of social cohesion.

Discoveries in neurophysiology (Porges, 2011, 2021) and development of somatic therapies (Levine, 2010; Riordan et al., 2019) are advancing attachment traumatology to induce a paradigm shift that includes the interpersonal neurosynchronistic dynamics of trauma ‘as a contagion’ to explain recent community psychopathology and its impact on human phylogenetics.

Attachment theorists (Schore, 2019a; Siegel, 2012) and traumatologists (Riordan, 2022; Riordan et al., 2017, 2019; Van der Kolk, 2014) have emphasised the importance of attachment relationships and the empathic witness (Levine, 2010) in the processes of SPA as a healing mechanism for the traumatised dyad. In this way, trauma as a contagion may be addressed with AF-SE to resolve dyadic trauma.

The evolving constructs introduced by Riordan (2022) offer new platforms to examine, understand and treat trauma in dyads and to elucidate on widespread community psychopathology. Riordan (2022) demonstrated that trauma is complicit in dysfunctional attachment styles and is socially destructive. Inferentially, trauma is therefore also complicit in the psychopathology of loneliness, social isolation and loss of social cohesion.

Riordan (2022) further demonstrated interpersonal neurobiological congruence and neurosynchronicity in a monozygotic twin pair confirming that the hypothesis that ‘trauma is contagious in dyads’ held true. These discoveries invite changes in the nomenclature of attachment traumatology to include SPA as a more nuanced and appropriate description of secure attachment to understand, categorise and codify trauma and its complex attachment-based components and to include INP as the neurosynchronistic mechanisms to explain SPA and community psychopathology emerging from dyadic trauma.

The construct that trauma exists only in the neurophysiology of the individual can no longer be held true and invites a paradigm shift for attachment traumatology to address trauma from the interpersonal neurosynchronistic perspective as a valid approach to further research in community psychopathology.

Attachment traumatology should therefore include AF-SE in treatment protocols incorporating interpersonal neurobiological features of attachment (Schore, 2019a; Siegel, 2012) now identified as INP as an essential component of trauma treatment.

Given the impact of trauma on neurological events in the brain (Porges, 2011, 2021; Riordan, 2022), neural synchronicity associated with attachment and trauma (Schore, 2019a) and behaviour changes associated with fear-vigilance and avoidance ideation of those with PTSD (Schore, 2019a), it is plausible to consider that trauma generates dyadic trauma which compromises SPA and contributes to widespread psychopathology, loneliness, social isolation and loss of social cohesion in our communities. The mechanism of transposition of trauma interpersonally, intergenerationally and phylogenetically, as a contagion, is M-INP.

Inferentially and conversely, SPA is therefore an essential component of individual and community wellness (Riordan et al., 2019) where loss of SPA is a precursor to individual and community psychopathology that may have life-long impact on relationships and intergenerational community psychopathology (Feldman, 2017; Flores & Porges, 2021; Van der Kolk, 2014).
Further investigation of the constructs of dyadic trauma, SPA, INP, dyadic completion, monozygotic attunement, monozygotic attachment, traumatic attachment and ‘trauma as a contagion’ in attachment dyads, and their relationship to community psychopathology is required.

Conclusions

The evidence invites the following observations:

- The theory of dyadic trauma indicates a paradigm shift in attachment traumatology to incorporate trauma as a contagion that contributes phylogenetically to community psychopathology.
- Secure phylogenetic attachment is the antithesis of dyadic trauma.
- Attachment focused-somatic experiencing is a heuristic treatment modality that requires a creative application of somatic neuropsychotherapy to resolve dyadic trauma and to reinstate SPA thereby reducing the phylogenetic consequences of dyadic trauma on communities.
- Interpersonal neurosynchronistic phylogenesis is a construct to describe and explain the neurosynchronicity of attachment rupture and repair associated with dyadic trauma, dyadic completion and recovery.
- Three unique categories of attachment are offered for further research namely SPA, traumatic attachment and monozygotic attachment.

Implications for practice

The implication for practitioners is to consider the nervous system in trauma presentations as a mechanism for treatment within the traumatised dyad and the neurosynchronistic dynamics of attachment. Epigenetics, neuroplasticity and neurogenesis need to be considered in theoretical constructs associated with dyadic trauma. Somatic and neurological interventions for trauma have emerged as alternatives to traditional cognitive-based treatments. Practitioners now have alternative treatment modalities and theoretical constructs to identify and categorise trauma in dyads and its contagious nature in communities.

Limitations and recommendations

The dominant limitation of this research is that there is sparse experimental, quantifiable information on the phenomena examined. The research relied heavily on a small number of recent publications to identify and explore the implications of the phenomena. Attachment focused-somatic experiencing requires more experimental research to expand the dialogue of the concepts presented above about trauma and attachment. Secure phylogenetic attachment and INP need to be assessed with functional magnetic resonance imaging to confirm the construct in direct neurophysiological, measurable terms over time and through life cycle events intergenerationally. This study hopes to inspire a discussion around the interpersonal neurobiological parameters of dyadic trauma and attachment theory and promote further inferential and quantifiable analysis regarding the interpersonal neurosynchronistic nature of trauma in attachment dyads.

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Competing interests

The author declares that they have no financial or personal relationship(s) that may have inappropriately influenced them in writing this article.

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J.P.R. declared sole authorship of this research article.

Ethical considerations

This article is a theoretical paper and therefore did not make use of any human subjects but focused on a review of the current body of literature.

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Data availability

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References


Siegel, D.J. (2012). The developing mind: How relationships and the brain interact to shape who we are. W.W. Norton.


