The Human Body and Psychological Trauma: Biological Explanatory Models. A review

The Human Body and Psychological Trauma: Biological Explanatory Models. A review

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ABSTRACT

Background

This review focusses on biological explanatory models expounding somatic dysfunction or symptoms after psychological trauma.

Methods

A Literature search was performed in PubMed with specific search terms. Limo and Web of Science were searched, using a funnel strategy. 1.115 articles were screened, of which 79 met the predisposed relevance criteria. 10 articles met all predefined criteria, and were assessed for quality. SWOT analysis was performed on the included studies.

Results

Eight explanatory models were found: Sympatho-adrenal theory, hypothalamic-pituitary-adrenocortical axis theory, Core Response Network theory, Preparatory Set theory, Toxic Mind theory, Brain Mechanism theory, Kindling theory, and Grounded Cognition theory.

Discussion and Conclusion

The average quality of the articles was moderate. Based on quality assessment The Core Response Network theory was currently evaluated as a comprehensive biological theory, explaining somatic symptoms after psychological trauma. However, implementing elements of other models should be considered to explain specific aspects of psychosomatic phenomena in trauma.

Keywords: trauma, psychological trauma, psychosomatic, somatic complaints, theory

BACKGROUND

When Reich (1972) defined the muscular armor as the experience-dependent development of a protective shell of muscle tension grown over time in response to a history of threat, anxiety and trauma, he not only coined a fundamental entry point in what would become body psychotherapy, he also elaborated on Freud's early trauma theory (Freud & Breuer, 1893a). Here an affect-quantum is thought to move, expand, reduce or shift back and forth between the psychic and somatic realm (Verhaeghe, 2006). Since trauma is a widely-used term in scientific and popular writings, it is worthwhile to contemplate on this concept. Throughout psychiatric history, the Diagnostic and Statistical Manual of Mental Disorders (DSM) has had a substantial influence on the different meanings of the concept 'trauma' and related disorders (Friedman, 2016). The concept of *trauma* in therapeutic context evolved from a well-defined, rather rare event to a more comprehensive concept with clear nuances. For a long period of time, trauma was categorised as an event that had to precede the development of a posttraumatic stress disorder (PTSD). In the DSM-IV, trauma was still defined as: "*The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others. The person's response to the event involved intense fear, helplessness, or horror" (American Psychiatric Association, 1994).* In this way trauma seems to concern an event that inflicts damage as a physical wound or the physical integrity of an individual. If a person is witness to such events, it is also considered as trauma. At least one of these three responses -intense fear, helplessness, and/or horror- must be present.

The DSM-V (American Psychiatric Association, 2013) recently renamed PTSD under 'Trauma and stressor-related disorders' whereby trauma is defined as a catastrophic event involving actual or threatened death, serious injury, or sexual violence in one of the following ways: *(1) directly experiencing the traumatic event(s); (2) or witnessing, in person, the event(s) as it occurred to others; (3) or learning that the traumatic event(s) occurred to a close family member or close friend - in cases of actual or threatened death of a family or friend, the event(s) must have been violent or accidental; (4) or experiencing repeated or extreme exposure to aversive details of the traumatic event(s)* (American Psychiatric Association, 2013). Although this definition provides more room for differences between direct and indirect forms of trauma, it is criticized by Briere and Scott (2015). They point out that the limitation of damage to the physical integrity and live-threatening situations in the DSM-V means that events with primarily emotional impact (so not being life-threatening) cannot be considered trauma. This implicates that bullying, verbal aggression, emotional neglect or humiliation are ruled out, which presumes a significant underestimation of the incidence of trauma and PTSD in general population (Briere & Scott, 2015). To date, DSM-V does not take these factors into account making the following question highly topical: *Could psychological trauma also cause physical symptoms or complaints which are not directly linked to an injury of the physical integrity?* Intuitively and based on experience, therapists and medical doctors will answer this question affirmatively. Scientific research is convincing, indicating correlations between psychological trauma in childhood and the development of functional somatic syndromes in adulthood, such as fibromyalgia, chronic generalized pain syndrome, irritable bowel syndrome, chronic fatigue syndrome and temporomandibular syndrome

(Afari et al., 2014; Ladd et al., 1996; Mohr & Fantuzzo, 2000; Plotsky & Meaney, 1993; Smith & Flannery-Schroeder, 2014). Further relations are shown between childhood abuse and low back-pain (Leisner et al., 2014), childhood maltreatment and migraine (Tietjen et al., 2010) and disturbed pain-processing (Tesarz et al., 2016; Tesarz et al., 2015). Moreover, Mohr and Fantuzzo (2000) point at a dysregulation of the parasympathetic nervous system after exposure to domestic violence. In addition, the mediating role of alexithymia in case of emotional maltreatment in childhood is well documented (Smith & Flannery-Schroeder, 2014). Animal studies on early traumatic experiences are also multiple and show amongst other effects, changes in hormonal response as well as lowered cortisol receptors in the hippocampus and elevated corticotrophin stimulating hormone in the hypothalamus (Ladd et al., 1996; Plotsky & Meaney, 1993). Both of which can contribute to a dysregulation of the hypothalamic-pituitary-adrenal axis (Ladd et al., 1996).

Strengthened by the latter and returning to the definition of trauma, Peter Levine (1977) describes trauma from a strong biological paradigm as an event that causes a long-term dysregulation of the autonomous nervous system and the extrapyramidal nervous system, emphasising the effect on the body and not on the event itself. This definition clarifies the fact that traumatic experience for one individual does not have to be traumatic for another individual. From a totally different stance and drawing on sound reviews, Verhaeghe and Vanheule (2005) reach the same conclusion, namely trauma is a necessary but insufficient condition for the development of PTSD. Moreover, research points out that no connection can be found between the objective severity of the traumatic event and the development of psychopathology, which leads Verhaeghe & Vanheule to state that "*whether PTSD develops is not so much determined by the trauma in itself; rather, there must be mediating factors of vulnerability and resilience." (Verhaeghe & Vanheule, 2005)*. So where biological explanatory models focus on brain models, neuro-anatomic pathways and hormonal processes, psychological theories deepen on cognition, emotion, behaviour or unconscious processing (Roelofs & Spinhoven, 2007). Agreeing with the above critique on the reduction of trauma in DSM-V (Briere & Scott, 2015) and referring to the prevalence of traumatic experiences in patients with psychological complaints, the main question underlying this review is which biological explanatory models are available for understanding somatic dysfunctioning after psychological trauma?". Regarding the above, as well as the complexity in human interactions, it is obvious that psychological trauma includes non-physical events inflicting the individual integrity or emotional impact in non-life-threatening situations. Motivated by Geuter (2015) who describes how in body psychotherapy patterns of experience and behaviour express themselves cognitively, affectively, imaginative (which in fact is the psychological dimension) as well as sensorial, motoric or vegetative in therapy (the biological dimension), this article aims to add also on a more clinical level. But as in the end, the authors would like to contribute to substantiate to the daily practice of body psychotherapy, a more detailed theoretical stance as will follow is necessary.

METHODS

Literature search

Initial search in Limo, PubMed, and Web of Science did not lead to sufficient relevant articles. Therefore, a funnel strategy was used. The term ‘psychosomatic AND trauma was used in the search engines Limo and Web of Science. Three relevance criteria were formulated: mentioning psychological trauma in the title or abstract; physical complaint or symptom caused by psychological trauma; and a description of a biological theorem, explaining this relation. In PubMed, following MeSh terms were used: (war-related injuries [MeSH Terms] OR adult survivors of child adverse events [MeSH Terms] OR battered child syndrome [MeSH Terms] OR compassion fatigue [MeSH Terms] OR trauma and stressor related disorders [MeSH Terms] OR psychological trauma [MeSH Terms] OR adult survivors of child abuse [MeSH Terms] OR spouse abuse [MeSH Terms] OR elder abuse [MeSH Terms] OR child abuse [MeSH Terms] OR sex offenses [MeSH Terms] OR intimate partner violence [MeSH Terms] OR trauma [Title/Abstract]) AND (theory* [Title/Abstract] OR concept* [Title/Abstract] OR somatic). Articles published between 2000 and 2016, in English and Dutch were withheld.

Selection criteria

After the formation of the search strategy, inclusion and exclusion criteria were formulated. The articles were equally divided among the researchers and independently screened and disagreements about inclusion of studies were resolved by discussion. Inclusion criteria: psychological trauma, somatic complaint or symptom after psychological trauma, biological theorems describing the relation between psychological trauma and somatic complaints or symptoms, articles in English or Dutch, Theorems about victims of psychological trauma, psychological trauma from birth. Exclusion criteria: animal studies; studies on cellular level; the term trauma exclusively used as medical term (physical damage without psychological impact); exclusive description of somatic complaints or symptoms as a direct effect of physical trauma, with or without mentioning psychological impact; incompletely described theorems; psychological theorems; statistical models; panic attacks, dissociation symptoms, cognitive symptoms, conversion symptoms as result of psychological trauma.

