COMENIUS UNIVERSITY IN BRATISLAVA
FACULTY OF MATHEMATICS, PHYSICS, AND INFORMATICS

POST-TRAUMATIC EMBODIMENT: PROPOSAL OF AN IMPROVED MODEL OF EMBODIMENT IN THE CONTEXT OF TRAUMA

Diploma Thesis

Bc. Matija Rupčić

Bratislava, 2023

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Study programme: Cognitive Science
Field of study: Computer Science
Supervising Department: Department of Applied Informatics
Supervisor: Prof.PhDr. Silvia Tomašková, CSc.

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Title: Post-traumatic Embodiment: Proposal of an Improved Model of Embodiment in the Context of Trauma

Annotation: Embodiment as a phenomenon in cognitive science follows a belief that cognitive processes are influenced by body morphology, emotions, and sensorimotor systems. The body regulates cognition by holding a feedback-driven role in cognitive processing and structures data to solve environment interacting problems and gives response in terms of complex behaviors. There is no perspective yet directed exclusively to the problem of disruptive nature such as trauma, negative life event that occurs in a position of relative helplessness. The overview of the existing data from various science fields can answer questions about trauma's effect on mental and physical health, as well as its somatic consequences.

Aim: Extend the current perspective of embodiment in cognitive science research and focus on how trauma affects the body in the long run, as well as its consequences for mental and medical health, and the interaction between the two.

Literature: Clark A. (2008) Supersizing the Mind: Embodiment, action, and cognitive extension. Oxford University Press.
Scaer R., Shapiro L. (2010). Embodied Cognition, in Oxford Handbook of Philosophy and Cognitive Science, E. Margolis et al (eds.), Oxford Univ. Press.
Shapiro L. (2010). Trauma Spectrum, Norton & Co. (eds.), New York, Norton Professional Books.

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ZADANIE ZÁVEREČNEJ PRÁCE

Meno a priezvisko študenta: Matija Rupčić
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Názov: Post-traumatic Embodiment: Proposal of an improved model of embodiment in the context of trauma
Posttraumatické stelesnenie: Náčrt inovovaného modelu stelesnenia v kontexte traumy

Anotácia: Vtelenie ako fenomén v kognitívnej vede vychádza z presvedčenia, že kognitívne procesy sú ovplyvnené morfológiou tela, emóciami a senzomotorickými systémami. Telo reguluje kogníciu tým, že zastáva úlohu riadenú spätnou väzbou v kognitívnom spracovaní a štruktúruje údaje na riešenie problémov s prostredím a poskytuje odpoveď v zmysle komplexného správania. Zatiaľ neexistuje perspektíva zameraná výlučne na problém rušivého charakteru, akým je trauma, negatívna životná udalosť, ktorá sa vyskytuje v pozícii relatívnej bezmocnosti. Prehľad existujúcich údajov z rôznych vedných oblastí môže zodpovedať otázky o vplyve traumy na duševné a fyzické zdravie, ako aj o jej somatických dôsledkoch.

Cieľ: Rozšírte súčasnú perspektívu stelesnenia vo výskume kognitívnej vedy a zamerajte sa na to, ako trauma ovplyvňuje telo z dlhodobého hľadiska, ako aj jej dôsledky na duševné a zdravotné zdravie a na interakciu medzi nimi.

Literatúra:
Clark A. (2008) Supersizing the Mind: Embodiment, action, and cognitive extension. Oxford University Press.
Scaer R., Shapiro L. (2010). Embodied Cognition, in Oxford Handbook of Philosophy and Cognitive Science, E. Margolis et al. (eds.), Oxford Univ. Press.
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vedúci práce

Declaration: I hereby declare that this thesis is written by myself, and investigated by myself, with help of relevant literature that had been properly cited and quoted where needed.

Bratislava, 2023 ....

Bc. Matija Rupčić

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I want to thank my program coordinator, Igor Farkas, FMFI faculty, and Comenius University for their patience, support, and for creating the opportunity for me to finalize this thesis and graduate. I'd like to thank my supervisor, Silvia Tomašková, in particular, for her patience and for sticking with me for so long.

This thesis was written with a special dedication to Rudolf Vig, who passed away in 2019, who was always there for me and loved me unconditionally in my pains and struggles, and to Vladimir Maslaric, who showed me that being a scientist means being yourself and not fitting supposed boxes.

Abstract

Embodiment research demonstrates that consciousness, agency, and social and environmental interactions are all dependent on the mind and body connection. This embodiment appears to be a unique sensorimotor interaction between an organism and its environment. A physical injury, a catastrophic event, or a psychological and physiological response to a negative experience are all examples of trauma. Trauma can have long-term consequences. Because psychology and psychiatry are the only explanations for trauma, this thesis defines trauma as a cognitive science construct so that science can investigate its effects from various angles. This dissertation investigates embodiment in philosophy and cognitive science, and then applies it to trauma. This necessarily requires the development of a new cognitive science model of post-traumatic embodiment. This thesis investigates embodiment in philosophy and cognitive science in the context of trauma, as well as defining trauma using cognitive science paradigms and concepts, by focusing on trauma consequences. Other issues to consider include which cognitive science paradigms or concepts can be used to define trauma, how they can explain trauma, and whether a model of post-traumatic embodiment can be developed as a result of redefined trauma and its effects on the body and mind. Trauma is defined by a predictive processing paradigm as a disruption or error in an agent's interaction with its environment, and trauma consequences are defined as a failure to cope with an unexpected environmental factor. A post-traumatic embodiment model describes how an agent's physical, psychological, and behavioral changes after trauma affect safety and reduce processing, learning, and decision-making.

Abstrakt

Štúdie o stelesnení uvádzajú závislosť tela na mysli v súvislosti s vedomím, sociálnymi interakciami a vonkajším prostredím. Zdá sa, že stelesnenie je jedinečným spôsobom, akým senzorimotorické schopnosti organizmu umožňujú interagovať s prostredím. Trauma sa obvykle chápe ako telesné zranenie, katastrofická udalosť alebo psychologická a fyziologická reakcia na negatívne skúsenosti. Predpokladá sa, že trauma bude mať dlhodobé následky na stav tela a mysle. Keďže nie sú k dispozícii žiadne iné vysvetlenia alebo prístupy k traume okrem tých v psychológii a psychiatrii, táto práca sa snaží definovať traumu ako kognitívnovedný konštrukt, ktorý umožňuje preskúmať jeho následky z viacerých než len v súčasnosti uplatňovaných perspektív. Predložená práca skúma význam a použitie pojmu stelesnenia tak ako sa aktuálne chápe v filozofii a kognitívnej vede, a snaží sa ho umiestniť do kontextu výskumu traumy. Pre dosiahnutie tohto cieľa sa navrhuje nový model posttraumatického stelesnenia v kognitívnej vede. Práca sa snaží odpovedať na otázky týkajúce sa stelesnenia, traumy a jej následkov. Ďalšie otázky sa týkajú toho, aké kognitívnovedné paradigmy alebo koncepty sa dajú použiť na definovanie a vysvetlenie traumy a akým spôsobom možno nastaviť model posttraumatického stelesnenia založený na novo definovanej traume a jej následkoch pre telo a myseľ. Navrhuje sa paradigma prediktívneho spracovania na vysvetlenie traumy ako narušenia alebo chyby pri interakcii agenta s prostredím a jej následkov, ako zlyhania zvládať neočakávaný environmentálny faktor. Popisuje sa model posttraumatického stelesnenia ako súhrn fyzických, psychologických a behaviorálnych zmien v osobe-agentovi po prežitku traumy, čo vedie k zmene pocitu bezpečia a zníženej schopnosti spracovávať informácie, učiť sa a rozhodovať.

List of figures

Abbreviations

ANS	autonomic nervous system
ACTH	adrenocorticotropic hormone
ASD	acute stress disorder
CBT	cognitive behavioral therapy
CNS	central nervous system
CORT	cortisol
CRH	corticotrophin-releasing hormone
DSM-III	The Third Edition of the Diagnostic and Statistical Manual of Mental Disorders
DSM-IV	The Fourth Edition of the Diagnostic and Statistical Manual of Mental Disorders
DSM-V	The Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders
EC	embodied cognition
HPA	hypothalamic-pituitary-adrenal
HPT	hypothalamic-pituitary-thyroid
PP	predictive processing
PFC	prefrontal cortex
PNS	parasympathetic nervous system
RL	reinforcement learning
PTSD	post-traumatic stress disorder
PVN	paraventricular nucleus
VR	virtual reality
SAM	sympathomimetic dorsal pathway
SNS	sympathetic nervous system
T3	triiodothyronine
T4	tetraiodothyronine

Contents

Introduction

In cognitive science, the now-famous concept of embodiment began with an attempt to explain the relationship between mind and body and to determine whether physical factors influence mental state. The body is the focus of embodiment studies in philosophy, which explain the dependence of the body on the mind in terms of consciousness, agency, and social and environmental interactions. Cognitive processing is considered embodied when it is dependent on elements of an actor's (person's) physical body, or when features of one's body play a causal or physical constitutive role in cognitive processing (Moya, 2014). The actor moves with orientation in a space based on the after-mentioned connection between body parts and cognition, providing a basis for engagement between organism and environment (Merleau-Ponty, 1980; Talero, 2005). This embodiment appears to be the unique way in which an organism's sensorimotor abilities enable it to interact with its environment. All views of the general embodied cognition thesis share the objective of developing cognitive explanations that capture how the mind, body, and environment interact and affect one another.

Trauma is known as a bodily injury, catastrophic event, or psychological and physiological reaction to a very negative experience, usually ending in helplessness. Psychological trauma is explained as a series of experiences that can have a significant impact on a subject's psyche, threatening the integration of thinking (Newbold et al., 1902). Trauma could be perceived as something abstract, an abstract idea in a sense, because of its novelty to one's previous experiences stored in a cognitive map. This abstraction then tries to be processed by the mind and body, but often, at least based on assumption, fails. In this sense, it is hard to integrate something abstract or find a quick explanation for it. According to Casasanto and Boroditsky (2008), it is through sensorimotor skill that humans represent abstract ideas. As a result, it is reasonable to expect that impairment in abstraction integration will also affect subsequent projections of the same abstraction.

For the purposes of this thesis, it is critical to first describe a philosophical view of the body and how it began to interact with the concept of the mind. It will be presented more narrowly how these body-mind interactions and the philosophical theories surrounding them led to the embodiment paradigm postulates we know in cognitive science today. Then a short introduction to the current definition and status of trauma is presented, along with a short but concise overview of trauma's impact on a body in terms of neuroscience, medical science, and (micro)biology. A simplified explanation of mental health consequences is then presented, with an accent on the stress response and a brief overview of the most common disorders associated with traumatization consequences in the long term. The thesis then finds connections between body responses in terms of interactions with surroundings and tries to provide answers with a representation of the vagus nerve and its functions, and then tries to find a sufficient frame in cognitive science paradigms or concepts in order to explain trauma in this context. The study focuses on predictive processing and reinforcement learning in this sense. Finally, a new model for post-traumatic embodiment is presented, which is based on all of the previous concepts in the study and on finding an explanation for trauma in the previously mentioned predictive processing and reinforcement learning frames, as well as the overall body-mind-environment connection, which is already well accepted in cognitive science.

Psychoneuroimmunology research indicates that stressful life events can contribute to health problems by dysregulating another critical system's inflammatory response (Segerstrom & Miller, 2004). Many of these diseases seem to be caused by changes and deregulation of critical stress response systems as a result of acute or excessive stress and PTSD. Stress is a response to a perceived danger that impacts all physiological systems (Kudielka & Kirschbaum, 2005). Cortisol is a hormone that assists in acute, short-term survival conditions, but when stress becomes chronic and prolonged, high quantities of cortisol can destroy tissue, increase blood pressure, and damage the heart (Hannibal & Bishop, 2014). There are a decent number of studies showing this connection. For example, people who receive repeated cytokine injections over the course of weeks are witnessing dramatic changes in stress responses and behavior (Dantzer et al., 2008). There is also a relationship between peripheral inflammation and the onset of major depressive disorder and other neurological disorders. Proinflammatory mediators could possibly also contribute to the symptoms of PTSD, as PTSD patients exhibit a moderate to substantial rise in levels of many proinflammatory cytokines (Schiepers et al., 2005). Research indicates that inflammation is a preexisting risk factor for the development of PTSD in trauma-exposed people (Tian et al., 2007; Kloet et al., 2005). Felitti et al. (1998) discovered that traumatized individuals were more likely to develop ischemic heart disease, cancer, stroke, chronic bronchitis, emphysema, and obesity than patients who had not been traumatized. Chronic pain syndrome specialists are also showing a strong connection between trauma and health (Stenager et al., 1991). Other research shows how defective neuroinflammation and excessive neuroinflammation due to inflammatory or infectious diseases or due to psychosocial stress are associated with symptoms of brain dysfunction, such as learning and memory impairment (Yirmiya & Goshen, 2011). Early adulthood sadness and psychosis are also strongly linked to childhood systemic inflammation (Kiverstein et al., 2022). This type of early childhood immunological stimulation can alter brain development, activate resident immune cells, and program neuroendocrine stress responses, which indicates the strong significance early stress has on people's overall development (Bierhaus et al., 2003).

Three parts of the brain that are mainly affected by trauma are the prefrontal cortex, the anterior cingulate cortex, and the amygdala (Perrotta, 2019), where the amygdala is most active. Stress and trauma activate the body's defense systems by activating regions of the brain that were evolved to respond automatically to threats—the autonomic nervous system (ANS) gets activated, a system that regulates hormones, heart rate, and digestion—when it detects danger. This is closely related to the acute stress response and occurs when a person's nervous system is put on alert in the absence of a threat (Bracha, 2004). Chronic stress and unresolved trauma inhibit the survival brain's sense of security. If the agent is not given the time to detect the threat and devise a reaction, not only will the fight-or-flight response intensify, but he or she will also be attempting to make sense of the trauma. Therefore, you may not have any time to comprehend the scenario. integrates into the body or neurological system.

The significance of the influence of the environment on the body and how the body and environment ultimately shape cognition stems from the fact that the brain is the organ that perceives the world and displays how the structure of the world will dictate the structure of the body (Shapiro, 2019). With this kind of notion, it is important to emphasize how embodiment focuses on body activities that influence how the brain interacts with and responds to the external environment. This goes in favor of the theory of dynamical systems explanation: the agent's (person's) system is self-organizing, and its behaviors stem from this self-organizational structure when components are coupled. Beer (2008) defines the body, mind, and environment as dynamic systems that interact continuously. This thesis will include this dynamic fluctuation in an explanation of trauma in a cognitive science context, as well as use this perspective in modeling the post-traumatic embodiment. A dynamic system's frame seems a valid and clear underline for agent-environment-action-reaction dynamics, which this thesis emphasizes in the explanation of how trauma affects embodiment and helps to narrow the choice for cognitive science paradigms and concepts to explain trauma first and foremost.

The most suitable paradigm to explain trauma, based on all the literature research conducted for this thesis, seems to be the predictive processing (PP) computing paradigm. PP is based on the theory that the brain continuously generates and updates mental models of the surrounding environment. According to the notion of predictive coding, our brain constantly generates and refines an internal representation of its external world. To comprehend the nature of chronic pain, familiarity with predictive coding seems to be required. According to Clark (2013), PP serves as a link between the human body, cognition, and the outside world, and it influences the environmental behavior of agents. For purposes of this thesis, the concept of nocioception seems to be of significant importance in understanding an agent's comprehension of pain in any form, as nociception is an injury detector and pain generator (Black, 2002; Kiverstein et al., 2022; McLaughlin et al., 2014). PP and nocioception are the frontlines of the agent's reaction to unexpected and/or stressful events, and this work tries to find novel connections and figure out conditions and details active in this integration of trauma in one's body and mind, and then explain possible reactions one could then give back into an environment based on them.

As of today, research is not very rich on the topic of trauma and dynamic systems. Current research is mostly done in the field of PTSD, and a few can be found in a more specific reinforcement learning framework. Trauma seems to still be widely researched and explained in the neuroscience part of cognitive science studies, and the approach from a dynamic and computational point of view seems to be rather novel, at least from what this thesis managed to find in the literature search. Most research is done on the benefits of treatments for PTSD. PTSD is believed to function through reinforcement learning, and such an approach is considered to assist patients in forming fresh, less traumatic memories of earlier painful events (Ross et al., 2018). There is evidence that persons with a history of

PTSD and child abuse suffer from conditioned fear memory erasure and reinforcement learning, and that there is a global deficit in fear learning (Acheson et al., 2015). Failures in reward learning also seem related to trauma and PTSD (Ross et al., 2018).

Trauma as an established term traces its roots to the Greek language where it originally referred to wounds created by slicing the skin apart (Kolaitis & Olff, 2017). Trauma obviously has consequences for a whole organism, first and foremost on a physical level, as the word itself implies. It inflicts a wound that needs to be treated and in most cases leaves scars. The word trauma first found its place in medicine as a part of defining and explaining injuries that the physical body endures but soon became recognized in psychiatry as not only psychiatry emerged from medical sciences, but also both philosophers and psychiatrists found a word and its meaning as a suitable metaphor for as well psychological injury and metaphysical/psychological suffering (Herman et al., 2016). In 17th century the phrase starts being used in science and psychiatry to point out how heavily dependent on a body trauma is, regardless of its nature (Talero, 2005). Therefore today's (mental, psychological) trauma found its explanation as the emotional, psychological, or bodily reaction to a severely unpleasant or disturbing incident (Sternberg et al., 1989). While there are multiple versions of this explanation, there is no known definition of trauma in any other context nor there is any known definition of the same in some more specified scientific frame, being it in any other science than medicine.

For now, cognitive science studies have bothered to research post-traumatic stress disorder, which is related to trauma but is more of a byproduct than an explanation of trauma itself (Resick & Schnicke, 1992). Considering cognitive science's interdisciplinary nature, it would be interesting and valuable to try to explain trauma in the context of cognitive science. When it comes to embodiment as a paradigm, trauma as a part of neuroscience and psychiatry includes embodiment as a way to explain the relationship between body and mind in terms of traumatic event consequences and is widely used as a frame of understanding in therapy work with traumatized individuals in a form of somatic and embodiment therapy (Gendlin, 1998; Levine, 1997). However, these approaches include the connection of a mind to a body and how the mind affects the body through the nervous system and brain functions, leaving the body with long-term health consequences (Hopper et al., 2007). Current therapeutic tools are attempting to reverse these negative effects in the body before affecting the mind to reframe trauma. Other classical approaches are finding ways to influence damage inflicted on the brain and nervous system through behaviors and changes of thinking. There is, however, no well-developed and known approach to traumatization that includes actively involving the body and mind's dependence on an environment, at least not in an embedded context such as cognitive science and embodiment research do.

In cognitive science, the concept of embodiment is becoming increasingly difficult to explain without mentioning this reliance on body-environment interaction. For purposes of this study and to simplify these dependencies and interactions, a dynamical systems approach is used to simplify a person by describing them as an agent with a body and mind that holds its agency and moves, behaves, and interacts with the environment. Such dynamical movement of these components (mind-body-environment) in a trauma context is regarded as a key to explaining embodiment that is entirely dependent on the environment—post-traumatic embodiment.

The main aim of this thesis is to explore the concept of embodiment as it is currently known in philosophy and cognitive science and frame it in the context of trauma by setting up a novel model of post-traumatic embodiment in cognitive science. A partial aim is to define trauma as a cognitive science construct by segregating it from its current definition in psychology and psychiatry. This thesis work explores the following objectives:

1) To offer a clear overview of embodiment concept from philosophy to cognitive science
2) To explain the current view of trauma and its consequences on a body and mind (brain) in terms of a stress response
3) To define trauma in the context of cognitive science paradigms or concepts
4) To set up a post-traumatic embodiment model that depends on newly defined trauma and currently known consequences of trauma on a body and mind (brain)

The proposed project aims at answering the following research questions:

1) How can we easily and clearly explain embodiment in cognitive science?
2) What is trauma and how it is currently defined?
3) What is research currently showing about the consequences of trauma on a body and mind**?**
4) What cognitive science paradigm or concepts can be used to define trauma?

5) How can trauma be explained based on previously mentioned paradigms or concepts?
6) Is it possible to set up a model of post-traumatic embodiment depending on a newly defined trauma and its consequences on a body and mind?

The previously mentioned explanation of trauma could open further windows to research trauma in cognitive science as well as to develop different approaches in psychiatry and psychology, which can not only give different insights and novel understandings of this phenomenon but also open opportunities to develop new therapeutical methods or improve existing ones.

By explaining an embodiment in terms of embodied cognition and the "mind is in the body, body with its mind is placed in an environment, and all three are in constant interaction" point that this thesis outlined, and explaining trauma by main predictive processing mechanisms and reinforcement learning activations, a dynamic model of the post-traumatic embodiment is proposed at the end, where the body as a traumatized body is seen as neuroscientific and cellulated. The simplification of complex phenomena such as trauma and focusing on agency and governance as a response to the consequences of such a distorting event may be a key to regaining functionality and reversing the damage, as well as complete healing or the holistic approach.

The model of post-traumatic embodiment that this thesis aims to suggest is a novel point of view on embodiment in terms of trauma studies and trauma therapy. To explain trauma solely in the context of cognitive science and to find a specific frame in the context of entire cognitive science studies is difficult and requires a lot of creativity and responsibility due to the risk such decisions and arguments carry. While the research process and contemplating a subject might be adventurous and challenging, it is questionable if such a model is actually sustainable and if it will fit the narrative of cognitive science research and methodology in the long run. Another limitation of this study is the limited time and maximum dissertation length that had to be adhered to; despite the fact that this topic has been researched for the past three years, the overall work revealed that this is insufficient time to properly and thoroughly research it. The volume of research that shows the consequences of trauma on a body and mental health is, in reality, very broad and possible to divide into even more categories of research. Overall, current research on this topic has been limited and narrowed. It is to expect that a long time and the possibility to research more deeply would give a more detailed representation of the phenomena of trauma but it is lightly possible that conclusions would be any different. It remains a question to be resolved as to how to analyze or determine the validity of this model, how to conduct research within its framework, and whether it is even possible to set up a methodology around this approach to a post-traumatic embodiment.

This is a theoretical thesis work that complies with a simple overview of existing research about trauma’s impact on a person's mind and body and an overview of how the concept of embodiment was developed through a philosophy-to-science timeline. The thesis uses parts of other paradigms and concepts in cognitive science that intuitively seem significant for modeling post-traumatic embodiment to frame a definition and explanation of trauma and its impact on a person in the context of cognitive science. For purposes of simplification and a clear introduction to a topic in a cognitive science context, an environment of computational approach is used with predictive processing being a pre-requisite for it. Such an approach is already seen in cognitive science PTSD studies where settings include reinforcement learning techniques in a direction of efficient therapy.

1.Philosophical views of embodiment

The development of embodiment we know today in cognitive science started with efforts to beat the challenge of the mind-body problem that has been active in philosophy until this day. The mind-body conundrum is concerned with the relationship between these two sets of qualities. Of most interest remained whether physical factors influence mental conditions, and it remained what most sciences are trying to answer. Central questions in a context of mind-body problems are: (a) What is the connection between the mind and the body (what is the relationship between mental and physical characteristics)? (b) Are all mental states physical, or vice versa, or are mental and physical states diametrically opposed? (c) Do emotional states have an impact on physical conditions, and how? (*Dualism and Mind | Internet Encyclopedia of Philosophy*, n.d.)

An embodiment scientific framework began with an emphasis on intentional movement. Philosophers such as Gassendi and Elizabeth were questioning how the mind actually moves the body. The central question was how can the non-extended mind provide an extended effect when wanting is a mode of the non-extended mind and body-part motion is a mode of the extended body (Descartes et al., 1996)? For some item to produce another's item motion, the two must be in contact, and given the mind's non-extended nature, voluntary body motions prevent mind-body communication. Due to the condition of two surfaces' contact, where surface is a characteristic of the physical body, it can be assumed the intellect has no surface that can move the body. If mind and body are separate, there's no way to explain voluntary physical movement. A similar dilemma occurs for feelings, or "body-to-mind causality." A visual object is a mind-only mode that is caused by a movement of undetectable items prompting eye and the optic nerve to move inside of the brain, resulting in the perceptual concept of an observed object. It is of great interest for that period this particular movement of fine items and how can they create a sensory concept when the mind couldn't receive motion due to its non-extended nature? No comprehensible explanation of feelings appears feasible since the mind lacks motion and a surface. The mind and body's distinct natures make causal interaction impossible (Descartes et al., 1996).

Such confusions opened doors for dualistic point of view that was proposed by Descartes and back and forth of standpoints about if mind and body are completely distinct or if they are in causal interaction that requires rejecting these separatist natures of both entities.

1.1. Body in philosophy and science

Body is a central axis of embodiment studies in philosophy and as a first point of research before its own connection and dependance to mind even came into question. Body as a term underwent a lot of metamorphosis and different contexts but somehow always circled back to its undeniable dependency to our mind, especially in terms of consciousness, agency, and overall governance of our social and environmental interactions. To truly understand body as a concept, especially in a context of Merleau-Pointy´s embodiment theory which is a central theory to understanding embodiment in this thesis, it is important to make a short overview of how it was understood and how it interacted with other philosophical constructs such as mind and society/environment through philosophical history.

In terms of a technique, the philosophy of the body rejects the dichotomy between the body and the mind. However, it is difficult to study the body while abandoning certain fundamental patterns of thought regarding the link between the individual and society, nature and culture. The body is mostly negotiated and validated as a product of the discourses from which it is produced (Stoliarova, 2016).

1.1.1. Descartes' dualism

Even though we are examining the connection between mind and body and how we (the embodied mind) interact with our environment (society), it is important to note how the dualistic view seems to be the basis of modern mind-body interaction insights, regardless of its separatist focus. We could assume that "putting a mind into a body" and developing an agency perspective of environment interactions (which will be discussed in later parts) would be difficult if body and mind were not first seen as two very different and separate forms: body as a physical one and mind as a metaphysical one. To investigate potential connections or interactions between such forms, it is first necessary to understand them as distinct entities with distinct properties. According to dualist philosophy, both the mental (mind) and the physical (body) are real, but neither can be reduced to the other. In other words, the nature of the mind is entirely separated from that of the body, and hence one may be existent without the other. In dualism, the entity mind is contrasted with the entity body.

According to René Descartes, mind and body are essentially separate entities, or essences (Descartes et al., 1996). This view, now commonly known as substance dualism, asserts that mind and body not only have distinct meanings but also relate to distinct types of entities. Consequently, dualists would reject any theory that equates the mind with the brain as a physical process. Although the mind and body interact closely, they are distinct beings with distinct essences. The body represents sensation, uncertainty, and contingency, whereas the intellect represents reason, truth, stability, and assurance in terms of the mind.

1.1.2. Body as a center of philosophy

In the 19th century, the relationship between the human body and spirit was reexamined by Nietzsche, who rethought the nature of reason, emotion, thought, and will. The term "body" became a term to fight the arbitrariness of the philosophy of consciousness and a major research topic in the second half of the 20th century because scientists shifted their focus from the inspected object to the subject of ideas and behaviors (Slatman, 2014). Since Nietzsche, the body has been central to philosophy. With the body as a starting point, philosophers started combining the body with society. Comparing society to a body makes its authoritative order more inevitable (Fei, 2020). The human body, in this period, has social and political significance as a natural component. This will later be required to understand the significance of environment in embodiment, particularly in terms of agency.

