



THE KEY TO UNDERSTANDING PTSD

Contrasting post-traumatic stress and
post-traumatic growth

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Abstract

Traumatic incidences happen all around the globe. Some of the people who experience trauma develop post-traumatic stress disorder (PTSD), while some do not. Even more interesting is that some also experience growth afterwards (post-traumatic growth; PTG). The purpose of this paper is to look at neural aspects of why some people develop PTSD and others PTG after a traumatic event. To fulfill the aim, both PTSD and PTG will be reviewed to create an image of the existing research in behavioral and neurological terms. In addition to looking at the constructs separately, a chapter will also look at studies where both PTSD and PTG are acknowledged collaterally in participants. When looking deeper into the theories of PTSD divisions occur, and more research is needed to establish the most prominent explanation of PTSD. PTG on the other hand has only been studied for a short period of time but yields important insights into trauma-related outcomes. These fields need to be submerged and new multidisciplinary definitions are needed for future research. The key to PTSD is suggested to emerge within the new field.

Keywords: trauma, post-traumatic growth, post-traumatic stress disorder, growth, emotion regulation, prefrontal cortex, mindfulness

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1. Introduction

“For months after the (knife) attack, I couldn't close my eyes without envisioning the face of my attacker. I suffered horrific flashbacks and nightmares. For four years after the attack I was unable to sleep alone in my house. I obsessively checked windows, doors, and locks. By age 17, I'd suffered my first panic attack.”

P.K. Phillips

1.1. Trauma Related Outcomes

Post-traumatic stress disorder (PTSD) causes loss, private and societal economic constraint, and impairment to the people affected (Kirkpatrick & Heller, 2014; Wu, Xu, & Sui, 2016). People with PTSD have nightmares, intense fear, and avoidance of situations that might trigger memories. Trauma is a situation that causes intense horror, helplessness, or near-death situations. Trauma does not include; ordinary stress, stressful events that have a high occurrence rate, or situations causing an intense emotional response (if it is not related to violence or accidents; Morrison, 2014). The lifetime prevalence of PTSD is 7-9% in the US (Morrison, 2014). In other words, this percentage of people do not recover after experiencing a traumatic event.

The fact that PTSD causes significant constraints on the people affected is clear, however it does not account for the other 91% that don't develop PTSD. Additionally, research has found that 69% of the general population experience a traumatic event during their life (Norris, 1992). In fact, many of those encountering trauma instead experience overall benefits, growing as individuals after the traumatic event, a phenomenon known as *post-traumatic growth* (PTG; Joseph & Hefferon, 2013).

Between 30 and 70% of people experiencing trauma report some growth after a traumatic event (Joseph & Hefferon, 2013). The question then becomes: after trauma, who develops PTSD and who develops PTG, and why? Some suggest that we need post-traumatic events to start a growth process (Calhoun & Tedeschi, 1998). Others suggest that those with the highest rate of PTSD are the most likely to subsequently develop PTG (Zhou, Wu, & Chen, 2015). However, the debate is still open.

Few studies have looked at the intersection between PTSD and PTG. Those who have looked at this interaction, suggest that it is complex (Blix, Hansen, Birkeland, Nissen, & Heir, 2013; Wu, et al., 2016; Zhou & Wu, 2016). The complex interaction suggests that different aspects of emotion regulation and factors that buffer against PTSD are of importance

for the development of both PTSD and PTG. Even though buffering factors exist, researchers rarely explain the reason behind this.

The amygdala is suggested to play a significant role in fear learning and other emotional responses (LeDoux, 2000). Other suggested brain areas involved in the processing of traumatic events are the hippocampus, the prefrontal cortex (PFC), and the cerebellum (Shin, Rauch, & Pitman, 2006). These areas have ascending or descending relations to the amygdala and are suggested to play a part and are considered relevant for fear-processing (LeDoux, 2000). However, due to its emotion-regulating properties the PFC is suggested in the development of PTG (Garland, Goldin, & Fredrickson, 2015).

For people to recover from the most horrible of events, and even to grow after them, the gap explained needs to be filled. When this is done, hopefully, we can help relieve PTSD and other related disorders. For this task, a focus on a deeper functional perspective is necessary, because the simple behavioral observations may not accurately reflect the base on which these behaviors are built upon.

1.2. The Aim of the Current Review

The aim of this review is to look at neural aspects of why some people develop PTSD and others PTG after a traumatic event. Questions to be addressed is:

1. Can we determine what type of individuals who will develop PTSD and PTG based on current research?
2. Can we by looking at both constructs find a neural base for the development of PTSD and PTG?
3. Are there any survival benefits of developing PTSD and PTG?

To fulfill the aim, it is of relevance to look at PTSD, and PTG separately. The discussion in this review will be done both from a behavioral perspective and from a neurological perspective. A section has been included that looks at PTG and PTSD in the same individuals and in relation to the same events. The review includes both traditional theories and methods, in addition to the approaches and functional findings from neuroscience. The hope is to add new thoughts and views of trauma-related outcomes, both to theory and practice.

This essay looks only at adult PTSD, even if there is extensive research also on children (Kilmer, et al., 2014; Meyerson, Grant, Carter, & Kilmer, 2011). However, PTSD in children and adults differs in their etiology and how to treat the disorder. PTSD were first

seen as exclusive to war-veterans (Oster, Morello, Venning, Redpath, & Lawn, 2017) and there is plenty of research on the topic from a PTSD perspective, but few studies have acknowledged PTG in this population which makes it hard to compare, and thus are excluded from this paper. Family violence, abuse and other domestic related incidents are also well researched, however, the development of complex PTSD is not included in the scope of this paper, because it involves several other aspects than the trauma itself (Messman-Moore & Bhuptani, 2017). There is also much research on cancer patients and relatives of cancer patients (Parikh, et al., 2015). Breast cancer is a subject under debate and not classified as a trauma, and thus excluded from this essay (Sumalla, Ochoa, & Blanco, 2009). In this essay a great extent of the research covers natural disasters and accidents, this is due to several reasons, mostly as the research are looking at both PTSD and PTG is in this segment of the field. It would have been a to complex and big essay to cover all aspects of PTSD.

2. An Overview of Post-traumatic Stress Disorder

PTSD is one of the few disorders with an onset event. This chapter includes a description of the characteristics of PTSD described in the diagnostics and statistical manual of mental disorders (DSM). It also includes a brief introduction to theories about PTSD and how to measure PTSD. Additionally, a discussion about treatment options and neural findings that are related to PTSD will be presented. The chapter will end with a presentation of the psychological construction approach and a summary of PTSD.

2.1 Characteristics of PTSD

The following list states the criteria of PTSD in DSM V (Morrison, 2014);

- A tendency to relive the stressful event, in the form of nightmares, daydreams, associations with sound or other triggers.
- Experience of distress or physical sensations when reminded of the event.
- The onset of PTSD can be delayed, but a beginning PTSD causes avoidance of reminders as far as possible. This avoidance can include to refuse watching or reading about the event or similar events or to push thoughts out of consciousness.
- Mood shifts and blaming oneself for what happened. They have thoughts that put themselves and the world at a negative view and show distrust in other people.
- Hyperarousal, including negative moods, low concentration, insomnia and an increased startle response (high intensity, novel stimulus) is present.

PTSD has a lifetime prevalence of 7-9% in the United States (Kirkpatrick & Heller, 2014). The prevalence of PTSD is higher among women, younger persons (including children), low socioeconomic status (SES) and minority groups, and it is common with PTSD in war veterans (Kirkpatrick & Heller, 2014; Whitaker, Gilpin, & Edwards, 2014). Studies show that almost everyone (40-90%) encounters at least one stressful event in their life, but only a few of those gets PTSD (Milani, Hoffmann, Fossaluza, Jackowski, & Mello, 2017). Today there is no explanation for the variance between the individuals experiencing traumatic events and those who get PTSD. However, some suggest that resilience factors and genes are important contributions to whether a person gets PTSD or not (Almli, Fani, Smith, & Ressler, 2014; Whitaker et al., 2014). There are differences in the variability in several of the factors suggested. (Almli, Fani, Smith, & Ressler, 2014; Whitaker et al., 2014). Risk

factors of PTSD can be divided into three categories (Kirkpatrick & Heller, 2014): pre-trauma, peri-trauma, and post-trauma.

- Pre-trauma factors contribute to the development of PTSD before the event has occurred. SES, parental neglect, gender, other psychiatric diseases and amount of social support are suggested factors.
- Peri-trauma factors are related to the event and include characteristics of the event (intensity, duration, severity, and frequency), characteristics of the person (e.g., how much they respond to the initial event), and how unpredictable or uncontrollable the event appeared.
- Post-trauma factors are related to the aftermath of the event. Social support, overall life stress, when the disorder is recognized and when treatment starts are factors in the post-trauma category. (Kirkpatrick & Heller, 2014).

PTSD is an incapability to cope with a traumatic event, and the person use negative coping strategies (such as avoidance). Fear learning and PTSD are associated, and a heightened fear response does not decrease with time (Kirkpatrick & Heller, 2014).

The traumatic event causes changes in the processing of information and the neural processing of the trauma (Kirkpatrick & Heller, 2014). Several theories about PTSD and its specific elements exist. PTSD includes processing in memory function, attention, dissociation, cognitive-affective reactions, beliefs, cognitive coping strategies and social support. Each function is part of the diagnostics criteria and affects PTSD in separate ways. Following is a brief description of these elements.

- Memory functions are impaired, and one explanation is that traumatic events are more frequently recalled, in addition to problems recalling auto-biographical memories (Brewin & Holmes, 2003). Memory functions are also involved in the flashbacks and reliving of the traumatic event when comparing to non-PTSD individuals. Negative memories in PTSD seem to be involuntary and part of the present rather than the past (Brewin & Holmes, 2003).
- Attention bias on the other hand have no evidence that people with PTSD shift toward specific things or situations, nor that it is specific to PTSD (Brewin & Holmes, 2003).
- Dissociation appears when our fundamental assumptions and beliefs are challenged, and typically causes emotional numbing, depersonalization and out of body experiences. Dissociation is linked to freezing behavior in animals. Animals in experiments tend to

play dead in situations where they cannot fight or flee (Brewin & Holmes, 2003). Ratings of fear, helplessness, and horror during the event predicts PTSD (Brewin, Andrews, & Rose, 2000).

- The emotional component is related to recovery. Individuals with high levels of negative emotions (e.g., anger towards others) show a slower recovery pace (Andrews, Brewin, Rose, & Kirk, 2000).
- One's beliefs about the self and the world are linked to PTSD. Traumatic events are thought to create a shattering of current assumptions and stress a need to build coherent beliefs about the world (Brewin & Holmes, 2003; Foa & Tolin, 2000).
- Negative rating and cognitive coping strategies are related to PTSD. Avoiding thoughts of the event is suggested to make them go away for a short amount of time and then come back stronger the next time (Brewin & Holmes, 2003).
- Social support is the last aspect of PTSD. Negative social support is related to PTSD, and more strongly for women than men (Brewin & Holmes, 2003).

PTSD is, in summary, a complex disorder involving several functional and behavioral aspects (e.g., emotion regulation, social support, beliefs, dissociation and attention biases). The key aspects of PTSD presented include four domains: a) re-experiencing the event, b) Avoidance, c) Negative cognitions and mood, 4) Hyperarousal (Kirkpatrick & Heller, 2014). Several impairments relate to PTSD, but not all are well established or specifically linked to PTSD only.

2.2 Theories about PTSD

This section includes a brief overview of theories about PTSD, with both early and more recent views. The first part will briefly cover some traditional theories, and the other part will consider evolutionary aspects of PTSD.

2.2.1 Traditional theories about PTSD. The early theories can be divided into three groups (Brewin & Holmes, 2003), each explaining one aspect of PTSD.

1. The social cognitive theories include thoughts about inner worlds, beliefs, and thoughts about how the world should be. Beliefs are shattered in a traumatic event.
2. The second group is the conditioning theories that say that learned behaviors can trigger avoidance.
3. The third group is about information processing. Theories in this group explain how memories are formed, stored and are retrieved. For example, fear memories are

different from other memories and are formed and triggered differently. The connections are stronger for fear memories than for neutral memories, causing a negative feedback loop (Brewin & Holmes, 2003).