Quality assessment

Since there was no questionnaire available for the evaluation of theoretical articles and narrative reviews, a self-developed questionnaire was used (Table 1). Articles were scored on twelve criteria, following a categorical format: adequate (score: plus 1), inadequate (score: plus 0), and not available (score: plus 0). On a scale of 12, scores from 0 and 4 were considered poor quality, scores from 5 to 8 moderate quality and scores from 9 to 12 high quality (Table 1). Two observational studies were assessed on quality by a standardized questionnaire, published by the Critical Appraisal Skills Programme ("Critical Appraisal Skills Programme," 2017). The articles are scored on a scale from 0 to 10, scores from 0 to 4 were considered poor quality, scores from 5 to 7 moderate quality, and scores from 8 to 10 high quality (Table 2).

Data Extraction

The aim of the data extraction was to extract all described biological theorems, explaining somatic symptoms after psychological trauma, from the included studies.

RESULTS

The funnel strategy resulted in 396 hits on Limo, and 213 hits on Web of Science. After applying the three relevance criteria on title/abstract, these numbers were reduced to respectively 20 and 13 relevant articles. The applied search terms on PubMed delivered 506 hits, which were reduced to 46 relevant articles. The 79 articles were screened in relation to the inclusion and exclusion criteria. 69 articles were excluded (Figure 1, excluded articles in appendix 1). 10 articles met all criteria (6 in PubMed, 2 in Web of Science, and 2 in Limo) and were assessed for quality. The included articles consisted of 5 theoretical articles, 2 observational studies, and 3 narrative reviews. Three articles were of high quality, four articles of moderate quality and three articles of poor quality. Strengths and weaknesses of each article were assessed via SWOT analysis (Table 3). Data extraction resulted in eight biological explanatory theories (Table 4).

Sympatho-adrenal theory

The sympatho-adrenal theory could be seen as the basis of the “fight-or-flight” theory and is also called the fast response to stress. This mechanism enables an individual to react to danger or threat but is also linked to the development of chronic complaints in patients with PTSD (Bedi & Arora, 2007; Saltzman et al., 2005). The underlying theory suggests a central role of the hypothalamus. In case of danger the hypothalamus signals the adrenal medulla, which produces (nor-) epinephrine. This results in an increase of sympathetic activity and a decrease in parasympathetic activity. Heart rate for example will increase and heart-rate-variability will decrease in patients with PTSD (Bedi & Arora, 2007; Saltzman et al., 2005). Patients also tend to get in a state of neuro-endocrine arousal much faster, which may result in panic (Perry, 1997). Traumatic experiences are shown to have a negative impact on the cardiovascular system and in turn, sympatho-adrenal hyperactivity contributes to the development of cardiovascular disease (Bedi & Arora, 2007; Rogosch et al., 2011; Saltzman et al., 2005). The elevated levels of catecholamines cause a change in function of platelets, resulting in a negative effect on vascularization and elevation in blood pressure (Goldstein, 1983).

Hypothalamic-pituitary-adrenocortical axis theory

The hypothalamic-pituitary-adrenocortical or HPA axis works slower in comparison to the sympatho-adrenal axis. The hypothalamus produces corticotropin-releasing hormone as a reaction to stress, which results in the release of adrenocorticotropic hormone in the anterior pituitary gland. In turn, this stimulates secretion of corticoid steroid cortisol from adrenal cortex cells (Bremner et al., 1999; Lamprecht & Sack, 2002). Elevated levels of cortisol aim to regulate other stress mechanisms of the hypothalamus and pituitary gland from chronic hyperactivity (Bremner et al., 1999). Saltzman

(2005) acknowledges that there is a controversy on cortisol levels measured after stress. Carrion et al. (2002) found an elevated level of cortisol in patients with PTSD, while others measured a decrease in cortisol levels in young girls after sexual abuse (King et al., 2001). Resnick et al. (1995) found a decrease in cortisol level and a weakened HPA axis response on acute trauma in women with a history of previous sexual abuse and higher levels of cortisol in women who had never been assaulted before.

Core Response Network theory

Payne, Levine and Crane-Godreau (2015) describe a theory in which experiencing a trauma or chronic stress causes a dysregulation of what they call the Core Response Network (CRN). This CRN is a subcortical network of deep regulatory systems in the central nervous system and consists of four parts: *the autonomic nervous system, the reticular arousal system, the emotional motor system, and the limbic system*.

The autonomic nervous system can intensify or calm the activity of the viscera, alter blood circulation, trigger hormonal and endocrine activity, change muscle tone, increase or decrease cognitive arousal, and contribute to emotional experience (Norman et al., 2014). The autonomic nervous system is divided to an ergotropic (energy seeking) and a trophotropic (nutrition seeking) system, which can be seen as an extension of the more traditional approach of the (para-)sympathetic nervous system described in the previous models above. The limbic system includes the amygdala, hippocampus and septal nuclei, central to fear- and pleasure and to the recall of emotion (Heimer & Van Hoesen, 2006). The reticular arousal system involves multiple networks which trigger arousal and controls alertness and orientation in different contexts. It also interfaces strongly with the other components of the CRN (Krout et al., 2002). The emotional motor system consists of the striatum, red nucleus and periaqueductal gray and is primarily involved in extrapyramidal movements such as vomiting, coughing, sneezing and breathing (Holstege, 2014) . The CRN provides a very quick and instinctive response to threatening stimuli, with little input from higher cortical structures (Porges, 2004) and enters various discrete functional and dysfunctional states (Payne et al., 2015). In general the stress response of the autonomic nervous system on stress or trauma is considered to be the trigger activating the CRN system as described by Levine (1977). Here, in mild stress reactions, the sympathetic increase and simultaneous decrease in parasympathetic activity, is followed by a parasympathetic rebound before returning to baseline. However, even in mild disturbance or chronic stress the sympathetic nervous system can keep functioning for longer periods of time on a higher level of activity, without returning to baseline. However, in extreme stress or trauma like life-threatening events, something paradoxically occurs. The initial increase in sympathetic activity is now followed by an even stronger co-activation of the parasympathetic nervous system (Gellhorn, 1964; Paton et al., 2006). This phenomenon is described as tonic immobility and occurs in humans confronted with extreme stress (Bovin et al., 2008). The clinical implications of this tonic immobility have been described by Levine as unpleasant, interoceptive and proprioceptive sensations such as stomach tension, shaking, increased or decreased blood pressure, increased or decreased heartrate, hyperventilation, cold hands and excessive sweating. In PTSD, tonic immobility can provide an

explanatory model for freezing and collapse symptoms (Halvorsen, 2014). However, a traumatic event in this theory is described in a strict biological manner as an event that causes a long-term dysregulation of the autonomic nervous system and the extrapyramidal nervous system.

Preparatory Set Theory

Payne and Crane-Godreau (2015) hypothesize that a response to a certain stimulus is always preceded by a preparatory phase or so called Preparatory Set. According to them, physical complaints after trauma are linked to processes in this preparatory phase. Generally, the reaction to a stimulus (both traumatic and non-traumatic) proceeds in three phases: *initial noticing and orienting* of the situation, *preparation* of response, and finally, *execution* of response. These three phases do not necessarily follow a chronological order. After an orienting phase, a new evaluation of the situation, can alter the initial noticing and orienting (Resulaj et al., 2009). The execution phase can take place immediately after the orienting phase, after a delay, or not at all. Proprioceptive and exteroceptive feedback will inform the organism about the successful completion of the action, following which a new preparatory set may form. (Gellhorn & Hyde, 1953; Payne & Crane-Godreau, 2015; Suetterlin & Sayer, 2014).