Michel Foucault says the source is flesh and all its embeddings, like food, climate, and earth. Just as the body develops desires, weaknesses, and flaws, all past experiences are linked in the flesh and sometimes clash (Foucault, 2019). Each body part's functions and operations are influenced and constrained by sociocultural settings, and its modes of activity and results are reflected in these environments. Foucault studied how personal body conditions and modes of activity have been constrained by social systems and regulations in various historical eras, how the body and social systems and regulations interact during the production process, and how social normalization and personal subjectivation fulfill the functions of each body part. He explained how personal demand and desire are tied to the maintenance and operation of the entire social system in terms of digestive and sexual organs. These activities appear to be part of physical existence, but they have social morality and cultural value, and they have been socialized and culturally signified by institutions and rituals over time.

Foucault's research showed how power mechanisms are directly related to the human body, its functions, physiological processes, sentiments, and enjoyment. The relationship between biological and social nature becomes complex; for Foucault, history gets a new meaning and becomes an important construct that gives the body meaning and value (Foucault, 2019). This particular view of history and its interaction with a body will later show itself to be of great importance in the philosophical and theoretical context of trauma as a collection of body experiences stored in various types of memories that then form an individual's meaning and value in terms of personality. Trauma response could be seen as a recollection of all an individual's or body's history and memories stored in the body and mind that have the power to be relived in the present moment.

Gilles Deleuze saw the body as an ascending, producing force, like Nietzsche. Deleuze expanded on Nietzsche's body theory by describing it as a producing force or desire machine. The body, as such a machine, can grow, evolve, and expand forever. Internally (a body without organs) and externally (a body without skin), desire is repressed by society. In this context, a body without organs is an antistructural, generative, and variable body. A body without organs exerts organ persecution or agent harassment (Deleuze, 2011; as cited in Fei, 2020). Decentralized, fractured, and dynamic describe desire. The movement of desire is not to find objects that can satisfy it but to find new connections and representations within its overflowing energy. Desire could be seen as what will later be described as "agency" or some motivational factor that makes agents head towards a goal set in their environment.

1.2. From phenomenology to psychology and cognitive science

1.2.1. Integrating mind into a body

In the second half of the 20th century, numerous factors contributed to the transition from phenomenology to psychology. Merleau-Ponty's phenomenology of the body was a philosophy-based psychology that replaced Descartes' dualistic view with the perceptual body-subject. Merleau-Ponty argues that perception must be fundamentally reinterpreted and examines the embeddedness of perception and the body's intentionality.

Merleau-Ponty saw topics such as time, space, the natural world, freedom, and intersubjectivity all in the wide context of the body. In this view, the body is the proprietor of vision and touch, not only their instrument. The body creates circumstances for conscious objects and perception-derived meanings. It observes everything and can observe itself. It is the most visible and noticeable item on its own (Merleau-Ponty, 2002). The body and the world coexist, and the world is shown to a person with all the components of his or her body through a similar link to that which exists between the parts of the body. The comprehension of the world begins with the comprehension of the body, as the world is not an objective existence and cannot be exhausted by rational knowledge. Through bodily experience and perception, one can access the world (Zavota, 2016).

George Lakoff and Samuel Johnson established this concept of perceiving the world through the body at the end of the 20th century. They challenge the notion that the physiological structure and physical experience of humans play a crucial part in the formation of meaningful concepts and reasoning. On this premise, they asserted that the construction of the concept of space is the consequence of continuous spatial experience; this most fundamental concept is the outcome of the interaction between persons and their natural environment (Lakoff & Johnson, 2003). Mind and cognition are inseparable from direct physical experience, which is achieved on the basis of a particular, vast, and profound culture. Culture is implicit in every experience because it is how we experience the world in which we live. Our experience is structured by the natural dimensions of our bodies and the essence of our natural and cultural contexts. Repetitive experience creates categories that experience gestalt with natural dimensions. These gestalts define experience's coherence. We understand experience directly when we rely on gestalts obtained directly from ourselves, our environment, and our interactions with the environment, and when we believe experience has coherent structures; "we understand experience metaphorically when we use a gestalt from one domain of experience to structure our experience of another domain" (Lakoff & Johnson, 2003).

Merleau-Ponty (2002) explains that the movements of organisms are constrained by external influences and that we can take actions that are entirely the result of the environment. In the same way, all stimulation from an organism is gotten through its prior movements. According to Merleau-Ponty, the body chooses the stimulation it wants to experience in the physical environment based on the type of receptor, the thresholds of its nerve center, and the organization's actions. Based on this, the theory of embodied cognition has established the following propositions: a) Cognition is dependent on the variety of sensory experiences generated by bodies with diverse sensory motions; b) the perception-movement capabilities of individuals are rooted in a larger biological, psychological, and cultural framework. These ideas underline that perception and movement processes, as well as consciousness and action in nature, are inseparable in cognition because they are linked in the individual not just by chance but through evolution and integration (Varela et al., 2017).

Growing research indicates that cognition is anchored and embodied (Fei, 2020). However, abstract notions remain a formidable theoretical obstacle. Shannon et al. (2009) argues that a good description of how language augments cognition must highlight its symbolic features and embrace a perspective of embodiment that acknowledges the fluid, multimodal, and task-related nature of action, emotion, and perception systems. Allen and Friston (2016) demonstrated how the free energy principle might alleviate tensions between internalist and externalist theories of cognition by offering a formal, synthetic account of how internal representations emerge via autopoietic self-organization. Thus, the free energy principle provides empirically fruitful process theories for driving discovery via formal modeling of the embodied mind. As an alternative to representational approaches to the imagination, Medina (2013) has proposed an enactivist method for investigating how the enactive imagination operates in animal cooperative behavior and animal communication. His method suggests that a person's active imagination is essential for cognitive, affective, and moral development. How, if at all, emotions and subcortical contributions fit into this evolving picture remains unknown. Miller and Clark (2017) have proposed a closely coordinated process of ongoing reciprocal causation that ties together bodily input and top-down predictions, creating a coherent understanding of what is out there and why it matters. The fundamental concept of the mind in the body is that the mind is not only a distinct entity or attribute but rather an act or physical action, and that all mental activity is rooted in physical activity. If we can refer to the mind regulated by dualism as the mind of an entity and of an epiphenomenon, then we can refer to the mind within the body as the body-mind of an embodied mind, or as the physical mind.

Substantial body of research indicates that our conceptions are frequently embodied and rooted in sensorimotor systems (Varela et al., 2017). In the process of investigating the mind and challenging dualism, the philosophy of the body contradicts the mind's alleged cognitive priority and supremacy. This denial has shifted the focus of research from consciousness to the body, and subsequent investigations take into account both the notion of belonging and the possibility of bodily independence (Gallagher & Zahavi, 2012). This leads to understand the relationships between individuals and the world in a new way - with meaning, sense, and the body serving as their foundations.

1.2.2. Embodiment of a mind

As advances in psychology were made in the 1970s, the cognitivist or symbolic approach began to dominate the field of study, particularly in the form of connectionism, which assumes neural network processing is similar to human cognitive activities via biological functions of the brain. In this era, the field of artificial intelligence (AI) emerged, which saw awareness as a method of information processing in the human brain.

Symbolism views human brains as symbolic operation systems and human thought as symbolic processing; intelligence can be modeled using static, sequential digital calculation models. Connectionism merges cognitive processes and human brain characteristics based on neurophysiology, where information is translated and subsymbols are processed in parallel using digital properties (Dyer, 1991). Centrally published commands direct undistributed systems. Distributed systems have multiple command-issuing organizations for each network layer. The distributed system is fault-tolerant, self-learning, associative, and fast. Typically, the components of awareness' pattern of great excitation occur alone. Consciousness is serial, but the brain processes information in parallel. Consciousness reflects the brain's most important event. The brain is a parallel information-processing system with awareness as its serial core (Horgan & Tienson, 1991).

Cognitive psychology's rich experimental results have changed the face of psychology, but they've been widely attacked, especially on the methodological level. Individual cognitive phenomena have been the focus of cognitive psychology research, to the exclusion of inter-individual, inter-cultural, and inter-institutional interactions. Cognitive psychologists often describe mental processes using computer metaphors. Programmers don't need to understand hardware, so cognitive psychologists perform behavioral analysis. Increasing numbers of psychologists recognize that human information processing differs from that of a digital computer in almost every significant way.

Mental representation and computation theory underpin modern cognitive psychology. The representation theory sees cognition as a replication of the object world, while the computation theory sees it as a process of computing information or manipulating symbols using formal systems or algorithmic principles. In either case, the computational mind is exposed, which is different from the experienced mind, even though human cognition must include both (Jackendoff, 2002). We haven't solved the consciousness enigma, but we know it will require multiple disciplines. Over the past three decades, cognitive psychology has emphasized the occurrence and development of psychological phenomena in daily life to establish a cognitive model consistent with the human experience of consciousness and that reveals cognitive activities through intuitive experience.

Due to the specificity and precision of quantitative research on psychological processes, nearly all modern cognition theories can be tested in the lab. In many ways, lab research doesn't replicate real life. In a psychological experiment, the stimulus indicates the experimenter's subjective response, not the subject's. This interference with independent variables may affect the subject's comprehension of the experimental scenario and role, as well as their motivational decisions. Participants show the so-called Hawthorn effect, or demand characteristics in response to the experimenter's hypotheses (Orne, 2002). Such experimental effects are evidence that the experimental scenario differs from the real world; because the former is a transient, artificial, and abnormal situation created by subjects and experimenters, extrapolating experiment results to a larger population is problematic. With more research, it's clear that experimental approaches are limited. The study of interests, needs, emotions, and hopes involves unrelated variables that are difficult to control. The experiment's artificial atmosphere affects the subject's psychological and behavioral responses, reducing its validity. Some studies don't control their subjects for ethical reasons, instead customizing each participant's setting. Neisser et al. (1996) have noted that the study of information-processing approaches has not investigated human nature outside the laboratory and that its fundamental assumptions have not gone beyond computer models. Human minds are sensitive organs, not general-purpose computers. Eysenck and Keane's (2010) caution is understandable. The more scientific the study of memory becomes, the further it strays from its intended goals because new phenomena lead to many sub-phenomena.

Although this perspective is negative, it's helpful to know how motivational and emotional shifts affect internal cognitive processes. Cognitive psychology will repeat behaviorism's errors if it focuses on inner psychological processes under strict control without considering daily perception and memory, becoming a narrow and specialized field with no broader significance. These reasons don't mean descriptive scientific concepts and procedures shouldn't be used in psychology, nor do they reject separating physical and spiritual experience. Modern psychology's physics-based biases must be viewed critically. It criticizes the use of empirical notions for direct description when such concepts have not been adequately analyzed and how descriptive and explanatory sciences are juxtaposed and viewed as equivalent interpretations.

Phenomenology studies how people perceive reality. Subjective emotions, not objective reality, rule phenomenology. What we experience isn't always the same as the objective world, and our behavior depends on that. Edmund Husserl believed that feelings provided direct knowledge of the world's original form, but that our intent can corrupt this authenticity. Distortion can range from an optical illusion to racial hatred. Scholars who emphasize phenomenology in psychology suggest supplementing scientific study with human experience-based phenomenology.

Their reasoning is that psychology is different from the natural or biological sciences because it studies self-aware humans. Human experience is alive and can be stated clearly in the first person, and consciousness is an important issue. This concern requires research methods different from traditional science and more suited to behavioral research (Gallagher & Zahavi, 2012), which eventually opened the door to broader approaches in research.

1.2.3. Mind-body interaction overtake in science

Experimental psychologists were focused on a fundamental understanding of brains and sensory organs. They believe that this understanding must be based on controlled studies conducted in simulated situations. Cognitive psychologists place their subjects in an experimental context in an effort to exclude specific cultural material in order to define and classify their subjects' performances. Their research stresses and investigates the universality and presence of fundamental cognitive structures. In contrast, the ecological paradigm of cognitive research stresses the cultural roots of cognitive functions. We recognize, for instance, that in the majority of cultures, the memories of men and women will differ due to differences in education and career; similarly, we observe that witnesses from very different cultural backgrounds have different recollections of the same events, particularly complex events that are recounted orally.

In the past two decades, ecological validity has received a growing amount of interest, gaining widespread acceptance among psychologists and becoming a crucial reference for experimental research and design. The ecological validity of a study in psychology measures how applicable its results are to everyday life. This idea stresses research on psychological processes and events that occur spontaneously and have functional importance. It is used to determine whether a theory or an experimental finding has practical relevance, and it identifies applicability to multiple populations, tasks, and stimuli as a prerequisite for external validity (Galotti, 2017). In other words, if a study lacks ecological validity, it simply reveals the psychological and behavioral responses of the subjects in the study, not their true representation in everyday life.

Hermann Ebbinghaus, an early cognitive psychologist, employed meaningless syllables as memory aids in order to simplify stimuli and isolate reactions. Consequently, he discovered the well-known Ebbinghaus memory curve. This type of formal research rarely examines behavior with daily relevance. Despite this, it offers a lot of benefits. It is grounded in the daily lives of its subjects; it demonstrates that the memory materials for descriptive recall patterns are distinct from those for meaningless syllables; it does not require any specific observation or recall techniques; and it does not impose time constraints.

In the middle of the twentieth century, Frederic Bartlett discovered that the advancement of psychology theory was dependent on research that solved practical problems. His work bridged the gap between theoretical and applied psychology, making psychological research more grounded in reality. He opposed Ebbinghaus' use of meaningless syllables isolated from reality and began studying memory in contexts resembling real-world settings. His materials were images and stories from everyday life, blended into what he termed the descriptive approach, the hieroglyph method, the series replication method, and the diary method, and he utilized them to analyze the entire process of memory as well as his own autobiographical memory.

In Bartlett and Burt (1933) opinion, mental processes tend to be contained in memory words in a realistic manner, i.e., as they truly occur in a normal individual, within or outside of a social group. In addition to considering the accuracy of the recall, the purpose of his methods was to analyze the subjects' free descriptions and their thoughts when answering the experimenters' test questions, thus examining the subjects' naturally occurring behaviors rather than those prompted by the psychologists.

Although Bartlett's hypothesis has many speculative elements and was never widely accepted, his research encouraged many individuals to consider the nature and dynamics of memories from an entirely new angle. Cognitive research was previously dominated by the computational-representational theory of the mind. In terms of this theory, representation is the format in which the brain receives and stores information. Both cognitivism and connectionism view cognition as a representation; however, cognitivism views symbol processing as the suitable bearer of representation, whereas connectionism views the complete emergent representation as the world's representation (Thagard, 2012). In part, the concept of representations is based on foundationalism and essentialism, the views that information and meaning are expressed through occurrences and that symbols are vehicles for exposing information or significance.

The generative perspective rejects representations as cognitive science's Archimedean vantage point and instead views cognition as an embodied action. It underlines that cognition is the combined enactment of the mind and the external environment on the basis of all human activities. Other researchers argue that human cognition is devoid of patterns and norms because the brain and environment constantly exchange information in a state of flux and that cognition is the result of the interplay between the cognitive subject and the environment. Cognition is a dynamic system that encompasses everything that is subject to change over time. In their opinion, symbolism and connectionism disregard the significance of time in the cognitive picture. In actuality, the state of the brain is continually evolving, as the state space of a non-computational dynamic system is constantly expanding. Merritt (2013) emphasizes the viewpoint of radical enactivism from the unique vantage point of the dance of meaning-making by highlighting how agency distinguishes meaningless movement from meaningful movement.

Phenomenology's psychological importance derives from its emphasis on the oneness of humans and the world. Spirit is an independent, self-contained existence, and the object that exists in consciousness is distinct from that of nature. The relevance of experience and the experience itself can be the subject of psychological study. Noting that phenomenology does not preclude empirical approaches and that phenomenological and empirical methods are interrelated (Yoshimi, 2021).

1.3. Embodiment in cognitive science

To perceive what being embodied would actually mean in Merleau-Ponty point of view, and how it can be easily understood, we would have to simplify it in every-day language levels. Following this logic, we can say that to *trust our bodies and truly feel linked to them* would mean that we are embodied. To make choices and selections of social environments, social or personal interactions, and movement or nutrition based on what select food and movement and friends and companions based mostly on what feels good and true and right in our bodies means we are embodied, following this logic.

Our physical body can also be seen to some extent, and our senses, if expanded, can understand the surrounding world by removing our grounding inside of senses (Merleau-Ponty, 1980). With these detachable organs, our senses are not limited to our physical bodies but extend to the environment and bridge the body's connection to it. Senses perceive information from the environment and help the mind make predictions about the world, then send signals to the body about interactions it should undertake. Merlau-Ponty's view is, in a way, simple and most suitable to take as a starting point in explaining embodiment in a cognitive science context.

1.4. Embodied Cognition

Cognitive science mentions the terms embodied cognitive science and embodied cognition, and the two are often interchangeable, even though some literature might show differences in definitions. Due to this fact, the mentioned two terms should be approached separately for purposes of deeper and more constructive research. For the purposes of this thesis, the context of embodied cognition will be used more to simplify the simple approach to understanding embodiment in cognitive science, particularly because of how well it explains the body's and mind's reliance on the environment.

Cowart et al. (2005) defines embodied cognition (EC) as a cognitive science program of analysis with an emphasis on the formative position of the surroundings in the creation and improvement of cognitive processes. Some cognitive process develops based on a central idea when an organism-environment connection with a clear direction toward a clear objective creates a well-coupled system in real time. How specific cognitive abilities develop is largely determined by the nature of the previously mentioned organism-environment interactions. By presenting embodied cognitive science, it is important to point out its link to situated cognition. As a paradigm within situated cognition, embodied cognitive science differs from both the study of embedded cognition and the theory of extended cognition. A person's cognitive processes are said to be embodied when they are heavily reliant on their physical body, or when aspects of their body other than their brain play a causal or physically constitutive role in their thought processes (Moya, 2014). Postulator to organism-environment engagement is the connection that can be seen as the foundation for an agent's activities and that allows the agent to move in an oriented manner in a specific region (Merleau-Ponty, 1980; Talero, 2005).

Gallagher and Zahavi (2012) point out how this reference to the environment is of great importance, as it entails understanding of the body's (the agent's) physical environment and surroundings and puts emphasis on events that are meaningful for a body. Meaning is made up of our embodied experience patterns and preconceptual structures. These embodied patterns are neither private nor unique to the one who experiences them. Many of our felt designs are interpreted and codified with the aid of our surroundings, which are engraved in us as cultural modes of experience and contribute to the determination of the character of our meaningful, coherent worldview.

Embodiment seems to be necessary prerequisite for cognition, and it can also be defined as the unique way in which an organism's sensorimotor capabilities enable it to interact efficiently with its surrounding environment. Furthermore, the goal of all iterations of the general embodied cognition thesis is to provide cognitive explanations of the interplay between an organism's cognition, behavior, and environment as they work together to foster adaptive success.

The emphasis of the modern cognitive science approach is on symbol manipulation. Embodiment is intended to emphasize physical actions that affect how the brain influences and reacts to the world. The brain is the one seeing the world and, by design, demonstrates how the structure of the world will determine the body type of organisms. That is how important the environment is for a body and how the body and environment ultimately produce cognition. Firstly, disembodied symbols need to be processed, which begins with a sensory system. The process then continues to the motor system, where ongoing interactions between the brain, body, and world happen (Shapiro, 2019). This subserves the theory of dynamical systems that describes the behaviors of self-organizing systems when their components are coupled: human bodies, their minds, and their environments are all viewed as dynamical systems that are constantly influencing one another (Beer, 2008). The image below (Figure 1.) shows this interaction between the agent's nervous system (brain), body, and environment. As can be seen, all those components are in constant interaction.

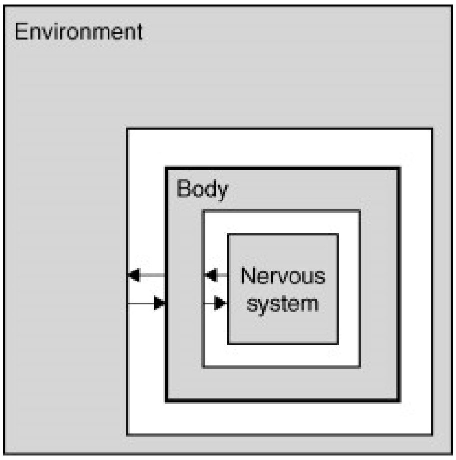

Figure 1. *An agent and its environment as coupled dynamical systems* (Beer, 2008). The agent is made up of dynamical systems: the body and nervous system. Nervous system and body interact with each other and body and environment interact as well, in the same time.

The ideas mentioned are compatible with EC theorists' views that we comprehend the world through our senses. According to Silver et al. (1982), thinking derives from an agent's embodiment, which includes the brain and cognitive systems that allow us to detect and move, construct conceptual frameworks, and reason. To understand reason, we must understand our visual, motor, and neural binding systems. According to Gallese and Lakoff (2005), conceptual knowledge is embodied. The world that an agent perceives is decoded and assigned a meaning in the nervous system (brain), where the concept of a world and full worldly experience that incorporates in the nervous system through mentioned visual, motor, and neural binding systems and conceptualizes

Another concept important to mention in the context of embodied cognition is abstract thought processing, which has potential importance for understanding trauma in this context. Casasanto and Boroditsky (2008) were figuring out the abstract idea of time, and they interpreted it metaphorically in terms of space and concluded that spatial representations underlie human time perceptions. This shows that humans represent abstract ideas through sensorimotor skill, which for purposes of this thesis can be taken as an underline for forming the meaning of trauma. Trauma can be seen as some abstract content, an event, perceived in the environment, and its meaning is hypothetically connected to time perception as it can be assumed it forms its meaning in terms of memory, being processed and stored in the limbic system.

2. Trauma

Trauma may be described as bodily harm, a catastrophic occurrence, or psychological and physiological reactions to an extraordinarily unfavorable experience, in most cases ending in a feeling of helplessness. Psychological trauma is considered to be a succession of events that, because of their unique qualities, can have a substantial impact on a subject's mind, putting at risk the integration of his or her thoughts (Newbold et al., 1902). The word "trauma" derives from its Greek origin, with its meaning referring to wounds caused by slicing the skin open, a terminology and meaning that has remained in a medical field to this day. The term was first used in science and psychiatry in the seventeenth century, but the mention of an open physical wound caused by injury is an indication and subtle reference to trauma's dependency on the body, or embodiment. When taken into account intuitively, it is clear that the word *trauma* is taken as a metaphor for wound, injury, and managing to heal a body once they inflicted an injury causing a scar -- it is already implied in the word's origin that trauma as itself is something that has consequences for a whole human system, predominantly the psyche, as psychiatry will be centered around the psyche concept for a while, but also a body. Regardless of psyche studies that highlight the origins of psychology as a philosophical and scientific context, it can be hypothesized that even psychiatrists used the term intuitively and approached the psyche in such a way that they concluded the body in this approach. Indirect evidence for it comes from the use of pharmacotherapy and attempts to understand the neurobiology of stress and mental disorders. Clinical psychology later became focused on figuring out how disproportionate the influence of an individual's resistance to stress is when confronted with a demanding stimulus.

The experience of being a witness to an event that poses a threat to one's life or physical safety can be traumatic. Extreme fear, helplessness, and anxiety are common reactions to these kinds of situations, suggesting that they may play a role in the onset of post-traumatic stress disorder (PTSD). The experience of being a witness to an event that poses a threat to one's life or physical safety can be traumatic. Extreme fear, helplessness, and anxiety are common reactions to these kinds of situations, suggesting that they may play a role in the onset of post-traumatic stress disorder (PTSD). Kloet (2012) points out that a person's response to trauma depends on aspects unique for the individual and not only the qualities of the stressor. For many people immediate threat causes acute, brief disruption. These disruptions are unpleasant and can be sorted into three groups by phenomena of a certain characteristics. Sherin and Nemeroff (2011) are categorizing them into following groups: “(a) reminders of the exposure (flashbacks, intrusive thoughts, and nightmares); (b) activation (hyperarousal, insomnia, agitation, irritability, impulsivity, and anger); and (c) deactivation (numbing, avoidance, withdrawal, confusion, derealization, dissociation, and depression)”. These responses are self-limiting and result in minimal damage in functional sense within course of a time. Opposite to this fact is the psychological trauma that appears as a consequence of a profound danger and has evidenced long-term consequences such as a longer-term illness that is dubbed in the PTSD clinical picture in the psychiatric literature - debilitating functional disability often associates to PTSD (Brown, 2000).

A traumatic encounter can be categorized as a single or type 1 (occasional, one-time happening), repeated, cumulative, or type 2 (repeated, cumulative, and multiple impacts over a period of time), with one or more intervening events being interdependent (Perrotta, 2019). The Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders covers a variety of disorders that emerge as a consequence of traumatic and stressful situations and assumes an etiological perspective. DSM-V (5th ed.; DSM–5; American Psychiatric Association, 2013) mentioned the following categories of disorders connected to trauma: reactive attachment disorder, schizoaffective disorder, schizotypal personality disorder, uninhibited social engagement disorder, post-traumatic stress disorder, acute stress syndrome, and adaptation disorders. This thesis will mention only the few most recognized ones in this context, the most general ones, and will not go deeply into psychotic symptoms and dissociation as the etiology and neurobiology of those disorders are connected to, yet particular to, the PTSD context. It is important to mention for the context of this thesis that if a person develops PTSD symptoms that are typical of the psychotic spectrum, such as depersonalization (where person feels like and outside observer of their body and feels detached from their mental state) or derealization (where person feels as the surrounding environment is partially unreal, either persistently or recurrently), the risk of a full-blown psychotic breakdown is high (Perrotta, 2019).

2.1. History of trauma in science

Until the late 19th century, there were no attempts to classify traumatic experiences or the clinical significance of such experiences. Pierre Janet and Sigmund Freud were the first to come up with certain explanations, put predominantly in the context of hysteria based on people's exposure to traumatic events, especially sexual ones (Herman et al., 2016). Pierre Janet described 591 cases, of which 257 had traumatic backgrounds (Janet, 1987, as cited in van der Kolk, 2000). Janet argued that vigorous emotions prevent individuals from processing painful experiences. According to him, the painful memories are separated from regular awareness and deliberate control. He observed that traumatic memories frequently resurface in the form of reenactments of overwhelming emotions, aggressive behavior, physical pain, and physical states. Janet discovered that traumatized people react to reminders of their traumatic experience with actions that were once effective but are now ineffective—reminders evoked images, emotions, and bodily sensations associated with the trauma. If a person has not effectively dealt with trauma, they will have difficulty processing new information. Trauma memory mechanisms impeded people's ability to act deliberately and gain insight from their experiences, so personal and professional performance declined until the fragmented components of the trauma became conscious (van der Kolk et al., 1989). In the 1880s, Sigmund Freud, in cooperation with Joseph Breuer, studied the motor and sensory abnormalities of hysterical patients and concluded that the traumatic memory (Lewis, 1992, as cited in van der Kolk, 2000) operates as a foreign body and must be treated as a persistent agent. Repressing an emotional reaction connects the emotion to the memory. According to Freud's notions, even if a traumatic memory is not eradicated, it will be assimilated into the network of associations and corrected by other ideas—a connected sensation can be eliminated through association.