The more recent theories have evolved with more research and new guidelines.

1. The *emotional processing theory* is related to a person's inner world, beliefs and thoughts. This theory does not explain how change and restructuring occur. However, it includes a comprehensive set of research on how to treat these characteristics (Foa & Tolin, 2000).
2. The *dual representation theory* is not as comprehensive as the emotional processing theory. Instead, it has explanatory power in how memories are formed and used. The theory states that traumatic memories are stored in a different memory system than ordinary memories. The amygdala has separate routes for input and can explain the different memory systems theory. The amygdala has one route involving the hippocampus and other routes that do not (Brewin, 2001). The input not passing the hippocampus have no spatial or temporal context and can be unconsciously brought up by trigger events, causing a feeling of direct danger (Ehler & Clark, 2000). The hippocampus has distinctive features in short and prolonged stress (non-traumatic), where the declarative memory enhances in short durations and breaks down in prolonged duration (Cahill & McGaugh, 1998). Memories not encoded with spatial and temporal context needs to be brought up into conscious processing so they can be tagged with these features to eliminate the feeling of present danger. This theory focuses primarily on memory, appraisals and emotions, it misses several of the other features of PTSD explained by the emotional processing theory. However, it adds valuable insight about traumatic memory formations.
3. Ehler and Clark's *cognitive model* (2000) explains the anxious features of PTSD. This cognitive model looks at aspects of why people are anxious about the future when the event is lying in the past. The link is missing contextual information and memories create a threat to safety or the self. Coping strategies that may interfere with PTSD, along with treatment implications are brought up in the model. The cognitive model is the most comprehensive theory but lacks some explaining of how these methods function in traumatic situations.

The difference seen is how the traumatic event affects memory encoding, and how this is related to recovery from PTSD. The emotional processing theory does not use several memory systems, while the others do. The dual representation theory focuses on how the brain reacts to trauma and not how the representation in memory is involved (Ehler & Clark, 2000). All three theories state that we need to integrate the traumatic event into the ordinary memory system, through the dampening of neural strengths or by consciously adding contextual information to the traumatic memory. A remaining question would be if memory formations are important for treatment.

Many people experiencing a traumatic event often make up strategies to avoid reminders. Most victims can gradually confront memories with time, but people not seeing a decrease of fear use safety behaviors and will eventually avoid anything that might remind them of the fear. The avoidance-behavior can cause occupation with avoiding fear and result in few safe reminders (Kirkpatrick & Heller, 2014).

2.2.2 Evolutionary Thoughts on Threat, Coping, Appraisals and Survival. PTSD as a diagnosis states that we have a reaction to a traumatic event, from which we don't get back to normal processing (Kirkpatrick & Heller, 2014). However, what is likely to be a traumatic event and how these discrepancies occur and are maintained in terms of adaptiveness is seldom explained. This section will explain some points to consider. From an evolutionary perspective everything a species focuses on is to recreate and to survive. In the light of this it is an important question why we develop PTSD, as it seems to have little adaptiveness for the individual.

The evolution of the human species can be divided into several time eras, in which we also can see some evolutionary adaptive functions (Bracha, 2006).

- In the Mesozoic era (140 million years ago), a fear circuitry evolved that we share with most other mammals. This era explains fears related to height and separations that can be seen across several species (Bracha, 2006).
- The Cenozoic era (20 million years ago) includes fear circuitry that we share with all higher order primates and includes fear of darkness, confined spaces, snakes, reptiles, immersions in water and fear of high CO_2 (oxygen-low air; Bracha, 2006).
- The mid and upper Paleolithic era (200 000 -12 000 years ago) is associated with human specific fears including compulsive behaviors and fear of blood, insects, mice and non-kin individuals (Bracha, 2006).

- The last era, Neolithic (12 000 years ago) is related to only some humans and thought to be a result of cultures or rare allele variants. In this segment of time we find “conversive” symptoms (e.g. communicative problems; Bracha, 2006).

Except for the evolution of fear related circuits there are also differences in the human cortex. The most recognized division stem from MacLean (MacLean, 1990) and his theory about the triune brain. MacLean proposed that the brain, due to its structure, could be divided in three different evolutionary parts. This includes the reptilian brain, the paleomammalian brain and the neomammalian brain.

Where the reptilian brain includes basic functions such as breathing and act for survival (Naumann, et al., 2015). The paleomammalian brain is linked to the concept limbic system, however, as this concept has contradictory support it is more relevant to see it as the structures of septum, amygdala, hypothalamus, hippocampus and cingulate cortex. The last part is the most recent evolved structure including the neocortex.

The important aspect with this theory and PTSD is that we can switch between the different layers of the cortex. Besides the connections between the layers it is also important to note that stress may interfere with the normal communications between different areas of the brain (Baldwin, 2013). This can be seen in the findings that stress reduces the communication between elements. The elements proposed by Baldwin (2013) are biologic and psychologic, including both internal/external, infection/predator and social/non-social. When considering survival, our more basic structures in our brain take over and inhibit the function of neocortex.

It is also important to note that activation of the Hypothalamic–pituitary–adrenal (HPA)-axis along with the sympathetic-adrenal-medullary system allow the body to gather energy to rise body temperature to fight infections. This means that all systems in interaction create bodily changes that are most suitable for the situation at hand using both central and peripheral aspects, even if how these aspects affect the brain is unclear (Baldwin, 2013).

A little story of survival; imagine a field on which you are gathering food for your tribe. Suddenly you hear a noise from the trees, you stop to look around. What is the noise, is it important to act on or can you go back to your previous activity, to gather food? It was nothing and you continue with the food. After a while you hear some other noise, you stop and look around. This time you sees a lion. You try to make yourself small and assess the situation, is the lion after you? Your thoughts are running through your head, what can you do? What are your options? Searching for any way to cope with the situation,

considering all your options.

The lion may represent a traumatic experience as it includes threat to your life. However, as we see in the story you react to other noises as well. LeDoux (1996) suggested that we have two different routes involving fear, one fast and one slow. The fast way includes the right amygdala, thalamus, hypothalamus, PAG and sensory organs, whereas the slow route also includes the frontal cortex and hippocampus. The fast way is involved in reflexive behaviors and the slow route makes it possible to inhibit an unwanted response and the ability to choose coping strategy (Baldwin, 2013; LeDeux, 1996). The conscious experience of fear also comes with the involvement of PFC and hippocampus. The slow and fast ways of reaction to novel stimulus also show differences in evolutionary and ontogenic origins, as well as neural cells. For example, the ventral fast route, goes mainly in the right hemisphere and is populated by granular cells. The memory formation associated with this system show connections to the limbic area. The dorsal route on the other hand goes mainly in the left hemisphere, is populated with pyramidic cells, whereas the connections in memory formation goes from the limbic areas. This suggests two separate routes and functions. In normal instances these two systems interact to create a current state involving both seeing the world as it is (dorsal) and scanning and reacting to external novel stimulus and survival pressure (ventral), but these connections can be disorganized under prolonged stress (Baldwin 2013)

An important discussion is threat and novel stimulus as the amygdala has been proposed in the ability to feel fear (Feinstein, Adolphs, Damasio, & Tranel, 2011). In our evolutionary environments there is a survival skill to consume as little energy as possible. Looking at PTSD, fear learning, and fear induction research often miss some important aspects of energy consumptions (Cantor, 2009). The most recognized fear response is the “fight and flight response” (Cannon, 1915). However, seen from a survival perspective this is very energy consuming and other more energy-preserving methods would be more beneficial. For example, Cantor (2009) suggests that we have the following six stages of methods for avoiding danger, and threat:

1. Avoidance of danger is the least energy consuming method, this is also highly present in PTSD. The subject therefore avoids any situation that may be harmful, or any trigger of harmful places. Avoidance is linked to the basal ganglion and evolved in the paleolithic-era, suggesting that we might not have subjective, but rather reflexive, behaviors in this method.

2. Attentive mobility is when we freeze and listen to our surrounding. This can be compared with hyper arousal and vigilance seen in PTSD. This state is related to hypervigilance and high arousal, stemming from a shift from parasympathetic to sympathetic activation through vagal changes (e.g. That the ventral vagal withdrawal lifts the vagal brake; (Baldwin, 2013).
3. Withdrawal can be to crawl away from a potential danger, or in the case of PTSD to stay in the apartment. Or to slowly crawl away from the lion in the story. This is different from avoidance as this state occurs after encounter of predators (Cantor, 2009).
4. Aggressive defense can be either to show how strong you are to the predator hoping the other part will go away, or it may be to fight. However, this kind of defense is energy consuming. In PTSD this shows as emotional disturbances. This state is regulated by the sympathetic nervous system, and can fluidly change from fight to flight mode, with change in blood flow (e.g. to legs or arms; (Baldwin, 2013).
5. Appeasement is when you go back to a person after being beaten down. The survival value lies in showing the attacker that you will be part of the group and will be loyal. This can be seen in abusive families. This is also seen in people captured, they might even protect their attacker (Cantor & Price , 2007).
6. The last defense stated is the tonic immobility, to play dead. If hurt or frightened with fear this response decrease heartrate and shut down the body. This response is regulated by the vagal nerve, through its dorsal aspects along with changes in sympathetic activation. Causing decreased heartrate (compared to fight) but strengthen heart contractions and increased blood flow (Baldwin, 2013).

However, Baldwin (2013) also includes another response which he calls collapse. This state is compared with giving up, or mental defeat. It also overlaps with tonic immobility. This is due to a shift to parasympathetic dorsal vagal activation. Which also leads to decreased heart rate.

In the relation to adaptiveness of specific reactions to different traumas, it is of interest to note that different traumas are more and less connected to the development of PTSD. For example, only 4-5% of the people exposed to fire develop PTSD in comparison to 22% for abuse and 39% of combat. This may have an evolutionary explanation as fires has been around for a longer time, whereas guns have a shorter evolutionary time frame. Thus, our brains and genes have been programmed for such

instances, and thus help us cope with it (Bracha, 2006).

In summary, the evolutionary approach to PTSD suggest that we have a more nuanced response pattern than the research suggest. This conclusion is based mostly on the energy consumption that is connected to the fight and flight response. From an evolutionary point of view every reaction is based on its survival properties, and a high energy consumption is suggested to not be adoptive.

2.3 How to measure PTSD?

PTSD is measured by questions covering the characteristics of the disease. Examples of measures are the PTSD checklist (PCL; Blanchard, Jones-Alexander, Buckley, & Forneris, 1996), the clinician-administered PTSD scale (CAPS; Weathers, Keane, & Davidson, 2001), the short post-traumaticstress disorder rating interview (SPRINT; Connor & Davidson, 2001) and the post-traumaticstress disorder symptoms scale-interview version (PSS-I; Foa & Tolin, 2000).

CAPS (Weathers, Keane, & Davidson, 2001) is a tool for measuring PTSD in an interview-based manner. CAPS is the gold standard (Foa & Tolin, 2000) of measuring PTSD but with its 17 items and corresponding severity question for each item, it is rather time-consuming to use (for the full review see: Weathers, et al., 2001). This measure also shows sensibility to clinical changes, thus measuring real behavioral changes (Weathers, et al., 2018).

The SPRINT is a measure of PTSD developed by Connor & Davidson (2001). It is a short interview-based measure consisting of 8 items to cover the full criteria of the DSM (Kim, et al., 2008). Comparing the SPRINT with the CAPS, Vaishnavi, Payne, Connor, & Davidson (2006) showed that these scales are comparable both in the sense of each cluster of criteria's and on the scales in total. However, the CAPS is time-consuming, and the SPRINT show a much shorter time frame in which to collect the answers. Fast scoring is important in situations where time is a limited resource, and many people need to be assessed (Kim, et al., 2008).

The PSS-I (Foa & Tolin, 2000) is a semi-structured interview and takes about half an hour to complete. Comparing the scales between CAPS and PSS-I, the correlation coefficient ranges from .63 to .87, showing that the items tend to overlap (Foa & Tolin, 2000).