Payne and Crane-Godreau describe different kinds of Preparatory Sets, depending on the situation for which preparation is made, for example the digestion of food or sports. In normal circumstances, these are adequately adapted to handle a situation or stimulus. However, a Preparatory Set can also be maladaptive as a response to a stimulus if the preparation was not adapted, but exaggerated for the situation. This could lead to a Preparatory Set that keeps on going, even long after the actions have been taken and mostly when the situation has passed. Disorganization of the Preparatory Set occurs when trophotropic and ergotropic reactions keep competing. This status equals the earlier described tonic immobility, of which the primary signs are physical immobility and muscular rigidity (Bovin et al., 2008; Bovin et al., 2014). Under normal circumstances an adaptive Preparatory Set is followed by an adequate reaction, which solves the situation and the state of alertness can be interrupted by the individual. In a mild stress situation, a normal increase of ergotropic arousal and a simultaneous decrease in trophotropic activity occurs. This is followed by a trophotropic rebound, and the ergotropic activity returns to baseline (Gellhorn, 1956). If situations do not get resolved, despite an adequate action, the Preparatory Set stays active and possibly becomes overwhelming for the individual. If this situation keeps persisting and the Preparatory Set gets disorganized (meaning a co-activation of the ergotropic and trophotropic system), the individual has a high probability of developing a PTSD after the situation (Bovin et al., 2008).

Toxic Mind Theory

The Toxic Mind theory (Van Winkle, 2000) describes a process in which repetitive suppression of emotions during fight-or-flight reactions in traumatic experiences results in atrophy and endogenous toxicosis in noradrenergic neurons. This can occur in early childhood trauma or experiencing trauma in later years. When thoughts and emotions are repetitively suppressed, nerve impulses through

noradrenergic neurons are diminished. This leads to a decrease in the concentration of synaptic norepinephrine - associated with symptoms of depression -, and atrophy of the noradrenergic neurons. Atrophy leads to an accumulation of metabolic waste products (endogenous toxicosis) in the cytoplasm. The Toxic Mind Theory assumes that each neuron has the inherent ability to initiate a detoxification process. This process is initiated through high levels of intracellular toxins and is called a 'detoxification crisis'. The increase of synaptic norepinephrine leads to overexcitement of postsynaptic neurons, causing excitatory symptoms. Excitatory symptoms that are sympathetically controlled and are fight or flight reactions, range from mild anxiety to violent behaviour. Ending the detoxification process occurs with an adaptive response to the excess norepinephrine in the synapse. Finally, excess endogenous toxins (which diffuse together with norepinephrine into the synapse) cause a saturation of postsynaptic receptors for norepinephrine. Unless the detoxification crisis is completed, it is likely to be followed by or relapse of depression. Saturation of noradrenergic receptors by endogenous toxins cause a change in neural transmission, which might lead to disturbed and compulsive thoughts, hallucinations, psychoses, and unwanted behaviour. Rage can be transformed into aggression, and sorrow into suicidal acts (Van Winkle, 2000).

Detoxification is an autonomic starting, self-limiting process. If detoxification occurs adequately, the pre- and post-synaptic neuron can be restored and the symptoms disappear. However, in case of inadequate detoxification or persistent suppression of emotions, inhibitory complaints arise, followed by a new periodical detoxification. Toxins accumulating in brain regions with a specific function, will generate symptoms related to those functions.

This theory provides a possible explanation of the transition between inhibitory and excitatory symptoms, which are observed in psychological, neurological, and psychosomatic disorders, but just equally in individuals without clinical pathology. If symptoms do not derive from organic damage, according to this theory they derive from an attempt of the nervous system to detoxify and can only be cured by treating the real cause (the cause of the toxification). This theory could explain physical complaints after psychological trauma with (repetitive) suppression of emotions.

Brain Mechanisms Theory

Atarodi and Hosier (2011) describe the initial brain mechanisms that are activated in emotional traumatization and consolidation of its memory. These consist of two important components: firstly, *amygdalic nuclei* located in the medial temporal lobe and secondly, *catecholamine neurotransmitters*, epinephrine, and norepinephrine. The process starts with an arousal-based input from sensory organs, which is lead to the thalamus and locus ceruleus. The locus ceruleus releases norepinephrine that enables the lateral nucleus of amygdala to evaluate the emotional value of the input. This is the start of encoding the trauma for consolidation in the memory. The lateral nucleus forwards a message to basolateral nucleus of amygdala. This basolateral nucleus has connections with other areas of the brain and this enables it to encode memory pathways and organize emotions, somatosensory processes, and motor action caused by the trauma (Atarodi & Hosier, 2011). The basolateral nucleus of amygdala sends signals to the central nucleus of amygdala. The central nucleus of amygdala produces fear and evokes the sympathetic nervous system, the HPA axis, the nucleus accumbens,

the hippocampus, and the prefrontal cortex. The nucleus accumbens creates motivation to action and the sympathetic nervous system initiates the fight or flight reaction (Ruden, 2005). Experiencing pain as a physiologic response to actual or potential tissue damage is called nociception. The same brain structures are involved in experiencing psychogenic and organic pain, but psychogenic pain has no sensory input (Bob, 2008). The central nucleus of amygdala has a nociceptive area that has an important role in the experience of psychogenic pain (Meeus & Nijs, 2007). The modulators of the central nucleus amygdala are the prefrontal cortex and the anterior cingulate cortex (Scaer, 2001). In threatening situations, the prefrontal cortex inhibits emotional processing of the limbic system. As a result, the individual can act in the best manner and gives the best response. However, if an inadequate response occurs, the individual will be traumatized (Lewis, 2007) and the prefrontal cortex which has a controlling effect on nociceptive stimuli, causes the formation of psychogenic pain (Bob, 2008).

Kindling Theory

The Kindling theory is used to explain the formation of chronic psychogenic pain (Rome & Rome, 2000), but is also linked to general psychosomatic symptoms following trauma (Atarodi & Hosier, 2011). The theory states that repetitive, low-level electrical or subthreshold stimuli of the brain could lead to changes in neural response. After a while, repeated stimulation causes increased neuronal excitability of the brain, because repeated stimulation can alter the function of the neural membrane, the synaptic activity, and the intracellular communication via neuroplastic mechanisms. The Kindling theory consists of three phases. *The developmental phase* in which repetitive electrical stimuli of a brain area exceed the threshold, resulting in focal neuronal activity and discharge. *The completed phase* in which any stimulation (subthreshold or threshold) causes a discharge. Finally, *the spontaneous phase* in which discharge takes place without any external stimulus (Atarodi & Hosier, 2011). The limbic system (including the amygdala) is a part of the brain susceptible to kindling (Adamec, 1990). Chronic pain and psychosomatic disorders develop through plastic changes via kindling in the limbic system and other supraspinal parts of the central nervous system. The development of psychogenic pain is described as follows: following trauma, the traumatic facts are stored in the memory via the lateral nucleus amygdala and the basolateral nucleus amygdala. Via kindling, the neural threshold is lowered for the processes, like the amygdala, that cause psychogenic pain, causing stimuli that are related to the trauma that can lead to psychogenic pain (Scaer, 2001).

Grounded Cognition Theory

Weston (2014) proposes the Grounded Cognition Theory to explain how traumatic events lead to the development of complaints or symptoms in the hyperarousal type of PTSD. The Grounded Cognition theory suggests that brain areas precipitating motor, sensory or other stimuli, can also mediate enduring representations of those stimuli in higher-level brain areas. For example, the sensory system is active in the registration of sensory stimuli and the storage in sensory centres. The sensory system is later reactivated when the corresponding memory representation is retrieved. This results in the fact that in case of reliving traumatic stimuli, a state of arousal is reached faster

(Barsalou et al., 2003; Meyer & Damasio, 2009). The amygdala plays a key role and is connected to the network that regulates sleep pattern and alertness and contains the midbrain, reticular formation, hypothalamus, solitary tract nucleus and the brainstem. Because of the amygdala's connection with these areas, the amygdala is anatomically positioned to influence sleep-wake regulation. The hypothesis is that this overactivity of the amygdala causes sleep disturbances in PTSD (Germain et al., 2008). Pain is also processed in the amygdala (Han & Neugebauer, 2004) and can cause pain syndromes in PTSD (Weston, 2014). The activity in the amygdala, inflicted by pain, causes a consolidation of nociceptive stimuli and representations of pain that contribute to a disturbed representation of pain in the brain, which leads to psychogenic pain in hyperarousal subtype of PTSD (Weston, 2014).