A therapist named Abram Kardiner wrote about the biological and psychological effects of trauma. He stated that individuals with traumatic neuroses are typically hypervigilant and hypersensitive to perceived environmental threats. This predates every accommodating mechanism in the chain of command and persists across the entire organization and battlefield. The trauma-related syndrome persists. He observed that these people were hypersensitive to heat, discomfort, and abrupt touches (Kardiner, 1941; as cited in van der Kolk, 2000). This hysteria-directed view was redirected, and the later trauma context was shaped by research into the experiences of male soldiers exposed to war. In the aftermath of World War I, researchers began studying PTSD and developing effective treatments for war veterans. An independent PTSD diagnosis was added to the DSM-III as a result of research into trauma reactions in both combat and interpersonal violence (DSM-III; APA, 1980; as cited in Jones & Cureton, 2014). Prior DSM editions recognized reactions to stress as a temporary situational disruption, meaning that in the absence of a preexisting mental disorder, the individual's psychological experiences would disappear as the stressor subsided (Jones & Cureton, 2014; Yehuda & LeDoux, 2007). The DSM-IV and DSM-IV-TR publications expanded the definition of trauma significantly. Automobile accidents, natural disasters, learning of a loved one's death, and even a particularly contentious divorce were all classified by DSM-V as traumatic experiences. Modern trauma theory depicts trauma and traumatic responses as something operating along a continuous spectrum in order to better understand the genesis, direction, body functions, and success of therapy in case of a traumatic response. Six symptoms from one of three core symptom clusters were used to make a diagnosis of PTSD in the DSM-V (American Psychiatric Association, 2013). There are needed at least one symptom connected to experiencing trauma again, three symptoms of avoiding or emotionally numbing, and two markers of increased arousal, that should last at least for one month after a traumatic event to proclaim possible PTSD. It could also be considered if there is rapidity, persistence, or tardiness of the onset of mentioned symptoms, and added into observation of possible diagnosis.

2.2. Trauma and brain

The brain can undergo plastic changes in anticipation of future experiences, similar to how it changes when those experiences actually occur. The brain is pliable and capable of growth. Trauma seems to impact mostly three regions: a) the prefrontal cortex; b) the anterior cingulate cortex; c) the amygdala (Perrotta, 2019). The primary role of the amygdala is to process all incoming sensory data to decide whether or not it poses a threat. If it detects the danger, it induces fear, which heightens our awareness and action readiness. The prefrontal cortex and anterior cingulate cortex are less active in the traumatic brain, while the amygdala is more active. The individual lacks a feeling of safety, which is linked to the hyperactivity of the amygdala. While the prefrontal cortex is less likely to be active in a traumatized brain, a trauma survivor may have difficulty focusing for extended periods and perceive a lack of clarity (Felitti et al., 1998; Perrotta, 2019).

Stress and trauma activate the body's intrinsic defense systems by stimulating the part of the brain that we used as an automatic response to danger in the past. Van der Kolk (2000) defines trauma as a terrible, irreversible illness caused by stress. In his book *Body Keeps the Score* (der Kolk, 2015), he discusses how stress and trauma are linked mentally and physically in the context of the previously mentioned prehistoric brain response. It is supposed that humans might have two brains: the neocortex, or *thinking brain*, which is important for reasoning, planning, and memory and is controlled intentionally, and the *survival brain*, which consists of the limbic system, brainstem, and cerebellum and controls unconscious survival functions like breathing, sleeping, and hunger in all mammals. The survival brain is the one that manages stress.

The human brain uses neuroception to detect internal and external hazards, allowing bodies to evolve a stress response—a biological mechanism capable of coordinating the body's key systems to protect us from injury (Porges, 2009). When the survival brain perceives a threat, it activates the autonomic nervous system (ANS), which governs hormones, heart rate, and digestion. The ANS has two divisions: the sympathetic nervous system (SNS), which activates the stress response, and the parasympathetic nervous system (PNS), which reduces it. The mind-body system engages the lines of defense when the SNS triggers the stress reaction, stimulating flight-or-fight responses. The stress reaction releases energy, increases physical strength, improves mental clarity, and even increases pain resistance. It is one of the primary reasons why people have survived for so long.

The stress response of the human body is comprised of two waves, based on Newport and Nemeroff insights (2003): (a) the sympathomimetic dorsal pathway (SAM) axis and (b) the hypothalamic-pituitary-adrenal (HPA) axis. The first wave is the fight-or-flight response generated by our brain's control center, the hypothalamus. Adrenaline is produced as a direct outcome of the sympathetic nervous system's rapid activation. The physiological effects of adrenaline release increase the heart rate, improve blood flow to the brain and limbs, release energy into the bloodstream, and activate the immune system to guard against infection. It also enhances the sense of smell and vision. The second wave occurs when the adrenaline surge causes the adrenal glands to create cortisol. Cortisol regulates the activity of the immune system by counteracting the action of adrenaline, and in its absence, the immune system would become overactive and damaging to the body.

Though it may have short-term benefits, chronic stress is harmful to our health (Gatchel et al., 2007). Cortisol and adrenaline may be poisonous to the body in large quantities. Chronically elevated cortisol levels might cause digestion troubles, weight gain, and memory loss. In contrast, adrenaline boosts blood pressure and can increase the risk of a heart attack or stroke (Hannibal & Bishop, 2014). Hyperarousal, commonly known as the "fight, flight, or freeze reaction," is a state of heightened activation. It occurs when a person's nervous system goes into high alert, even when there is no threat present, and is closely related to the acute stress response (Bracha, 2004). It is frequently triggered by a perceived threat, traumatic experiences, or certain emotions. It is also one of the key symptoms of PTSD. Symptoms of hyperarousal include rage, fear, anxiety, overwhelming emotion, panic, hypervigilance, and muscle tension. People who are hyperaroused frequently struggle to develop healthy sleeping habits, control their emotions, and focus effectively. Physically, their body may appear taut and on the verge of exploding, which can lead to furious outbursts and antagonism (van der Kolk, 2003).

As van der Kolk (2015) further explains, hypoarousal is also known as the shutdown or collapse reaction. It is frequently triggered by feeling threatened, recalling painful memories, or experiencing emotions connected with prior trauma. Even a perceived danger might cause a client to shut down or dissociate. Hypoarousal symptoms can include depression, numbness, emptiness, the feeling that the body is flaccid, a blank stare, the inability to talk, and dissociation. Hypoarousal occurs when a person has insufficient arousal as a result of an overburdened parasympathetic nervous system. It can disrupt sleep and food patterns, leaving them emotionally numb, socially isolated, and unable to express themselves.

The survival brain manages stress recovery by restoring the brain's, hormones', immune system's, and neurological system's health through allostasis. Chronic stress and unresolved trauma might keep the survival brain from feeling safe. In such a case, the body puts its focus on immediate safety rather than long-term wellness because its mechanisms of responding to stress are permanently activated and unable to release allostatic burden by releasing short-acting stress hormones such as cortisol and endorphins while decreasing growth and sex hormones (Vermetten & Bremner, 2002). The body and brain become dysregulated when neurological, hormonal, and immune system processes are damaged.

2.3. Stressors

As seen above, trauma is the overall collection of consequences of some stressful event, or, in other words, the body and mind's response to it. Trauma is not the event itself. This unexpected or intense event that causes stress, which eventually transgresses into trauma, is called a stressor. It may be physical or psychological, acute or persistent. A person's subjective assessment of the stressor and their assessment of their available coping mechanisms are both crucial in determining the full extent of the stressor's influence on that person (Ursin & Eriksen, 2010).

Trauma causes mental health problems that are classified as trauma- and stress-related in psychiatry and psychology. When a person has difficulty adjusting to a recent stressor—anything that directly or indirectly increases a person's bodily or psychological requirements and existing resources are insufficient to make a positive outcome, the result is helplessness (Ursin & Eriksen, 2010). This is seen as a threat, real or imagined. Many people may face the same difficulties in their lives, but only a small number will need psychological help.

2.3.1. Psychological stress

When the body perceives a threat, it reacts physiologically to stress (Marshall & Garakani, 2002) and triggers higher levels of cortisol which aids survival in immediate, life-threatening situations. Cortisol is produced in response to chronic and prolonged stress and is harmful to the body and can cause tissue destruction, high blood pressure, and heart damage.

To understand how stress modulates the immune system in the brain, it is crucial to understand the distinction between short-term and long-term stress. Because it prepares the body to face obstacles, short-term stress can be protective and immune-enhancing, whereas long-term stress When the body perceives a threat, it reacts physiologically to stress (Marshall & Garakani, 2002) and triggers higher levels of cortisol, which aids in survival in immediate, life-threatening situations. Cortisol is produced in response to chronic and prolonged stress and is harmful to the body. It can cause tissue destruction, high blood pressure, and heart damage. Chronic stress is harmful and suppresses the immune system (Straub & Cutolo, 2017). On the other side, recent research (Cutolo et al., 2006; Herrmann et al., 2000; Lee et al., 2016; Walker et al., 1999; Straub & Cutolo, 2017) suggests that systemic lupus erythematosus, rheumatoid arthritis, and juvenile idiopathic arthritis are all examples of chronic autoimmune-inflammatory illnesses that may be exacerbated by psychological stress. Most stressful circumstances can set off the inflammatory response, which is what causes most chronic inflammatory disorders. A stress-induced illness flare-up could be exacerbated by one or two additional factors: the interplay between psychological stress and an additional proinflammatory factor (Straub, 2014).

The stress response of the human body is influenced by both the HPA axis and the SAM axis. The fight-or-flight response is triggered by the hypothalamus, the command and control center of the brain. Adrenaline is produced when the sympathetic nervous system is rapidly stimulated. Adrenaline stimulates the cardiovascular system, increases oxygen delivery to the brain and limbs, releases stored energy into the bloodstream, and primes the immune system to combat infection. As a result, the senses of smell and vision improve. The second phase of the stress response occurs when the adrenal glands produce cortisol in response to the adrenaline surge. This hormone regulates immune system activity and serves as an antidote to adrenaline. A hyperactive immune system is detrimental to the body, and without cortisol, it would become exactly that.

2.3.2. Short overview of stress disorders

2.3.2.1 Post-traumatic stress disorder (PTSD)

When the mind and body react negatively to a traumatic experience, post-traumatic stress disorder (PTSD) may develop (Daffin, 2018). This includes being present at the occurrence of a traumatic incident, hearing about the painful experience of a close relative or friend, or experiencing a sequence of unpleasant occurrences. The estimated prevalence of post-traumatic stress disorder (PTSD) in the overall population is 7% (McLaughlin et al., 2014), with a much higher incidence among groups sensitive to serious psychological trauma (Mota et al., 2016). In the general community, only a small fraction of those who have experienced trauma go on to acquire severe PTSD (Breslau, 2009).

Each of PTSD's four clusters of symptoms, as described in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V) (American Psychiatric Association, 2013), must have begun or been significantly exacerbated after exposure to the traumatic event: intrusive recollection of the original traumatic event, avoidance of trauma-related reminders, negative changes in cognition and mood, and alterations in arousal or reactivity. Recurring symptoms must be unique to the traumatic event or its aftermath in order to be diagnosed with PTSD. Recurrent bouts may last anywhere from a few seconds to several days. They are frequently triggered by similar bodily experiences or environmental cues, such as a specific location. People PTSD tend to avoid triggers and anything that could reactivate traumatic memories. As a coping mechanism, individuals with PTSD may suppress negative emotions and/or thoughts. Avoiding the places, people, activities, and events that evoke unpleasant memories is one way to prevent them from returning. Due to PTSD, affected individuals may exhibit altered thought processes or emotional states. Those with PTSD frequently report having a negative worldview.

The mood and frustration of PTSD patients can lead to hostile verbal and physical behavior. These responses may be the result of a heightened awareness of potential threats, particularly if the new threat is similar to one the individual has previously encountered. PTSD patients frequently experience hyperawareness and heightened senses. Symptoms can result in significant social, occupational, and other difficulties. At least one month of distressing symptoms must be present before post-traumatic stress disorder can be diagnosed. Those who have been missing for a month may have ASD.

2.3.2.2. Acute stress disorder (ASD)

Symptoms of Acute Stress Disorder (ASD) are similar to those of Post-Traumatic Stress Disorder (PTSD), but they must first appear within three weeks to one month.

Post-traumatic stress disorder is diagnosable if symptoms last longer than a month. A person does not have acute stress disorder if their reactions to a traumatic event subside within three days.

There are at least nine symptoms that must be present from the following five categories to diagnose ASD: intrusion, negative mood, dissociation, avoidance, and arousal. Hyperawareness, inattention, and sleep disruptions are common symptoms. For a diagnosis of acute stress disorder, a patient must exhibit nine symptoms that are either extremely distressing or severely impair daily functioning.

2.3.2.3. Maladjustment disorder

An adjustment problem stems from a recent stressful event. This stressor may exist in the form of a single occurrence or a chain of occurrences. There is no set group of symptoms necessary to diagnose adjustment disorder. For stress to be considered a causal factor, the individual's symptoms must be both stress-related and severe enough to cause significant impairment in social, occupational, or other important areas of functioning.

3. Neurobiology of trauma

The majority of studies conducted since the early 1990s have demonstrated that psychological trauma can have negative effects on brain function that are persistent and might change later neurodevelopmental patterns which results in long-term consequences (Weber & Reynolds, 2004).

Trauma, as a form of chronic stress, has detrimental effects on the body and the brain, particularly on the immune system, cardiovascular system, and bones. The trauma responses of the hippocampus and other brain regions and stress regulating factors that regulate a stress response of a body system are well evidenced in neuroscience (De Kloet et al., 1998; Herbert et al., 2006; McEwen, 2007; Wolf, 2008).

3.1. Etiology of traumatic event

When an organism becomes aware of a threat for the first time, the hypothalamus in the brain stem produces corticotrophin-releasing hormone (CRH). At the base of the skull, the pituitary gland converts this hormone into adrenocorticotropic hormone (ACTH). The blood transports ACTH to the adrenal glands, which are located in the adipose tissue above the kidneys. The adrenal glands secrete cortisol, which has widespread effects throughout the body by increasing a blood pressure and a heart rate, and in same time lowering organ blood flow. The objective is to heighten your awareness of the present moment in order to respond appropriately.

The HPA axis may contribute to the onset of trauma and stress-related disorders. The HPA axis might be responsible for the development of trauma symptoms since it is involved in the fear-producing response. The amygdala is responsible for integrating the physiological response to a traumatic or stressful environment. The amygdala's response is meant to get you ready to either fight or flee. The HPA axis is responsible for the release of the stress hormones adrenaline and cortisol in response to potential danger (Stahl & Wise, 2008). Evidence suggests that epinephrine raises a person's blood pressure, heart rate, acuity, and muscle tension. Once the danger has passed, the body's homeostasis is restored thanks to the hormone cortisol. Image below (Figure 2.) shows the interplay between hormones and the hypothalamus.

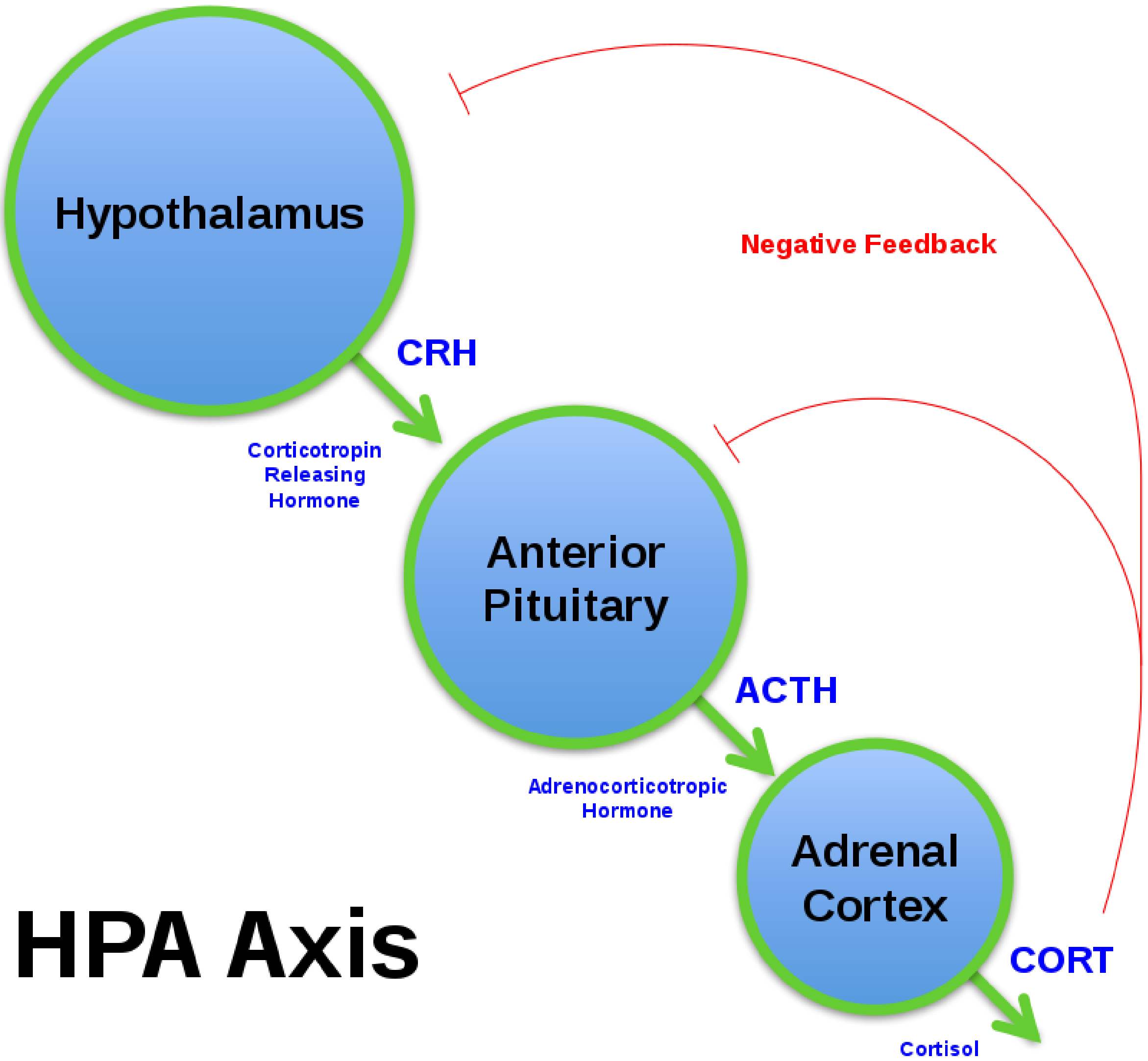

Figure 2. *Schematic of the HPA axis* (User:BrianMSweis - Wikimedia Commons, 2012). Basic hypothalamic–pituitary–adrenal axis summary: CRH - corticotropin-releasing hormone; ACTH - adrenocorticotropic hormone, CORT - cortisol

The coping theories on which the cognitive activation theory of stress is based suggest that excessive activation without adequate coping may lead to pathological stress responses (Ursin & Eriksen, 2010). It is hypothesized that psychobiological sensitization of pain and illness-related cognitive networks play a similar role in the maintenance of chronic pain and recurrent subjective health problems. Anxiety, rumination, and anticipating stress are all symptoms of perseverative cognition, a similar concept that has been associated with disease (Brosschot et al., 2006). Anxiety, ruminating, and anticipatory stress have been associated with increased cardiovascular, endocrinological, immune, and neurovisceral activity (Ursin & Eriksen, 2010).

In PTSD patients, studies of the amygdala and HPA axis have revealed heightened sensitivity to stress and traumatic stimuli (Sherin & Nemeroff, 2011). Individuals with PTSD are less able to overcome their fears, and they exhibit a generalized increase in tension even in non-stressful situations. Schmidt et al. (2013) say that these results could explain why PTSD patients have a higher startle response and are more sensitive to trauma. In PTSD patients, studies of the amygdala and HPA axis have revealed heightened sensitivity to stress and traumatic stimuli (Sherin & Nemeroff, 2011). Individuals with PTSD are less able to overcome their fears, and they exhibit a generalized increase in tension even in non-stressful situations. Schmidt et al. (2013) say that these results could explain why PTSD patients have a higher startle response and are more sensitive to trauma.

The HPA axis has been linked to the development of rheumatoid arthritis and other chronic inflammatory diseases (Sternberg et al., 1989). Trauma or stress affects the hormones involved in the HPA axis, thereby impairing its ability to regulate and cooperate. The HPA axis is responsible for rheumatoid arthritis and other chronic rheumatic disorders (Apostolova et al., 2012), but it also affects the sympathetic nervous system (SNS), which comprises the adrenal medullary system and sympathetic nerve fibers that transport adrenaline and noradrenaline to practically every area of the body (Neisser et al., 1996; Sternberg et al., 1989). The negative feedback processes of the hypothalamus, the anterior pituitary's hormones, and cortisol result in decreased synthesis of CRH and ACTH. The HPA axis is thought to be very important in the stress response, acting as a first line of defense against the effects of trauma on the body. As discussed in the following chapter, HPA dysregulation is at the root of inflammation and chronic immune issues that arise as a result of traumatic events caused by the aforementioned hormonal imbalance, which results in dysregulated hormonal activations at all levels.

3.2. Posttraumatic brain development

The limbic system and frontal lobes remain relatively stable throughout a person's life, but the DNA determines the brain's fundamental neuroanatomical structure. There is a relationship between a mother's style of caregiving and the gene expression involved in both endocrine and behavioral responses to stress (Meaney, 2001). Maternal care influences synaptogenesis in the hippocampus. This effect, according to Meaney, contributes to differences in stress reactivity and cognitive attitude. It has been shown that maternal stress increases the susceptibility of children to stress.

The brains of adolescents internalize and organize new information contextually (Perry et al., 1995): Trauma-induced neurobiological abnormalities of normal development affect the brainstem, locus coeruleus, memory systems (hippocampus, amygdala, frontal cortex), and executive function regions. The HPA axis, which connects the hypothalamus, pituitary gland, and adrenal glands, is especially susceptible to trauma's effects (Stanton et al., 1988; Wiener et al., 1992).

The use-dependent architecture of brain regions involved in emotion and interpersonal understanding is shaped by intensity, frequency, and the patterns of neural information that informs about self in opposition to its environment, where this information is acquired during crucial phases of brain development. The manner in which a person processes information depends on the structure of their brain. There may be issues with executive function, such as long-term planning and neuronal inhibition (Mezzacappa et al., 2001). Disruptions in neurobiological control have far-reaching repercussions.

Teenagers whose brains developed post-trauma have difficulties in the classroom, difficulty paying attention, difficulty comprehending time and space, and sensorimotor developmental impairments (Streeck-Fischer & van der Kolk, 2000). Extreme emotional reactivity among young children is furthermore predictive of depression, low academic achievement, impulsivity, and criminal behavior. Body and environment seem to be in codependency even in terms of higher cognitive functions not only those of nervous system.

Bellow image (Figure 3.) outlines visually the brain structures that get impaired in PTSD mostly: PF, HPA axis, amygdala, hippocampus, locus coeruleus, and periaqueductal gray. Image further schematically shows how all these structures might get triggered and how they are connected in case when sensory organs detect possible danger, dividing structures into fast and slower response in such case. Overall purpose of this image is to depict possible implications that post-traumatic stress disorder might have in long-term abnormal anxiety and fear creation, indicating that impairment of mentioned structures is assumed or proven to be existent in exploration of negative long-term trauma consequences. Details of how exactly trauma affects these structures will be explained in further text in more details.

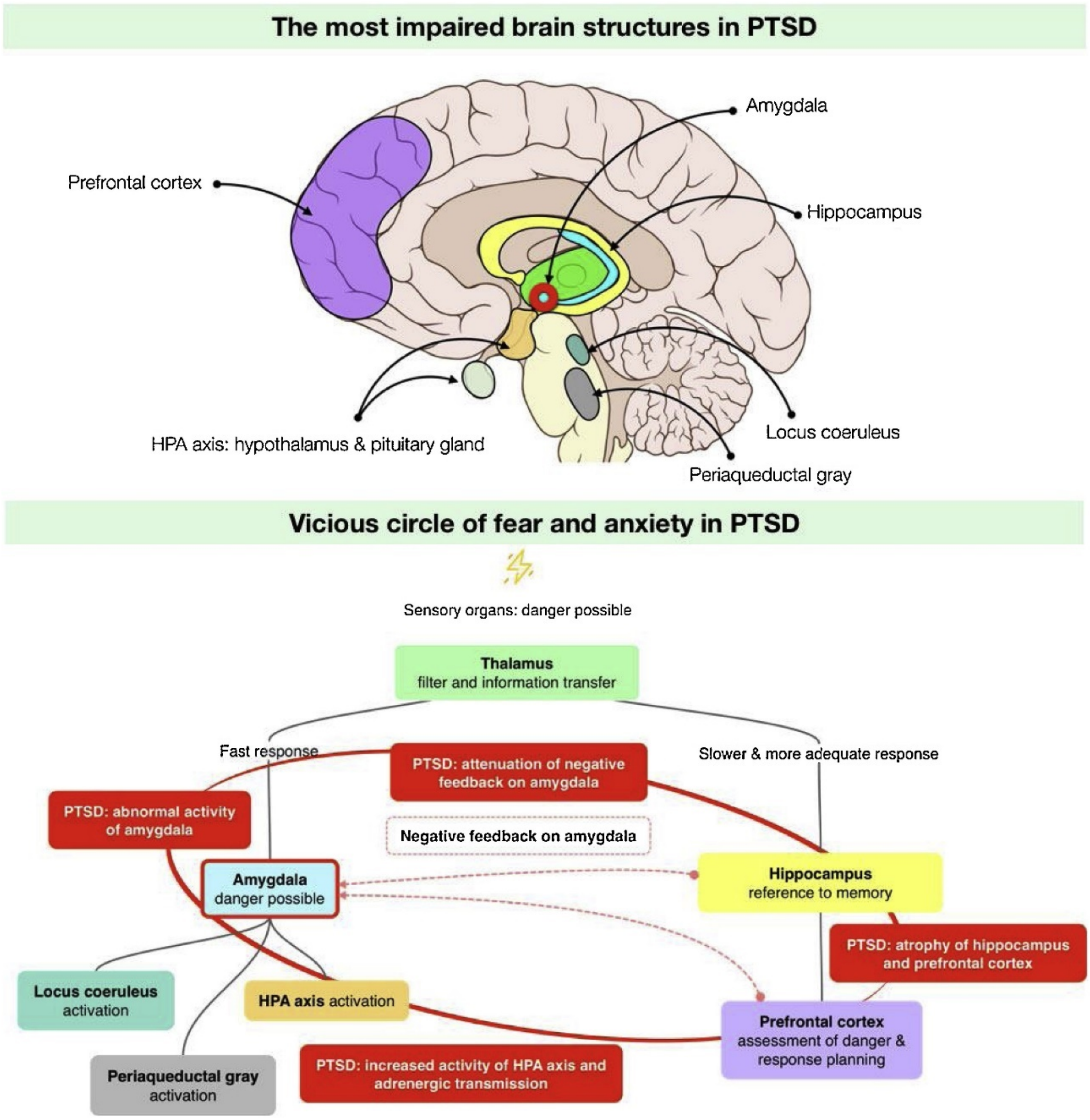

Figure 3. *Short overview of neurobiological response to trauma* (Malikowska-Racia & Salat, 2019). The most impaired structures in PTSD; circle of fear and anxiety in PTSD. Image indicates possible implications of PTSD role in abnormal anxiety and fear creation.