The PCL (Weathers, Keane, & Davidson, 2001), is a questionnaire consisting of 20 items measuring post-traumatic stress symptoms and can also be used to measure clinical change over time (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996; Bovin, et al.,

2016).

CAPS is the golden standard for measuring PTSD; however, it is time-consuming, and several other methods may be better suited. These other measures use both semi-structured interviews and questionnaires, in comparison to the CAPS that is in a full interview format. All the presented measures show an overlap, suggesting that they measure the same construct, but differ in the length of assessment.

2.4 Neural Underpinnings of PTSD

This section will look more closely into the brain and activation of specific brain areas regarding PTSD and other disorders. First an overview of different disorders overlapping with PTSD. Secondly a discussion on areas of interest in PTSD.

2.4.1 Disorders that overlap with PTSD. Looking at different diagnosis in the same study can yield some insights into the differences in underlying both biological and neurological differences. Rachel Yehuda (2000) summarized some differences in biological markers between ordinary stress and a PTSD stress response. Stress often relates to increased levels of cortisol. This cortisol release seems to be related both to the intensity of the stressor and be equal to the doses of Adrenocorticotrophic hormone (ACTH), which in turn relates to the doses of Corticotrophin-releasing factor (CRF). However, as one look at these biological markers of PTSD, the hormone doses are not presenting the same picture. In PTSD there seems to be a low dose of cortisol and a high dose of CRF, this indicates that these hormones in PTSD have a different function than in everyday stress. Also, administration of synthetic cortisol, show differences in the response between acute stress and PTSD. Underlining that acute stress and PTSD are different in their neural markers and epigenetic influences, even if the behavioral characteristics are similar (Gilboa, 2015; Yehuda, et al., 1993).

In a study by McFarlane, Atchison, and Yehuda (1997), they found that patients with low levels of cortisol immediately after the traumatic event (car accident) were more likely to develop PTSD. These results indicate that sensitivity to cortisol release may have a role in the development of PTSD. These findings also have support by a review article by Yehuda, McFarlane, and Shalev (1998), where they summarized findings from cortisol analyses in the emergency room, and its relation to the development of PTSD. In short, they found that the response of those who developed PTSD included increased heart rate and lower cortisol levels than those who did not develop PTSD.

PTSD in comparison to anxiety disorder and specific phobia show differences in biological response. A meta-analysis by Etkin and Wager (2007) on neural activation among

anxiety, phobia, and PTSD also found significantly different markers of the different disorders. In anxiety disorders and specific phobia, they found hyperactivation in the amygdala and the insula, whereas this was not found in PTSD as consistently. However, the PTSD patients were the only ones showing hypoactivation (Etkin & Wager, 2007).

Areas with hypoactivation in PTSD were the inferior occipital gyrus, ventromedial PFC, parahippocampal gyrus, lingual gyrus, dorsal amygdala, anterior hippocampus, orbitofrontal cortex, putamen, middle occipital gyrus, dorsomedial PFC, dorsal anterior cingulate cortex and middle cingulate cortex. Correlation analysis showed several areas correlating with severity of PTSD symptoms (Etkin & Wager, 2007). The hypoactivity in the rostral anterior cingulate cortex, dorsal anterior cingulate cortex, thalamus and ventromedial PFC were seen more frequently in PTSD than any of the other disorders. Whereas hypoactivation (in contrast to hyperactivation) of thalamus were found more frequently in PTSD than controls (Etkin & Wager, 2007).

In summary, there is several biomarkers involved in the reaction to trauma. Several of these markers can be seen directly after the trauma, and therefore highlight at risk people. Also, other disorders have insights to add, as the brain activation differ between the disorders.

2.4.2 Areas of Interest in PTSD Research. In a review by Shin and colleagues (2006) they reviewed regions of interest in PTSD. The regions studied were the amygdala, the medial PFC, and the hippocampus. Here is a presentation of the areas in previously presented order.

Shin and colleagues (2006) found that there are contradictory results regarding the amygdala, even if five of the reviewed studies found that the amygdala was hyperactive. The amygdala has also been discussed by Etkin and Wager (2007) in their meta-analysis in which they found that the amygdala was hyperactive in anxiety and phobias, but not consequently in PTSD, indicating a controversy in the involvement of the amygdala in PTSD. They also discuss that the amygdala was hyperactive in PTSD, anxiety, and phobia, in addition to fear conditioning in healthy subjects. Also, Suvak and Barrett (2011) discussed the involvement of the amygdala in PTSD and concluded that the amygdala is present in almost any emotion, both positive and negative, and probably is involved in the detection of novel stimuli rather than fear or stress.

Suvak and Barrett (2011) also question if the amygdala is needed for fear at all. Several of the mouse models put forth have not taken all the potential fear responses into account when examining the role of the amygdala. In humans, recognition of fearful faces

does not depend only on amygdala functions. Even if the amygdala is activated regarding the perception of fear (Suvak & Barrett, 2011). The amygdala has been proposed to serve a function in novelty detection, vision, and memory, besides the herein discussed involvement in all emotion processing. Thus, suggesting that the amygdala is a core structure, that is involved in many different tasks and as such may not be connected to fear, fear processing or PTSD-impairments per se, but rather causes emotions that are involved in these states (Suvak & Barrett, 2011).

The amygdala has also been studied in human case studies, including the case of a person called S.M (Feinstein, Adolphs, Damasio, & Tranel, 2011). S.M. is unique due to a bilateral amygdala lesion and show no signs of fear or reported fear in any of the experiments. This may suggest that the amygdala plays an important role in fear detection and fear memories. S.M. also have intact emotions and brain structures beside from the amygdala lesions (Feinstein, Adolphs, Damasio, & Tranel, 2011).

The amygdala has been proposed to be involved in fear, however new insights question these thoughts. The amygdala is involved in several functions including monitoring and reactions to novel stimulus, in addition to the feeling of fear. However, the involvement of the amygdala seems to be complex and contradictory.

The second region of interest suggested by Shin and colleagues (2006) is the medial PFC. They suggest that functional studies have not been able to detect increased activation of the medial PFC, specifically the anterior cingulate cortex and the medial prefrontal gyrus. Shin and colleagues also review a link between the amygdala and the medial PFC even if they state that the causality is yet to be determined.

Etkin and Wager (2007) also review areas included in the medial PFC along other areas, suggesting that areas of the anterior cingulate cortex and PFC are hypoactivated in PTSD. However, as there has been no distinction of the function of specific areas of the medial PFC, this is needed in future research and suggest that these areas are related to executive control and emotion generalization, as well as emotion regulation. They state that they “propose that hypoactivation of the rostral anterior cingulate cortex and ventromedial PFC in patients with PTSD reflects a deficit in reflexive emotion regulation processes...” (Etkin & Wager, 2007, p. 1484). Thus, suggesting that the involvement of the medial PFC is due to the regulative properties in any emotion.

Suvak and Barrett (2011) discuss parts of the medial PFC suggesting that the hypoactivation in these regions can be due to a complicated relationship between inhibitory and excitatory regions, including the spinal cord and the brainstem. This relationship suggests

that this causes a “reduced cortical oversight of bodily responses” (Suvak & Barrett, 2011, p. 7). Suvak and Barrett also put forth other tasks where the dorsal and ventromedial PFC are involved, such as emotion, person perception, object perception and memory functions. Thus, saying that the involvement of PFC may be due to another task, and not specifically related to fear or PTSD (Suvak & Barrett, 2011).

The PFC is a relatively big area, thus the results from this region can be mixed due to more specific functions of smaller parts. The amygdala and part of the medial PFC is connected. Several other areas are also connected to the medial PFC suggesting that is important of some functions of fear.

The last region suggested by Shin and colleagues (2006) to be important in PTSD is the hippocampus. Based on the research that Shin and colleagues had reviewed, they suggest that the hippocampus does have decreased volume, but mixed results when it comes to the functional analyses. However, looking at Etkin and Wager’s (2007) meta-analysis, they found hyperactivation of the parahippocampal gyrus when looking at emotional processing.

Besides the areas suggested by Shin and colleagues (2006) other areas have also been suggested in the involvement of PTSD. The insula is one area related to fear-learning, however with mixed results (Suvak & Barrett, 2011; Etkin & Wager, 2007). Suggesting that insula may be important in some other function parallel to fear-processing. Also, Lanius and colleagues (2001) suggest other areas, including decreased activation in the thalamus and the anterior cingulate cortex in PTSD.

When comparing brain activation between subjects with and without PTSD, increased activation was seen in anterolateral PFC (Bremner, et al., 1999), posterior cingulate cortex (Bremner, et al., 1999), motor cortex (Bremner, et al., 1999), left ventral/subcallosal (Thomaes, et al., 2011), left dorsal anterior cingulate cortex (Thomaes, et al., 2011), and the dorsomedial PFC (Thomaes, et al., 2011). Decreased activation was seen in the subcallosal gyrus and the parietal cortex (including supramarginal gyrus, right hippocampus, visual association cortex, fusiform gyrus and dorsolateral PFC (Bremner, et al., 1999). However, some areas also show increased activation in both groups, suggesting some other parallel function (incl. cerebellum, thalamus, uncus, left inferior frontal gyrus and the temporal pole (Bremner, et al., 1999).

Neural findings regarding PTSD has shown inconsistencies in the involvement of brain areas. However, previously thought areas may not be of relevance for trauma processing, but rather other more common processes.

2.5 Treatment Options of PTSD

PTSD has two first-line treatments including behavioral therapy and drug treatment, with selective serotonin reuptake inhibitors (SSRI) and serotonin-norepinephrine reuptake inhibitors (SNRI; Kirkpatrick & Heller, 2014). There are several other drug therapies included in the treatment of PTSD, but they often have significant side effects and as the etiology of PTSD is not fully understood they may harm treatment in the long run, rather than curing the patient (Kirkpatrick & Heller, 2014). Behavior therapy includes cognitive behavior therapy and exposure therapy (Kirkpatrick & Heller, 2014). However, the treatment with behavioral techniques require psychological stability to be effective, and thus the patients may need a pretreatment period with other exercises to get psychologically stable (Kirkpatrick & Heller, 2014). This section will discuss treatment options that are effective in the treatment of PTSD.

PTSD has its onset six months after the trauma occurred (Morrison, 2014). Therefore, it may be beneficial to find interventions to treat PTSD before it has its onset. More than 78% of patients with acute stress disorder (ASD) develop PTSD (Kearns, et al., 2015). However, not all people that got PTSD had ASD. There is as such no perfect predictor in the prevention of PTSD. For example, Zohar and colleagues (2011) found that treatment with hydrocortisone immediately (in 6h) after the traumatic event reduced both ASD and later PTSD onset.

Several medications have been tested but also here with mixed results. Some examples are benzodiazepines, morphine, propranolol, and norepinephrine. Taken together these studies seldom meets criteria for randomized controlled trials and show mixed results. Some studies show significant results and others show no results or even worsening of symptoms. So, these lines of prevention need more examination before they can be used to prevent PTSD (Kearns, et al., 2015).

Psychological debriefing (PD) is a technique where the victim talks about the event in a specific manner, depending on the framework of PD. It has received critique as the results are mixed. More research is needed to understand when and why we should use these different versions of PD. One such critique showed that the results of PD on motor vehicle accident victims were depending on their initial levels of impact the event had on the participants (Mayou, Ehlers, & Hobbs, 2003). This study shows that there is a more complicated view of PD, than have been investigated so forth, and concludes that a one session intervention may cause more harm than help (Mayou, et al., 2003).

Extinction of learned fear and habituation have been proposed to be one vital

part of PTSD. Animal models have suggested that the length between the traumatic event and onset of extinction have a significant role in the effect of extinction training. The relevance of time between a fearful event and its extinction has also been suggested in fear conditioning (to pair fearful stimulus to a response) in humans. Participants that got extinction training (to reduce learned fear) 10 minutes after conditioning had lower levels of fear-potentiated startle (stress) than those who got extinction training 72 hours after conditioning (Kearns, et al., 2015).

In sum, the treatment of PTSD has different approaches, including both pharmacological and behavioral aspects. The pharmacological aspects may not be as well studied to be used. Whereas the behavioral aspects have different properties, and it is useful to find ways to use individual treatment plans.