DISCUSSION AND CONCLUSION

The quality of the articles discussed in this review were moderate to high for the HPA axis and sympatho-adrenal theory, moderate for the Core Respons Network, Brain Mechanisms, Kindling, and Grounded Cognition theory, poor for the Preparatory Set theory and Toxic Mind theory, Although differing on several levels, these theories can be regarded as corresponding or complementary. The HPA axis and the sympatho-adrenal theory, describe the acute reactions of the nervous system and hormonal system on a traumatic event. Increased sympathetic activity was linked to cardio-vascular disease, increased rest heart-rate, decrease in heart-rate variability, and faster reaching a state of arousal. The HPA axis also provides an explanatory model for post-traumatic physical complaints or symptoms. Both theories focus on the basal level of regulation, without involving higher brain structures. The complaints of a traumatic patient are strongly stress related and sympathetically tuned. In that way these theories can be regarded as a peripheral expression of higher-order central processes as discussed by Atarodi and Hosier (2011). In their Brain Mechanism theory, brain structures are described which are activated as a reaction to trauma and account for the activation of previously mentioned acute responses. The Grounded Cognition theory (Weston, 2014) states that traumatic experiences can exert an effect on specific areas of the brain. The nature of the symptoms depends on the areas of the brain in which the effect occurs. The disadvantage of this theory is the fact that it was only described for the hyperarousal type PSTD and therefore the findings cannot be generalized. This region specificity also returns in the Toxic Mind theory (Van Winkle, 2000). Both theories describe changes in the brain after traumatic events, although the Grounded Cognition focuses on cognition while the Toxic Mind zooms in on the underlying biological substrate, namely a structural change based on toxicosis of specific noradrenergic neurons. So, both Toxic Mind and Kindling theory describe processes that take place on inter- and intraneuronal level, but with a different physiological mechanism (toxification versus neuroplasticity). It is interesting that the Toxic Mind theory tries to explain the periodicity of complaints or symptoms by means of an autonomic starting and self-limiting process, initiated by a natural response of a neuron on toxification. Not the traumatic event itself causes the toxification, but the suppression of emotions during the traumatic event. In this, the Kindling theory differs. It states that the event and associated stimuli are the cause of kindling and cause a lower threshold for complaints or symptoms. In the Toxic Mind theory

complaints and symptoms are described as part of an automated process, whereby in Kindling Theory, complaints and symptoms develop after experiencing trauma related stimuli (with exception of the spontaneous phase, where these can occur without external stimuli). It is the theory of Brain Mechanisms (Atarodi & Hosier, 2011) that highlights an important concept: consolidation and memory. Like the Grounded Cognition theory, traumatic stimuli are projected to specific brain areas. But in Atarodi and Hosier's theory, a mechanism is described how stimuli can cause long-lasting effects on psychogenic pain. The Grounded Cognition theory also describes this in the development of pain in the hyperarousal subtype of PTSD. Interesting is the description that the specific brain areas, responsible for memories of traumatic experiences, also contribute directly to the basal stress reaction mechanisms such as the HPA-axis and the sympatho-adrenal theory and describes this as a physiological reaction on the experience. On the one hand, these brain structures cause consolidation of the trauma, leading to complaints in the long run, on the other hand, they provide a direct physiologic response to a traumatic event. Structurally the limbic system, including the amygdala, delivers an important contribution to the different theories. Both in general trauma patients and in hyperarousal PTSD patients this neural circuit plays an important role. The limbic system also plays a role in the Core Response Network theory and Preparatory Set Theory, although the specific role of the amygdala is not specifically described in the development of complaints or symptoms. In these models, symptoms are understood from two angles. On the one hand they are considered as a direct effect from stagnation in a disorganized state of arousal (Payne & Crane-Godreau, 2015), on the other hand, they are seen as a dysregulation of the Core Response Network (Payne et al., 2015). Both angles converge in the mechanism of tonic immobility. This paradoxical increase in both sympathetic and parasympathetic activity occurs in extreme stress, trauma or life-threatening events. In both theories, clinical symptoms of tonic immobility such as unpleasant, interoceptive and proprioceptive sensations like stomach tension, shaking, increased or decreased blood pressure, increased or decreased heartrate, hyperventilation, cold hands and excessive sweating are described.

Based on the implemented quality assessment two contiguous theories - the Core Response Network and the Preparatory Set - provide a most comprehensive biological model, explaining somatic symptoms after psychological trauma. However, additional and more detailed neurophysiological insight in trauma mechanisms is offered by other of the discussed explanatory theories, such as the Toxic Mind Theory, Kindling Theory and Brain Mechanism Theory. Concerning a more clinical implication, this review provides further ground to work with trauma patients from an integrated perspective, such as body psychotherapy does. It weighs existing biological models making somatic dysfunction after psychological trauma more understandable. Therefore it appeals to a more profound and extended approach of trauma whereby *"trauma memory is as much in the sensory receptors, in the skin and in the muscles as it is in the brain.*" (Fogel, 2009). Finally and similarly, this article tries to add on a more conceptual level, to the need of a trans-disciplinary view on trauma whereby trans-disciplinarity has been described as a practice that transgresses and transcends disciplinary boundaries and seems to have the most potential to respond to new demands and imperatives (Russell et al., 2008). Although further clinical research is indispensable, it seems that from the early

days of Reichs' body armor, body psychotherapy established itself as a genuine trans-disciplinary approach on trauma.

BIOGRAPHIES

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REFERENCES

Adamec, R. E. (1990). Does kindling model anything clinically relevant? *Biol Psychiatry, 27*(3), 249-279.

Afari, N., Ahumada, S. M., Wright, L. J., Mostoufi, S., Golnari, G., Reis, V., & Cuneo, J. G. (2014). Psychological trauma and functional somatic syndromes: a systematic review and meta-analysis. *Psychosom Med, 76*(1), 2-11. doi:10.1097/PSY.0000000000000010

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders* (Fourth ed.). Washington, DC.

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Disorders* (Fifth ed.). Arlington, VA.

Atarodi, N., & Hosier, S. (2011). Trauma in the Mind and Pain in the Body: Mind-Body Interactions in Psychogenic Pain. *Human Architecture: Journal of the Sociology of Self-Knowledge, 9*(1), 111-131.

Barsalou, L. W., Kyle Simmons, W., Barbey, A. K., & Wilson, C. D. (2003). Grounding conceptual knowledge in modality-specific systems. *Trends Cogn Sci, 7*(2), 84-91.

Bedi, U. S., & Arora, R. (2007). Cardiovascular manifestations of posttraumatic stress disorder. *J Natl Med Assoc, 99*(6), 642-649.

Bob, P. (2008). Pain, dissociation and subliminal self-representations. *Conscious Cogn, 17*(1), 355-369. doi:10.1016/j.concog.2007.12.001

Bovin, M. J., Jager-Hyman, S., Gold, S. D., Marx, B. P., & Sloan, D. M. (2008). Tonic immobility mediates the influence of peritraumatic fear and perceived inescapability on posttraumatic stress symptom severity among sexual assault survivors. *J Trauma Stress, 21*(4), 402-409. doi:10.1002/jts.20354

Bovin, M. J., Ratchford, E., & Marx, B. P. (2014). Peritraumatic dissociation and tonic immobility:clinical findings. In F. Ulrich, U. Lanius, S. Paulsen, & F. Corrigan (Eds.), *Neurobiology and Treatment of Traumatic Dissociation: Towards an Embodied Self* (pp. 51-67). New York, NY: Springer.

Bremner, J. D., Southwick, S. M., & Charney, D. S. (1999). *The neurobiology of posttraumatic stressdisorder: A n intergration of animal and human research*. Needham Heights, MA: Allyn & Bacon.

Briere, J., & Scott, C. (2015). *Principles of trauma therapy: A guide to symptoms, evaluation, and treatment (DSM-5 Update)* (Second Edition ed.). Washington, DC: SAGE Publications, Inc.

Carrion, V. G., Weems, C. F., Ray, R. D., Glaser, B., Hessl, D., & Reiss, A. L. (2002). Diurnal salivary cortisol in pediatric posttraumatic stress disorder. *Biol Psychiatry, 51*(7), 575-582.

Critical Appraisal Skills Programme. (2017). Retrieved from http://www.casp-uk.net/checklists

Fogel, A. (2009). *The psychology of self-awareness*. New York: W.W. Norton.

Freud, S., & Breuer, J. (1893a). Über den psychischen Mechanismus hysterische Phänomene, Vorläufe Mitteilung. *Neurologisches Zentralblatt, 12*.

Friedman, M. J. (2016). *PTSD History and Overview*. Retrieved from https://www.ptsd.va.gov/professional/ptsd-overview/ptsd-overview.asp.

Gellhorn, E. (1956). Analysis of autonomic hypothalamic functions in the intact organism. *Neurology, 6*(5), 335-343.

Gellhorn, E. (1964). Cardiovascular Reactions in Asphyxia and the Postasphyxial State. *Am Heart J, 67*, 73-80.

Gellhorn, E., & Hyde, J. (1953). Influence of proprioception on map of cortical responses. *J Physiol, 122*(2), 371-385.

Germain, A., Buysse, D. J., & Nofzinger, E. (2008). Sleep-specific mechanisms underlying posttraumatic stress disorder: integrative review and neurobiological hypotheses. *Sleep Med Rev, 12*(3), 185-195. doi:10.1016/j.smrv.2007.09.003

Geuter, U. (2015). *Körperpsychotherapie: Grundriss einer Theorie für die klinische Praxis* (1 ed.). Berlin: Springer.