3.3. Parasympathetic nervous system

The parasympathetic nervous system is the other half of the autonomic nervous system. This mechanism is commonly referred to as the "rest and digest system" because it reduces energy expenditure by easing the workload on the heart and digestive system. The parasympathetic nervous system controls stress-induced involuntary responses. When this portion of the autonomic nervous system is activated, our heart rate and respiration decrease naturally, allowing us to relax and recover from stress. After experiencing trauma, the functioning of two distinct regions of the brain is typically altered. The hippocampus is the primary structure for memory and learning. The second component is the amygdala, which regulates our emotions and our capacity to experience fear.

In traumatized children and adults, modulation of the parasympathetic nervous system has been studied less frequently, in part due to the complexity of the research methods required to examine it (Porges, 1992; Sahar et al., 2001). Vagal fibers in the brain stem regulate emotional and behavioral responses to stress (Porges, 1992; Porges, 2001; Porges, 2003). The parasympathetic nervous system's vagal nerves can exit the spinal cord in either the ventral or dorsal position. The mammalian dorsal vagal complex controls the digestive, gustatory, and hypoxic responses; it regulates intestinal tone and facilitates digestion. If stimulated, the dorsal vagal complex might have a role in the development of ulcers and colitis. The ventral vagal complex regulates the larynx, trachea, esophagus, and heart (Porges, 2001; Sahar et al, 2001).

The myelinated motor channels of the ventral vagal that travel to visceromotor organs (heart, bronchi) and somatomotor structures (larynx, throat, esophagus) provide precise control and rapid response. The vagus nerve suppresses the sympathetic nervous system's activation. The rapid regulation of the vagal brake enables rapid environmental participation and withdrawal. The ventral vagal complex responds strongly to parental cues (Porges, 2001; Porges, 2021). An insufficiency of ventral vagal modulation of the early development stress response may be responsible for the association between altered early attachment patterns and problems with emotion regulation and comfort responsiveness in adulthood. The sympathetic system is relevant in trauma response as it gets overactivated as a prolonged response to staying in trauma response. As a result, an individual may become stuck in a sympathetic response over time. However, the overactivation is a direct reaction to blockages in parasympathetic nervous system activation, so for purposes of this thesis, PNS is discussed more, as well as due to its perspective on the vagus nerve, which will be discussed in more detail in further chapters.

Sympathetic-parasympathetic nervous systems balance can be measured by power spectral analysis of heart rate variability to reveal autonomic hyperarousal in PTSD patients (Porges, 1992). Sympathetic nervous system is expectedly more active and parasympathetic nervous system weaker when resting heart rate is low (Cohen et al., 2002). Similar to how some individuals overreact to traumatic memories, those with PTSD also overreact to nonthreatening stimuli.

Adults and children with PTSD have impaired vagal modulation of their heart rate in response to cognitive challenges. Abnormalities in brainstem arousal regulation seem to contribute to PTSD. Children who have experienced trauma may have impaired autonomic responses and low vagal inhibition, which increases the likelihood that they will misinterpret social cues and behave inappropriately in social interactions (Boyce et al., 2001).

3.4. Limbic System

The limbic system is responsible for survival, reproduction, childrearing, and play. It regulates the activity of the hypothalamus and brainstem and acts as a filter for incoming sensory data before it is processed by the brain. The amygdala mediates emotional intensity to evaluate the existential significance of complex information and plan actions for self-protection. The fight, flight, or freeze responses are triggered by signals from the amygdala that increase heart rate and blood pressure. The limbic assessment system bypasses the superior cerebral system and enables immediate responses in times of crisis.

To protect itself from potential threats, the amygdala interprets sensory input and releases a flood of emotional and hormonal messages (LeDoux, 1993). Sympathetic and parasympathetic reactions are triggered by the amygdala, which sends projections to the rostral ventral medulla and the lateral hypothalamus. It's possible that autonomic arousal is regulated by the amygdala's parasympathetic responses. The HPA axis is activated by a central amygdala projection to the bed nucleus of the stria terminalis.

In the vast majority of studies examining fear perception, the amygdala constistently seems to play a critical role in identifying cues of danger. Even in masked fear faces (Whalen et al., 1998), the amygdala is activated in fearful faces, in contrast to neutral, happy, or disgusted faces (Plotsky & Meaney, 1993; de Bellis et al., 1994). During memories of traumatic events, people who have experienced trauma show higher activity in the right amygdala and lower activity in Broca's area (Rauch et al., 1997). When the amygdala gets active as a response to traumatic stimuli, it misinterprets neutral cues as dangerous, leading to harmful fight, flight, or freeze responses. This impairs the system's ability to learn from experience and promotes a reflexive response to small irritations.

3.4.1. Amygdala

The amygdala is responsible for emotional responses and in a brain is visible as a small structure that looks similar to almond. It stores memories and helps us process emotions - it plays a role in how we process and remember information about trauma. The amygdala is also involved in the fight or flight response, which prepares our body for danger by releasing hormones such as adrenalin, cortisol and norepinephrine. When we experience trauma, this part of the brain becomes overactive and can potentially cause depression, anxiety, or PTSD (Joëls & Baram, 2009). The amygdala is one of the first parts to develop and it’s one of the last parts to stop developing as we grow older. Stress increases lateral amygdala activity, anxiety behaviors, and fear learning (Rosenkranz et al., 2010).

Most studies of amygdala morphology in humans have focused on individuals with PTSD (Gerritsen et al., 2015; Lupien et al., 2011; Cacciaglia et al., 2017). Same studies demonstrate that if people have been exposed to traumatic stress their amygdala would become enlarged, and amygdala-dependent learning may increase. Chronic stress, current stress, and trait anxiety show in PTSD patients (Cacciaglia et al., 2017; McLaughlin et al., 2014; Schmidt et al., 2013).

3.4.2. Hippocampus

The hippocampus is a limbic system structure in the medial temporal region of the brain involved in the storage and retrieval of memories (Apostolova et al., 2012). It plays a major role in the formation of long-term memories and spatial navigation and when damaged by traumatic events can cause a lot of cognitive symptoms such as memory loss or even amnesia. Physiological hyperarousal during and after exposure to a stressful event may make it harder to manage memories.

The aberrant processing of memories may result in either overrepresentation, such as persistent thoughts and nightmares, or suppression, the inability to recall memories, or selective amnesia. These cognitive symptoms show that the hippocampus is involved in the pathophysiology of PTSD, particularly in terms of learning from past experience. Corticosterone, the animal equivalent of the human hormone cortisol, which may be neurotoxic if released in high concentrations (Dhikav & Anand, 2012). In case a person develops PTSD, they will have smaller hippocampal volume than those without PTSD. The hippocampus reacts to stressful events by releasing cortisol, which aids in memory formation and retrieval for short-term memories, but also causes a reduction in hippocampal volume as time goes on because it damages cells in this area of the brain. Sperlagh and Sylvester Vizi (2011) explain how cortisol regulates the body's response to stress and trauma and increased levels of cortisol may cause problems with sleep, appetite, and moods. This hormone may also affect how well someone's immune system works and how they respond to medications and its role in trauma response is very important.

In investigations with mice, the number of hippocampal cells destroyed by corticosterone exposure was correlated with the severity of learning losses (Apostolova et al., 2012). In human investigations of individuals with Cushing's illness, which is characterized by excessive cortisol release over lengthy periods, changes in hippocampus volume have been seen and connected with verbal declarative memory problems (Dhikav & Anand, 2012). Similarly, in epileptic patients who had surgical resection of the hippocampus, the reduction in left hippocampal volume was connected with verbal and visual memory performance abnormalities according to findings of Bremner (2006); lesions to the hippocampus eliminate this context-dependent kind of memory. The hippocampus' high density of glucocorticoid receptors suggests it regulates emotions. Glucocorticoids affect hippocampus neurons. Large dosages of hydrocortisone impair human memory, but cortisol may improve memory.

Reductions in hippocampus volume have been linked to PTSD (Bremner et al., 1997). Abused people with PTSD seem to have smaller left hippocampus compared to healthy population, while the right hippocampus and other regions of the brain seem to be normal in size. Stein et al. (1997) discovered anomalies in the left hippocampus in abused women, where if those women were depressed they also tend to have a reduced hippocampal capacity.

The size of the right hippocampus was much larger than that of the left. The severity of symptoms is inversely related to the size of the left hippocampus. Because of the anomalies in hippocampus function, trouble learning from negative experiences is common. The hippocampus of people who had significant childhood trauma shrank over time (Sherin & Nemeroff, 2011). Studies on hippocampal atrophy show that increased cortisol causes cell death in the hippocampus, leading to shrinkage. Patients with reduced hippocampus function have poor context-regulating function as involvement of hippocampi in psychopathology is the most evident in the emotional information processing (Sherin & Nemeroff, 2011). Impaired hippocampal function has been linked to an increase in the activation and failure to terminate stress reactions, a reduction in the ability to extinguish conditioned fear, and a lack of ability to distinguish between safe and harmful environmental conditions.

3.5. Prefrontal cortex

The frontal cortex is the region of the brain that controls our emotions, behavior, and thinking. It is also responsible for making connections between stimulus and response. The prefrontal cortex manages cognitive processes such as attention, memory, and language. The prefrontal cortex is a part of the brain in charge of many cognitive processes including attention, memory and language. A traumatic event can cause a person to experience stress which can lead to an unhealthy reaction from the frontal cortex. This can lead to post-traumatic stress disorder (PTSD).

One thing that changes after a traumatic event are connections between the stimulus and response in the brain due to the fact PFC's dependency on a environment in terms of neurochemistry and how important it is to be in homeostasis to function properly (Arnsten, 2009). For example, when a person sees something bad happen to someone else it triggers a response in us that we would not normally have if we were not exposed to this event. The frontal cortex is the part of the brain that helps regulate responses to stress.

The prefrontal cortex (PFC) is active during many cognitive processes, such as reasoning, planning, and problem solving (Arnsten, 2009). It is also thought to be the part of the brain responsible for personality development. Traumatic events can have serious consequences on the frontal cortex. This can lead to lasting changes in personality, emotional responses, and cognitive function (Arnsten, 2009).

PFC is also essential for establishing response-reinforcement connections and promoting goal-directed behavior (Chafee & Goldman-Rakic, 2000): people with PTSD may struggle to maintain focus and are more likely to become distracted. In addition, they may have difficulty controlling their fear responses and suppressing intrusive memories of the trauma, such as flashbacks or nightmares. These behavioral symptoms of PTSD overlap with PFC function, indicating that the PFC is involved in the pathophysiology of PTSD and associated learning difficulties. Motivational control is associated with sympathetic regulation of postural-driven low-frequency heart rate variability, whereas executive control is associated with vagal regulation of respiratory-driven high-frequency heart rate variability (Mezzacappa et al., 2001). Enhanced vagal modulation correlates with enhanced executive function. This is consistent with the observation that male adolescents who have experienced trauma have impaired vagal modulatory function and executive control (Williamson et al., 2015). In response to fear, children's bodies release noradrenaline before their frontal lobes can evaluate the situation. Hyperarousal has detrimental effects on sensory discrimination, learning, and problem-solving.

According to neuropsychological, neurologic, and brain imaging studies, prefrontal disorders are associated with antisocial and aggressive behavior (Sherin & Nemeroff, 2011). A loss of inhibitory control over aggressive, violent acting out may be the result of prefrontal dysfunction brought on by the demands of late adolescent social and executive function. A number of brain systems, including an increase in subcortical sensitivity and a decrease in frontal lobe performance, are required for an effective response to varying levels of stress.

Lupien et al., (2009) discovered that people with PTSD had dysfunctional working memories. Higher activation was observed in the bilateral inferior parietal lobes, whereas lower activation was observed in the inferior medial frontal lobe, bilateral middle frontal gyri, and right inferior temporal gyrus, indicating an abnormal functional connection pattern. Adults with post-traumatic stress disorder have trouble concentrating, manipulating their thoughts, learning new information, and correcting past errors.

It is by now discovered that children with a history of abuse were more likely to develop PTSD. According to Arnsten (2009) children with PTSD performed worse on tests of attention, abstract reasoning, and executive function, and they were more easily distracted and impulsive than their peers who had not experienced trauma.

3.6. Neuroendocrinology of stress

By definition, stress occurs when a person perceives a real or imagined threat to their equilibrium, necessitating the development of a corresponding coping mechanism (De Kloet et al., 2005, McEwen, 2007). The source of stress is known as a stressor, as mentioned earlier. It could be physical or psychological. A stressor may be either short-term or long-term, such as an upcoming oral exam. Key components in determining an individual's response to a stressor are subjective assessment of the stressor and evaluation of coping resources (Ursin & Eriksen, 2010). An organism releases neuroendocrine mediators in response to a stressor. Hormonal interactions between cognitive and affective systems that foster adaptability (De Bosscher et al., 2003; McEwen, 2007; Sternberg et al., 1989; Wolf, 2008).

Allostasis, or maintaining homeostasis under fluctuating conditions, is adaptive and advantageous in the short term, but can place a strain on peripheral and central systems over time (McEwen, 2007; Wolf, 2008). The SNS is responsible for the immediate response. The hypothalamus initiates a chain reaction in which spinal cord neurons transmit messages to the adrenal medulla. Both adrenaline and noradrenaline are released simultaneously. These chemicals cause the physiological responses we refer to as stress. Although adrenaline and noradrenaline cannot cross the blood-brain barrier directly, they can increase noradrenergic tone by stimulating the vagus nerve, which then acts on the brain stem. These regions stimulate a number of regions of the brain, most notably the amygdala (Roozendaal et al., 2006).

The HPA axis regulates a delayed, secondary reaction in terms of hormonal response. At this location, the hypothalamic paraventricular nucleus secretes into the portal blood circulation corticotrophin-releasing hormone (CRH) and vasopressin. By acting as a neurotransmitter in regions of the central nervous system other than the hypothalamus, CRH modulates the anxiety system (Berridge & Dunn, 1990). The pituitary gland secretes adrenocorticotrophin (ACTH) into the bloodstream in response to circulating CRH. Activated ACTH triggers the release of glucocorticoids (equivalent of cortisol in humans) from the adrenal cortex. Multiple brain regions, including the hippocampus, amygdala, and prefrontal cortex, as well as the pituitary and hypothalamus, are negatively affected by increases in cortisol (De Bosscher et al., 2003; Sorrells & Sapolsky, 2007; Sternberg et al., 1989). As with all steroid hormones, natural glucocorticoids (GCs) may be able to cross the blood-brain barrier, whereas catecholamines cannot. Different GCs have distinct distributions and affinities for distinct brain intracellular receptor types (Sorrells & Sapolsky, 2007). GCs regulate not only cholinergic neurotransmitter systems, but also noradrenergic, serotonergic, and dopaminergic systems (Sorrells & Sapolsky, 2007). In addition, the effects of GCs on the central nervous system are regulated at multiple points. In conclusion, GCs have the potential to alter both the short-term and long-term structure and function of the brain and they play an important role in stress response.

3.6.1. Endocrine factors of trauma

The research is divided as to whether PTSD is associated with abnormal regulation of cortisol and thyroid hormones, two crucial endocrine characteristics of the disorder. Traumatic brain injury is characterized by endocrine imbalance, specifically in the hypothalamus and pituitary gland. The hypothalamic-pituitary-adrenal (HPA) axis, as we saw by now, has important role in trauma response and has received a great deal of attention in studies of PTSD.

The anterior pituitary and the adrenal glands make up the hypothalamic-pituitary-adrenal (HPA) axis. Stress causes neuronal activity in the paraventricular nucleus (PVN) of the hypothalamus, which then releases CRH into the hypothalamo-hypophyseal portal circulation via nerve terminals in the median eminence. This entire process has been detailed at length elsewhere. It is however important to mention glucocorticoid production by the adrenal cortex which in addition to immunological and brain function, affect metabolism as they coordinate physiological and organismal responses to stress. The activity of the HPA axis is simultaneously regulated by multiple neural circuits in the brain. Amygdala and aminergic neurons in the brain stem stimulate CRH neurons in the PVN, whereas the hippocampus and prefrontal cortex inhibit them (Bernhagen et al., 1996;

Sorrells & Sapolsky, 2007). In addition, glucocorticoids regulate hippocampal and PVN neurons to modulate HPA axis activity. Long-term glucocorticoid exposure has deleterious effects on hippocampus neurons, including reduced dendritic branching, loss of dendritic spines, and impaired neurogenesis. Numerous variables, such as the severity and timing of trauma, signs and symptoms, co-occurring disorders, and personality traits, may affect the outcomes of a traumatic event (Black, 2002).

Studies utilizing low doses of dexamethasone indicate that hypocortisolism in PTSD is associated with an increased susceptibility to negative feedback from the HPA axis. Increased glucocorticoid receptor binding and activity in PTSD patients suggests hypersensitive inhibition of negative feedback (Bierhaus et al., 2003). Cerebrospinal fluid (CSF) levels of the hormone CRH were higher in patients with PTSD. ACTH responses to stimulation of pituitary CRH receptors are diminished. Patients with PTSD have a suppressed HPA axis because their hippocampus is smaller as PTSD is associated with a dysfunctional HPA axis, according to neuroendocrine research.

Prospective studies link PTSD to decreased cortisol levels following exposure to psychological trauma (Calandra et al., 1995; Sorrells & Sapolsky, 2007). Hypocortisolism makes stress responses such as PTSD irrational. Following psychological stress, hydrocortisone reduces the risk of developing PTSD. Hydrocortisone mimics the normal cortisol cycle in order to treat post-traumatic stress disorder. Uncontrolled stress reactivity and fear processing can be traced back to dysfunction in the HPA axis. Glucocorticoids, which inhibit the formation of traumatic memories in the brain, may alleviate PTSD symptoms.

Promotion of health and disease prevention includes the hypothalamic-pituitary-thyroid (HPT) axis, whose primarily function is to maintain normal levels of thyroid hormones that are responsible for immunity (Crossin et al., 1997; de Bosscher et al., 2003). It regulates metabolic and anabolic states by influencing thyroid hormone levels. Due to the fact that trauma can cause thyroid abnormalities, the HPT axis may be involved in stress disorders. The HPT axis and PTSD have received inadequate research. The T3 and thyroxine levels of Vietnam veterans with PTSD were found to be elevated (T4). Increased peripheral deiodination is observed when T3 is significantly greater than T4. Mentioned research suggests importance of thyroid gland and its hormones in stress response mechanism.

According to McFarlane et al. (2011), cortisol, a stress hormone, is secreted by the body when it is under stress. Being called a stress hormone functions as an alarm clock in nature. Stabilized glucose levels, reduced inflammation, and enhanced memory are all desirable outcomes that are controlled by cortisol. In response to stress, the stress hormone cortisol increases heart rate, blood pressure, blood glucose, respiration rate, and muscle contraction while shutting down the non-essential digestive and reproductive systems. It is crucial to comprehend the role of cortisol in trauma, as it serves as a developmental marker. Cortisol aids fetal brain and lung development. These three systems are impacted by this. During pregnancy, there are both positive and negative influences on the fetus. The health of both the mother and the fetus can be negatively affected by stress during pregnancy. The fetus can detect the mother's anxiety.

The cognitive and motor development of a child are adversely affected by prenatal stress. Ingesting stress hormones such as cortisol during pregnancy can be harmful to the developing fetus. A person's odds of acquiring PTSD, effective conflict management skills, and satisfying interpersonal connections all rise after experiencing trauma. Abused men, for example, have abnormally low cortisol concentrations (Carpenter et al., 2007). Carpenter and colleagues also found that women who reported childhood physical abuse had lower cortisol levels. Cortisol is less sensitive to childhood trauma (McFarlane et al., 2011).

In severe depression, stress response sensitivity and HPA axis dynamics are altered (Heim & Nemeroff, 1999) and this can go vice versa. Suicide was also discovered to be correlated with cortisol levels by O'Connor et al. (2016; Keilp et al., 2016). Lindqvist et al. linked low cortisol to suicidal behavior in suicide attempters. Researchers discovered that those who attempted suicide had lower cortisol levels than controls or those who had suicidal thoughts. It has been demonstrated that HPA activity can predict depressive relapse and suicide attempts (Keilp et al., 2016; Varela et al., 2017), but little is known about dysregulation in suicidal individuals. In severe depression, serotonin levels become unbalanced and decline significantly, which can affect cortisol levels (Keilp et al., 2016; McFarlane et al., 2011).

3.7. Creation of traumatic memories

Traumatic memories are in general considered that: (a) they are primarily imprinted in sensory and emotional modes and that a semantic representation of the memory may coexist with sensory flashbacks; (b) these sensory experiences often remain stable over time and unaltered by other life experiences; (c) they may return, triggered by reminders, at any time during a person's life with a vividness as if the subject were having the experience all over again; and (d) these sensory imprints tend to occur in a mental state in which victims may be unable to precisely articulate what they are feeling and thinking. (Van der Kolk & Fisler, 1995; van der Kolk, 2000)

According to Van der Kolk (2015), PTSD sufferers may experience flashbacks and other sensory reexperiences without any updates or connections to other experiences. If something from the past is triggered, it can be experienced again with the same level of intensity, both emotionally and sensorily, as if it were happening for the first time. Individuals who suffer from PTSD frequently become fixated on reliving or thinking about their traumatic experiences. Trauma's sensorial components persist as intrusive flashbacks and nightmares, altered states of consciousness in which the trauma is relived, unintegrated with a comprehensive sense of self in the present moment. The disjointed quality of traumatic memories is consistent with the hypothesis that the central nervous system (CNS) is unable to integrate the sensations associated with the trauma during states of high emotional arousal.

Sensory and perceptual perceptions of a traumatic incident might recur automatically after a person has experienced psychological trauma (Iyadurai et al., 2019). Intrusive memories, also called flashbacks, can manifest as images or take the form of other sensory cues like sounds, smells, tastes, and bodily sensations. Intrusive memories are found to be associated with peri-traumatic detachment, data-driven processing, and a lack of self-referential processing, all of which are highlighted in cognitive models of PTSD.

3.7.1. Hippocampus as a memory storage

While dealing with trauma, a person's self-esteem and sense of direction are damaged, as traumatic memories may resurface as a result of emotional memory alteration (Liotti, 2004). Short-term memory loss is also common following trauma, and long-term memory impairments are common. Emotional or personal memories could be so severely affected that they could even be erased by comprehending traumatic experiences (van Der Kolk & Fisler, 1995) . Trauma's impact can be heightened by the formation of new procedural memories. On the other side, it is possible for a single incident to permanently alter short-term memory.

Reconstructing the past to satisfy current needs, worries, or interests is in a way reconstruction of memory as memory is based on one's own experiences, such as autobiographical or episodic experiences that integrate as these same types of memories (Moore & Zoellner, 2007). According to a psychobiology, the body maintains track of PTSD and it does so following memories logic. Traumatic memories are always involved in diagnosis and are closely related to dissociation (Liotti, 2004). This reminiscence of trauma may cause physical and mental pain. It is common for people with PTSD to exhibit psychophysiological activity that mirrors their subjective experience. Even if specifics fade, trauma survivors are more likely to recall what happened. Memory loss and retention can be caused by traumatic experiences (Byrne et al., 2001). A person who has been traumatized will experience both symptoms. When it comes to traumatic events, many details seem to be imprinted in the brain, never to be forgotten even though time and subsequent experience have passed. Traumatic memories, according to trauma studies, are distinct from ordinary memories (Byrne et al., 2001). Traumatic memories may be encoded differently than regular memories, possibly as a result of changes in attentional focusing or acute emotional arousals interacting with hippocampus memory systems.

For purposes of this thesis, it is important to understand how memory is constructed from information collected from environment and is actually a data storage, that, among other things, helps construct cognitive maps and knowledge to be used in future. That is why memory might inflict agent’s interaction with environment in future, in case some environmental trigger starts a memory without agent’s control.

3.7.2. Insufficiency of traumatic memory

Trauma, especially amnesia, might be hard to research, as children who experience trauma are unable to construct a logical narrative of a traumatic event (van Der Kolk & Fisler, 1995). We perceive the world more intuitively and even lack certain stages of environmental processing, and it is possible for traumatized children to lack cultural and personal remembrance because of this. Dissociation and memory gaps may make it difficult for individuals to remember the past and the present, and such disturbances are more common when it comes to childhood trauma. These people might be vulnerable to suggestion and the manufacture of trauma-related explanations because they lack autobiographical memory, they tend to experience chronic dissociation, and they form meaning schemes that incorporate victimhood, helplessness, and betrayal.

Trauma-suffering persons experience uncontrollable flashbacks of traumatic experiences (van der Kolk, 2000) When the nervous system is overworked, it causes a flashback. A person's brain is able to tell the difference but doesn't realize it. Because of this, the nervous system experiences trauma all over again. Emotional and physical cues are essential to the formation of associative memories (van der Kolk, 2003). Our mental frames are influenced by our emotional states. Many rape, domestic violence, and child abuse survivors are able to live normally if they suppress the awful experiences they've experienced in the past. New emotional or sensory stimuli can cause trauma in some people. People who have been traumatized may experience feelings of dread, need, intimacy, and sexual arousal.

Traumatic situations can cause forgetfulness in many people. Sexually abused children had amnesia in 19–38 % of cases before their memories returned (van der Kolk, 2003). Emotional and cognitive forgetting are more likely to occur in those who have experienced long-term trauma. After a painful emotional event, amnesia moves backward in terms of time, and the traumatized person can become forgetful of their own feelings. When overwhelmed, people might be incapable of forming narrative memory because of being stressed out and memory might simply get overrid by intensity of emotion and information might not be able to integrate.

Feeling of fear to remember could reject people from recalling terrible memories in a moment of now. There is an intuitive, subconscious awareness that trauma memories are not possible to eliminate without the inclusion of a personal narrative, and this in itself is stressful and worrisome. Because of this direct personal narrative inclusion, working on trauma and trauma regressions tend to be hard. The ego gets stimulated and energized by small levels of fear and fury, which in turn improves performance of recall. Emotional arousal builds up until the ego is crippled by constant stress. The assumption is that these are all possible reasons why sense of self and identity get so intact when there is trauma involved in person’s life, as memory (autobiographical) is considered to be a basis of personality, stored in hippocampus. (van der Kolk, 2003)

PTSD's biobehavioral abnormalities may be caused by a failure to integrate traumatic memories, as integration faces many difficulties due to pain that accompany those memories. It is possible for a person who has been traumatized to identify the emotional impact of a stimuli, but not why they feel or act in a certain way. These memories are stored in the form of sensations, thoughts, or behavioral reenactments (van der Kolk, 2000). Although traumatized individuals may not be able to recall the event itself, they may be able to access their implicit memories, which are indirect and often subtle.