2.6 Introducing the Psychological Construction Approach to PTSD

The standard of theories has long been to look at a specific task, or a specific region of interest. However, as the equipment and mathematical models get more and more robust, new ways and possibilities emerge. This evolvement has led to the evolvement of network-based theories and looking at projections, connections and clusters of areas that are involved in different tasks. This can be compared with the evolutionary shift in focus where Darwin wrote “On the origin of species”, where he noted that there is variation in populations, rather than visual characteristics that separated individuals (Barrett, 2013). In the science of emotions this can be seen in topological thinking (e.g. labels of fear, sadness etc.) versus the psychological construction approach that think more of categories that do not share labels rather they share basic functional core systems. The core systems then create byproducts that we observe as emotions (Barrett, 2013). In PTSD the fear conditioning paradigm has been the most prominent after the stress response theory. However, this has several flaws and cannot explain all the criteria of PTSD. As a response to these matters, Suvak and Barrett (2011) have put forth a framework from which one can understand PTSD and other emotion regulation related diseases. They call their theory the *psychological construction approach*, a brief introduction to their thinking and the neural support for this approach will follow.

According to Suvak and Barrett (2011), three assumptions need to be accounted for in order to understand the theory. First, fear as a mechanism allowing us to detect and respond to dangerous situations may be different between individuals and situations. Most of the research done on fear and fear conditioning uses paradigms that cause specific behaviors

(mostly in animals) that we label as a fear response. However, there are more responses to fear than those accounted for in fear conditioning research.

Secondly, we need to think of fear as an emotional response to something, internal or external. With this said increased heart rate may be the result of fear, stress, or excitement. Moreover, several other changes from homeostasis in the body create a similar pattern of hormonal and neural changes. Thus, we can also question if what we know of specific brain areas are involved in the suggested tasks. For example, is the amygdala necessary for fear perception, or can we have fear-perception without the amygdala?

Third, the psychological construction approach suggests that we shall not look at specific functions but instead look at the interplay of more basic functions and that these connections that form what we call emotions. In their early model they challenge several points in the current state of research; including the heterogeneity of PTSD, the unspecific frames in categorizing disorders, that emotions and affect is not what we search for (rather we search for what build emotions) and that these areas are parts of a broader network doing other things that create emotions. The most significant contribution of this theory is thus to distinguish between the psychological concepts about affect and the corresponding neurological parts of an emotion.

In the psychological construction approach, however, they have expanded their early model saying that a psychological construct will not tell the various parts that it is built from. Evidence for this notion is that several areas throughout the brain are engaged in several functions. For example, the amygdala that is engaged in the detection of salient stimuli and memory, along with the insula that is involved in task switching and memory. Their notion is as such that several areas may be involved in the creation of emotion, as they form a network of other functions.

Suvak and Barrett (2011) use the term "*core affect*", which is the affect felt by the individual. For example, every emotion can be broken down into its physiological parts (e.g., arousal, heart rate, and all other functions of the nervous system). This core-affect is then built by networks that put together information from within the body, with information from environmental and external information, and then creates a behavioral response to this information. The response-network then creates a feeling of comfort/discomfort mixed with different degrees of arousal, going from specific input to a multimodal integration of the state of the body. This response is the sum of its parts and differs from situation and individuals. The response is further thought to be a learned response, so if a situation triggers the same physiological cascade of responses several times, it becomes habituated (Quigley & Barrett,

2014).

This core-affect network is also involved in restore and regulates the homeostasis of the body, in response to salient stimuli. This is further suggested to be involved in first impressions, emotion regulation, moral decision and the emotional experience and perception. These networks continuously receive input and build a new coherent picture of what the state of the moment is and affect occurs throughout this process. This information can also change with a change in executive functions, conceptual activation, and focus, or any number of combinations among these changes (Suvak & Barrett, 2011). In this contextual frame of autonomic nervous systems response, there is some support for the notion that similar responses are present across emotions (Quigley & Barrett, 2014), their summary of evidence reviewed is that more research is needed, and that consistency is more robust than specificity at this point.

The PTSD diagnosis is characterized by intrusive thoughts, emotion dysregulation, avoidance and negative mood (Morrison, 2014). The psychological construction approach looks at impairments in neural processing, and the mix of information is interpreted as important to act upon or not. Due to an increased focus on internal information, this change in focus they say may be due to heightened activation of the amygdala (saying cues are important to act on) and reduced cortical oversight of autonomic responses. The processing in PTSD is therefore not related to fear per se, but rather an effect of the working networks within the brain and the increased response to stimuli as salient and important to act upon. These networks are supposed to include the amygdala, among other regions. Also, the insula is involved in creating the representation of affect and emotions that arise from internal somatosensory cues.

This theory also explains the link between hyperarousal and the intrusive thoughts saying that persons with PTSD are experiencing pictures from the past that are uncontextualized and thus creates both arousal and intrusive thoughts, which is supported by increased activation in the hippocampus and the parahippocampal gyrus (Etkin & Wager, 2007). As such this framework, even if it is in its infancy, explain several features of PTSD. Further support is that increased arousal may lead to intrusive thoughts (Nixon & Bryant, 2005) Also, research of neural networks support notions of this hypothesis, including the default mode network and the network for executive attention (Suvak & Barrett, 2011). However, more research is needed within this framework to establish its accuracy (Suvak & Barrett, 2011).

2.7 Summarizing PTSD

Recent research and models of PTSD have started to challenge the stress and fear frameworks in which much of the PTSD research has its roots. The studies summarized show some controversies where neither the amygdala nor the insular is involved in the evolution of PTSD. More research is needed to find relevant connections and interactions between areas that supposedly are involved in PTSD and the maintenance of the same. The psychological construction model is the most comprehensive theory, as it involves almost all features of PTSD. However, this model does not take treatment into account and is still in its infancy.

3. An Overview of Post-traumatic Growth

Not all the people that experience a traumatic event get PTSD. However, traumatic events are common. For example, a study in the US found that 21% of the 1,000 participants had experienced a traumatic event in the past year and 69% had experienced at least one traumatic event in their lifetime (Norris, 1992). However, all these 69% do not get PTSD, which further highlights the importance of understanding the other outcomes of trauma. In answer to this, recent research has focused more on these individuals that do not get diagnosed with PTSD and the reasons behind that. When looking at positive outcomes of trauma somewhere between 30 and 70 % report that they have had some benefit or growth from the trauma or adversity they experienced (Joseph & Hefferon, 2013). It is here important to note that trauma can cause both positive and negative outcomes in different or the same timeframe. This chapter will cover a definition of post-traumatic growth (PTG), some studies and reviews using PTG as a construct in addition to constructs overlapping with PTG, a discussion about the mindfulness to meaning theory, how to measure PTG, and neurological findings.

3.1 Characteristics of PTG

PTG is a subjective positive psychological change because of a traumatic experience and thus leads to withstanding cognitive and affective changes that follow this traumatic event (Nakagawa, et al., 2016). The traumatic event causes a person to rethink its assumptions about life (Nakagawa, et al., 2016). PTG has several names such as adversal growth, benefit finding, stress-related growth among others. However, Calhoun and Tedeschi (1998) coined the term PTG, which is the name used in this review.

PTG and outcomes after trauma are related to several factors that increase or decrease the result of the event (e.g., increase and decrease PTG and PTSD symptoms). Some of these factors are: (a) the severity of the trauma, both as an objective and a subjective measure and (b) reactions of the person immediately after the trauma (Blix, et al., 2013). Further, age also affects the severity of trauma (Kirkpatrick & Heller, 2014).

PTG has been seen to involve several areas of optimal functioning. There is no uniform definition, and researchers have proposed different models. For example, (Picoraro, Womer, Kazak, & Feudtner, 2014) has found five domains in which growth occur after trauma:

1. Greater appreciation for life
2. Better relations with other people

3. A greater strength within the self
4. A better understanding of new ways to live life
5. Greater spirituality and religious beliefs.

Moreover, Shaw, Joseph, and Linley (2005) state that there are changes in three dimensions: [1] enhanced relations, [2] changes in their self-view, and [3] alterations in life-view. Also, there are overlapping domains, and these may or may not reflect the same underlying constructs involved in PTG. This is also seen when looking at the different definitions of PTG.

There is, however, no precise and uniform definition of what the term PTG is and what it includes. Therefore, it is also hard to establish how many that evolve PTG after a traumatic event (Shand, Cowlshaw, Brooker, Burney, & Ricciardelli, 2015). Linley and Joseph (2004) suggest that one person can have both PTSD and PTG, but at separate times following the event, such as PTSD may trigger PTG and that PTG may reduce PTSD. There may also be possible that these constructs can co-occur. For example, Calhoun & Tedeschi (2004) argue that the different areas may be correlated to different events, or at different time points. Linley & Joseph (2004) found mixed results when correlating subscales with different traumatic experiences.

There are also further discussions on how one should define this term. PTG may be the opposite of PTSD (e.g., to grow from traumatic experiences as defined by DSM characteristics) or it may be a broader term (e.g., growth from stressful events). However, using the broader term, it cannot be contrasted with PTSD as there are not the same triggering events. If the narrower definition is used, there is no growth due to stress even if it is the same neurological mechanism that underlies the growth. However, given that the definition is not clear this essay will have to focus on them separately.

PTG and post-traumatic stress symptoms (PTSS) do not correlate in a linear manner, but rather as an inverted u-shaped curve (Nakagawa, et al., 2016). There is a stronger relation between PTSD-symptoms and PTG for younger individuals, but the results are mixed and do not conclude a precise definition on what relates to PTG (Wu, et al., 2016). PTG as defined above is not merely the opposite of PTSD, but rather a complement. PTG and PTSD rest upon two separate set of assumptions, where PTSD is a disorder. Whereas most research on PTG shows it as a process rather than a state, however, PTG can be both a state, trait and process. Most people investigating these constructs do not separate or discuss this further in the operationalization of their construct.

To summarize, PTG is not the opposite of PTSD. These constructs can be seen together or alone in the same individual over time. PTG is from now on seen as a process rather than a state, even if some of the articles put forth in the following does not state that explicitly. PTG divides into several aspects that may overlap to some extent. There may be three or more construct, however; they fall into three categories:

1. reevaluation of the self (values, priorities, and schemata),
2. the relations to others,
3. the relation to the world (physical and spiritual).

Following will be a deeper discussion about constructs that may overlap or will be affecting the development of PTG in some way or the other.

3.2 PTG in Relation to Overlapping Constructs.

Calhoun & Tedeschi (2004) argue that their measure of post-traumatic growth (the post-traumatic growth inventory; PTGI) has five dimensions, and each of these dimensions are separate from the other. They further suggest that different traumas may affect the different dimensions in a different manner (Calhoun & Tedeschi, 2004). It is therefore essential to look at another dimension of well-being and to which extent these other dimensions overlap with the construct of PTG. In this session, a brief discussion about some other dimension will be made, including religion, spirituality, optimism, resilience, mindfulness, rumination, reappraisal and social support.

Starting with religion and spirituality. Religion is “the tendency to adhere to religious beliefs and to engage in religious practices”, while spirituality “more specifically [is] a concern for God and a sensitivity to religious experience, which may include the practice of a particular religion but may also exist without such practice” (American Psychology Association, 2018). Religion have far-reaching history and several of the religions present today has a belief about trauma and the meaning-making after such an event (Shaw, et al., 2005). Shaw and colleagues (2005) in their review found that spiritual and religious goals were related both to recovery and meaning derived from the trauma. However, religion can be divided into several aspects, including internal religion (the relationship with God) and extrinsic religiousness (the people and places connected to engaging in religious practices). It can also be a coping strategy to pray. Jeon, Park, and Bernstein (2017) looked at different coping strategies and found that those who prayed (or visited a health specialist) had higher scores of PTG, an indication that prayer as a coping strategy is important for growth

after trauma. Religion as a coping strategy was also investigated by Prati & Pietrantonio (2009) in their meta-analysis. They found that both religious coping and spirituality were related to growth (strong and moderately, respectively). In their discussion, they argue that this affect may be due to their meaning-making properties. The results of religion may also be due to the social support and engagement it brings to the person (Prati & Pietrantonio, 2009).