Goldstein, D. S. (1983). Plasma catecholamines and essential hypertension. An analytical review. *Hypertension, 5*(1), 86-99.

Halvorsen, L. A. (2014). *Understanding Peritraumatic Dissociation: Evolution-Prepared Dissociation, Tonic Immobility, and Clinical Dissociation.* (Ph.D.), Antioch University, New England.

Han, J. S., & Neugebauer, V. (2004). Synaptic plasticity in the amygdala in a visceral pain model in rats. *Neurosci Lett, 361*(1-3), 254-257. doi:10.1016/j.neulet.2003.12.027

Heimer, L., & Van Hoesen, G. W. (2006). The limbic lobe and its output channels: implications for emotional functions and adaptive behavior. *Neurosci Biobehav Rev, 30*(2), 126-147. doi:10.1016/j.neubiorev.2005.06.006

Holstege, G. (2014). The periaqueductal gray controls brainstem emotional motor systems including respiration. *Prog Brain Res, 209*, 379-405. doi:10.1016/B978-0-444-63274-6.00020-5

King, J. A., Mandansky, D., King, S., Fletcher, K. E., & Brewer, J. (2001). Early sexual abuse and low cortisol. *Psychiatry Clin Neurosci, 55*(1), 71-74. doi:10.1046/j.1440-1819.2001.00787.x

Krout, K. E., Belzer, R. E., & Loewy, A. D. (2002). Brainstem projections to midline and intralaminar thalamic nuclei of the rat. *J Comp Neurol, 448*(1), 53-101. doi:10.1002/cne.10236

Ladd, C. O., Owens, M. J., & Nemeroff, C. B. (1996). Persistent changes in corticotropin-releasing factor neuronal systems induced by maternal deprivation. *Endocrinology, 137*(4), 1212-1218. doi:10.1210/endo.137.4.8625891

Lamprecht, F., & Sack, M. (2002). Posttraumatic stress disorder revisited. *Psychosom Med, 64*(2), 222-237.

Leisner, S., Gerhardt, A., Tesarz, J., Janke, S., Seidler, G. H., & Eich, W. (2014). [Childhood abuse experiences and chronic low back pain. Direct and mediated effects of childhood abuse in different pain dimensions of nonspecific chronic low back pain]. *Schmerz, 28*(6), 600-606. doi:10.1007/s00482-014-1487-2

Levine, P. A. (1977). *Accumulated Stress, Reserve capacity and disease (Doctoral Thesis).* (PhD in medical biophysics Doctoral Thesis), University of California, Berkeley.

Lewis, R. (2007). Robert Scaer's Neurobiological Model for PTSD and Psychosomatic Illness. Retrieved from bodymindcentral website: http://www.bodymindcentral.com/pdf/pubs/LewisPub_Scaer_PTSD.pdf

Meeus, M., & Nijs, J. (2007). Central sensitization: a biopsychosocial explanation for chronic widespread pain in patients with fibromyalgia and chronic fatigue syndrome. *Clin Rheumatol, 26*(4), 465-473. doi:10.1007/s10067-006-0433-9

Meyer, K., & Damasio, A. (2009). Convergence and divergence in a neural architecture for recognition and memory. *Trends Neurosci, 32*(7), 376-382. doi:10.1016/j.tins.2009.04.002

Mohr, W. K., & Fantuzzo, J. W. (2000). The Neglected Variable of Physiology in Domestic Violence. *Journal of Aggression, Maltreatment & Trauma, 3*(1), 69-84. doi:10.1300/J146v03n01_06

Norman, G. J., Berntson, G. G., & Cacioppo, J. T. (2014). Emotion, Somatovisceral Afference, and Autonomic Regulation. *Emotion Review, 6*(2), 113-123. doi:doi:10.1177/1754073913512006

Paton, J. F., Nalivaiko, E., Boscan, P., & Pickering, A. E. (2006). Reflexly evoked coactivation of cardiac vagal and sympathetic motor outflows: observations and functional implications. *Clin Exp Pharmacol Physiol, 33*(12), 1245-1250. doi:10.1111/j.1440-1681.2006.04518.x

Payne, P., & Crane-Godreau, M. A. (2015). The preparatory set: a novel approach to understanding stress, trauma, and the bodymind therapies. *Front Hum Neurosci, 9*, 178. doi:10.3389/fnhum.2015.00178

Payne, P., Levine, P. A., & Crane-Godreau, M. A. (2015). Somatic experiencing: using interoception and proprioception as core elements of trauma therapy. *Front Psychol, 6*, 93. doi:10.3389/fpsyg.2015.00093

Perry, B. D. (1997). *Incubated in terror: Neurodevelopmental factors in the "cycle of violence."* (J. D. Osofsky Ed.). New York: Guilford Press.

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. *Brain Res Mol Brain Res, 18*(3), 195-200.

Porges, S. W. (2004). Neuroception: a subconscious system for detecting threaths and safety. *Zero to Three, 24*(5), 19-24.

Reich, W. (1972). *Character analysis*. New York: Touchstone.

Resnick, H. S., Yehuda, R., Pitman, R. K., & Foy, D. W. (1995). Effect of previous trauma on acute plasma cortisol level following rape. *Am J Psychiatry, 152*(11), 1675-1677. doi:10.1176/ajp.152.11.1675

Resulaj, A., Kiani, R., Wolpert, D. M., & Shadlen, M. N. (2009). Changes of mind in decision-making. *Nature, 461*(7261), 263-266. doi:10.1038/nature08275

Roelofs, K., & Spinhoven, P. (2007). Trauma and medically unexplained symptoms towards an integration of cognitive and neuro-biological accounts. *Clin Psychol Rev, 27*(7), 798-820. doi:10.1016/j.cpr.2007.07.004

Rogosch, F. A., Dackis, M. N., & Cicchetti, D. (2011). Child maltreatment and allostatic load: consequences for physical and mental health in children from low-income families. *Dev Psychopathol, 23*(4), 1107-1124. doi:10.1017/S0954579411000587

Rome, H. P., Jr., & Rome, J. D. (2000). Limbically augmented pain syndrome (LAPS): kindling, corticolimbic sensitization, and the convergence of affective and sensory symptoms in chronic pain disorders. *Pain Med, 1*(1), 7-23. doi:10.1046/j.1526-4637.2000.99105.x

Ruden, R. A. (2005). A Neurological Basis for the Observed Peripheral Sensory Modulation of Emotional Responses. *Traumatology, 11*(3), 145-158. doi:doi:10.1177/153476560501100301

Russell, A. W., Wickson, F., & Carew, A. L. (2008). Transdisciplinarity: Context, contradictions and capacity. *Futures, 40*(5), 460-472. doi:https://doi.org/10.1016/j.futures.2007.10.005

Saltzman, K. M., Holden, G. W., & Holahan, C. J. (2005). The psychobiology of children exposed to marital violence. *J Clin Child Adolesc Psychol, 34*(1), 129-139. doi:10.1207/s15374424jccp3401_12

Scaer, R. C. (2001). The neurophysiology of dissociation and chronic disease. *Appl Psychophysiol Biofeedback, 26*(1), 73-91.

Smith, A. M., & Flannery-Schroeder, E. C. (2014). Childhood Emotional Maltreatment and Somatic Complaints: The Mediating Role of Alexithymia. *Journal of Child & Adolescent Trauma, 6*(3), 157-172. doi:10.1080/19361521.2013.811456

Suetterlin, K. J., & Sayer, A. A. (2014). Proprioception: where are we now? A commentary on clinical assessment, changes across the life course, functional implications and future interventions. *Age Ageing, 43*(3), 313-318. doi:10.1093/ageing/aft174

Tesarz, J., Eich, W., Treede, R. D., & Gerhardt, A. (2016). Altered pressure pain thresholds and increased wind-up in adult patients with chronic back pain with a history of childhood maltreatment: a quantitative sensory testing study. *Pain, 157*(8), 1799-1809. doi:10.1097/j.pain.0000000000000586

Tesarz, J., Gerhardt, A., Leisner, S., Janke, S., Treede, R. D., & Eich, W. (2015). Distinct quantitative sensory testing profiles in nonspecific chronic back pain subjects with and without psychological trauma. *Pain, 156*(4), 577-586. doi:10.1097/01.j.pain.0000460350.30707.8d

Tietjen, G. E., Brandes, J. L., Peterlin, B. L., Eloff, A., Dafer, R. M., Stein, M. R., . . . Khuder, S. A. (2010). Childhood maltreatment and migraine (part II). Emotional abuse as a risk factor for headache chronification. *Headache, 50*(1), 32-41. doi:10.1111/j.1526-4610.2009.01557.x

Van Winkle, E. (2000). The toxic mind: the biology of mental illness and violence. *Med Hypotheses, 55*(4), 356-368.