3.7.3. Trauma memory integrating as reality

External reality is accepted unquestionably by the unconscious ego. This means the agent perceives the environment and receives all the stimuli from it intuitively and automatically. It integrates this information into its nervous system, processes it, and stores it in memory and body memory without having any doubts about the stability of this reality or the world. The mind is not questioning the stimuli itself, especially if it is really rapid, extreme or intense and doesn't give much space to think and estimate the situation.

Traumatic embodiment includes nociception, which will be discussed in further chapters. There are still certain rules and certain sets of order when it comes to estimating danger and pain and the most suitable response to the possibility of stress or trauma. It is assumed that the event happens unexpectedly. If it does not resonate with the agent's already existing and knowledgeable cognitive map and is intense, it can result in a lack of readiness to process the information. The information therefore gets processed unconsciously and intuitively - automatically by default of agent-environment interaction. If there is not enough time for agents to perceive a threat and find ways to act upon it, not only will the fight or flight response be more intense, but there might not be any time at all to perceive a situation as traumatic until the information has already tried to integrate into the nervous system or the body. This is the moment when the struggle of integrating a stressful or traumatic event starts. So, in this case, it can skip the step of perceiving traumatic events and then dealing with them while trying to process them.

Therefore, somatic memory integration and overall memory integration regarding the event are severely distracted, but they can also be completely dismissed because the mind didn't have enough time to perceive and process stimuli as toxic after it passed the threshold and started integrating. It might be too much of an overload for the body and mind themselves to deal with it and the mind might just let the body integrate it into its nervous system without questioning. We can associate this with the mechanism of how repressed memories are created. We can assume that memories will get repressed if they get integrated with skipping conscious in-moment confrontation and processing of traumatic event and might cause severe damage in future. Because of the way they're integrated, if something skips conscious and awake processing of information, it could be hypothetically triggered on subtle cell levels and be causing inflammation or behaviors driven by the deep subconscious that may cause issues in social interactions, where the trigger would be not only situations that resemble traumatic events but also any other stressful situation or concept that causes stress, which on cellular memory levels might trigger the same response as in trauma experienced. This could also be a backend regression. All this remains creative theory and assumption based on all the facts already mentioned in this thesis, as no current research yet goes in this direction. However, it is an interesting direction to take.

The second approach begins with the agent recognizing something as a potential threat. Then a mind tries to deal with possibility of threat and level of danger and tries to process it while it is happening or after it happened. Attack coming from the environment are perceived by sensory preceptors and based on already existing cognitive maps about the world; if the event or situation is not already stored in a cognitive map or is stored but labeled as a danger, the agent will either not have any knowledge of it and not be able to defend, or it will react based on knowledge of prior integrated responses to danger. In this case, integration flows in direction of predictive processing, that will be mentioned in future chapters in more detail. Predictive processing might get disturbed, and already instructed information might get stored in the nervous system and later in the body and overall memory.

A traumatic event gets stored in memory as a traumatic event itself and less as abstract sensations, and one's struggle becomes comprehending and integrating the experience itself. This happens not only with something unexpected but even with something extreme and possibly violent, endangering, or life-threatening. Trauma in this sense becomes a consequence of failed integration and processing, a direct reaction to mind and body trying to cope with this danger and failing in doing so. Traumatic memory is stored as failure to cope with traumatic stimuli and this memory, of course, can later cause many damage, especially for one's health, both mental and physical. In a previous case trauma could be described as distorted and abstract integration of a shocking experience, where this is also seen as a direct response or consequence of a happening. All the future reactions that come from triggers are coming from triggers from the environment reminding an agent of the event itself or difficulty or danger level of the event.

The secondly mentioned scenario above is more likely to be connected to PTSD. And it is a better explanation of how memories are stored and how they work in the case of PTSD. However, we should not discard the first situation at any moment, as the unconscious and subconscious integration of trauma are very possible as well. There is a big lack of studies in science when it comes to this particular phenomenon, and we cannot assume how connected to PTSD this scenario or any of these assumptions are or how significantly.

There are PTSD issues connected to both mentioned types of integration. Both scenarios, though, include memory and memory integration in the nervous system and body overall, and how the mind tries to cope with those memories. Possible memory recall can imply PTSD sufferers' brain activity in the Broca's region as decreased (Rauch et al. 1997). Emotions and imagery in the right hemisphere likewise elicit in this situation. The strength with which memories are associated during arousal is used by organisms to interpret sensory input even if time has passed; emotional memory retrieval is accelerated by future bouts of elevated arousal. In times of stress, those who have experienced trauma are more sensitive to memory traces associated with the incident, and they may recall the trauma even though it has no relevance to their current situation (Pitman & Delahanty, 2005).

3.7.4. Framing a trauma

According to numerous studies, trauma is carved into the long-term memory of the brain. (van der Kolk, 1989). Traumatic memories are commonly remembered as fragments of the event's sensory components, such as visual images, olfactory sensations, auditory sensations, kinesthetic sensations, or overwhelming waves of feelings, according to psychologists who work with traumatized individuals (Southwick, 1993). It is an interesting notion that there are no linguistic components to traumatic memories.

Regardless, storytelling is always used to talk about a tragedy. Everyday storytelling seems to make good use of all kinds of sensory cues. Post-trauma linguistic framing and explanation of the event are necessary so that a person could integrate trauma in its personal narrative. Writing in a form of regression diary or simply writing about events is sometimes used in therapy, but it is also a well-structured part of court witness stands or reporting violent crimes. The difference is in therapy writing being guided and observed by a professional as well as being assigned in a very controlled setting with a set of rules, while in the second case it is forced and dysregulated. These are just two examples, but they are here to show how powerful giving a verbal explanation, context, and meaning can be. By talking about trauma and dissecting details linguistically, a mind has an opportunity to go deep into memory and extract and interpret all information stored through sensory input and sensations by logical framing within the verbal meaning of those sensations, which then gives new meaning to memories and gives a person the opportunity to even reframe the meaning of a whole experience and then reintegrate this into their nervous system or body. When such a process is guided it commonly results in healing but when it is forced it can cause disruptions in functioning as a person might extract and interpret memories they are not ready to give a meaning to. This is an important notion for anyone going through the process, as currently there is a lack of awareness of the need for professional attention during such a process.

A third example could be everyday interactions, friendships, and opening up to people in our surroundings. There should be a high level of awareness about the readiness to frame trauma verbally so that severe, uncontrollable body and mind reactions can be avoided. On the opposite side, non-traumatic experiences are weaved into personal narratives without any efforts and therefore we can talk about ourselves in these healthy concepts effortlessly. There is, however, no evidence that telling a story about a traumatic event eliminates the accompanying memories, as people stay haunted by memories of the event's sensory components and emotional states.

3.8. Trauma's chronic harmful consequences

3.8.1. Health consequences

Negative childhood experiences, such as physical and emotional abuse or neglect, a parent with a mental illness, or an environment that does not feel safe due to violence or drug use, can also cause individuals to carry a chronic level of tension in their bodies (Harris, 2015). A child who has been abused might experience pain, numbness, or dissociation as a result of the trauma. The body will encase and isolate the trauma's effects in order to survive and continue to function normally. Occasionally, a child compartmentalizes their life without even realizing it. They may encounter a general dulling of emotions and senses. In people with PTSD there may be difficulty experiencing normal emotional responses to everyday events, leading to inflicting harm on themselves or others. It is possible to simultaneously experience chronic and acute trauma. It is discovered that those who had never experienced trauma are better able to recover from the trauma of war, whereas those who had experienced trauma as children were more likely to suffer from PTSD symptoms such as nightmares, flashbacks, and dissociation fifty years later. When the human body is in a state of equilibrium and optimal health, all its parts are in constant communication with one another. In response to any trauma or injury, the immune system rushes resources to the affected area to initiate the healing process. The area has been sectioned off in a very specific manner to permit healing while containing damage. Immediate reintegration follows the completion of the recovery process. If the stressor is extremely intense and persists, the body will fortify itself to protect itself from further harm.

There is a substantial body of evidence demonstrating the negative health effects of allowing trauma to persist unresolved for decades (Maté, 2011). Numerous persistent health issues may stem from past or current traumatic experiences - multiple sclerosis, lupus, scleroderma, and other incapacitating autoimmune diseases. When our bodies retain physical, emotional, or mental stress, the normal give and take of our cells is disrupted. One effect of unresolved trauma is prevention through a fair assessment of the person's current circumstances. Being held back by the memories of the past and concerns about the future will make living in the present impossible. When the navigational system that delivers information about the present moment is damaged, blocked, or captivated by trauma memories, people disregard warnings of imminent danger or the possibility of making a certain decision.

This implies that returning to the present moment in an environment of trust is the initial step toward reclaiming one's cells and overall body. The research of Dr. Stephen Porges demonstrates that the vagus nerve is the primary transmitter of parasympathetic nervous system signals associated with feelings of safety and social connection (Porges, 2003). This process, which is normally quite seamless and imperceptible when functioning at peak efficiency, is severely impaired in trauma survivors and other high-risk groups. His findings validate my classroom and clinic observations. As a result, I have spent the better part of the last three decades investigating new techniques for facilitating present-moment, whole-body feelings in others.

PTSD, anxiety disorders, schizophrenia, personality disorders, and mood disorders are only some of the mental illnesses that can occur from trauma (Misiak et al., 2017). Brain structure is altered, immune system function is altered, and mental capacity is diminished all as a result of trauma (Assogna et al., 2020). Diabetes, obesity, anxiety, depression, and substance abuse are just some of the health problems that have been linked to long-term stress (Bielawski et al., 2022).

Trauma has a deleterious effect on the cognitive capacity of both psychotic patients and healthy individuals who have encountered trauma in the past (Schenkel et al., 2005; Shannon et al., 2009). Corticosteroids have a deleterious influence on multiple brain regions involved in sensory identification, memory, and learning (hippocampus, amygdala, medial prefrontal cortex, and hypothalamus) during both acute and chronic stress (Bielawski et al., 2022). Overall, trauma has severe consequences for a person's physical and mental health. They are not limited to stress and psychiatric disorders and extend to physical distortions, which hold their roots in inflammation.

3.8.2. Inflammation

Inflammation becomes severe when it is systemic and when it becomes chronic. It can affect all organs and tissues and gets triggered by overactivation of stress hormones and dysregulations in endocrine system which is connected to neurotransmitter connections. Inflammation of the brain is a common complication of systemic inflammation, particularly when it is severe or persistent and may alter mood and behavior (Sankowski et al., 2015). In contrast, the brain has an impact on the immune system. It does so by sending an electrical signal along the vagus nerve to the spleen, the organ responsible for regulating the immune system. Chronic inflammation can be caused by a number of factors, such as autoimmune diseases, aging, obesity, and stress, and it wreaks havoc on our bodies and minds, despite the fact that inflammation is a vital component of the immune system (Goverse et al., 2016; Lu & Wu, 2021; Tracey, 2007). Every emotion, thought, and deliberation are affected.

Due to chronic muscular tension caused by rushing, the inability to be fully present and calm is rooted in the body (Sankowski et al., 2015). Overactivity can result in constriction of the digestive tract, which can lead to sleep disturbances and other gastrointestinal problems. In case of brain inflammation, neurotransmitter transmission is inhibited. For instance, nerve cells utilize the amino acid tryptophan to produce serotonin. Inflammatory cytokines induce neurons to use tryptophan to produce neurotoxic proteins, such as kynurenine, which aid in the inflammation process but are neurotoxic in the long run. Kynurenine may reduce serotonin levels in the brain by reducing serotonin's building blocks.

If inflammation in other parts of the body gets prolonged, it can spread easily to the brain (Pavlov & Tracey, 2012). In contrast, the brain has an impact on the immune system. It does so by sending an electrical signal along the vagus nerve to the spleen, the organ responsible for regulating the immune system. Chronic inflammation can be caused by a number of factors, such as autoimmune diseases, aging, obesity, and stress. Inflammatory diseases of the immune system, such as rheumatoid arthritis, lupus, and Hashimoto's, can also contribute to chronic inflammation. In recent years, it has been discovered that a large number of diseases that were once thought to have unknown causes are actually autoimmune diseases, caused by the body's immune system's abnormal response to some of its own structural components.

Diabetes is the result of the immune system's attack on the pancreatic cells responsible for insulin production. Genetics, certain environmental factors, and lifestyle choices can cause chronic inflammation even in individuals without traumatic experiences (Sankowski et al., 2015). The levels of both inflammatory cytokines and CRP tend to increase with age. A risk factor for inflammation is being overweight. Sixty percent of adipose tissue cells are macrophages, and the levels of cytokines and C-reactive protein are elevated in obese individuals (Tateya et al., 2013). This may also explain why overweight individuals are more susceptible to depression. Extreme stressors such as poverty, debt, and social isolation have been linked to elevated cytokine and CRP levels (Kalupahana & Moustaid-Moussa, 2011; Tateya et al., 2013).

Social stresses, such as delivering a speech in front of an audience, may temporarily stimulate the immune system. Body has a stress response, a biological mechanism that activates in times of extreme danger and coordinates the body's vital systems to keep us safe. As a result of the stress response, energy is released, physical strength is enhanced, mental clarity is improved, and pain tolerance is increased. As such, it is one of the primary factors that have allowed humans to persist for such a long time. It is however not natural or normal to endure prolonged levels of stress as hormones released during stress response can damage the body if they continue being active in a body and in wrong places in organs in a wrong time, meaning when threat is over. It is expected on subtle body levels that a body reacts to a threat once a situation is perceived as such, and the evolutionary role of the stress response is exactly in that comprehension of stressful situations.

4. Embodiment in a context of trauma

4.1. Environment-body interaction

The nervous system evaluates risk by sifting through information acquired from the environment and internal organs (LeDoux, 2012). Subcortical limbic circuits seem to be involved in the neurological evaluation of risk without a conscious awareness, the same neural areas that showed to be important, if not even crucial, in overall bodily trauma response, as well as CNS. Risk assessment seems to be natural first step in body and mind preparing to deal with a potential threat and firstly a situation has to be perceived in a form of sensory information by bodily senses (Porges, 2007). Agent then has to differentiate between benign, damaging, and potentially lethal components of its own surroundings and decide whether to react out of automized nervous system response (Siegel, 1999). Such processes and a person's (agent's) interaction with the environment in the sense of perceiving and avoiding or dealing with danger can be easily understood through the concept of neuroception (Porges, 2004; Siegel, 2012). Neuroception is a cognitive process that aids humans and other mammals in avoiding dangerous social situations. It is hypothesized to mediate the manifestation and disruption of prosocial behavior, emotional regulation, and somatic equilibrium and is suggested they may be provoked by sensors that involve temporal cortex regions which in this process connect to the central nucleus of the amygdala and the periaqueductal gray (Porges, 2007).

The vast majority of people have a neurological system that accurately assesses danger and adjusts their neurophysiological state to match the level of actual danger exposure (Porges, 2007). When people feel safe in an environment, their protective limbic systems shut down, allowing for more social interaction and positive emotions. In individuals with disturbances of homeostasis, such as PTSD anxiety, or another psychological issue, the nervous system incorrectly interprets the environment as dangerous (Schore, 2003). As a result, people experience physiological states that encourage defensive behaviors (fighting, fleeing, and freezing), and positive interaction with others and the environment is discouraged (Gramsch et al., 2014). According to the hypothesis, in order for the social engagement system to facilitate efficient social communication, defensive circuits must be suppressed. A disturbance can be much worse with people who endured severe traumatization in past, especially childhood, as by physiological states of fight-flight-freeze they might repeat a state of traumatization both mentally and physically. In other words, the body of a traumatized individual might experience flashbacks as the defense state makes the body go to repeated state of their traumatization. This mechanism also works in reverse, where the body locks into a defensive state and causes constant issues for the individual. Neuroception is not influenced only by environmental hazards; a person's evaluation of anything is influenced by emotions, and for social engagement to be reciprocal, the engaged individual must reside in a state of accessibility (Irwin, 2008; Porges, 2004). Insula seems to play a role in regulating neuroception, as it has a significant role in the perception of pain and other emotions. Insula reflects internal body states and contributes to subjective feeling states. A mobilized individual may respond to the same engaging behavior with antisocial traits such as withdrawal or aggression. To keep control over the defensive response and adaptively manage the autonomic state and avoid falling into the SNS response, the body engages the vagal nerve, which the novel polyvagal theory (Porges, 2007) shows as an important mediator between the environment and the body.

The vagus nerve, according to Porges, has both dorsal and ventral branches. SNS is the one that provoked the fight-or-flight response. Mobilization is fear-induced paralysis when a person enters hibernation when under attack to conserve energy. When the vagus nerve is stimulated, it relaxes the muscles and reduces blood pressure. With fearless immobilization, a person may feel safer and more comfortable in close quarters. Healthy rivalry is caused by the release of endorphins and the activation of the ventral vagal system. Many individuals get stuck in the mobilization or immobilization phases, characteristic of the trauma response. Fear paralysis has effects on both the mind and the body due to the fact that the dorsal vagus nerve causes a heightened response to perceived danger (Gramsch et al., 2014). Due to anxiety, the dorsal vagus nerve becomes immobilized, resulting in a person being exhausted, irritable, and feeble. A healthy autonomic nervous system facilitates growth and recuperation (Porges, 2007). The sympathetic, parasympathetic, and dorsal vagal pathways are in autonomic balance when a person is in a ventral vagal state. When the ventral vagus is dysfunctional, the autonomic nervous system serves to protect as opposed to heal.

During the fight or flight response, sympathetic activity increases mobilization and decreases digestion. While perceiving and experiencing danger, response of SNS triggers and if mind and body don't manage to cope with the event properly and process it - integrate into cognitive map, store in experience into bodily functions and system without disturbances on biochemical levels, the body might get stuck in overactivated symphaticus. By decreasing sympathetic activity, dorsal vagal activation can be enhanced, which can improve the feeling of safety. In other words, it is needed to enhance autonomic response in order to regain homeostasis, as the most of visceral organs are getting information from both the parasympathetic and sympathetic branches of the autonomic nervous system (Irwin, 2008; Porges, 2004).

The importance of the vagus lies in its function; it controls an essential function in the body. It links the brainstem to the rest of the body and connects the brain to the organs. Sensory input from the vagus nerve regulates brain structures, the vagus nerve regulates the activity of brain regions, and the vagus branch plays a phylogenetic role. Porges (2003) explains the detailed functions of the vagus and how it is connected with the rest of the body. The vagus consists of a brainstem regulator, visceral afferent input, and target organs that receive efferent output. The vagal regulator in the brainstem is both a gateway to and an input to other neural systems, with nuclei distributed to numerous peripheral tissues, such as the nucleus ambiguous, which controls the facial, cardiac, and pulmonary muscles. Dorsal motor nucleus vagal fibers regulate gastrointestinal function, while the vagus nerve transmits sensory information from the periphery to the medullary nucleus tractus solitarius, which is connected to both the forebrain and brainstem (Porges, 2001). Less obvious pathways regulate autonomic function and facial striate muscles. Vagal afferent stimulation modifies the activity of higher brain structures and influences the function of organs such as the face, heart, intestines, and pancreas. Social interaction is influenced by somatosensory and visceromotor processes (McCabe et al., 2014). While the myelinated vagus controls the heart and bronchi, the visceral efferent circuits command the striated facial and head muscles. Polyvagal Theory provides a unique approach which explains how trauma changes the nervous system and defends evolutionary approaches to it (Cozolino, 2017; Porges, 2001). In this context, trauma can be explained as a reaction of the nervous system to a stressful and unexpected event and the coding of this in the nervous system and body in general. In other words, trauma is an impairment in communication between the body in terms of the nervous system and the environment in terms of social interactions. The image below shows the Social Engagement System constructed from a somatomotor and a visceromotor component, illustrating the logic of communication between cortex and a brainstem, and the brainstem's relationship with cranial nerves and which parts of the body they communicate with - the cortex communicates with the environment through the cranial system.

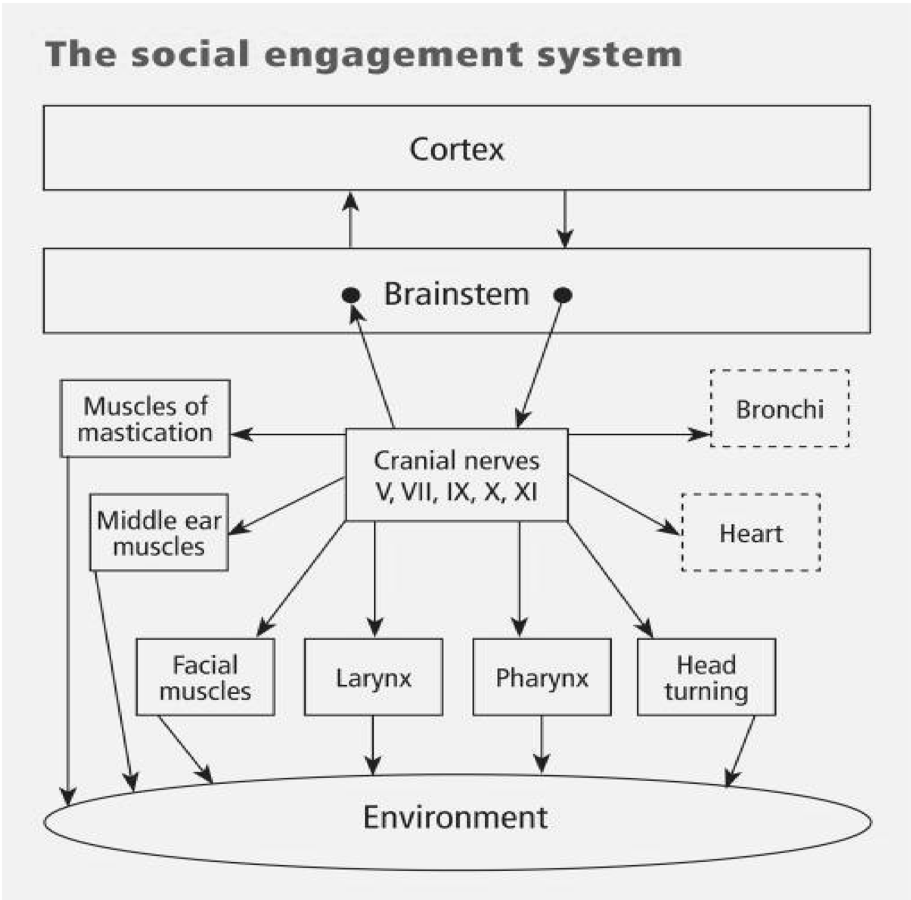

Figure 4. *The Social Engagement System* (Porges, 2003). The cortices control the medullary nuclei through corticobulbar pathways, hence determining the nature of social communication. There are two parts to what we call the "Social Engagement System": the somatomotor (i.e., visceral efferent pathways that control facial and head muscles) and the visceromotor (the rest of the body's muscles) (i.e., the myelinated vagus that regulates the heart and bronchi).

Highly embodied agents display novel behaviors and coping mechanisms in response to their environment and such a notion is a good explanation on the role of embodiment in behaviors - the more embodied the agent is, the better they interact with the environment (Brooks, 1991). It is therefore important how much "in the body" agent feels and how much agency over their body they have. It is also possible to assume that embodiment of an agent will suffer if there is a severe distortion in the environment, such as trauma, and that less the agent is embodied, the harder they will deal with stress from the environment, and it will cause more issues for a body as the aftermath. This remark is small but notable - embodiment seems to be of a big importance in trauma context and needs to be investigated more.

Cognition has evolved from previous successes and is essential to begin educating children at a young age. Survival and reproduction require the capacity to detect and adapt to one's environment, and according to EC theorists, perceptual and motor control are the foundations of all higher cognitive activities (Clark, 2013). According to Rodney Brooks (1991) once being and reacting are understood, problem-solving behavior, language, expert knowledge and application, and reasoning emerge. Trauma could be seen as a disturbance in this evolution that blocks or disturbs growth in cognitive processes, as it is an occurrence out of the ordinary in the environment's context. The nature of the mind is dynamic and not easily computationalized from the standpoint of radical embodied cognitive science (Clark, 2013). Nonlinear dynamic systems are the ones that model agents and their operating environments, which would mean that both agent and environment are fluid interactive constructs.

Cognitive processes are not restricted to the body and brain (Clark, 2013) and as perceived in embodied cognition theory, the brain ejects cognitive load to the body and environment - bodies are tools that are operated by our conscious brains. Representations are what agent's cognitive system is mostly composed of and these representations rarely are a confident presentation of objective reality (Tolman et al., 1992). This can be seen in agent's behaviors, as the agent reacts in environment based on representation of environment it stored as knowledge based on information from it (environment). These behaviors are referred to as a cognitive map, "a rich internal model of the world that accounts for the relationships between events and predicts the consequences of actions" (Tolman et al., 1992). Every environment an agent has experienced has its cognitive-mapping system which is dependent mainly on placing locality and space in its system.

There should be social and physical contact between an agent and its surroundings for embodiment to occur. It is clear that traumatic event would be considered a complex situation that would demand a complex response or even one-dimensional response which is complex enough in its backend so that it can handle complexity of a situation with a simple solution. In this context behavior would be fight or flight responses, which also include freeze state. Trauma can be observed from behavior alone as an extreme response in a stressful situation that is registered by the body as a danger alarm. It is also connected to how the response to the event is stored in nervous system, as memory of environment experience is stored in hippocampus (Tolman et al., 1992). EC has long held that behavioral complexity is a reflection of the environment and not the other way around, which is evidence that environment directly influences agent in terms of cognitive maps as well as behavior (Clark, 2013). It is not known how social and physical contact these two components of an organism's environment relate to one another, nor how different social settings may give rise to different experiences, nor how different experiences may be significant to the categories developed as a result of such experiences.

4.2. Embodiment and trauma

The embodiment paradigm suggests that experiences of trauma can be stored in the body and may have long-term neurobiological and health consequences. Neurobiological research has shown that traumatic events lead to changes in the brain's neural pathways and structures, which can result in over-active amygdala and low-active prefrontal cortex. These neurological changes can lead to physical symptoms such as anxiety, depression, and insomnia, including higher risk of cardiovascular and metabolic diseases. Trauma can also affect the body's stress response systems, leading to issues with the immune system, digestive system, and endocrine system. These physical and mental health consequences can have a lasting impact on an individual's well-being.

Embodiment examines the role of the body in cognition, decision-making, and behavior, as well as the influence of physical elements of the body on cognitive processes (Gallagher & Zahavi, 2012). It sheds light on the interconnections between physical and mental processes (Lakoff & Johnson, 2003). All this suggests that our physical actions, perceptions, and interactions with the environment can inform and influence our cognitive processes (Shapiro, 2019). Thus, the body and environment can be considered cognitive mediators, as they provide a context for the emergence and development of ideas and thoughts. In cognitive research, embodiment refers to the idea that cognition is grounded in the physical body and its interactions with the environment (Chemero, 2011). This suggests that the physical structure of the body, its movements, and its ability to interact with the environment are essential for understanding the cognitive processes of the mind.