Zoellner and Maercker (2006) looked instead at meaning-making. They state that those who were prone to search for meaning in the trauma had a higher amount of PTG when also reaching criteria for PTSD further underlining the importance of religion as a multifaceted construct about PTG. Also, Calhoun, Cann, Tedeschi, and McMillan (2000) found that openness to religions had a positive effect on PTG development.

Optimism is defined as “the attitude that good things will happen and that people’s wishes or aims will ultimately be fulfilled” (American Psychology Association, 2018). Research on optimism and PTG suggest that they overlap, but is distinct (Zoellner & Maercker, 2006). However, as an extension on this statement, Prati & Pietrantonio (2009) argued that even if they are overlapping this may be due to the ability for flexible coping and threat appraisal as optimism does not link to meaning-making (Prati & Pietrantonio, 2009; Zoellner & Maercker, 2006). However, more research is needed, as the results are mixed (Zoellner & Maercker, 2006).

Resilience is “the process and outcome of successfully adapting to difficult or challenging life experiences, especially through mental, emotional, and behavioral flexibility and adjustment to external and internal demands” (American Psychology Association, 2018). Resilience is a dynamic developmental process, whereby PTG is transformative and due to a traumatic event. Jeon and colleagues (2017) in their study looked at Korean Americans and found that those low in resilience had lower levels of PTG, suggesting a link between these constructs.

Rumination is “obsessional thinking involving excessive, repetitive thoughts or themes that interfere with other forms of mental activity” (American Psychology Association, 2018). On the other hand, it is this thought-process that tries to make sense of the external world. Calhoun and Cann (2000) state that rumination is a recurrent event related to thinking and involves sense-making, reminiscence, problem-solving, and anticipation. Rumination can be deliberate or intrusive. The deliberate rumination is characterized by trying to reconceptualize what has happened (Tedeschi & Blevins, 2015), which is important for the recovery after the trauma, as has also been suggested in the PTSD literature (Foa, Riggs,

Dancu, & Rothbaum, 1993). Intrusive rumination, on the other hand, is the negative dwelling in the event. As such it is related to negative emotions and inability to cope with the stressor (Cann, et al., 2011).

Zoellner and Maercker (2006) argue that the literature on the relation between rumination and PTG are very shallow. The review articles did not separate between various kinds of rumination, as such there is not any good evidence, even if there seems to be a beneficial aspect of rumination. Calhoun and colleagues (2000) regression model found that two of the variables significantly affected the PTGI scores, including positive rumination and how open a person was to religious change.

Re-appraisal is the ability to restructure event and is a part of the rumination process and mindfulness model. Tedeschi and Blevins (2015) state that the ability to shift between deliberate and intrusive rumination is through mechanisms of mindfulness and reappraisal, where reappraisal is the ongoing restructuring of the event. Also, Zoellner & Maercker (2006) include reappraisal in their review suggesting that positive reappraisal is related to PTG. Moreover, this is one suggested route between trauma and PTG. Furthermore, the relation of reappraisal to growth can be linked to the interaction between mindfulness and reappraisal, which over time strengthens with practice (Garland, Kiken, Faurot, Palsson, & Gaylord, 2017)

The last aspect related to PTG to discuss in this section is social support (for a more comprehensive review see: Prati & Pietrantonio, 2009; Zoellner & Maercker, 2006). Firstly, it is important to note that social support has several benefiting aspects, including perceived support, seeking support among other aspects (such as quality and positive support). In their meta-analysis, Prati and Pietrantonio (2009) found that both social support and seeking social support is related to PTG. Seeking support, they argue, may be due to the increased number of relations and better quality in these relations. Social support may be beneficial in dealing with trauma. However, studies have found that the importance of others is related to different timeframes after the event (Prati & Pietrantonio, 2009).

PTG is a construct in its infancy, but as more research has focused on this construct, it can tell that PTG is a product of changes in beliefs or worldview. A sense of getting along with the traumatic event and find something that makes us continue and develop as a person. Religion, spirituality, optimism, resilience, rumination, reappraisal and social support are all factors that contribute to the development of PTG but are distinct in its definition and underlying processes, as seen in the correlations that are present/absent at different time points.

3.3 How to Measure PTG?

Several different scales that measure PTG is in use, including the Change in Outlook Questionnaire (COQ; Joseph, et al., 2005), Post-Traumatic Growth Inventory (PTGI; Tedeschi & Calhoun, 1996), Stress-Related Growth Scale (SRGS; Park, Cohen, & Murch, 1996), Revised version of the Stress-Related Growth Scale (SRGS-R; Armeli, Gunthert, & Cohen, 2001), Perceived Benefits Scale (PBS; McMillen & Fisher, 1998) and Psychological well-Being Post-Traumatic Change Questionnaire (PWB-PTCQ; Joseph, et al., 2012).

- The COQ measures positive and negative outcomes after trauma, on a 26-item questionnaire. This scale involves two subscales, one positive and one negative (Joseph, et al., 2005).
- The PTGI measures 21 items divided into five subscales. The subscales included is perceived changes in self, development of close relationships, changes in life philosophy, changed priorities, and enhanced spiritual beliefs (Tedeschi & Calhoun, 1996).
- The SRGS measure 50 items, have one factor: growth (Park, Cohen, & Murch, 1996). This test has been revised (SRGS-R), the updated version includes 43 items on five subscales. The subscales are relating to others, new possibilities, personal strength, spiritual change, and appreciation of life. The PTGI and the SRGS-R have both five subscales and measure similar constructs. No comparison between the psychometric properties has been found.
- The PBS (McMillen & Fisher, 1998) measure 38 items on eight subscales. The subscales include enhanced self-efficacy, community closeness, spirituality, compassion, faiths in people, lifestyle changes, family closeness, and material gain. This scale involves several of the subscales included in other measurements. However, it also includes some other subscales (e.g., material gain and compassion).
- The PWB-PTCQ is derived from Ryff's psychological well-being theory and suggests that growth after trauma is related to an increase in psychological well-being (Joseph, et al., 2012). The scale consists of 18 items measuring six subscales including environmental mastery, autonomy, purpose in life, positive relations, and self-acceptance.

However, there is no unified definition of what is involved in PTG, neither of the number of dimensions (Linley & Joseph, 2004). Linley and Joseph (2004) highlight some further notions for the study of these constructs as most of these presented questionnaires only

measure positive events following trauma (except revised stress-related growth scale and outlook questionnaire). To fully understand what happens after trauma there needs to be a picture including as many aspects as possible, both positive and negative. The measures of PTG is not compared directly, and thus there is no answer if they measure the same or different constructs.

3.4 Neural Aspects of PTG

The neural underpinnings of PTG are still in its infancy, and research looking at these aspects is rare. Therefore, this section includes only three different studies.

Nakagawa and colleagues (2016) investigated the dorsolateral PFC and its association on the levels of relation to others (in PTGI). They found that delta-rGMV (regional grey matter volume) in the DLPFC correlated with total scores on the PTGI and the relation to other dimension (positive). A regression model also showed that this correlation was due to the PTGI scores and “relation to others” dimension (Nakagawa, et al., 2016). They also found increased grey matter volume in the right DLPFC correlated positively with the PTGI sub score of relating to others and negatively related to post-traumatic stress symptoms. However, this study found no correlation with areas of ACC, ventromedial PFC or the left middle temporal gyrus, which indicates that these regions may not be important for PTG, even if other studies (Nakagawa, et al., 2016) has related PFC and dorsolateral ACC activation to resilience score.

Fujisawa, Jung, Kojima, Saito, Kosaka, and Tomoda (2015) found correlations between the scales of depression and PTG and between post-traumatic stress symptoms and depression in healthy individuals. However, they did not find a significant correlation between PTG and PTSD (measured in symptoms). When looking at correlations between scales and brain regions they found that the PTGI correlated positively with activation of the rostral PFC and the superior parietal lobule, indicating the default mode network as relevant, and no other networks reached significant correlations. However, they found that the activation was stronger when the PTGI score was higher. They also performed a seed-based analysis which showed that stronger connectivity between the superior parietal lobule and the supramarginal gyrus were correlated with the scores on PTGI (Fujisawa, et al., 2015).

Wei, Han, Zhang, Hannak, Dai, and Liu (2017) used functional near-infrared spectroscopy to examine prefrontal activation, in addition to heart rate variability in explosion victims. Further, Wei, Han, Zhang, Hannak, Dai et al., (2017) suggest an association between PTGI and increased activation in the left dorsolateral PFC, in comparison to participants with

PTSD, and controls. They also found that the heart rate variability was different between groups, suggesting that people high in PTG process positive stimuli in a different manner than both controls and PTSD individuals (Wei, Han, Zhang, Hannak, Dai et al 2017).

Neural studies do indicate that the PFC plays a significant role in the development of PTG. However, more research and a more precise definition of PTG is needed, and so are a standard for using the data and correlation to different subscales before any conclusions can be drawn.

3.5 Introducing the Mindfulness to Meaning Theory to PTG

Mindfulness has been widely used to relieve psychological distress. However, the link between mindfulness and positive functioning have been paid less attention. Garland and colleagues (2017) proposed a model of which mindfulness is a key in positive functioning, due to its ability to create a sense of living in the present. Their model called the *mindfulness to meaning theory* (MMT), also suggests that periods of mindfulness are mixed with periods of rumination and thus create a sense of the world. In this section, the discussion will cover a more detailed description of this model and its relevance to PTG.

Garland and colleagues (2017) argue that mindfulness affect distress through the mechanism of reappraisal. However, Tedeschi and Blevins (2015) expand this model also to be relevant for PTG. They argue that trauma is important for the initiation of rumination, and as such relevant for the growth process. Tedeschi and Blevins (2015) argue that re-appraisal shares features with rumination and has further divided rumination into two categories (see rumination section above). Further Garland and colleagues (2017), argue that mindfulness is important to bring the negative event back to a low arousal state (as previously discussed in PTSD chapter), which makes it a likely process in recovery. In short Garland and colleagues (2017) provides three suggestions.

1. That negative mood tends to dampen the ability to look at the world positively, and that the possibilities come with creating a state of low arousal. As such, mindfulness is suggested to be the key switch between these states of mind.
2. The second thing Garland and colleagues state is that while this process is ongoing, it also affects states of well-being. More specifically they argue that the eudaimonic emotions (the deeper feelings of well-being) are changeable and broadened with the ongoing process. The mindful low arousal state is connected to more eudaimonic emotions, whereas the high arousal negative state is related to low eudaimonic well-being. As this process progress the low arousal state becomes more frequent, and as

such also the eudaimonic emotions broaden and deepen. Hedonic emotions (feelings of joy and happiness) are suggested to play a part as well, even if these only strengthen the eudaimonic emotions.

3. Garland and colleagues' (2015) third aspect are that mindfulness modulates this process via re-appraisal. Reappraisal in this sense is the processes of making stressful events seem more benign or meaningful (e.g., positive reappraisal). They state that "...mindfulness enables reflective processes to magnify the affective benefits of positive re-appraisal and generate eudaimonic well-being" (pp.296). On this note, re-appraisal is the key to turning negative appraisals, via new evaluations of the stressful event into positive, benign or meaningful appraisals.

To underline the importance of this process, they summarize some neuroscientific evidence. For example, that the dorsal and ventral aspects of the PFC associate with evaluating self-relevance. Further evidence is that the ventral PFC functionally connects with the amygdala, nucleus accumbens and the insular, that previously in this review is linked with emotions in PTSD. Also, the ventral PFC is related to affective judgments and that the dorsal PFC is involved in the monitoring without judgmental aspects (Garland, Kiken, Faurot, Palsson, & Gaylord, 2017). Mindfulness is also related to several other aspects like increased sensory representations and a shift to decrease the affective salience of situations (through a changed pattern of dorsal and ventral PFC activation). As such the attentive focus on, for example, the breath forces the individual to readjust the neural activation pattern to a more focused and nonjudgmental state.