Verhaeghe, P. (2006). *Tussen hysterie en vrouw. Van Freud tot Lacan: een weg door honderd jaar psychoanalyse*. Leuven: Acco.

Verhaeghe, P., & Vanheule, S. (2005). Actual neurosis and PTSD: The impact of the other. *Psychoanalytic Psychology, 22*(4), 493-507.

Weston, C. S. (2014). Posttraumatic stress disorder: a theoretical model of the hyperarousal subtype. *Front Psychiatry, 5*, 37. doi:10.3389/fpsyt.2014.00037

Table 1: Quality assessment of included theoretical and narrative reviews (n=8)

Legend: AE = as expected, NAE = not as expected, NA = not available

	The preparatory set: a novel approach to understanding stress, trauma and the bodymind therapies	Chronic idiopathic utricaria and post-traumatic stress disorder (PTSD): An	The toxic mind: the biology of mental illness and violence	Posttraumatic Stress Disorder Revisited	Posttraumatic stress disorder: a theoretical model of the hyperarousal subtype	Somatic experiencing: using interoception and proprioception as core elements of trauma	Cardiovascular Manifestations of Posttraumatic Stress Disorder	Trauma in the Mind and Pain in the Body: Mind-Body Interactions in Psychogenic Pain
First author	Payne P	Gupta M	Van Winkle E	Lambrecht F	Weston E	Payne P	Bedi U	Atarodi S
Year of publication	2015	2012	2000	2002	2014	2015	2007	2011
Journal	Frontiers in human neuroscience	Clinics in dermatology	Medical hypothesis	Psycho-somatic Medicine	Frontiers in psychiatry	Frontiers in psychology	Journal of the national medical association	Journal of the sociology of self-knowledge
Article type	Theoretical article	Narrative review	Theoretical article	Narrative review	Theoretical article	Theoretical article	Narrative review	Narrative review
Triangulation								
1) data	NA	NA	NA	NA	AE	AE	AE	AE
2) theory	NAE	NAE	AE	NAE	NAE	NAE	AE	AE
3) researcher	NA	NA	NAE	NA	NAE	AE	AE	AE
Content article								
Did the study address a clearly focused issue?	AE	AE	AE	AE	AE	AE	AE	AE
Did the study formulate a clear research question?	AE	AE	AE	AE	AE	AE	AE	AE
Was psychological trauma clearly described as an event?	NAE	NAE	NAE	NAE	AE	AE	AE	AE
Was the search strategy well defined?	NAE	NAE	NAE	NAE	NA	NA	NA	NA
Was there a clear quality assessment described in the articles?	NAE	NAE	NAE	NAE	NA	NA	NA	NA
Was there mentioning of their own limitations?	NAE	NAE	NAE	NAE	AE	NA	NA	NA
Was the theory clearly described?	AE	AE	AE	NAE	AE	AE	AE	AE
Has the question been answered in a clear and concrete manor?	NAE	AE	NAE	NAE	AE	AE	AE	AE
Did the article formulate a summary conclusion?	NAE	AE	NA	NAE	AE	AE	AE	AE
Assessment of quality **score 0-4= poor** **score 5-8 = moderate** **score 9-12= high**	poor	moderate	poor	poor	moderate	moderate	high	moderate

Table 2: Quality assessment of included observational studies (n=2)

	Child Maltreatment and Allostatic Load: Consequences for Physical and Mental Health in Children from Low-Income Families Rogosch FA, Dackis MN & Cicchetti D (2011)	The Psychobiology of Children Exposed to Marital Violence Saltzman KM, Holden GW & Holahan CJ (2005)
Did the study address a clearly focused issue?	YES	YES
Did the authors use an appropriate method to answer their question?	YES	YES
Were the cases recruited in an acceptable way?	YES	YES
Were the controls selected in an acceptable way?	YES	YES
Was the exposure accurately measured to minimize bias?	YES	YES
What confounding factors have the authors accounted for?	Child age and gender	Demographic results were still different
Have the authors taken account of the potential confounding factors in the design and/or in their analysis?	YES	YES
What are the results of this study?	- Maltreatment and allostatic load predicting a lower score on the health assessment - Maltreatment and allostatic load predicting a lower score on internal, external and total score - Maltreatment and allostatic load predicting a lower score on attention - Higher allostatic load gave higher cognition problems in children who were maltreated	As expected, a high proportion (48%) of all mothers of children exposed to marital violence reported that their children had been the direct target of physical violence from a parent or parent figure. **Trauma symptomatology**: children exposed to marital violence had significantly higher trauma symptom checklist for children total scores than did control groups **Heart rate:** children exposed to marital violence had higher pre-interview heart rates than did those who were not exposed. Post-interview heart rates of those exposed to martial violence were higher than those who were not. Heart rate response to a physical challenge did not differ between the 2 groups when controlling for total child abuse score. **Blood pressure**: there was no main effect of exposure to martial violence on pre-interview systolic blood pressure, pre-interview diastolic blood pressure or post-interview systolic blood

		pressure. There was a main effect of exposure to martial violence on post interview diastolic blood pressure. **Salivary cortisol:** exposure to martial violence was associated with higher levels of pre-interview salivary cortisol.
How precise are the results? How precise is the estimate of risk?	5% significant level No drop out = good	All means reported are unadjusted. There was a total population of 47 children. All the data were extracted by a standard protocol. = good
Do you believe the results?	YES	YES
Can the results be applied to the local population?	NO	YES
Do the results of this study fit with other available evidence?	YES	YES
Conclusion article = quality assessment **Score 0-4 = poor** **Score 5-7= moderate** **Score 8-10 = high**	High	High

Table 3: SWOT analysis of included studies (n=10)

Author(s), year	Strengths	Weaknesses
Atarodi S & Hosier S (2011)	- very comprehensive for psychogenic pain after trauma - includes comprehensive definition and physiology of pain - comprehensive brain model in the emergence of psychogenic pain - comprehensive kindling theory, explaining psychosomatic disorders after trauma - relates theory with treatment - describes the initial phase of reaction to trauma for the sympatho-adrenal axis and HPA axis - describes brain model for psychogenic pain	- describe the biological model of how kindling can cause psychogenic pain and state that this theory can also be used to explain general psychogenic complaints, without giving a detailed description
Bedi US & Arora R (2007)	- Good quality - clinical implication are described (cardiovascular disease, …) - very comprehensive article in relation to PTSD	- Results only applicable on PTSD patients
Gupta MA & Gupta AK (2012)	- comprehensive description of specific physical symptom (urticaria) - theory clarified with clinical examples	- results only applicable on PTSD patients - no correlation to other physical complaints or symptoms
Lamprecht F & Sack M (2002)	- comprehensive history of emergence and evolution of PTSD included - refers to treatment, mainly psychosomatic therapy	- poor quality - brief description of theories (HPA axis and sympatho-adrenal axis theory) - results only applicable on PTSD patients

Payne P & Crane-Godreau MA (2015)	- comprehensive theorem on tonic immobility - easy explicable theory to patients - refers to treatment, especially body-mind therapies	- poor quality
Payne P, Levine PA & Crane-Godreau MA (2015)	- very comprehensive theory, with description of the biggest number of structures involved in somatic complaints after trauma - comprehensive theorem on tonic immobility - clinical implications are described - refers to treatment - theory is clarified with a clinical example	- No clear description on how the 3 other parts of Core Response Network are influenced by the autonomic nervous system, only an interaction is described
Rogosch FA, Dackis MN & Cicchetti D (2011)	- observational study of high quality	- brief description of theories (HPA axis and sympatho-adrenal theory) - results only applicable on child abuse
Saltzman KM, Holden GW & Holahan CJ (2005)	- observational study of high quality - comprehensive description of HPA axis and symptho-adrenal theory - performed an observational study to test the hypotheses, derived from theories, which gave a clarification of the theories	- no inventory of non-PTSD related complaints in children, possible influence of group differences through psychiatric comorbidities could not be determined
Van Winkle E (2000)	- describes a comprehensive neural theorem - refers to treatment	- poor quality - theorems are strongly correlated with different pathologies (addiction, psychosomatic disorders, …), but little empirical evidence is mentioned - unclear design and title follow-up
Weston CS (2014)	- very comprehensive theory - explains most complaints of the hyperarousal type PTSD, not only physical (not included)	- only applicable on the hyperarousal subtype PTSD