While undeniably interdependent, embodiment slightly differs from embodied cognition. Embodied cognition investigates the notion that the mind and body are intricately intertwined and that the body influences and mediates cognitive processes such as learning, memory, problem-solving, and decision-making (Clark, 2013). It promotes a more comprehensive theory of cognition that takes physical, environmental, and social factors into account, and while embodiment is focused on interactions between mind, body, and environment. Embodied cognition is rather focused on its integration, where body is integrated and part of its environment, and mind is inside a body, where by default this means it is part of that same already mentioned environment. Regardless, this thesis for purposes of understanding rather translated even embodied cognition in terms of interaction, as this mentioned integration includes all these components cooperating and interacting.

4.3. Importance of predictive processing

The concept of computing known as predictive processing (PP) is predicated on the theory that the brain continually constructs and updates a mental model of the surrounding environment (Hohwy, 2014). This theory posits that through a highly selective and adaptive process, our perceptions are determined by the predictions that have proven to be the most accurate thus far. According to the theory of predictive coding, our brains create and refine a constant internal representation of the external environment (Kühn & Gallinat, 2014). The brain continually predicts the future and attempts to align its predictions with actual world events (Hohwy, 2014). The brain, or as in this thesis seen as mind, uses predictions to generate expectations and continuously modifies these expectations in response to actual events. This hypothesis is founded on the idea that the brain strives to minimize surprise and uncertainty by actively monitoring the environment, forming predictions, and revising those predictions as new information is acquired. The predictive coding concept seems to be essential for understanding the nature of chronic pain. For example, the brain feels pain if you move immediately after the injury, and since this is a negative experience, it works to avoid it in the future (Clark, 2013). Neural circuits in the brain establish a connection between an external stimulus and an internal response as part of the predictive coding process (Friston, 2005).

PP is in a way a bridge between the human body, human consciousness, and the external world and it governs agent's behavior in environment. Our mental and physical states could also influence the external environment and provide agent with new information about it. Agent is always influenced by its surroundings and PP helps to make good decisions and avoid possible negative effects based on what agent learned in a past (Hohwy, 2014). Idea of this concept can due to this fact easily be put in a context of embodiment - PP is designed for agent to minimize a processing error from information it receives from environment and helps mind to make decision about how will body act in this environment. If the long-term average prediction error of the agent increases as a result of the accommodation process, then the agent does not conform to the standard and fails to validate the model, where the model is a prediction schema agent has about a world and based on which it propagates errors (Hohwy, 2014). Opposite, a system that minimizes its prediction error successfully collects evidence consistent with the model used to explain it - the evidence agent collects supports the model if and only if it is consistent with the system's predictions. Data from the environment is collected to validate the model (Kühn & Gallinat, 2014). Steps in this validation are selecting, modifying, and refining the model so that it makes precise predictions that are useful for an accurate sampling of the world. This is where Bayesian inference comes in place and its role is to increase its own confidence in its predictions or decrease long-term error by approximating Bayesian inference (Clark, 2013).

The mind or brain stores sensory-perceived information after receiving it to cognitive maps, mental representations of the spatial organization and spatial relationships of an environment. They are produced and updated by sensory experience, enabling humans and other animals to form mental representations of their environments in order to use them for movement planning and navigation. Cognitive maps enable an individual to make predictions about their environment and the outcomes of their actions within it. The relationship between predictive processing and cognitive maps in cognitive research is close. Predictive processing utilizes previous experiences to predict and interpret the present (Friston, 2010) where cognitive maps guide behavior in a particular situation. PP updates and rewrites cognitive maps, allowing an individual to make predictions based on their environment and previous experiences. Consequently, cognitive maps become more precise and refined over time, allowing the individual to make more accurate judgments and engage in more fruitful actions. Reinforcement learning employs rewards and punishments to facilitate learning, whereas predictive processing (Körding, 2007) predicts future events on the basis of past events. Cognitive maps allow a person, or in this context an agent, to mentally depict a comprehension of a particular environment or problem and enable them to make decisions based on their awareness of the surrounding environment. As already mentioned, reinforcement learning is a method of machine learning in which an agent interacts with its environment and learns from the rewards and punishments it receives and, in this context, creating a cognitive map is comparable to how a person may learn from their surroundings (Doya, 2000). RL employs rewards and punishments to facilitate learning and agent can discover how to maximize its rewards by understanding its environment and the actions that will result in rewards or punishments. As the brain constantly predicts the future and attempts to align its predictions with actual world events (Friston, 2005), it uses predictions to produce expectations and constantly adjusts these expectations based on actual events.

Consequently, it is believed that this predictive processing enables us to make quick and effective decisions (Hohwy, 2014). Those who experienced a traumatic event have stronger predictive processing abilities than those who had not experienced trauma (McTeague et al., 2017) but at the same time, those with higher levels of predictive processing have higher levels of PTSD symptoms (Massazza et al., 2021). PP abilities that studies showed as trauma-specific and high in trauma survivors are threat anticipation, threat detection, and threat vigilance. Same persons also have stronger predictive processing abilities related to the ability to form adaptive responses to the environment, such as threat avoidance, attentional control, and emotional regulation (McTeague et al., 2017). As a result of the life-or-death nature of traumatic experiences, individuals develop risk-predicting attitudes thereafter, based on Kube et al. (2020). They also propose a significant hypothesis that traumatized individuals are more likely to make potentially hazardous assumptions about their surroundings. These ideas may cause people to feel constantly on edge, which can contribute to post-traumatic stress disorder symptoms. According to this theory, traumatic memories become more intrusive when disconformity in contextual information or indicating a safe setting is not used for updating, as this improves the accuracy of incoming sensory data (Kube et al., 2020). Wilkinson et al. (2017) add that intrusive re-experiencing of trauma may be more strongly associated with predictive processing following direct trauma. The impact of trauma-related thoughts on interoceptive emotional states is induced by intrusive re-secondary experiences. Thoughts of a horrific occurrence may prompt a person to adhere to their established routines and avoid trying anything new. Due to this form of biased sampling, it is less likely that trauma beliefs will be found and corrected (Linson & Friston, 2019).

Studies have suggested that predictive processing may help to explain the maladaptive cognitive patterns in people with PTSD, such as overgeneralizing, rumination, and avoidance (McTeague et al., 2017). Such studies provide valuable insights into the cognitive processes underlying the development and maintenance of PTSD symptoms and suggest that predictive processing may be a key factor in understanding how traumatic experiences can lead to the development of PTSD and that interventions targeting predictive processing may be beneficial in treating the disorder. According to the predictive processing paradigm, individuals with PTSD tend to keep trauma-related beliefs that are fixed. Those who do not maintain their trauma knowledge run the risk of developing post-traumatic stress disorder. Those who have difficulty controlling their emotions and behaviors or have a limited repertoire of coping mechanisms are more susceptible to acquiring posttraumatic stress disorder (PTSD) after enduring multiple stressful situations (Levy-Gigi et al., 2015). The inability to process negative experiences productively is a component that contributes to post-traumatic stress disorder. If negative reinforcements are not balanced by positive reinforcements, PTSD symptoms may become worse.

4.3.1. Reinforcement learning and embodiment

PTSD is described in this context as pervasive abnormalities in reinforcement learning mechanisms and alterations in learning connections with non-fear-related stimuli (Ross et al., 2018). Anxiety and stress can be brought on by an exaggerated assessment of the severity and possibility of impending negative events. In reinforcement learning (RL) a signal is connected with an anticipated outcome in order to construct a prediction model (Ross et al., 2018). Standard PTSD treatment includes exposure therapy, which is believed to function via reinforcement learning to assist the patient in forming new, less traumatic memories of a previously painful stimulus (Foa, 2011; Rothbaum & Davis, 2003; Ross et al., 2018). It is possible to assume how individuals with PTSD and anxiety utilize reinforcement learning to manage their fears (Ross et al., 2018). There is evidence that individuals with PTSD and a history of childhood maltreatment struggle with reinforcement learning and erasure of conditioned fear memories and overall, they have deficits in fear learning and extinction. What defines PTSD in this context is pervasive abnormalities in reinforcement learning mechanisms and changes in learning connections with non-fear-related stimuli.

4.4. Defining trauma in a context of cognitive science

According to the American Psychiatric Association (2013), trauma is the emotional, psychological, or physical response to a severely upsetting or unpleasant event that causes the affected individual extreme emotional agony and distress. Trauma can be the result of a single incident, such as a car crash or natural disaster, or it can be the result of repeated or chronic abuse, neglect, or violence (Courtois et al., 2009). It can cause long-lasting emotional and physical difficulties, such as anxiety, sadness, flashbacks, insomnia, and pain. Trauma can also alter a person's interpersonal relationships and daily functioning. Trauma can result in shock, terror, helplessness, and/or worry, as well as physical symptoms such as a rapid heartbeat, trouble breathing, and profuse perspiration. The trauma experienced by individuals is deeply connected to their bodies and the environment they inhabit; it is not simply a mental or emotional experience but a physical one as well, as agency, embodiment, and enactment are defined in psychosomatic theory and practice (Kirmayer & Gómez-Carrillo, 2019). The body and mind are intimately connected to the environment, and the environment can influence both physical and psychological experience.

Through the embodiment paradigm, it can be concluded that trauma is both an embodied experience and a product of the environment in which it occurs (Kirmayer & Young, 1998). Traumatic experiences are extremely difficult to predict (Chamberlin, 2019). As it is seen in the previously mentioned definitions of trauma, it is hard to predict as it always involves some unexpected event that agents do not possess any methods on how to deal with, as such events do not exist in the cognitive map of an agent and the agent has not yet learned how to deal with such an environment, which results in a breakdown of normal predictive processing (Chamberlin, 2019). This disruption may result in a variety of unpleasant psychological and physiological effects, including intrusive memories, fear, avoidance, and difficulty regulating emotions (Chamberlin, 2019). The predictive processing model of trauma postulates that the brain attempts to predict the environment and make sense of the traumatic event but is unable to do so due to the unpredictable nature of the event (Chamberlin, 2019; Letkiewicz et al., 2022). As a result, the brain continues to attempt to predict the environment despite its inability to do so, resulting in a number of negative consequences associated with trauma.

Trauma can be easily perceived through lenses of reinforcement learning and predictive processing and based on what we said about both in a previous text, it can be explained as a type of negative reinforcement experienced by an agent when it encounters unexpected or uncontrollable events and/or environments. It can be assumed that trauma can cause an agent to become preoccupied with survival, resulting in behavioral changes such as avoiding certain stimuli or attempting to flee the situation. it can also reduce an agent's capacity to learn or process information, as well as impair its decision-making abilities (Letkiewicz et al., 2022). In the meaning and importance of the predictive processing paradigm trauma could be explained in more detail a dysregulation of a person's physical, psychological, and emotional states following exposure to a threat or perceived threat (van der Kolk, 2003). This dysregulation can cause permanent changes in the brain, body, and behavior, which was previously mentioned and overviewed as proved neurobiological, physical health, and mental health consequences of a traumatic event. Trauma can also be caused by a lack of predictability or control, leaving the individual feeling unsafe and unable to process and integrate the experience effectively (Yeager & Dweck, 2012).

4.5. Proposal for Post-traumatic Embodiment model

Considering all previously mentioned, embodiment would include the utilization of prior knowledge and experience to anticipate and predict future sensory inputs. This requires the integration of ongoing sensory information with prior experience, enabling the organism to comprehend and respond meaningfully to its environment. Thus, embodiment would be the capacity to interpret the environment within the context of one's own body and capabilities. So, in the predictive processing paradigm frame, embodiment is viewed as the utilization of prior knowledge and experience to anticipate and predict future sensory inputs. In a sense of reinforcement learning, embodiment refers to the notion that an artificial agent can learn by interacting with its environment and that its physical structure, or body, can aid in its learning.

Trauma, embodiment paradigm, and predictive processing are all interconnected within the context of cognitive science (Wilkinson et al., 2017). This thesis explained among other consequences as well that trauma affects cognitive processes, resulting in a variety of cognitive impairments. The embodiment paradigm, which is based on the notion that the body and its environment interact to shape cognition, has been utilized to explain how traumatic experiences can disrupt cognitive processes. Trauma can lead to an impaired sense of body ownership and a disrupted sense of self (Kirmayer & Gómez-Carrillo, 2019), which in turn can lead to mentioned cognitive impairments. Trauma is related to the embodiment in that changes in the physical body can affect how an individual responds to trauma. Predictive processing is associated with trauma in that it can lead to the formation of certain schemas and cognitive biases that can influence how an individual interprets and responds to the world, including traumatic situations because as we can assume, trauma might lead to an increased reliance on top-down processes, which might lead to an impaired ability to make accurate predictions.

The interaction between these three concepts is exemplified by the fact that trauma can lead to changes in a person's perception and response to the world, which in turn can result in physical changes. A person who has experienced trauma, for instance, may become more guarded and have difficulty connecting with others, which can result in physical changes such as increased muscle tension and decreased physical activity. These physical changes can further affect how a person perceives the world and how they respond to it, leading to changes in the way a person processes information, as evidenced by the development of certain cognitive biases. The influence of these cognitive biases can then extend to an individual's embodied experience and behavior.

Post-traumatic embodiment could be defined as the physical, psychological, and behavioral changes that occur in an agent following exposure to an unexpected or uncontrollable event or environment, resulting in an altered sense of safety, dysregulation of physical, psychological, and emotional states, and a diminished capacity to process, learn, and make decisions. In a particular case of post-traumatic embodiment, it is the trauma experienced by the agent and its effect on the predictive processing paradigm that has caused these modifications. Post-traumatic embodiment refers to the long-term, cumulative effects of trauma on a person's mind and body, resulting in changes to their behavior, physical and mental health, and overall functioning. It is the process by which an individual's trauma-related responses become embodied, resulting in a state of hypervigilance, anxiety, and dysregulation. This manifestation of trauma can manifest itself in a variety of ways, including through avoidance behaviors, physical symptoms, psychological distress, cognitive impairment, and altered self-perception. Post-traumatic embodiment reflects the individual's effort to adapt and cope with their trauma and can have profound, long-lasting effects on their quality of life, some of them assumingly being avoidance, modifications to the learning and decision-making processes, and alterations to the way the body and mind interact with the environment.

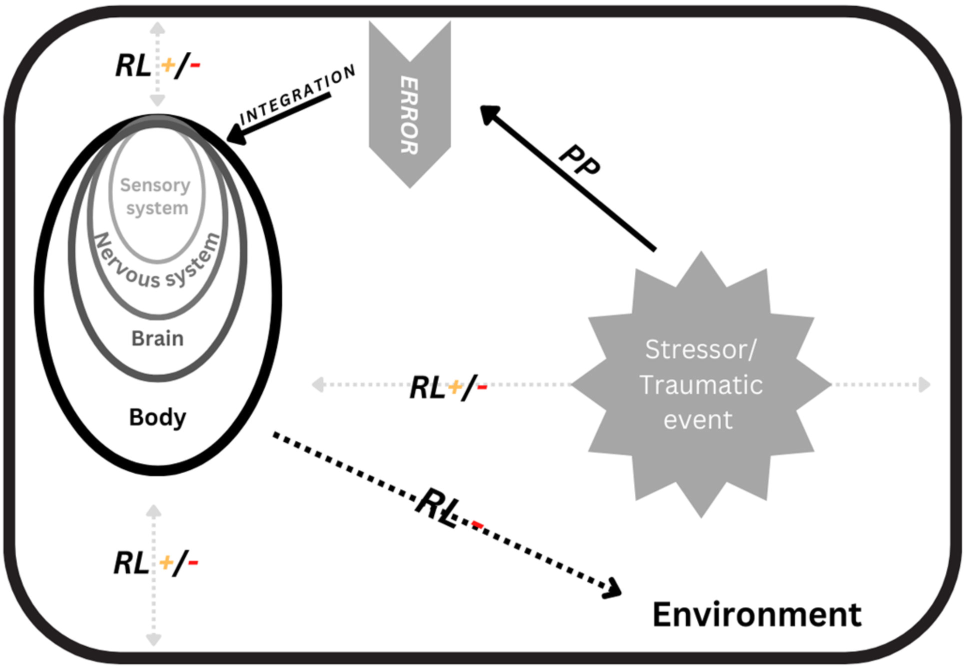

Figure 5. *Simplified schematic display of post-traumatic embodiment model.* Schematic model to show graphically logic of post-traumatic embodiment, created

based on the conclusions and assumptions made in this thesis. The agent (oval shape) that interacts with environment based on RL principles, then encounters a stressor or traumatic event that then has influence on PP, creating an error that then influences the whole agent's system.

The above schematic display (Figure 5.) shows an agent, consisting of a sensory system, nervous system, brain, and body, that has all these components embedded and working together in order to function properly and interact with its environment. The agent interacts with the environment constantly, following the RL logic of rewards and punishments (positive and negative feedback), and stores learned feedback in its cognitive map. In the future, it makes decisions and interacts with the environment based on this stored information, making predictions about what can be expected and how to behave (PP). When a traumatic event or stressor unexpectedly appears in the environment, the agent's PP process now cannot adequately respond to the event based on the RL and PP logic previously described and responds intuitively in order to avoid the immediate negative consequences of the event. This gives negative feedback in terms of RL logic, and this will be integrated in the future as something where agents can expect negative consequences or punishment if they encounter the same situation or similar settings. On the PP level, the impairment shows due to the unexpectedness of the event, as the mind or brain does not have adequate information about such events and does not know how to handle a situation it has not yet encountered. A traumatic event then creates an error in processing as the brain realizes it does not have the proper tools to protect the agent from possible danger or to interact with that novel situation. This error then integrates into the nervous system, and because it is constantly interacting with the brain, it eventually integrates into the entire body. The agent continues to interact with the environment in the future, but it is now possible to assume that the agent will respond to environments, details, or the same or similar event as the traumatic one in the same or identical way that it responded to a traumatic event that embedded into the body as a result of an error that integrated.

A bellow image (Figure 6.) shows a process of the previously explained flow of interactions and shows steps and how these parts of process are interconnected. It is supposed that the agent interacts with environment based on the RL principles, which then leads to process of PP, with a mediation of sensory system information that integrates into cognitive maps. Each arrow type represents possible flow or possible next step.

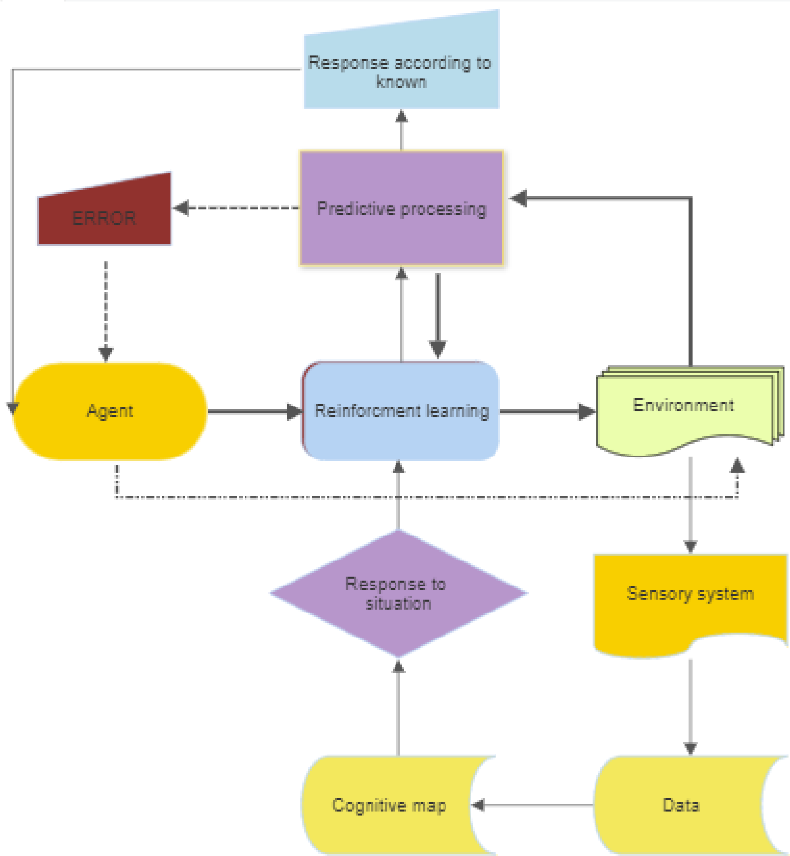

Figure 6. *A representation of the process of post-traumatic embodiment model.* A Process model that is made for purposes of this thesis as a result of all gathered information. Colors of each box are paired categorically – dark and bright yellow are connected to the agent, bright and dark blue are connected to RL, purple to PP, green to surroundings/environment, and red to error in processing. Arrows show the flow of steps, and all their possibilities. Each arrow type describes a particular and possible steps' flow. Details of this process are described in above text.

4.6. Implications of a model for science and therapy

RL is commonly used in cognitive science research settings for the treatment of PTSD and trauma, and it has shown promising results for the reduction of symptoms and improvement of psychological functioning. In some studies, RL techniques were used in a cognitive behavioral therapy (CBT) program designed to treat PTSD in veterans (Karstoft et al., 2015), and it was shown that those who received the CBT program with reinforcement learning had greater reductions in PTSD symptom severity and improved psychological functioning than those who received only traditional CBT. RL interventions are associated with greater improvements in PTSD symptom severity, sleep quality, and depression, as well as better functioning in social and occupational domains than traditional treatments (Galatzer-Levy et al., 2017). Cognitive science research has enabled novel approaches to research methods as well as the testing of technology tools with potential in trauma therapy, such as virtual reality (VR). A study conducted by Difede and Cukor (2007) found that the use of VR exposure therapy in combination with reinforcement learning techniques was more effective in treating PTSD than VR alone. Participants in the RL group were provided with rewards, such as points and virtual currency, for the successful completion of VR scenarios. The results showed that those in the RL group exhibited greater reductions in symptom severity and avoidance behavior than those in the VR-only group. Other studies (Karstoft et al., 2015) indicate that the RL rewards system exhibits greater reductions in PTSD symptom severity and avoidance behavior compared to the usual treatment of trauma, as well as potentially reducing PTSD symptoms such as symptom severity, sleep quality, and depression, as well as better functioning in social and occupational domains than traditional treatments (Rizvi et al., 2009).

PP employs cognitive and behavioral strategies to assist individuals in recognizing, anticipating, and preparing for their reactions to trauma, thereby reducing the likelihood of future distress (Rizvi et al., 2009). Predictive processing assists individuals in identifying their emotional and physical reactions to trauma and in developing coping mechanisms to manage these reactions (Schumm et al., 2015). In addition, it assists people in identifying their triggers and developing effective coping mechanisms to manage them. PP is an effective method for aiding individuals in the healing process and trauma recovery. Using reinforcement learning, individuals who have experienced trauma can learn to regulate their emotions and responses in a more positive and adaptive manner. This could be especially important in embodied situations, as reinforcement learning enables humans to recognize and respond to environmental stimuli in a manner that is more congruent with their desired goals. A trauma survivor may learn to detect and respond more adaptively to cues that are associated with feelings of fear or anxiety, such as by focusing on their breath or engaging in other calming activities.

Individuals can learn to perceive and respond to these cues in a manner more congruent with their goals by reinforcing these behaviors. This can aid people in overcoming trauma and living more fulfilling lives. RL can be used in trauma recovery therapy based on an embodiment framework to help patients learn to be in the present moment and strengthen their connection to their bodies. RL can help patients recognize and control their emotions, modify their behavior in response to stimuli, and develop more adaptive coping skills. RL can assist patients in learning to recognize, express, and manage their emotions through the use of an embodied approach. In addition, an embodied approach to RL can assist patients in exploring and comprehending the physical sensations associated with their emotions, as well as how to use those sensations to guide their behavior. This can help patients gain a better understanding of how their bodies are reacting to challenging situations and how to effectively cope with them. RL can provide a safe and supportive environment for patients to practice new behaviors and develop the necessary skills to help them process and heal from their trauma.

Future implications for cognitive science studies of embodiment and trauma could include exploring the ways that trauma affects our ability to form meaningful connections with our physical environment, as well as how our physical bodies respond to traumatic experiences. Research could also look at how the physical and psychological impacts of trauma can be better managed and treated. Additionally, cognitive science could explore the ways in which embodiment and trauma can be used to inform interventions or treatments for mental health conditions like anxiety or depression. Finally, cognitive science could investigate the ways that trauma-informed interventions can help individuals better process difficult or overwhelming emotions. By examining the physiological, neurological, and psychological reactions to trauma, researchers could develop a better understanding of how the body stores and expresses trauma and the ways in which the environment can either buffer or exacerbate the effects of trauma. Such research could include examining the effects of cognitive-behavioral therapies and psychotherapies on the body-mind-environment interaction as well as the potential intergenerational transmission of trauma through the body-mind-environment interaction.

Research could also look at the relationship between trauma, agency, and environment. For example, how does the physical environment shape our experience of trauma, and how can we use the environment to foster resilience?

Research could look at how trauma can affect an individual’s sense of self and how this can affect the way they interact with the environment. Further potential approaches include examining the role of cognitive flexibility in a trauma survivor's ability to perceive and respond to their environment, as well as how their sense of self and identity may be impacted by traumatic experiences. Additionally, the research could explore how different types of trauma may affect the way in which people use their bodies to interact with their environment and how this could have an impact on their sense of agency.

One very important but often forgotten benefit of upgrading current cognitive science approaches to trauma is how trauma can affect our physical and sensory experiences immediately during traumatic events and retrospectively in terms of long-term consequences. Sensory experience is at the forefront of perceiving a specific interaction or happening, and it is how the nervous system and brain learn about what and how is going on. It is a vague assumption, but it is interesting to note that such research could lead to clearer research on how trauma can be embodied through the use of technology, such as virtual reality, and how this can be used to support healing. Some of the research questions that this thesis evokes and opens doors to ask ourselves about are the following: How do the physical body and its environment, such as the presence of other people, affect the psychological processes associated with trauma? For example, how does the level of physical comfort and safety in an environment influence the recovery rate of trauma victims? How does the physical environment influence the intensity and duration of post-traumatic stress disorder (PTSD) symptoms? Another research question could focus on the effects of embodiment on the cognitive processes associated with trauma, such as memory and emotion. Specifically, how does the physical body interact with memories of traumatic events, and how does this interaction shape the experience of post-traumatic stress? Finally, how does one’s physical environment, including the presence of friends and family, shape the way in which one processes and heals from trauma?

As it is visible, there are many more questions and problems that remain unanswered when it comes to trauma and embodiment, and it is obvious why it would be of great scientific value to explain embodiment in the context of trauma and further explore it in an environment of cognitive science.

Conclusion

This thesis has explored the interconnectedness of trauma, embodiment, and predictive processing in the field of cognitive science (Blonder, 1999; Brown, 2000). It has highlighted how trauma can lead to physical and psychological changes in the body (Damasio, 2010; van der van der Kolk et al., 1989), which can lead to changes in the way one perceives and responds to the world, and how predictive processing can lead to the development of certain schemas and cognitive biases which further influence one’s embodied experience and behavior (Clark, 2013; Mur et al., 2012). Ultimately, this research has provided further insight into how trauma, embodiment, and predictive processing are interconnected in the field of cognitive science, which can be used to develop more effective interventions and treatments for those who have experienced trauma.