Garland and colleagues (2017) did a study to test their theory, recruiting participants for an 8-week mindfulness-based stress reduction program to examine both state-mindfulness and reappraisal. In sum, they saw that deepen state-mindfulness were associated with increased use of positive re-appraisal. They also suggested, through their models that these two trait-like constructs strengthen each other with time (Garland, Kiken, Faurot, Palsson, & Gaylord, 2017).

The MMT suggests that we make sense of the world around us in small shattered pieces, alternating mindfulness, and rumination, which causes a sense of meaning and processing which the individual can accept and build a new assumptive world.

3.6 Summarizing PTG

PTG can be a state, or a trait or a process, which remains to be clarified. There are several definitions, theories and measurements about PTG. This includes several different

names and overlaps with other constructs that causes growth not related to trauma. Defining PTG is an open question in which it could be the opposite of PTSD, or it can be another concept than PTSD, or it can be different forms of trauma included. What can be said is that PTG is not optimism, resilience or religiousness. Research needs to focus more on a common ground to stand on when looking at these aspects of trauma to find a relevant frame. There is also a need to establish a unified definition, and to divide neural processing between PTG and other related constructs. However, time will bring answers to this ongoing debate.

4. What we can Learn from the Intersection of PTSD and PTG

A gap exists between PTSD and PTG, and current research investigates how and why the gap exists. What affects PTSD prevalence and how we can cure PTSD when it has occurred, is important. The following section will examine the differences found when examining PTSD and PTG in the same person, based on specific traumatic events. The two earthquakes Wenchuan and Yaán, the explosion in Tianjin and the Oslo bombing. The last subsection includes a discussion about various traumas.

4.1 Findings from Earthquakes

Wenchuan was registered at 8.0 on the Richter's scale and located in the county of Wenchuan in southwest China. In this catastrophe about 70000 people died, 400 000 people were injured, and 18 000 were missing, and caused serious economic problems and losses. In a study investigating the result on PTSD and PTG in survivors of the earthquake Wenchuan, Wu and colleagues (2016) had the aim of looking at coexistence of PTSD and PTG. They investigated age, gender, income, amount of exposure to the earthquake, ethnicity, education and current housing status. Wu also measured resilience and social support. In the investigated sample, 40% were classified with PTSD according to the PCL-C, and 51.1% showed moderate levels of PTG using the PTGI. Measures were gathered a year after the earthquake. Results show that several of these factors caused changes in both PTSD and PTG, including that those high in PTSD symptoms also were high in PTG score. Confounding factors that were related to having both PTSD and PTG were level of income, current housing status and amount of social support. The participants showing elevated levels of both PTSD and PTG were more likely to be aged 31-50 and had a temporary house, lower income, and less support compared with the other groups (Wu, et al., 2016).

By looking at the aftermath of Wenchuan Jin, Xu, Liu, and Liu (2014) tried to answer the question how the state of PTSD and PTG were after one year. PTSD was more likely to occur in females, young individuals (18-30y) and those with a lower level of education. The youngest age group were also those with most PTG (Jin, et al., 2014). A regression analysis found that gender, age, education and exposure levels were all predictors of PTSD, while age and exposure level were the only predictors of PTG. Further correlation analysis found that PCL and PTGI correlated almost among all subscales and total scores, with a few exceptions. Jin and colleagues (2014) also found that women were more affected than men, using the same sample and measurements.

Longitudinal attempts are made to capture changes in PTSD and PTG. Zhou and

colleagues (2015) gather data for a three-point analysis: after 3.5 years (T1), 4.5 years (T2) and 5.5 years (T3) of the earthquake. The study aims to make a longitudinal projection to examine the evolvement of PTSD and PTG. The results from Zhou and colleagues (2015) work showed that PTSD at T1 correlated with PTG at all three points. However, PTSD at T2 did not correlate with PTG at T1, and PTSD at T3 was not correlated with PTG at T2, showing a somewhat complicated relation between the timepoints that decrease with time. Zhou and colleagues (2015) state that these relations indicate that PTSD predicts PTG but not the other way around. Suggesting that post-traumatic distress is a crucial part of the development of later PTG, even if they with time develop on different rather than shared factors.

Du, Ma, Ou, Jin, Ren, and Li (2018) also made a longitudinal attempt to capture changes in PTSD and PTG over time. The prevalence of PTSD eight years after the earthquake was 1.9% in the generally affected area (away from the epicenter) and 2.7% in the severely affected area (near the epicenter; Du, et al., 2018). The study found that ethnicity, loss of house, social support and negative coping were predictors of PTSD in the generally affected area, and that gender, ethnicity, psychological aid, loss of house, to be buried, social support, positive and negative coping were significant risk factors for the severely affected area. When looking at PTG, there were differences in PTGI total score between the areas, the generally affected area had a mean PTGI score of 39.93, and the severely affected area 54.09. The significant factors for PTG were gender, ethnicity, psychological aid, anxiety, loss of house, social support, positive and negative coping (Du, et al., 2018). Correlations between the PTGI and PCL were seen more in the generally affected area than in the severely affected area (Du, et al., 2018).

Taken together, the research related to Wenchuan found that PTSD and PTG are correlated and found in the same individuals. Longitudinal attempts found that PTSD and PTG were related but that the relation decrease with time and that PTSD predicted PTG but not the other way around. Eight years after Wenchuan, the severity of the initial event was related to the development of PTG. Risk factors included gender, age, aid, ethnicity and the severity of the exposure.

After the Yaán earthquake, Zhou, Wu, and Zhen (2017) tried to expand the knowledge they had derived from the Wenchuan earthquake. They hypothesized that emotion regulation plays a role in the development of PTSD and PTG. Zhou found a correlation between trauma exposure and both PTSD and PTG. Further, they found that social support was correlated with cognitive reappraisal and PTG and negatively correlated with expressive

suppression. Emotion regulation was correlated with measures of PTSD and PTG, however, the relationship between PTG and expressive suppression was only a trend toward significant. In this sample, PTSD and PTG did not correlate, in contrast to the previous discussion (Zhou, et al., 2017). Further analyses found that social support had an indirect effect on both PTSD and PTG through emotion regulation. Other significant effects were seen between trauma exposure and expressive suppression, social support, PTSD and PTG, but not cognitive reappraisal. However, cognitive reappraisal was affecting PTSD and expressive suppression. Social support was only affected by trauma exposure, even if it did affect all the other variables (Zhou, et al., 2017).

To further expand on their notion Zhou and Wu (2016), made a longitudinal attempt to examine PTSD and PTG after the Yaán earthquake. Yaán earthquake was placed in the south of China, in the Lushan county and reached a magnitude of 7. This earthquake resulted in about 200 dead people, 93 missing and 15 500 injured. Zhou and Wu (2016) used a three measurement points method (T1: 6 months, T2: 12 months, T3: 18 months after the earthquake; Zhou & Wu, 2016). The primary aim was to assess rumination and its relation to PTSD and PTG. In this sample, they found that intrusive rumination at T1 were predicting intrusive rumination at T2 and PTSD at T3, (but not PTG at T3). However, intrusive rumination at T2 predicted both PTSD at T3 and deliberate rumination at T2. Further deliberate rumination at T2 was predicting both PTSD and PTG at T3 (Zhou & Wu, 2016).

Taken together, findings from the Yaán earthquake adds to the previous research by extending the predictors of PTSD and PTG to rumination and emotion regulation, which both seem to play a role in the development and maintenance of PTSD and PTG along with social support.

4.2 Findings from Explosion Incidents

Findings from the Oslo bombing incident show that the amount of reaction and exposure were related to more PTG. The Oslo bombing occurred in 2011, with a car bomb from a terrorist exploding near the government building (Blix, et al., 2013). In this incident, eight people were killed and additional 209 people were injured as a result. Blix and colleagues (2013) found that PTSD was related to impaired work, social adjustments and ratings of life satisfaction. Perceived growth is related to the amount of life satisfaction, even if there were a similar amount of PTSD (Blix, et al., 2013).

In Tianjin, a series of explosions occurred at the port, 165 people died, and an additional 700 people were injured. Wei, Han, Zhang, Hannak, and Liu (2017) looked at the

aftermath of the Tianjin explosion and found a correlation between PTSD and PTG. The study showed that after this explosion PTSD rate was 16.9% and that PTG rate was 8.4%. They found that direct exposure correlated with PTSD. Explosion related stressors correlated with PTSD, PTG and direct exposure. Loss is correlated to all other variables. Further, they divided the sample into three groups and showed that reaction times were significantly different. Results indicate that both positive and negative priming (“the effect in which recent experience of a stimulus facilitates or inhibits later processing of the same or a similar stimulus”; American Psychology Association, 2018) affected the PTSD group significantly more than the other two groups (Wei, Han, Zhang, Hannak, and Liu, 2017).

Taken together the research on explosion victims showed that the more severe the exposure, the more PTG (in the Oslo sample), but exposure where also correlated to the both PTSD and PTG in the Tianjin sample. The reaction times for both positive and negative priming where longer for those with PTSD.

4.3 Correlations of PTSD and PTG in Other Traumas

Lowe and colleagues (2013) made a longitudinal attempt to look at PTG and post-traumatic stress syndrome (PTSS). They only enrolled participants screened for optimism, psychological distress and purpose 12 months before the hurricane Katrina (T1), and were assessed on the pre-disaster variables, post-traumatic stress, and event-related aspects after the hurricane (T2). At a third measurement point, three years after the hurricane, measures of PTG along with the previous measurements were made (T3; Lowe, Manove, & Rhodes, 2013). Their findings suggest that PTG at T3 were correlated with PTSS at T2 and T3, purpose at T1 and T2, along with optimism at T2. PTSS at T2, on the other hand, correlated with PTSS at T3, psychological distress at T1 and optimism at T1 and T2. Psychological distress at T1 is also correlated with PTSS at T2 and T3 (Lowe, Manove, & Rhodes, 2013).

Larsen and Berenbaum (2015) found that PTG is significantly correlated with emotional processing and meaning-making. Distress correlated with expression, suppression, and meaning-making, indicating different emotion regulation strategies for the PTSD and PTG. Further, a regression model found that meaning-making where a predictor of both distress and PTG, whereas suppression was a predictor of distress (Larsen & Berenbaum, 2015).

A systematic review by Schubert, Schmidt, and Rosner (2016) will summarize this section. They were investigating PTG in participants with PTSD and reviewed 19 studies.

Included traumas were war, medical disasters, sexual trauma, natural disasters, accidents and some studies with mixed traumas. Five studies found more growth in PTSD participants than in non-PTSD participants. However, two studies found no difference and one study found higher scores for a group without PTSD. They also found that 3 out of 6 longitudinal studies showed increased PTG with treatment. Schubert and colleagues (2016) conclude that PTG increase with time and PTSD decline with time. The methodologies are very different between the studies included. Optimism, personality, and social support were other aspects included in the systematic review, and they found that none of the factors were related to PTG unless it was dependent on the severity of PTSD (Schubert, et al., 2016).

4.4 Summary of PTSD and PTG

Summarized, the presented research suggests that PTG depends upon the amount of PTSS over longer timeframes, that PTG relates to emotion processing and meaning-making and that PTG develops if there are initial PTSD symptoms (Lowe, Manove, & Rhodes, 2013; Larsen & Berenbaum, 2015). PTS, on the other hand, is seen to relate to the amount of initial distress and optimism over time. These findings suggest that distress and post-traumatic symptoms are beneficial in the growth process (Schubert, et al., 2016; Larsen & Berenbaum, 2015). Some have even suggested that negative emotions are necessary to bring growth. However, the amount of helplessness and inability to cope and control the treatment process may dampen this growth process (Schubert, et al., 2016).

PTG and PTSD seem to occur at both different and the same time points, suggesting that they have a mutual relation even if they with time have different survival purposes. PTSD ends in PTG when the emotion regulation is effective, including optimism, positive rumination, low suppression and decreased amount of distress over time. PTG and PTSD seem to share several features, both behaviorally and neurologically, suggesting that the same areas are involved in the development of both PTSD and PTG.