Table 4: Data-extraction of included studies

Author(s) (year)	Title	Theorem
Atarodi S & Hosier S (2011)	Trauma in the Mind and Pain in the Body: Mind-Body Interactions in Psychogenic Pain	Brain mechanisms and Kindling theory: amygdala leading to triggering of various brain structures in trauma
Bedi US & Arora R (2007)	Cardiovascular Manifestations of Posttraumatic Stress Disorder	Hypothalamic-pituitary-adrenocortical axis theory and sympatho-adrenal theory: start and course of the axis's is described
Gupta MA & Gupta AK (2012)	Chronic idiopathic utricaria and post-traumatic stress disorder (PTSD): An under-recognized comorbidity	Hypothalamic-pituitary-adrenocortical axis theory and sympatho-adrenal theory: explanation for the emergence of chronic idiopathic utricaria
Lamprecht F & Sack M (2002)	Posttraumatic Stress Disorder Revisited	Hypothalamic-pituitary-adrenocortical axis theory and sympatho-adrenal theory: start and course of axis's is described
Payne P & Crane-Godreau MA (2015)	The preparatory set: a novel approach to understanding stress, trauma and the bodymind therapies	The Preparatory Set theory: explains how a state of arousal can lead to somatic complaints
Payne P, Levine PA & Crane-Godreau MA (2015)	Somatic experiencing: using interoception and proprioception as core elements of trauma therapy	Core Response Network theory: description of the Core Response Network and inducement to somatic complaints
Rogosch FA, Dackis MN & Cicchetti D (2011)	Child Maltreatment and Allostatic Load: Consequences for Physical and Mental Health in Children from Low-Income Families	Hypothalamic-pituitary-adrenocortical axis theory and sympatho-adrenal theory: start and course of axis's is described
Saltzman KM, Holden GW & Holahan CJ (2005)	The Psychobiology of Children Exposed to Marital Violence	Hypothalamic-pituitary-adrenocortical axis theory and sympatho-adrenal theory: start and course of axis's is described. A few somatic effects are summarized
Van Winkle E (2000)	The toxic mind: the biology of mental illness and violence	Toxic Mind theory: clarification of toxification and detoxification and how this can lead to somatic complaints
Weston CS (2014)	Posttraumatic stress disorder: a theoretical model of the hyperarousal subtype	Grounded Cognition theory: clarification of consolidation and memory of trauma and its consequences

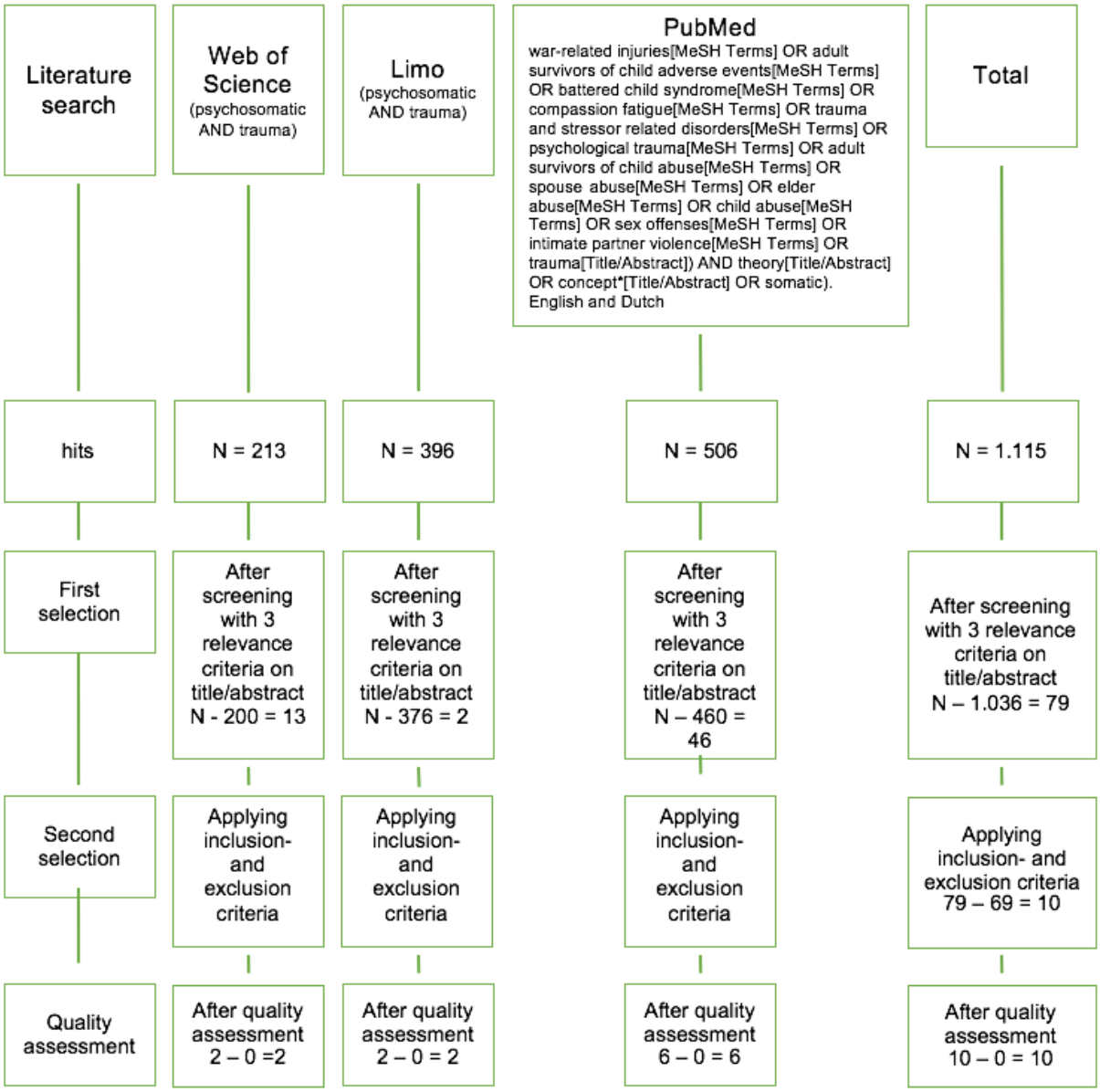

Figure 1: Flow Chart search strategy

Appendix 1: Overview of excluded studies and reason of exclusion

Excluded articles PubMed (n=40)

Nr	First author	Title	year	Reason of exclusion
1	Alvarez J	The relationship between child abuse and adult obesity among California women	2007	No biological theorem described
2	Andelic N	Disability, physical health and mental health 1 year after traumatic brain injury.	2010	No psychological trauma described
3	Armour C	The underlying dimensionality of PTSD in the diagnostic and statistical manual of mental disorders: where are we going?	2015	No model for physical complaints described
4	Axmacher N	Natural memory beyond the storage model: repression, trauma, and the construction of a personal past	2010	No physical complaint, handles about memory
5	Ben-Ezra M	Psychosomatic symptoms among hospital physicians during the Gaza War: a repeated cross-sectional study	2011	No biological theorem described
6	Ben-Ezra M	Psychosomatic symptoms among hospital physicians during the Gaza War: a repeated cross-sectional study	2011	No biological theorem described
7	Bentley JA	The indirect effect of somatic complaints on report of posttraumatic psychological symptomatology among Somali refugees	2011	No biological theorem described
8	Bob P	Chaos, brain and divided consciousness	2007	No model for physical complaints described
9	Brand BL	Where are we going? An update on assessment, treatment, and neurobiological research in dissociative disorders as we move toward the DSM-5	2012	No physical complaints, but dissociative complaints described
10	Carr ER	PTSD, depressive symptoms, and suicidal ideation in African American women: a mediated model	2013	No biological theorem described
11	Chakhssi F	Early maladaptive cognitive schemas in child sexual offenders compared with sexual offenders against adults and nonsexual violent offenders: an exploratory study	2013	No physical complaints described
12	Chen Y	Concepts and strategies for clinical management of blast-induced traumatic brain injury and posttraumatic stress disorder	2013	Only traumatic brain injury as physical complaint
13	Chen Y	Non-impact, blast-induced mild TBI and PTSD: concepts and caveats	2011	Only traumatic brain injury as physical complaint
4	Cohen H	An association between stress-induced disruption of the hypothalamic-pituitary-adrenal axis and disordered glucose metabolism in an animal model of post-traumatic stress disorder	2009	Animal study
5	Cordero H	Evidence for biological roots in the transgenerational transmission of intimate partner violence	2012	Animal study
6	Diebel LN	Organ failure in the obese adipocytes prime polymorphonuclear cell inflammation under stress conditions	2013	Cell study
7	Dutton MA	Resilience and crime victimization	2010	No physical complaints described
8	Engel CC	Terrorism, trauma, and mass casualty triage: how might we solve the latest mind-body problem?	2007	No biological theorem: psychological theorem
9	Franzke I	Dissociation as a mediator of the relationship between childhood trauma and nonsuicidal self-injury in females: a path analytic approach	2015	Handles about self-mutilation behavior, no physical complaints described
10	Frewen P	Trauma-related altered states of consciousness in women with BPD with or without co-occurring PTSD	2014	No physical complaints, describes alterations in the state of consciousness
11	Frewen P	Trauma-related altered states of consciousness: exploring the 4-	2014	No physical complaints,