Post-traumatic embodiment reflects the individual’s attempt to adapt to and cope with their trauma and can have profound, long-term impacts on their quality of life. Understanding the effects of trauma on the mind and body is necessary in order to support those who have experienced traumatic events and to create effective interventions for individuals suffering from post-traumatic embodiment. It is very important to explain how the embodiment paradigm is a framework for understanding the nature of cognition in which the physical and social environment play a vital role in shaping the ways in which organisms think, feel, and behave (Shapiro, 2019). It suggests that the body is an integral part of the cognitive process and that physical experiences are inextricably linked to cognitive ones (Barsalou, 2008). EC, on the other hand, is the idea that cognition is grounded in the physical body and its interactions with the environment (Kiefer & Pulvermüller, 2012; Niedenthal et al., 2005). This means that cognition is a result of the body's interactions with the outside world rather than an internal cognitive process. This theory suggests that thinking is partly a result of embodied experiences and that the body plays an important role in shaping how we think. However, this study has focused more on the mentioned postulates of the embodiment paradigm than embodied cognition's grounding processes, as the whole concept of post-traumatic embodiment in cognitive science as a specific construct that can be researched in the future is new, and this thesis is simply exploring its possibilities and establishing foundations for further research and definitions.

This thesis offered a clear overview of embodiment concept from philosophy to cognitive science and consequently clearly explained embodiment in cognitive science as a unique sensorimotor interaction between an organism and its environment that includes sense of being in one's body. The current definition of trauma was included, where trauma is defined as any bodily harm, a catastrophic occurrence, or psychological and physiological reactions to an extraordinarily unfavorable experience, in most cases ending in a feeling of helplessness. A short overview of trauma's consequences on a body and mind/brain in terms of a stress response was outlined successfully. Current body of research shows that trauma severely inflicts physical and mental health and provenly results in long-term negative consequences (Bielawski et al., 2022; Sankowski et al., 2015).

After some contemplation and exploration of different cognitive science paradigms and concepts, this thesis focused on predictive processing paradigm and reinforcement learning principles, as they show a logical connection to trauma research and PTSD mechanisms. In this context, trauma was defined as a type of negative reinforcement experienced by an agent when it encounters unexpected or uncontrollable events and/or environments.

Finally, a post-traumatic embodiment model was proposed, following an explanation that post-traumatic embodiment is the physical, psychological, and behavioral changes that occur in an agent following exposure to an unexpected or uncontrollable event or environment, resulting in an altered sense of safety, dysregulation of physical, psychological, and emotional states, and a diminished capacity to process, learn, and make decisions, where unexpected or uncontrollable event is explained as trauma that we defined in a context of cognitive science.

It is of essential importance to further expand the construct to include embodied cognition and even explore a post-traumatic embodiment in a wider range of enactivism, a theoretical approach to understanding cognition that emphasizes the active, embodied nature of the mind and holds that cognition is an embodied, situated, and adaptive process. This thesis did not explain or try to connect enactivism with post-traumatic embodiment, even though this approach challenges traditional cognitive science models of the mind as an information processing system. Enactivism posits that the mind is an embodied and embedded system that actively engages with its environment (Chemero, 2011), and because of this fact, it would be important to explore trauma in this context in future research. It would also be interesting to explore a concept of post-traumatic embodiment in a context of broader research ideas that would include more environment and social surroundings interactions with the agent, as observations of trauma consequences that are not yet properly explored.

Overall, regardless of a complexity of a given subject and main aims, this thesis successfully proposed the novel model of post-traumatic embodiment, successfully defined trauma in a context of cognitive science, and in the end, successfully set a ground in a theoretical way for future exploration of this model in metrical and more robust sense.

References

Acheson, D., Geyer, M., Baker, D., Nievergelt, C., Yurgil, K., & Risbrough, V. (2015). Conditioned fear and extinction learning performance and its association with psychiatric symptoms in active duty Marines. *Psychoneuroendocrinology*, *51*, 495–505. https://doi.org/10.1016/j.psyneuen.2014.09.030

Allen, M., & Friston, K. J. (2016). From cognitivism to autopoiesis: towards a computational framework for the embodied mind. *Synthese*, *195*(6), 2459–2482. https://doi.org/10.1007/s11229-016-1288-5

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5(TM))* (5th ed.). American Psychiatric Publishing.

Apostolova, L. G., Green, A. E., Babakchanian, S., Hwang, K. S., Chou, Y. Y., Toga, A. W., & Thompson, P. M. (2012). Hippocampal Atrophy and Ventricular Enlargement in Normal Aging, Mild Cognitive Impairment (MCI), and Alzheimer Disease. *Alzheimer Disease &Amp; Associated Disorders*, *26*(1), 17–27. https://doi.org/10.1097/wad.0b013e3182163b62

Arnsten, A. F. T. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience*, *10*(6), 410–422. https://doi.org/10.1038/nrn2648

Assogna, F., Piras, F., & Spalletta, G. (2020). Neurobiological Basis of Childhood Trauma and the Risk for Neurological Deficits Later in Life. *Childhood Trauma in Mental Disorders*, 385–410. https://doi.org/10.1007/978-3-030-49414-8_18

Barsalou, L. W. (2008). Grounded Cognition. *Annual Review of Psychology*, *59*(1), 617–645. https://doi.org/10.1146/annurev.psych.59.103006.093639

Bartlett, F. C., & Burt, C. (1933). Remembering: a study in experimental and social psychology. *British Journal of Educational Psychology*, *3*(2), 187–192. https://doi.org/10.1111/j.2044-8279.1933.tb02913.x

Beer, R. D. (2008). The Dynamics of Brain–Body–Environment Systems. *Handbook of Cognitive Science*, 99–120. https://doi.org/10.1016/b978-0-08-046616-3.00006-2

Bernhagen, J., Bacher, M., Calandra, T., Metz, C. N., Doty, S. B., Donnelly, T., & Bucala, R. (1996). An essential role for macrophage migration inhibitory factor in the tuberculin delayed-type hypersensitivity reaction. *Journal of Experimental Medicine*, *183*(1), 277–282. https://doi.org/10.1084/jem.183.1.277

Berridge, C. W., & Dunn, A. J. (1990). DSP-4-induced depletion of brain norepinephrine produces opposite effects on exploratory behavior 3 and 14 days after treatment. *Psychopharmacology*, *100*(4), 504–508. https://doi.org/10.1007/bf02244003

Bielawski, T., Drapała, J., Krowicki, P., Stańczykiewicz, B., & Frydecka, D. (2022). Trauma Disrupts Reinforcement Learning in Rats—A Novel Animal Model of Chronic Stress Exposure. *Frontiers in Behavioral Neuroscience*, *16*. https://doi.org/10.3389/fnbeh.2022.903100

Bierhaus, A., Wolf, J., Andrassy, M., Rohleder, N., Humpert, P. M., Petrov, D., Ferstl, R., von Eynatten, M., Wendt, T., Rudofsky, G., Joswig, M., Morcos, M., Schwaninger, M., McEwen, B., Kirschbaum, C., & Nawroth, P. P. (2003). A mechanism converting psychosocial stress into mononuclear cell activation. *Proceedings of the National Academy of Sciences*, *100*(4), 1920–1925. https://doi.org/10.1073/pnas.0438019100

Black, P. H. (2002). Stress and the inflammatory response: A review of neurogenic inflammation. *Brain, Behavior, and Immunity*, *16*(6), 622–653. https://doi.org/10.1016/s0889-1591(02)00021-1

Blonder, L. X. (1999). *The Emotional Brain: The Mysterious Underpinnings of Emotional Life*. Joseph LeDoux. *The Quarterly Review of Biology*, *74*(4), 505–505. https://doi.org/10.1086/394219

Boyce, W. T., Quas, J., Alkon, A., Smider, N. A., Essex, M. J., & Kupfer, D. J. (2001). Autonomic reactivity and psychopathology in middle childhood. *British Journal of Psychiatry*, *179*(2), 144–150. https://doi.org/10.1192/bjp.179.2.144

Bracha, H. S. (2004). Freeze, Flight, Fight, Fright, Faint: Adaptationist Perspectives on the Acute Stress Response Spectrum. *CNS Spectrums*, *9*(9), 679–685. https://doi.org/10.1017/s1092852900001954

Bremner, J. D. (2006). Traumatic stress: effects on the brain. *Dialogues in Clinical Neuroscience*, *8*(4), 445–461. https://doi.org/10.31887/dcns.2006.8.4/jbremner

Bremner, J. D., Innis, R. B., Southwick, S. M., Staib, L., Zoghbi, S., & Charney, D. S. (2000). Decreased Benzodiazepine Receptor Binding in Prefrontal Cortex in Combat-Related Posttraumatic Stress Disorder. *American Journal of Psychiatry*, *157*(7), 1120–1126. https://doi.org/10.1176/appi.ajp.157.7.1120

Bremner, J. D., Randall, P., Vermetten, E., Staib, L., Bronen, R. A., Mazure, C., Capelli, S., McCarthy, G., Innis, R. B., & Charney, D. S. (1997). Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse—a preliminary report. *Biological Psychiatry*, *41*(1), 23–32. https://doi.org/10.1016/s0006-3223(96)00162-x

Breslau, N. (2009). The Epidemiology of Trauma, PTSD, and Other Posttrauma Disorders. *Trauma, Violence, &Amp; Abuse*, *10*(3), 198–210. https://doi.org/10.1177/1524838009334448

Brooks, R. A. (1991). Intelligence without representation. *Artificial Intelligence*, *47*(1–3), 139–159. https://doi.org/10.1016/0004-3702(91)90053-m

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research*, *60*(2), 113–124. https://doi.org/10.1016/j.jpsychores.2005.06.074

Brown, J. P. (2000). Psychobiology of posttraumatic stress. *Australian and New Zealand Journal of Psychiatry*, *34*(s1), A10–A10. https://doi.org/10.1080/000486700559

Byrne, C. A., Hyman, I. E., & Scott, K. L. (2001). Comparisons of memories for traumatic events and other experiences. *Applied Cognitive Psychology*, *15*(7), S119–S133. https://doi.org/10.1002/acp.837

Cacciaglia, R., Nees, F., Grimm, O., Ridder, S., Pohlack, S. T., Diener, S. J., Liebscher, C., & Flor, H. (2017). Trauma exposure relates to heightened stress, altered amygdala morphology and deficient extinction learning: Implications for psychopathology. *Psychoneuroendocrinology*, *76*, 19–28. https://doi.org/10.1016/j.psyneuen.2016.11.012

Calandra, T., Bernhagen, J., Metz, C. N., Spiegel, L. A., Bacher, M., Donnelly, T., Cerami, A., & Bucala, R. (1995). MIF as a glucocorticoid-induced modulator of cytokine production. *Nature*, *377*(6544), 68–71. https://doi.org/10.1038/377068a0

Carpenter, L. L., Carvalho, J. P., Tyrka, A. R., Wier, L. M., Mello, A. F., Mello, M. F., Anderson, G. M., Wilkinson, C. W., & Price, L. H. (2007). Decreased Adrenocorticotropic Hormone and Cortisol Responses to Stress in Healthy Adults Reporting Significant Childhood Maltreatment. *Biological Psychiatry*, *62*(10), 1080–1087. https://doi.org/10.1016/j.biopsych.2007.05.002

Casasanto, D., & Boroditsky, L. (2008). Time in the mind: Using space to think about time. *Cognition*, *106*(2), 579–593. https://doi.org/10.1016/j.cognition.2007.03.004

Chafee, M. V., & Goldman-Rakic, P. S. (2000). Inactivation of Parietal and Prefrontal Cortex Reveals Interdependence of Neural Activity During Memory-Guided Saccades. *Journal of Neurophysiology*, *83*(3), 1550–1566. https://doi.org/10.1152/jn.2000.83.3.1550

Chamberlin, D. E. (2019). The Predictive Processing Model of EMDR. *Frontiers in Psychology*, *10*. https://doi.org/10.3389/fpsyg.2019.02267

Chapman, C. R., Tuckett, R. P., & Song, C. W. (2008). Pain and Stress in a Systems Perspective: Reciprocal Neural, Endocrine, and Immune Interactions. *The Journal of Pain*, *9*(2), 122–145. https://doi.org/10.1016/j.jpain.2007.09.006

Chemero, A. (2011). *Radical Embodied Cognitive Science (A Bradford Book)* (Reprint). Bradford Books.

Clark, A. (2013). Whatever next? Predictive brains, situated agents, and the future of cognitive science. *Behavioral and Brain Sciences*, *36*(3), 181–204. https://doi.org/10.1017/s0140525x12000477

Cohen, J. A., Perel, J. M., Debellis, M. D., Friedman, M. J., & Putnam, F. W. (2002). Treating Traumatized Children. *Trauma, Violence, &Amp; Abuse*, *3*(2), 91–108. https://doi.org/10.1177/15248380020032001

Courtois, C. A., Norton-Ford, J. D., Herman, J. L., & Van Der Kolk, B. A. (2009). Treating complex traumatic stress disorders: An evidence-based guide. *The Guilford Press EBooks*. http://ci.nii.ac.jp/ncid/BA89147024

Cowart, M., Fieser, J. eds., & Dowden, B. eds. (2005). *Embodied Cognition*. Internet Encyclopedia of Philosophy. https://iep.utm.edu/embodied-cognition/

Cozolino, L. (2017). *The Neuroscience of Psychotherapy: Healing the Social Brain (Norton Series on Interpersonal Neurobiology)* (Third). W. W. Norton & Company.

Crossin, K. L., Tai, M. H., Krushel, L. A., Mauro, V. P., & Edelman, G. M. (1997). Glucocorticoid receptor pathways are involved in the inhibition of astrocyte proliferation. *Proceedings of the National Academy of Sciences*, *94*(6), 2687–2692. https://doi.org/10.1073/pnas.94.6.2687

Daffin, L. A. B. A. W., Jr. (2018, January 5). *Abnormal Psychology – Simple Book Publishing*. Pressbooks. https://opentext.wsu.edu/abnormal-psych/

Damasio, A. R. (2010). Self comes to mind: constructing the conscious brain. *Pantheon Books*. https://ci.nii.ac.jp/ncid/BB04203350

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: when the immune system subjugates the brain. *Nature Reviews Neuroscience*, *9*(1), 46–56. https://doi.org/10.1038/nrn2297

de Bellis, M. D., Chrousos, G. P., Dorn, L. D., Burke, L., Helmers, K., Kling, M. A., Trickett, P. K., & Putman, F. W. (1994). Hypothalamic-Pituitary-Adrenal Axis Dysregulation in Sexually Abused Girls. *Obstetrical &Amp; Gynecological Survey*, *49*(7), 487–489. https://doi.org/10.1097/00006254-199407000-00022

De Bosscher, K., Vanden Berghe, W., & Haegeman, G. (2003). The Interplay between the Glucocorticoid Receptor and Nuclear Factor-κB or Activator Protein-1: Molecular Mechanisms for Gene Repression. *Endocrine Reviews*, *24*(4), 488–522. https://doi.org/10.1210/er.2002-0006

de Kloet, E. R., Joëls, M., & Holsboer, F. (2005). Stress and the brain: from adaptation to disease. *Nature Reviews Neuroscience*, *6*(6), 463–475. https://doi.org/10.1038/nrn1683

Descartes, R., Cottingham, J., & Williams, B. (1996). Meditations on First Philosophy. *Descartes: Meditations on First Philosophy*, 1–11. https://doi.org/10.1017/cbo9780511805028.006

Dhikav, V., & Anand, K. (2012). Hippocampus in health and disease: An overview. *Annals of Indian Academy of Neurology*, *15*(4), 239. https://doi.org/10.4103/0972-2327.104323

Difede, J., & Cukor, J. (2007). Virtual Reality Exposure Therapy for the Treatment of Posttraumatic Stress Disorder Following September 11, 2001. *The Journal of Clinical Psychiatry*, *68*(11), 1682–1689. https://doi.org/10.4088/jcp.v68n1102

Doya, K. (2000). Reinforcement Learning in Continuous Time and Space. *Neural Computation*, *12*(1), 219–245. https://doi.org/10.1162/089976600300015961

Dualism and Mind | Internet Encyclopedia of Philosophy. (n.d.). https://iep.utm.edu/dualism-and-mind/

Dyer, M. G. (1991). Connectionism Versus Symbolism in High-Level Cognition. *Connectionism and the Philosophy of Mind*, 382–416. https://doi.org/10.1007/978-94-011-3524-5_17

Eysenck, M. W., & Keane, M. T. (2010). *Cognitive Psychology: A Student's Handbook, 6th Edition* (6th ed.). Psychology Press.

Faap, M. T. M. K., Faap, M. H. A. M., Faap, M. D. C. E., Faap, M. T. D. G., Faap, F. J. M. M., & Faap, P. M. D. K. M. (2016). *American Academy of Pediatrics Textbook of Pediatric Care* (Second). American Academy of Pediatrics.

Fei, D. (2020). From 'the mind isolated with the body' to 'the mind being embodied': Contemporary approaches to the philosophy of the body. *Cultures of Science*, *3*(3), 206–219. https://doi.org/10.1177/2096608320960242

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M. P., & Marks, J. S. (1998). Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults. *American Journal of Preventive Medicine*, *14*(4), 245–258. https://doi.org/10.1016/s0749-3797(98)00017-8

Foucault, M. (2019). Nietzsche, Genealogy, History. *Language, Counter-Memory, Practice*, 139–164. https://doi.org/10.1515/9781501741913-008

Friston, K. (2005). A theory of cortical responses. *Philosophical Transactions of the Royal Society B: Biological Sciences, 360*(1456), 815–836. https://doi.org/10.1098/rstb.2005.1622

Friston, K. (2010). The free-energy principle: a unified brain theory? *Nature Reviews Neuroscience, 11*(2), 127–138. https://doi.org/10.1038/nrn2787

Full and partial posttraumatic stress disorder: findings from a community survey. (1997). *American Journal of Psychiatry*, *154*(8), 1114–1119. https://doi.org/10.1176/ajp.154.8.1114

Galatzer-Levy, I. R., Ma, S., Statnikov, A., Yehuda, R., & Shalev, A. Y. (2017). Utilization of machine learning for prediction of post-traumatic stress: a re-examination of cortisol in the prediction and pathways to non-remitting PTSD.

Translational Psychiatry, *7*(3), e1070–e1070. https://doi.org/10.1038/tp.2017.38

Gallagher, S., & Zahavi, D. (2012). *The Phenomenological Mind* (2nd ed.). Routledge.

Gallese †, V., & Lakoff, G. (2005). The Brain's concepts: the role of the Sensory-motor system in conceptual knowledge. *Cognitive Neuropsychology*, *22*(3–4), 455–479. https://doi.org/10.1080/02643290442000310

Galotti, K. M. (2017). *Cognitive Psychology In and Out of the Laboratory* (6th ed.). SAGE Publications, Inc.

Gatchel, R. J., Peng, Y. B., Peters, M. L., Fuchs, P. N., & Turk, D. C. (2007). The biopsychosocial approach to chronic pain: Scientific advances and future directions. *Psychological Bulletin*, *133*(4), 581–624. https://doi.org/10.1037/0033-2909.133.4.581

Gavish, M. (1996). Altered Platelet Peripheral-Type Benzodiazepine Receptor in Posttraumatic Stress Disorder. *Neuropsychopharmacology*, *14*(3), 181–186. https://doi.org/10.1016/0893-133x(95)00078-r

Gendlin, E. T. (1998). *Focusing-Oriented Psychotherapy: A Manual of the Experiential Method* (1st ed.). The Guilford Press.

Goverse, G., Stakenborg, M., & Matteoli, G. (2016). The intestinal cholinergic anti-inflammatory pathway. *The Journal of Physiology*, *594*(20), 5771–5780. https://doi.org/10.1113/jp271537

Gramsch, C., Kattoor, J., Icenhour, A., Forsting, M., Schedlowski, M., Gizewski, E. R., & Elsenbruch, S. (2014). Learning pain-related fear: Neural mechanisms mediating rapid differential conditioning, extinction and reinstatement processes in human visceral pain. *Neurobiology of Learning and Memory*, *116*, 36–45. https://doi.org/10.1016/j.nlm.2014.08.003

Hannibal, K. E., & Bishop, M. D. (2014). Chronic Stress, Cortisol Dysfunction, and Pain: A Psychoneuroendocrine Rationale for Stress Management in Pain Rehabilitation. *Physical Therapy*, *94*(12), 1816–1825. https://doi.org/10.2522/ptj.20130597

Harris, N. B. (2015, February 17). *How childhood trauma affects health across a lifetime*. TED Talks. https://www.ted.com/talks/nadine_burke_harris_how_childhood_trauma_affects_health_across_a_lifetime

Hart, J., Gunnar, M., & Cicchetti, D. (1996). Altered neuroendocrine activity in maltreated children related to symptoms of depression. *Development and Psychopathology*, *8*(1), 201–214. https://doi.org/10.1017/s0954579400007045

Heim, C., & Nemeroff, C. B. (1999). The impact of early adverse experiences on brain systems involved in the pathophysiology of anxiety and affective disorders. *Biological Psychiatry*, *46*(11), 1509–1522. https://doi.org/10.1016/s0006-3223(99)00224-3

Herman, J. L., Bevrani, K., & Bevrani, S. (2016). *Trauma and Recovery: The Aftermath of Violence--From Domestic Abuse to Political Terror (Persian Edition)* (Persian ed.). Kaveh Bevrani.

Herrmann, M., Schölmerich, J., & Straub, R. H. (2000). Stress and rheumatic disease. *Rheumatic Disease Clinics of North America*, *26*(4), 737–763. https://doi.org/10.1016/s0889-857x(05)70167-8

Hohwy, J. (2014). *The Predictive Mind* (1st ed.). Oxford University Press.

Hopper, J. W., Frewen, P. A., van der Kolk, B. A., & Lanius, R. A. (2007). Neural correlates of reexperiencing, avoidance, and dissociation in PTSD: Symptom dimensions and emotion dysregulation in responses to script-driven trauma imagery. *Journal of Traumatic Stress*, *20*(5), 713–725. https://doi.org/10.1002/jts.20284

Horgan, T., & Tienson, J. (Eds.). (1991). Connectionism and the Philosophy of Mind. *Studies in Cognitive Systems*. https://doi.org/10.1007/978-94-011-3524-5

Irwin, M. R. (2008). Human psychoneuroimmunology: 20 Years of discovery. *Brain, Behavior, and Immunity*, *22*(2), 129–139. https://doi.org/10.1016/j.bbi.2007.07.013

Iyadurai, L., Visser, R. M., Lau-Zhu, A., Porcheret, K., Horsch, A., Holmes, E. A., & James, E. L. (2019). Intrusive memories of trauma: A target for research bridging cognitive science and its clinical application. *Clinical Psychology Review*, *69*, 67–82. https://doi.org/10.1016/j.cpr.2018.08.005

Jackendoff, R. (2002). The Mind Doesn't Work That Way: The Scope and Limits of Computational Psychology (review). *Language*, *78*(1), 164–170. https://doi.org/10.1353/lan.2002.0024

Joëls, M., & Baram, T. Z. (2009). The neuro-symphony of stress. *Nature Reviews Neuroscience*, *10*(6), 459–466. https://doi.org/10.1038/nrn2632

Jones, L. K., & Cureton, J. L. (2014). Trauma Redefined in the DSM-5: Rationale and Implications for Counseling Practice. *The Professional Counselor*, *4*(3), 257–271. https://doi.org/10.15241/lkj.4.3.257

Kalupahana, N. S., & Moustaid-Moussa, N. (2011). The renin-angiotensin system: a link between obesity, inflammation and insulin resistance. *Obesity Reviews*, *13*(2), 136–149. https://doi.org/10.1111/j.1467-789x.2011.00942.x

Karstoft, K. I., Statnikov, A., Andersen, S. B., Madsen, T., & Galatzer-Levy, I. R. (2015). Early identification of posttraumatic stress following military deployment: Application of machine learning methods to a prospective study of Danish soldiers. *Journal of Affective Disorders*, *184*, 170–175. https://doi.org/10.1016/j.jad.2015.05.057

Keilp, J. G., Stanley, B. H., Beers, S. R., Melhem, N. M., Burke, A. K., Cooper, T. B., Oquendo, M. A., Brent, D. A., & John Mann, J. (2016). Further evidence of low baseline cortisol levels in suicide attempters. *Journal of Affective Disorders*, *190*, 187–192. https://doi.org/10.1016/j.jad.2015.10.012

Kiefer, M., & Pulvermüller, F. (2012). Conceptual representations in mind and brain: Theoretical developments, current evidence and future directions. *Cortex*, *48*(7), 805–825. https://doi.org/10.1016/j.cortex.2011.04.006

Kirmayer, L. J., & Gómez-Carrillo, A. (2019). Agency, embodiment and enactment in psychosomatic theory and practice. *Medical Humanities*, *45*(2), 169–182. https://doi.org/10.1136/medhum-2018-011618

Kirmayer, L. J., & Young, A. (1998). Culture and Somatization. *Psychosomatic Medicine*, *60*(4), 420–430. https://doi.org/10.1097/00006842-199807000-00006

Kiverstein, J., Kirchhoff, M. D., & Thacker, M. (2022). An Embodied Predictive Processing Theory of Pain Experience. *Review of Philosophy and Psychology*. https://doi.org/10.1007/s13164-022-00616-2

Kloet, R. D. E. (2012). *Stress Hormones and Post Traumatic Stress Disorder: Basic Studies and Clinical Perspectives*. Elsevier Science.

Kolaitis, G., & Olff, M. (2017). Psychotraumatology in Greece. *European Journal of Psychotraumatology*, *8*(sup4). https://doi.org/10.1080/20008198.2017.1351757

Kolk, V. B. der, MD. (2015). *The Body Keeps the Score: Brain, Mind, and Body in the Healing of Trauma* (Reprint). Penguin Publishing Group.

Körding, K. (2007). Decision Theory: What “Should” the Nervous System Do? *Science*, *318*(5850), 606–610. https://doi.org/10.1126/science.1142998

Kube, T., Berg, M., Kleim, B., & Herzog, P. (2020). Rethinking post-traumatic stress disorder – A predictive processing perspective. *Neuroscience &Amp; Biobehavioral Reviews*, *113*, 448–460. https://doi.org/10.1016/j.neubiorev.2020.04.014

Kudielka, B. M., & Kirschbaum, C. (2005). Sex differences in HPA axis responses to stress: a review. *Biological Psychology*, *69*(1), 113–132. https://doi.org/10.1016/j.biopsycho.2004.11.009

Kühn, S., & Gallinat, J. (2014). Brain Structure and Functional Connectivity Associated with Pornography Consumption. *JAMA Psychiatry*, *71*(7), 827. https://doi.org/10.1001/jamapsychiatry.2014.93

Lakoff, G., & Johnson, M. (2003). *Metaphors We Live By* (1st ed.). University of Chicago Press.