5. Discussion

PTSD and PTG are both prevalent after a traumatic event. The picture evolves from fear-learning into emotion regulation, and several aspects of PTSD, PTG and the co-occurrence between them is studied. The early theories about PTSD suggest a disorder that rests upon failed recovery (Kirkpatrick & Heller, 2014). However, as the field of neuroscience has emerged, PTSD seems to be difficulties in emotion regulation. Emotion regulation is linked to the activation pattern of the PFC (Brewin & Holmes, 2003), but the activation of PFC does not explain all the four domains of PTSD; re-experiencing the event, avoidance, negative cognition and mood, and hyperarousal (Kirkpatrick & Heller, 2014). We need more functional research regarding the neural underpinning of PTSD as an emotion regulation deficit or as an intense internal focus (Suvak & Barrett, 2011).

PTG as a construct is not defined, and more research is needed to fully understand which aspects are involved and how they relate to each other. Linley and Joseph (2004) state that various elements of PTG are related to different traumas. If growth evolves differently after different traumas, we need to divide traumas into categories and be cautious when generalizing findings to the other subgroups, or types of events. In other words, we do not know enough about trauma outcomes to either clarify the construct or understand how individuals react to various kinds of traumas. Could it be that the event itself is not important, but rather how the individual cope with the event?

When considering PTSD and PTG together, new aspects evolve. The longitudinal attempts are cross-sectional, and several directions of future research appear. The most prominent finding in this regard is that we need a certain amount of PTSS to initiate the PTG process (Zhou, et al., 2015). PTSD show different processing of positive emotional stimuli, probably due to impaired or altered neural processes (Wei, Han, Zhang, Hannak, Dai et al., 2017). Differences in processing are essential to understand and find the least common ground for PTSD and PTG. The research shows that some factors contribute to PTG, others to PTSD, while some even contribute to both PTSD and PTG. Especially the factors contributing to increased PTG and decreased PTSD should be studied more closely, e.g., social support and rumination (Calhoun, et al., 2000; Jeon, et al., 2017; Tedeschi & Blevins, 2015).

The following three sections will discuss the relevance of the findings presented. First by elaborating on how we view trauma, threat and appraisals through the lens of adaptiveness. Secondly by comparing the psychological construction model with the MMT, and last by discussing clinical relevance and treatment practices regarding the full trauma

outcomes model. The chapter ends with is a discussion about limitations and further research questions.

5.1 Presenting the Full Trauma Outcomes Model

Even if this review has no clinical applications per se, this section will elaborate upon the findings to find further research areas and treatment practices. The area of PTSD is emerging and includes new insights, from the area of neuroscience, in practices and guidelines. Depicted in figure 1 is several factors proposed to play a role in the development and maintenance of PTSD and PTG. The model suggested here includes four timeframes, that is derived from the reviewed articles. For example, Kirkpatrick and Heller (2014) suggest that there are three timeframes (pre, peri, and post-trauma).

The full trauma model is different from previously presented theories in at least two aspects. First, it is divided into four time-frames, including pre, peri and two post frames (0-6 months, over 6 months). Secondly, it includes factors contributing to both PTSD and PTG, in comparison to Kirkpatrick and Heller (2014), that only includes factors contributing to PTSD. The model does not suggest that four timeframes are enough to draw a full picture, in the sense of looking from both sides of trauma outcomes.

The first time-frame is related to the time before trauma onset. Parental neglect (Kirkpatrick & Heller, 2014), social support (Jeon, et al., 2017; Prati & Pietrantonio, 2009), and resilience (Whitaker, et al., 2014) are all factors in this frame. The mentioned factors can be the target of psychological interventions and part of the education system. Thus, suggesting that we can build individuals that have a buffer against trauma before it has even occurred. Gender cannot be changed but it is possible to change how we view the different gender roles, suggesting that we can also build this factor into a buffer against trauma. The time before trauma can be long or short, and it may be beneficial to divide into even smaller timeframes. Small children may need other interventions than older children or adolescents. Adults also differ in emotion processing and regulation, in comparison to younger individuals. If prevention can target several groups and at different times it is beneficial for the creation of psychologically strong individuals.

The second timeframe is the trauma phase. This time can last from seconds to years depending on the nature of the traumatic event. Ranging from earthquake to assault, rape, war, and abuse, all these have a traumatic nature but differ in length. Several of the factors contributing to PTSD or PTG is not possible to change, for instance, the intensity or duration of an earthquake (Blix, et al., 2013; Kirkpatrick & Heller, 2014). Other factors such

as the initial evaluation of the traumatic event can be a target of intervention (Brewin & Holmes, 2003). Martin Seligman has proposed one such intervention targeting learned optimism, saying that we can change the way we talk to ourselves (for a summary see Seligman, 2002). If individuals can learn optimism before the trauma, this may dampen the initial response to the traumatic event. Further, hormonal responses can be a target for interventions (Yehuda, et al., 1998). If all people that show low cortisol levels were administered cortisol, would this mean that fewer people developed PTSD? Cortisol administration may, however, need a systematic approach and would probably cost money.

The third timeframe proposed in the full trauma model is between trauma and PTSD onset (six months after trauma). The relevance of having such a phase is to diminish the initial response of trauma, considering the results presented earlier about ASD. Results showed that we could, with the administration of cortisol, decrease the rates of PTSD (McFarlane, et al., 1997). Next, the length between trauma and treatment onset is relevant for the development of PTSD. However, to decrease time requires a considerable structure in assessing and following up people at risk. It also requires systematic ongoing work to decrease the risk for PTSD. In regard to PTG a psychiatrist, the act of praying and religious acts all increase PTG (Jeon, et al., 2017). The results presented raise the question if it is possible to teach good social support and spirituality in wider perspectives as a buffering factor against PTSD.

The fourth timeframe is after PTSD onset and includes several different subgroups, that are grouped for the scope of this review. Further, good quality longitudinal studies are missing and no definitive time sections in the development of PTSD and PTG are

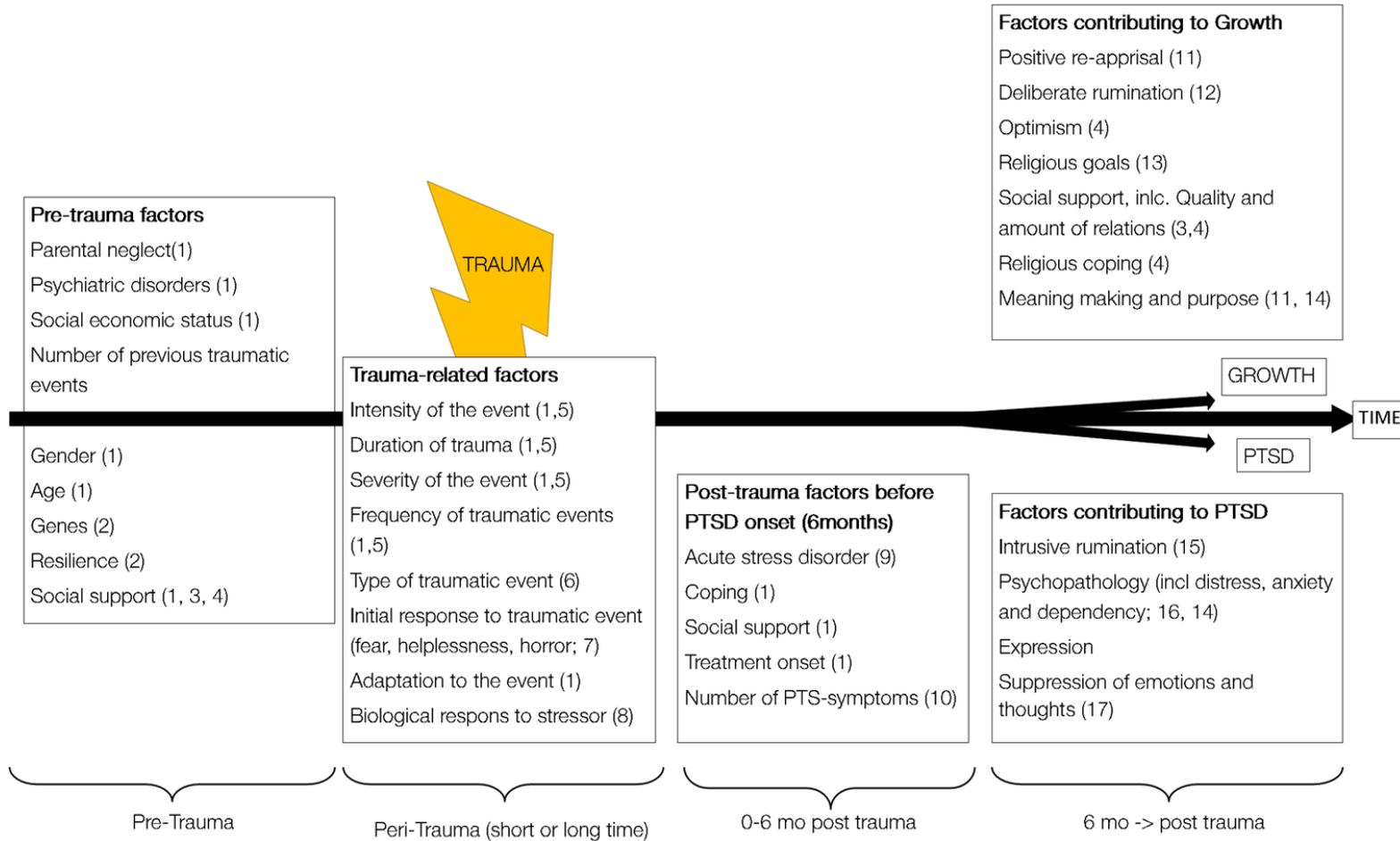


Figure 1: The full trauma model. A theoretical division of the current state of the development of PTSD and PTG. 1.
 Kirkpatrick, & Heller. (2014) 2. Whitaker, Gilpin, & Edwards (2014). 3. Jeon, Park, & Bernstein. (2017). 4. Prati, & Pietrantonio. (2009). 5. Blix, Hansen, Birkeland, Nissen, & Heir. (2013). 6. Linley, & Joseph. (2004). 7. Brewin, C. R., & Holmes, E. A. (2003). 8. Yehuda, McFarlane, & Shalev. (1998). 9. Zohar, Yahalom, Kozlovsky, Cwikel-Hamzany, Matar, Kaplan, . . . Cohen. (2011). 10. Zhou, Wu, & Chen. (2015). 11. Zoellner, & Maercker. (2006). 12. Tedeschi, & Blevins. (2015). 13. Shaw, Joseph, & Linley. (2005). 14. Lowe, Manove, & Rhodes. (2013). 15. Calhoun, Cann, Tedeschi, & McMillan. (2000). 16. Shand, Cowlshaw, Brooker, Burney, & Ricciardelli. (2015). 17. Larsen, & Berenbaum. (2015). 18. Calhoun, & Tedeschi. (1998).

solid. The last time-frame is relatively easy to build treatment practices around, as factors contributing to PTG is easily accessible, and interventions promoting these factors already exist in the literature (Seligman, 2002; Seligman, Steen, Park, & Peterson, 2005). There are, however, still questions about the best possible practice. All individuals are unique, and therefore all treatment plans should also be unique.

The question that remain is not whether there are effective treatment practices but rather how to view trauma; is the aim of treatment and prevention to relieve distress or build strong individuals? That is not to say that we should not relieve distress, but rather that we need to broaden the scope of our focus and include other aspects as well. The literature is partly existing but needs to be integrated, and the intersection that emerges needs to be studied in more detail. The solution to finding relevant thoughts and actions between PTSD and PTG lies in the understudied aspects between these constructs. Based on the research in this review, it would be better to prevent rather than to cure. There is a knowledgebase about the interaction of several brain areas and how these areas are relevant for the development of PTSD and PTG. This research has not fully considered the underlying mechanisms or areas involved. On the other side is the knowledge that builds strong individuals and would be more accessible to start from. Not to say that prevention would be easier to use, but rather that the knowledge about prevention is closer to the people with trauma related difficulties.

The full model of trauma outcomes suggests several timeframes and factors contribute to the development of both PTSD and PTG. This model does not consider different traumas or subcategories of PTG. As such, this model cannot be generalized to real samples without accounting for the aspects of trauma, emotional response and criteria for PTG. This brings a question if there is underlying neural processing involved in several subareas or assumptions. More longitudinal research is needed to understand the relationships between different subscales of PTG. Some might even disagree that trauma-related outcomes are based on emotion regulation.