		D model		describes alterations in the state of consciousness
12	Gao J	Glutamate and GABA imbalance promotes neuronal apoptosis in hippocampus after stress	2014	Animal study
13	George ME	Hypothermia is associated with improved outcomes in a porcine model of hemorrhagic shock	2010	No psychological trauma described
14	Hinton DE	Worry, worry attacks, and PTSD among Cambodian refugees: a path analysis investigation	2011	Physical complaint not obtained by trauma, but about how worrying influences physical complaints
15	Hinton DE	Khyâl attacks: a key idiom of distress among traumatized Cambodia refugees	2010	Physical complaints: panic attacks, exclusion because of the significant psychological aspect
16	Hinton DE	The 'multiplex model' of somatic symptoms: application to tinnitus among traumatized Cambodian refugees	2008	No full text available
17	Holleman M	The relationships of working conditions, recent stressors and childhood trauma with salivary cortisol levels	2012	Only a reference, no complete description of a theorem formulated
18	Ilin Y	Enriched environment experience overcomes learning deficits and depressive-like behavior induced by juvenile stress	2009	Animal study
19	Jia M	Corticosterone mitigates the stress response in an animal model of PTSD	2015	Animal study
20	Jia M	Biomarkers in an animal model for revealing neural, hematologic, and behavioral correlates of PTSD	2012	Animal study
21	Karatzias T	Cognitive appraisals and physical health in people with posttraumatic stress disorder (PTSD)	2009	No biological, but psychological theorem: how cognitive judgements can lead to physical complaints
22	Lanius RA	Trauma-related dissociation and altered states of consciousness: a call for clinical, treatment, and neuroscience research	2015	No physical complaints: describes alterations in the state of consciousness
23	McCall-Hosenfeld JS	The association of interpersonal trauma with somatic symptom severity in a primary care population with chronic pain: exploring the role of gender and the mental health sequelae of trauma	2014	No biological theorem described, only a statistical model described
24	Pietrzak RH	Support for a novel five-factor model of posttraumatic stress symptoms in three independent samples of Iraq/Afghanistan veterans: a confirmatory factor analytic study	2012	No biological theorem described, describes a new classification of PTSD and the compatibility in Afghanistan and Iraq veterans
25	Price CJ	Changes in posttraumatic stress symptoms among women in substance use disorder treatment: the mediating role of bodily dissociation and emotion regulation	2013	Only psychological symptoms described
26	Rorabaugh BR	Sex-dependent effects of chronic psychosocial stress on myocardial sensitivity to ischemic injury	2015	Animal study
27	Roth MK	Effects of chronic plus acute prolonged stress on measures of coping style, anxiety, and evoked HPA-axis reactivity	2012	Animal study
28	Tsai J	Examining the dimensionality of combat-related posttraumatic stress and depressive symptoms in treatment-seeking OEF/OIF/OND veterans	2011	Only description of a statistical model

29	Wörtwein G	Effects of maternal separation on neuropeptide Y and calcitonin gene-related peptide in "depressed" Flinders Sensitive Line rats: a study of gene-environment interactions	2006	Animal study
30	Zoellner T	Posttraumatic growth in clinical psychology - a critical review and introduction of a two-component model	2006	No physical symptoms described. Describes the psychological growth after traumatic experience

Excluded articles Web of Science (n=11)

Nr	First author	Title	year	Reason of exclusion
1	Axmacher N	Natural memory beyond the storage model: repression, trauma, and the construction of a personal past	2010	No physical complaints, describes memory
2	Ben-Ezra M	Psychosomatic symptoms among hospital physicians during the Gaza War: a repeated cross-sectional study	2011	No biological theorem described
3	Charles E	Links between life events, traumatism and dementia; an open study including 565 patients with dementia	2006	No physical complaints described, discusses dementia after trauma
4	De Graaf T	A Personal Sensitization Factor (PSF) mediating between life events and post-traumatic psychiatric or psychosomatic disease in adult life	1996	No biological theorem described
5	Ferri P	The analyst's body as relational space in the treatment of seriously ill children	2013	No biological theorem described, only psychodynamic theorems described
6	Hirsch M	Body dissociation because of trauma	2010	No physical complaint, discusses dissociation
7	Jasiukevicien e L	Chronic fatigue syndrome in cardiology neurohumoral changes	2006	No psychological trauma described
8	Katz A	Healing the Split Between Body and Mind: Structural and Developmental Aspects of Psychosomatic Illness	2010	No biological theorem, only psychodynamic theorems described
9	Tosevski DL	Stressful life events and physical health	2006	No full text available
10	Zimmermann P	Psychogenic disorders in German soldiers during World War I and II. A comparison from a psychotraumatologic perspective	2005	Article in German
11	Zwickl S	The association between childhood sexual abuse and adult female sexual difficulties	2014	No biological theorem, only theorems described regarding incidence of complaints based on the study

Excluded articles Limo (n=18)

Nr	First author	Title	Year	Reason of exclusion
1	Anand KJS	Gastric suction at birth associated with long-term risk for functional intestinal disorders in later life	2004	No biological theorem described
2	De Masi F	The psychodynamic of panic attacks: A useful integration of psychoanalysis and neuroscience	2004	Physical complaints: panic attacks, exclusion because of the significant psychological aspect
3	Farina B	Does a dissociative psychopathological dimension exist? A review on dissociative processes and symptoms in developmental trauma spectrum disorders	2013	No biological theorem, Psychodynamic theorems described
4	Hallberg RM	Health consequences of workplace bullying: experiences from the perspective of employees in the public service sector	2006	No causal theorem, only theorems described regarding incidence of complaints based on the study

5	Hopper JW	Preliminary evidence of parasympathetic influence on basal heart rate in posttraumatic stress disorder	2006	No biological theorem described
6	Katz A	Healing the Split Between Body and Mind: Structural and Developmental Aspects of Psychosomatic Illness	2010	No biological theorem described, only psychodynamic theorems
7	Kozlowska K	Intergenerational Processes, Attachment and Unexplained Medical Symptoms	2013	No biological theorem described, discusses a psychological theory
8	Lind AB	Struggling in an emotional avoidance culture: A qualitative study of stress as a predisposing factor for somatoform disorders	2013	No complete description of a biological theorem, only a brief hypothesis on results in conclusion
9	Manolopoulos S	Early traumas in psychosomatic patients: Splitting and integration	2006	Psychodynamic theorem, no biological theorem described
10	Martin P	Grief that has no vent in tears, makes other organs weep.' Seeking refuge from trauma in the medical setting	2012	Psychodynamic theorem, no biological theorem described
11	McCall-Hosenfeld S	The association of interpersonal trauma with somatic symptom severity in a primary care population with chronic pain: Exploring the role of gender and the mental health sequelae of trauma	2014	No biological theorem described
12	Oliner M	The Minefield of Emotions	2010	Psychodynamic theorem, no biological theorem described
13	Smith A	Childhood Emotional Maltreatment and Somatic Complaints: The Mediating Role of Alexithymia	2014	No biological theorem described
14	Spitzer C	Gender-specific association between childhood trauma and rheumatoid arthritis: A case–control study	2013	No biological theorem described
15	Stone J	The role of physical injury in motor and sensory conversion symptoms: A systematic and narrative review	2009	Discusses conversion, is an exclusion criterion
16	Von Kanel R	Momentary stress moderates procoagulant reactivity to a trauma-specific interview in patients with posttraumatic stress disorder caused by myocardial infarction (Report)	2010	No biological theorem described
17	Von Kanel R	Posttraumatic stress disorder and soluble cellular adhesion molecules at rest and in response to a trauma-specific interview in patients after myocardial infarction (Report)	2010	No biological theorem described
18	Zwickl S	The association between childhood sexual abuse and adult female sexual difficulties	2014	No causal theorem, only theorems described regarding incidence of complaints based on the study