LeDoux, J. (2012). Rethinking the Emotional Brain. *Neuron*, *73*(4), 653–676. https://doi.org/10.1016/j.neuron.2012.02.004

LeDoux, J. E. (1993). Emotional memory systems in the brain. *Behavioural Brain Research*, *58*(1–2), 69–79. https://doi.org/10.1016/0166-4328(93)90091-4

Lee, Y. C., Agnew-Blais, J., Malspeis, S., Keyes, K., Costenbader, K., Kubzansky, L. D., Roberts, A. L., Koenen, K. C., & Karlson, E. W. (2016). Post-Traumatic Stress Disorder and Risk for Incident Rheumatoid Arthritis. *Arthritis Care &Amp; Research*, *68*(3), 292–298. https://doi.org/10.1002/acr.22683

Letkiewicz, A. M., Cochran, A. L., & Cisler, J. M. (2022). Frontoparietal network activity during model-based reinforcement learning updates is reduced among adolescents with severe sexual abuse. *Journal of Psychiatric Research*, *145*, 256–262. https://doi.org/10.1016/j.jpsychires.2020.10.046

Levine, P. A. (1997). Waking the tiger - healing trauma - the innate capacity to transform overwhelming experiences. *North Atlantic Books*. http://ci.nii.ac.jp/ncid/BA69201502

Levy-Gigi, E., Richter-Levin, G., Okon-Singer, H., Kéri, S., & Bonanno, G. A. (2015). The hidden price and possible benefit of repeated traumatic exposure. *Stress*, *19*(1), 1–7. https://doi.org/10.3109/10253890.2015.1113523

Linson, A., & Friston, K. (2019). Reframing PTSD for computational psychiatry with the active inference framework. *Cognitive Neuropsychiatry*, *24*(5), 347–368. https://doi.org/10.1080/13546805.2019.1665994

Liotti, G. (2004). Trauma, dissociation, and disorganized attachment: Three strands of a single braid. *Psychotherapy: Theory, Research, Practice, Training*, *41*(4), 472–486. https://doi.org/10.1037/0033-3204.41.4.472

Lu, J., & Wu, W. (2021). Cholinergic modulation of the immune system – A novel therapeutic target for myocardial inflammation. *International Immunopharmacology*, *93*, 107391. https://doi.org/10.1016/j.intimp.2021.107391

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, *10*(6), 434–445. https://doi.org/10.1038/nrn2639

Malikowska-Racia, N., & Salat, K. (2019). Recent advances in the neurobiology of posttraumatic stress disorder: A review of possible mechanisms underlying an effective pharmacotherapy. *Pharmacological Research*, *142*, 30–49. https://doi.org/10.1016/j.phrs.2019.02.001

Marshall, R. D., & Garakani, A. (2002). Psychobiology of the acute stress response and its relationship to the psychobiology of post-traumatic stress disorder. *Psychiatric Clinics of North America*, *25*(2), 385–395. https://doi.org/10.1016/s0193-953x(01)00005-3

Massazza, A., Joffe, H., Hyland, P., & Brewin, C. R. (2021). The structure of peritraumatic reactions and their relationship with PTSD among disaster survivors. *Journal of Abnormal Psychology*, *130*(3), 248–259. https://doi.org/10.1037/abn0000663

Maté, M. G. (2011). *When the Body Says No: Understanding the Stress-Disease Connection* (1st ed.). Wiley.

McCabe, P., Schneiderman, N., Field, T. M., & Wellens, R. A. (2014). *Stress, Coping, and Cardiovascular Disease (Stress and Coping Series)* (1st ed.). Psychology Press.

McEwen, B. S. (2007). Physiology and Neurobiology of Stress and Adaptation: Central Role of the Brain. *Physiological Reviews*, *87*(3), 873–904. https://doi.org/10.1152/physrev.00041.2006

McFarlane, A. C., Barton, C. A., Yehuda, R., & Wittert, G. (2011). Cortisol response to acute trauma and risk of posttraumatic stress disorder. *Psychoneuroendocrinology*, *36*(5), 720–727. https://doi.org/10.1016/j.psyneuen.2010.10.007

McLaughlin, K. A., Busso, D. S., Duys, A., Green, J. G., Alves, S., Way, M., & Sheridan, M. A. (2014). Amygdala response to negative stimuli predicts PTSD symptom onset following a terrorist attack. *Depression and Anxiety*, *31*(10), 834–842. https://doi.org/10.1002/da.22284

McTeague, L. M., Huemer, J., Carreon, D. M., Jiang, Y., Eickhoff, S. B., & Etkin, A. (2017). Identification of Common Neural Circuit Disruptions in Cognitive Control Across Psychiatric Disorders. *American Journal of Psychiatry*, *174*(7), 676–685. https://doi.org/10.1176/appi.ajp.2017.16040400

Meaney, M. J. (2001). Maternal Care, Gene Expression, and the Transmission of Individual Differences in Stress Reactivity Across Generations. *Annual Review of Neuroscience*, *24*(1), 1161–1192. https://doi.org/10.1146/annurev.neuro.24.1.1161

Medina, J. (2013). An enactivist approach to the imagination: Embodied enactments and "fictional emotions." *American Philosophical Quarterly*, *50*(3), 317–335. https://philpapers.org/rec/MEDAEA

Merleau-Ponty, M. (1980). The nature of perception. *Research in Phenomenology*, *10*(1), 9–20. https://doi.org/10.1163/156916480x00037

Merleau-Ponty, M. (2002). *Phenomenology of Perception (Routledge Classics) (Volume 85)* (2nd ed.). Routledge.

Merritt, M. (2013). Thinking-is-moving: dance, agency, and a radically enactive mind. *Phenomenology and the Cognitive Sciences*, *14*(1), 95–110. https://doi.org/10.1007/s11097-013-9314-2

Mezzacappa, E. S., Kelsey, R. M., Katkin, E. S., & Sloan, R. P. (2001). Vagal Rebound and Recovery from Psychological Stress. *Psychosomatic Medicine*, *63*(4), 650–657. https://doi.org/10.1097/00006842-200107000-00018

Miller, M., & Clark, A. (2017). Happilly entangled: prediction, emotion, and the embodied mind. *Synthese*, *195*(6), 2559–2575. https://doi.org/10.1007/s11229-017-1399-7

Misiak, B., Krefft, M., Bielawski, T., Moustafa, A. A., Sąsiadek, M. M., & Frydecka, D. (2017). Toward a unified theory of childhood trauma and psychosis: A comprehensive review of epidemiological, clinical, neuropsychological and biological findings. *Neuroscience &Amp; Biobehavioral Reviews*, *75*, 393–406. https://doi.org/10.1016/j.neubiorev.2017.02.015

Moore, S. A., & Zoellner, L. A. (2007). Overgeneral autobiographical memory and traumatic events: An evaluative review. *Psychological Bulletin*, *133*(3), 419–437. https://doi.org/10.1037/0033-2909.133.3.419

Mota, N., Tsai, J., Kirwin, P. D., Harpaz-Rotem, I., Krystal, J. H., Southwick, S. M., & Pietrzak, R. H. (2016). Late-Life Exacerbation of PTSD Symptoms in US Veterans. *The Journal of Clinical Psychiatry*, *77*(03), 348–354. https://doi.org/10.4088/jcp.15m10101

Moya, P. (2014). Habit and embodiment in Merleau-Ponty. *Frontiers in Human Neuroscience*, *8*. https://doi.org/10.3389/fnhum.2014.00542

Mur, M., Ruff, D. A., Bodurka, J., De Weerd, P., Bandettini, P. A., & Kriegeskorte, N. (2012). Categorical, Yet Graded - Single-Image Activation Profiles of Human Category-Selective Cortical Regions. *Journal of Neuroscience*, *32*(25), 8649–8662. https://doi.org/10.1523/jneurosci.2334-11.2012

Neisser, U., Boodoo, G., Bouchard, T. J., Boykin, A. W., Brody, N., Ceci, S. J., Halpern, D. F., Loehlin, J. C., Perloff, R., Sternberg, R. J., & Urbina, S. (1996). Intelligence: Knowns and unknowns. *American Psychologist*, *51*(2), 77–101. https://doi.org/10.1037/0003-066x.51.2.77

Newbold, W. R., Janet, P., & Carson, C. R. (1902). The Mental State of Hystericals. *The Philosophical Review*, *11*(6), 656. https://doi.org/10.2307/2177034

Newport, D. J., & Nemeroff, C. B. (2003). Neurobiology of Posttraumatic Stress Disorder. *Focus*, *1*(3), 313–321. https://doi.org/10.1176/foc.1.3.313

Niedenthal, P. M., Barsalou, L. W., Winkielman, P., Krauth-Gruber, S., & Ric, F. (2005). Embodiment in Attitudes, Social Perception, and Emotion. *Personality and Social Psychology Review*, *9*(3), 184–211. https://doi.org/10.1207/s15327957pspr0903_1

Orne, M. T. (2002). On the social psychology of the psychological experiment: With particular reference to demand characteristics and their implications. *Prevention &Amp; Treatment*, *5*(1). https://doi.org/10.1037/1522-3736.5.1.535a

Pavlov, V. A., & Tracey, K. J. (2012). The vagus nerve and the inflammatory reflex—linking immunity and metabolism. *Nature Reviews Endocrinology*, *8*(12), 743–754. https://doi.org/10.1038/nrendo.2012.189

Perrotta, G. (2019). Psychological Trauma: Definition, Clinical Contexts, Neural Correlations and Therapeutic Approaches Recent Discoveries. *Curr Res Psychiatry Brain Disord: CRPBD-100006*.

Perry, B. D., Pollard, R. A., Blakley, T. L., Baker, W. L., & Vigilante, D. (1995). Childhood trauma, the neurobiology of adaptation, and “use-dependent” development of the brain: How “states” become “traits.” *Infant Mental Health Journal*, *16*(4), 271–291. https://doi.org/10.1002/1097-0355(199524)16:4

Pitman, R. K., & Delahanty, D. L. (2005). Conceptually Driven Pharmacologic Approaches to Acute Trauma. *CNS Spectrums*, *10*(2), 99–106. https://doi.org/10.1017/s109285290001943x

Plotsky, P. M., & Meaney, M. J. (1993). Early, postnatal experience alters hypothalamic corticotropin-releasing factor (CRF) mRNA, median eminence CRF content and stress-induced release in adult rats. *Molecular Brain Research*, *18*(3), 195–200. https://doi.org/10.1016/0169-328x(93)90189-v

Porges, S. W. (1992). Vagal Tone: A Physiologic Marker of Stress Vulnerability. *Pediatrics*, *90*(3), 498–504. https://doi.org/10.1542/peds.90.3.498

Porges, S. W. (2001). The polyvagal theory: phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology*, *42*(2), 123–146. https://doi.org/10.1016/s0167-8760(01)00162-3

Porges, S. W. (2003). The Polyvagal Theory: phylogenetic contributions to social behavior. *Physiology &Amp; Behavior*, *79*(3), 503–513. https://doi.org/10.1016/s0031-9384(03)00156-2

Porges, S. W. (2004). Neuroception: A Subconscious System for Detecting Threats and Safety. *Zero to Three*, *24*(5), 19–24. http://frzee.com/neuroception.pdf

Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology*, *74*(2), 116–143. https://doi.org/10.1016/j.biopsycho.2006.06.009

Porges, S. W. (2009). The polyvagal theory: New insights into adaptive reactions of the autonomic nervous system. *Cleveland Clinic Journal of Medicine*, *76*(4 suppl 2), S86–S90. https://doi.org/10.3949/ccjm.76.s2.17

Porges, S. W. (2021). Cardiac vagal tone: a neurophysiological mechanism that evolved in mammals to dampen threat reactions and promote sociality. *World Psychiatry*, *20*(2), 296–298. https://doi.org/10.1002/wps.20871

Rauch, S. L., Savage, C. R., Alpert, N. M., Fischman, A. J., & Jenike, M. A. (1997). The functional neuroanatomy of anxiety: a study of three disorders using positron emission tomography and symptom provocation. *Biological Psychiatry*, *42*(6), 446–452. https://doi.org/10.1016/s0006-3223(97)00145-5

Resick, P. A., & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology*, *60*(5), 748–756. https://doi.org/10.1037/0022-006x.60.5.748

Rizvi, S. L., Vogt, D. S., & Resick, P. A. (2009). Cognitive and affective predictors of treatment outcome in cognitive processing therapy and prolonged exposure for posttraumatic stress disorder. *Behaviour Research and Therapy*, *47*(9), 737–743. https://doi.org/10.1016/j.brat.2009.06.003

Roozendaal, B., Okuda, S., de Quervain, D. F., & McGaugh, J. (2006). Glucocorticoids interact with emotion-induced noradrenergic activation in influencing different memory functions. *Neuroscience*, *138*(3), 901–910. https://doi.org/10.1016/j.neuroscience.2005.07.049

Rosenkranz, R., Weber, C., & Rosenkranz, S. (2010). Validity of the Actical accelerometer step count function in children. *Journal of Science and Medicine in Sport*, *13*, e97. https://doi.org/10.1016/j.jsams.2010.10.667

Ross, M. C., Lenow, J. K., Kilts, C. D., & Cisler, J. M. (2018). Altered neural encoding of prediction errors in assault-related posttraumatic stress disorder. *Journal of Psychiatric Research*, *103*, 83–90. https://doi.org/10.1016/j.jpsychires.2018.05.008

Sahar, T., Shalev, A. Y., & Porges, S. W. (2001). Vagal modulation of responses to mental challenge in posttraumatic stress disorder. *Biological Psychiatry*, *49*(7), 637–643. https://doi.org/10.1016/s0006-3223(00)01045-3

Sankowski, R., Mader, S., & ValdÃ©s-Ferrer, S. I. (2015). Systemic Inflammation and the Brain: Novel Roles of Genetic, Molecular, and Environmental Cues as Drivers of Neurodegeneration. *Frontiers in Cellular Neuroscience*, *9*. https://doi.org/10.3389/fncel.2015.00028

Schenkel, L. S., Spaulding, W. D., DiLillo, D., & Silverstein, S. M. (2005). Histories of childhood maltreatment in schizophrenia: Relationships with premorbid

functioning, symptomatology, and cognitive deficits. *Schizophrenia Research*, *76*(2–3), 273–286. https://doi.org/10.1016/j.schres.2005.03.003
Schiepers, O. J., Wichers, M. C., & Maes, M. (2005). Cytokines and major depression. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *29*(2), 201–217. https://doi.org/10.1016/j.pnpbp.2004.11.003
Schmidt, U., Kaltwasser, S. F., & Wotjak, C. T. (2013). Biomarkers in Posttraumatic Stress Disorder: Overview and Implications for Future Research. *Disease Markers*, *35*, 43–54. https://doi.org/10.1155/2013/835876
Schore, A. N. (2003). *Affect Regulation and The Repair Of The Self*. W. W. Norton.
Schumm, J. A., Dickstein, B. D., Walter, K. H., Owens, G. P., & Chard, K. M. (2015). Changes in posttraumatic cognitions predict changes in posttraumatic stress disorder symptoms during cognitive processing therapy. *Journal of Consulting and Clinical Psychology*, *83*(6), 1161–1166. https://doi.org/10.1037/ccp0000040
Segerstrom, S. C., & Miller, G. E. (2004). Psychological Stress and the Human Immune System: A Meta-Analytic Study of 30 Years of Inquiry. *Psychological Bulletin*, *130*(4), 601–630. https://doi.org/10.1037/0033-2909.130.4.601
Shannon, C., Douse, K., McCusker, C., Feeney, L., Barrett, S., & Mulholland, C. (2009). The Association Between Childhood Trauma and Memory Functioning in Schizophrenia. *Schizophrenia Bulletin*, *37*(3), 531–537. https://doi.org/10.1093/schbul/sbp096
Shapiro, L. (2019). *Embodied Cognition (New Problems of Philosophy)* (2nd ed.). Routledge.
Sherin, J. E., & Nemeroff, C. B. (2011). Post-traumatic stress disorder: the neurobiological impact of psychological trauma. *Dialogues in Clinical Neuroscience*, *13*(3), 263–278. https://doi.org/10.31887/dcns.2011.13.2/jsherin
Siegel, D. J. (1999). The Developing Mind: Toward a Neurobiology of Interpersonal Experience. *Guilford Press EBooks*. http://ci.nii.ac.jp/ncid/BA4190950X
Siegel, D. J. (2012). The developing mind: how relationships and the brain interact to shape who we are. *Choice Reviews Online*, *50*(02), 50–1164. https://doi.org/10.5860/choice.50-1164
Silver, M., Lakoff, G., & Johnson, M. (1982). Metaphors We Live by. *Leonardo*, *15*(4), 323. https://doi.org/10.2307/1574760

Slatman, J. (2014). Multiple dimensions of embodiment in medical practices. *Medicine, Health Care and Philosophy*, *17*(4), 549–557. https://doi.org/10.1007/s11019-014-9544-2

Sorrells, S. F., & Sapolsky, R. M. (2007). An inflammatory review of glucocorticoid actions in the CNS. *Brain, Behavior, and Immunity*, *21*(3), 259–272. https://doi.org/10.1016/j.bbi.2006.11.006

Southwick, S. M. (1993). Abnormal Noradrenergic Function in Posttraumatic Stress Disorder. *Archives of General Psychiatry*, *50*(4), 266. https://doi.org/10.1001/archpsyc.1993.01820160036003

Sperlagh, B., & Sylvester Vizi, E. (2011). The Role of Extracellular Adenosine in Chemical Neurotransmission in the Hippocampus and Basal Ganglia: Pharmacological and Clinical Aspects. *Current Topics in Medicinal Chemistry*, *11*(8), 1034–1046. https://doi.org/10.2174/156802611795347564

Stahl, S. M., & Wise, D. D. (2008). The Potential Role of a Corticotropin-Releasing Factor Receptor-1 Antagonist in Psychiatric Disorders. *CNS Spectrums*, *13*(6), 467–483. https://doi.org/10.1017/s1092852900016709

Stanton, M. E., Gutierrez, Y. R., & Levine, S. (1988). Maternal deprivation potentiates pituitary-adrenal stress responses in infant rats. *Behavioral Neuroscience*, *102*(5), 692–700. https://doi.org/10.1037/0735-7044.102.5.692

Stenager, E., Knudsen, L., & Jensen, K. (1991). Acute and chronic pain syndromes in multiple sclerosis. *Acta Neurologica Scandinavica*, *84*(3), 197–200. https://doi.org/10.1111/j.1600-0404.1991.tb04937.x

Sternberg, E. M., Hill, J. M., Chrousos, G. P., Kamilaris, T., Listwak, S. J., Gold, P. W., & Wilder, R. L. (1989). Inflammatory mediator-induced hypothalamic-pituitary-adrenal axis activation is defective in streptococcal cell wall arthritis-susceptible Lewis rats. *Proceedings of the National Academy of Sciences*, *86*(7), 2374–2378. https://doi.org/10.1073/pnas.86.7.2374

Stoliarova, O. (2016). MiIlieu, Embodiment, and Cultural Studies of Science. *Epistemology &Amp; Philosophy of Science*, *47*(1), 78–83. https://doi.org/10.5840/eps201647133

Straub, R. H. (2014). Stress in RA: a trigger of proinflammatory pathways? *Nature Reviews Rheumatology*, *10*(9), 516–518. https://doi.org/10.1038/nrrheum.2014.110

Straub, R. H., Bijlsma, J. W., Masi, A., & Cutolo, M. (2013). Role of neuroendocrine and neuroimmune mechanisms in chronic inflammatory rheumatic diseases—The 10-year update. *Seminars in Arthritis and Rheumatism*, *43*(3), 392–404. https://doi.org/10.1016/j.semarthrit.2013.04.008

Straub, R. H., & Cutolo, M. (2017). Psychoneuroimmunology—developments in stress research. *Wiener Medizinische Wochenschrift*, *168*(3–4), 76–84. https://doi.org/10.1007/s10354-017-0574-2

Straub, R. H., Glück, T., Cutolo, M., Georgi, J., Helmke, K., Schölmerich, J., Vaith, P., & Lang, B. (2000). The adrenal steroid status in relation to inflammatory cytokines (interleukin-6 and tumour necrosis factor) in polymyalgia rheumatica. *Rheumatology*, *39*(6), 624–631. https://doi.org/10.1093/rheumatology/39.6.624

Streeck-Fischer, A., & van der Kolk, B. A. (2000). Down will Come Baby, Cradle and All: Diagnostic and Therapeutic Implications of Chronic Trauma on Child Development. *Australian &Amp; New Zealand Journal of Psychiatry*, *34*(6), 903–918. https://doi.org/10.1080/000486700265

Stress hormones protect against traumatic disorder. (2012). *Nature India*. https://doi.org/10.1038/nindia.2012.144

Talero, M. (2005). Perception, Normativity, and Selfhood in Merleau-Ponty: The Spatial 'Level' and Existential Space. *The Southern Journal of Philosophy*, *43*(3), 443–461. https://doi.org/10.1111/j.2041-6962.2005.tb01962.x

Tateya, S., Kim, F., & Tamori, Y. (2013). Recent Advances in Obesity-Induced Inflammation and Insulin Resistance. *Frontiers in Endocrinology*, *4*. https://doi.org/10.3389/fendo.2013.00093

Thagard, P. (2012). *The Brain and the Meaning of Life* (Reprint). Princeton University Press.

Tian, N., Moore, R. S., Braddy, S., Rose, R. A., Gu, J. W., Hughson, M. D., & Manning, R. D. (2007). Interactions between oxidative stress and inflammation in salt-sensitive hypertension. *American Journal of Physiology-Heart and Circulatory Physiology*, *293*(6), H3388–H3395. https://doi.org/10.1152/ajpheart.00981.2007

Tolman, E. C., Ritchie, B. F., & Kalish, D. (1992). Studies in spatial learning. I. Orientation and the short-cut. *Journal of Experimental Psychology: General*, *121*(4), 429–434. https://doi.org/10.1037/0096-3445.121.4.429

Tracey, K. J. (2007). Physiology and immunology of the cholinergic antiinflammatory pathway. *Journal of Clinical Investigation*, *117*(2), 289–296. https://doi.org/10.1172/jci30555

Ursin, H., & Eriksen, H. R. (2010). Cognitive activation theory of stress (CATS). *Neuroscience &Amp; Biobehavioral Reviews*, *34*(6), 877–881. https://doi.org/10.1016/j.neubiorev.2009.03.001

User:BrianMSweis - Wikimedia Commons. (2012, December 27). Retrieved January 8, 2023, from https://commons.wikimedia.org/wiki/User:BrianMSweis

van der Kolk, B. (2000). Posttraumatic stress disorder and the nature of trauma. *Dialogues in Clinical Neuroscience*, *2*(1), 7–22. https://doi.org/10.31887/dcns.2000.2.1/bvdkolk

van der Kolk, B. A. (2003). The neurobiology of childhood trauma and abuse. *Child and Adolescent Psychiatric Clinics of North America*, *12*(2), 293–317. https://doi.org/10.1016/s1056-4993(03)00003-8

van der Kolk, B. A., Brown, P., & van der Hart, O. (1989). Pierre Janet on post-traumatic stress. *Journal of Traumatic Stress*, *2*(4), 365–378. https://doi.org/10.1002/jts.2490020403

van Der Kolk, B. A., & Fisler, R. (1995). Dissociation and the fragmentary nature of traumatic memories: Overview and exploratory study. *Journal of Traumatic Stress*, *8*(4), 505–525. https://doi.org/10.1002/jts.2490080402

Varela, F. J., Thompson, E., Rosch, E., & Kabat-Zinn, J. (2017). *The Embodied Mind, revised edition: Cognitive Science and Human Experience (The MIT Press)* (revised edition). The MIT Press.

Vermetten, E., & Bremner, J. D. (2002). Circuits and systems in stress. I. Preclinical studies. *Depression and Anxiety*, *15*(3), 126–147. https://doi.org/10.1002/da.10016

Walker, J. G., Littlejohn, G. O., McMurray, N. E., & Cutolo, M. (1999). Stress system response and rheumatoid arthritis: a multilevel approach. *Rheumatology*, *38*(11), 1050–1057. https://doi.org/10.1093/rheumatology/38.11.1050

Weber, D. A., & Reynolds, C. R. (2004). Clinical Perspectives on Neurobiological Effects of Psychological Trauma. *Neuropsychology Review*, *14*(2), 115–129. https://doi.org/10.1023/b:nerv.0000028082.13778.14

Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., & Jenike, M. A. (1998). Masked Presentations of Emotional Facial Expressions Modulate Amygdala Activity without Explicit Knowledge. *The Journal of Neuroscience*, *18*(1), 411–418. https://doi.org/10.1523/jneurosci.18-01-00411.1998

Wiener, S. G., Lowe, E. L., & Levine, S. (1992). Pituitary-adrenal response to weaning in infant squirrel monkeys. *Psychobiology*, *20*(1), 65–70. https://doi.org/10.3758/bf03327163

Wild, J., Warnock-Parkes, E., Grey, N., Stott, R., Wiedemann, M., Canvin, L., Rankin, H., Shepherd, E., Forkert, A., Clark, D. M., & Ehlers, A. (2016). Internet-delivered cognitive therapy for PTSD: a development pilot series. *European Journal of Psychotraumatology*, *7*(1), 31019. https://doi.org/10.3402/ejpt.v7.31019

Wilkinson, S., Dodgson, G., & Meares, K. (2017). Predictive Processing and the Varieties of Psychological Trauma. *Frontiers in Psychology*, *8*. https://doi.org/10.3389/fpsyg.2017.01840

Williamson, J. B., Porges, E. C., Lamb, D. G., & Porges, S. W. (2015). Maladaptive autonomic regulation in PTSD accelerates physiological aging. *Frontiers in Psychology*, *5*. https://doi.org/10.3389/fpsyg.2014.01571

Wolf, O. T. (2008). The influence of stress hormones on emotional memory: Relevance for psychopathology. *Acta Psychologica*, *127*(3), 513–531. https://doi.org/10.1016/j.actpsy.2007.08.002

Yeager, D. S., & Dweck, C. S. (2012). Mindsets That Promote Resilience: When Students Believe That Personal Characteristics Can Be Developed. *Educational Psychologist*, *47*(4), 302–314. https://doi.org/10.1080/00461520.2012.722805

Yehuda, R., & LeDoux, J. (2007). Response Variation following Trauma: A Translational Neuroscience Approach to Understanding PTSD. *Neuron*, *56*(1), 19–32. https://doi.org/10.1016/j.neuron.2007.09.006

Yirmiya, R., & Goshen, I. (2011). Immune modulation of learning, memory, neural plasticity and neurogenesis. *Brain, Behavior, and Immunity*, *25*(2), 181–213. https://doi.org/10.1016/j.bbi.2010.10.015

Yoshimi, J. (2021). The Husserlian Mind. In *Phenomenological psychology as philosophy of mind* (pp. 446–458). Routledge.

Zavota, G. (2016). Expanding the Extended Mind: Merleau-Ponty's Late Ontology as Radical Enactive Cognition. *Essays in Philosophy*, *17*(2), 94–124. https://doi.org/10.7710/1526-0569.1558