5.1.1 The full Trauma Model in Relation to Adaptiveness. When considering evolution, our ancestral environment and the dangers presented in this era needs to be accounted for. The most prominent danger was wild animals and death-related factors such as water (Bracha, 2006). A trauma in this context could have been attack from animals or other tribes, or perhaps also the death of a loved one through an accident. Adaptive reactions such as freezing or fleeing would have been beneficial to non-detection from an encountered danger. Avoidance of water and other situations (such as open spaces) would have served

good for the avoidance of encountering dangerous situations. The criteria for PTSD involve such behaviors and would have served an evolutionary point of survival.

Also, the two memory systems related to threat and safety/relaxing stimulus would be explained by evolutionary theories. It is important to avoid situations and reminders that is connected to threat and decreased chances of survival. These memories are therefore stored out of our consciousness, to ensure the information will be used as a reflex rather than a voluntary action. According to Janoff-Bulman (1992) in their theory of a shattered world they state that a trauma shatters the held beliefs about the world, recovery occur when these pieces are rebuilt, and a new coherent picture evolves. On this note initial paralysis, avoidance and other behaviors can be explained (e.g. high PTSS). When we start to process what happened it is likely to be shame and guilt involved, and the individual have no programmed response for such situations. Slowly the person starts to create a new schemata including a beneficial response. According to this view the criteria for PTSD seems like a normal response, even if it is not normal to have continuous hardships.

Learning would also seem to be a plausible way of explaining the normal processing of trauma, as it requires a new schemata and a new response encoded into our brain. Learning can be coupled or compared with the concept of rumination. While ruminating on a traumatic experience it includes looking at this event from different angles, discussing different benefits and trying to find meaning or sense in the event (Cann, et al., 2011). In this process it is also important with tribe-members. Human as such use relations and group-belonging as a buffer against stress and survival (Prati & Pietrantonio, 2009). From our evolutionary environments a human alone is an easy prey and a group of humans are harder to beat. This might be a problem in a society who encourage individualistic approaches as we lose our evolutionary survival programs.

A simple line of happenings could look like this:

Encounter of traumatic situation/event → initial response from our survival-systems → rumination and positive relations → learning and memory systems code optimal response for next time → psychological well-being, specifically environmental mastery.

Psychological well-being can be defined as feelings beyond positive and negative emotions and satisfaction with life. According to Ryff (1989), there are 6 aspects including autonomy, environmental mastery, positive relations, purpose in life, personal growth and self-

acceptance.

When encountering a traumatic experience, we need to handle the situation to survive. When we escaped this situation, feeling safe again, we get increased environmental mastery and personal growth. This can be seen in a new successful repertoire of actions for survival. Any trauma causes constraints and stress to the body, both psychological and biological. When we experience prolonged danger, alterations occur (Radley, Morilak, Viau, & Campeau, 2015).

However, there are also suggestions that we have different processes activated during positive and negative bodily states. Positive feelings, for example, have been proposed to increase our solution orientation (Fredrickson, 2001). Further it is suggested that positive emotions increase our immune-function and build resilience against forthcoming negative events (Fredrickson, 2001). Stress (psychological and biological, internal and external) on the other hand causes imbalance and restructuring of resources (Yehuda, 2000). Prolonged experience of stress alters brain structure and hormone-release patterns (Milani, Hoffmann, Fossaluzza, Jackowski, & Mello, 2017). Ryff and colleagues (Ryff, et al., 2006) showed that several biomarkers were different between well-being and, what they call, ill-being (psychopathology). In total seven of the investigated biomarkers showed that they differ in involvement between well- and ill-being, whereas two of the markers showed the opposite working pattern. This further suggests that we have different hormonal functions in stress and well-being.

What this theoretical summary shows is that PTSD may not be contradictory, but rather logical from a survival point of view, whereas PTG is the optimal product of the processes initiated by a stressful or traumatic experience.

In our evolutionary environment it was never a question of prolonged stress. Either we escaped danger, or we died. If we died it did not matter at all how much deteriorating hormones that were released, if we survived we got back to our tribe. Getting back to the tribe probably meant rest and friends, social support and a good environment for rumination and building additional resources for the next danger.

An interesting question to ask is whether mental disorder is created by the structure of society, working against the origins of the human species.

5.2 Comparing the Psychological Construction Model and MMT

The psychological construction model stems from the PTSD literature and the MMT stems from the PTG literature. It is therefore interesting to study the findings from

these theories, to find similarities and differences in neural processing. This section has two suggestions; firstly, that we by comparing these two theories can find a common ground for processing after trauma, secondly that this common ground is outside of the fear-related areas.

As will be discussed more in the following section PTSD and PTG can occur in the same person in the same or different timeframes. This finding makes it important to look more closely from different perspectives. Also, findings from the positive psychological literature have shed some light on this. For example, the theory of the mental health continuum, suggests that we have different axis for well-being and ill-being. However, this thought is not new, already in the 1980s the development of the scale PANAS (positive affect negative affect schedule), suggested that positive and negative affect should be measured separately. This resulted in two subscales, one positive and one negative. As such is it important to acknowledge both the pathological and beneficial side of PTSD to find the neural processes involved.

Further, the choice of comparing these studies is its properties. The psychological construction model argues for a shift in view by addressing several unanswered questions and contradictory results (for example why the amygdala frequently show decreased activation in PTSD, that the amygdala cannot explain all four domains in PTSD and that learned fear may not involve all aspects of human fear), and highlights plenty of support for their theory, with is also in line with the evolving brain networks (e.g. DMN).

The MMT is chosen as it explains several of the gaps presented (that we may develop PTG after a time with PTSD and explain all aspects of PTSD). However, this theory also elaborates further on positive functioning mainly from a broaden and build (Fredrickson, 2001) theoretical perspective, but also from the science of mindfulness (Garland, Goldin, & Fredrickson, 2015), where they suggest that we by broadening our scope of attention and affect can see more positively, which in turn increase health, and positive functioning. Both theories further have a neural base in their reasoning, which makes it easy to compare neural processing, even if these theories may not be perfect. The conclusion from both theories is that the PFC is important for the development of PTSD and PTG. This gives the next suggestion; that the PFC is a central structure in the aftermath of trauma.

The two theories suggest that the PFC is a part in the regulatory process after trauma (Garland, Goldin, & Fredrickson, 2015), showing decreased activation to salient stimuli and inhibited neural activation (Suvak & Barrett, 2011). The only thing separating good from bad is our interpretation of the stimuli. If the evaluation process is mainly from judgmental processes, it causes negative feedback, and the opposing process for a positive or

nonjudgmental process, depending on ventral and dorsal PFC activation, respectively (Garland, Goldin, & Fredrickson, 2015).

It is important to note that the psychological construction model and the MMT rest upon different assumptions. The psychological construction model takes a network approach, saying that the whole brain interacts to create a present state of the body. The MMT on the other hand takes a topologic approach looking at specific areas. However, in the following discussion findings that support the same conclusion, in addition to some differences will be addressed. Findings from these theories include; [1] alternative explanations for the involvement of the amygdala, insular and hippocampus, [2] that the PFC is central in the processing of fearful situations and that it rebuilds assumptions.

The amygdala has long been suggested to be the core structure in fear. Case studies have shown that lesions in the amygdala affect the subjective feeling of fear (Feinstein, Adolphs, Damasio, & Tranel, 2011). The psychological construction model suggests that fear is an emotion, and thus a byproduct of other brain-processes. The fact that the amygdala shows decrease, rather than increase, activation during fear processing is one supportive finding (Etkin & Wager, 2007). Suvak and Barrett (2011) also suggests that the amygdala is involved in detection of novel stimuli.

Throughout this essay it has been argued that the involvement of the amygdala is more complex than is previously thought. One such claim is that inconsistent activation suggests a whole brain-approach of communication and all areas are regulating each other depending on the input. Saying that we need to take a whole-brain network approach to fully understand what parts of the brain that are engaged in any emotional state at any given time.

Suvak and Barrett (2011) suggest that the amygdala is involved in detection of novel stimuli. Further, they suggest that the brain creates a current state image in any given situation. To create an image, the brain uses input from the senses and decides if this state needs a reaction or not. For example, there is always input from several sources, including visual, somatosensory, internal organs, hormones, nervous system, and so on. When combining all data (that comes from specific areas of the brain), a current state of the body emerges. The psychological construction model also states that all our emotions are a mix of several factors, including a feeling of comfort/discomfort, and a state of arousal (Suvak & Barrett, 2011). If there is no specific region of emotions, it means that we might look at answers in the wrong place. If emotions are not entities by themselves, no activation will tell us where emotions arise. As the amygdala is involved in detecting novel stimuli, this structure should be engaged in every situation of fear. This is, however, contradictory as it is decreased

activation in the amygdala (Etkin & Wager, 2007) in PTSD. Suggesting that we should react less to novel stimulus, even if intrusive thoughts might not be novel.

Suvak and Barrett show that the ventral PFC has projections on the amygdala, nucleus accumbens and the insular. The regulatory process of the amygdala is proposed earlier (Gilboa, 2015), saying that the medial PFC usually decrease amygdala activation.

The PFC also shows descending connections with the insular, which is another area that is proposed in fear and fear processing. Suvak and Barrett (Suvak & Barrett, 2011) suggest that the insula is involved in the process of task switching and memory.

Garland and colleagues (2015) suggest that mindfulness is the key to the regulatory process between the PFC and other areas. The MMT (Garland, Goldin, & Fredrickson, 2015) suggest that the PFC (ventral and dorsal) are involved in processes with self-relevance. More specifically the dorsal PFC is related to non-judgmental monitoring of the state, and the ventral PFC is engaged in active judgments and ratings of self-relevance (Garland, Goldin, & Fredrickson, 2015). So, when we get a situation the PFC is involved as it decides if this input is self-relevant or not. We can therefore adjust and focus our brain activation, through mindfulness, on dampening or strengthening emotional interpretation.

The evaluation process, which is regulated by prefrontal activation, decides if a response is necessary (Garland, Goldin, & Fredrickson, 2015). This can be as the mindfulness approach changes patterns in the PFC to more dorsal activation. Changed activation patterns in the PFC can be due to switching between salient stimuli and mindful breathing (or other objects). The mindful experience deepens and opens patterns of a more nonjudgmental state and increased somatosensory representations (Garland, Goldin, & Fredrickson, 2015). The somatosensory representations are involved in the psychological construction model suggesting that individuals with PTSD have increased focus on internal cues. The mindfulness process is a key aspect in the treatment of PTSD, and other emotion regulation deficits. Garland and colleagues (2015) propose that even if reappraisal techniques are not thought in mindfulness, this practice enhances positive reappraisal and the eudaimonic well-being. The MMT, therefore, states that mindfulness practice through reappraisal is the key not only to relieve distress, but also to increase eudaimonic well-being (Garland, Goldin, & Fredrickson, 2015).

The theories included are in their infancy. However, they point in a direction to be acknowledged in further research. These theories have evolved from two different standpoints and in the end, suggest shared features and aspects to consider about trauma. Mindfulness may be the first line treatment after any traumatic event in the future. It is also

important to note that the construct of mindfulness is emerging, and need more research underpinning its existence. Is mindfulness important for all emotion processes or is it only important for some? Another question emerges: to what extent is mindfulness part of any growth process, no matter if it involves rumination, social support, or any other construct? However, these theories open doors for reflection, how much do we know about basic emotional processing? Do the used methods capture the actual state, or are they capturing what is wanted to be seen? Can neuroimaging use regions of interest in their research, or is it excluding what is activated?

These theories suggest that we need to take the full scale of existence into account when treating, probably any, disorders related to evaluating situations, including fear and phobias. What implications different approaches have for the regulation in PTSD and PTG is still unknown, and thus a plausible route for further investigation. The description presented in this section underlines the importance of looking at constructs from a whole-person view. This comparison only involves two sides of trauma, and it might be relevant to add other disciplines as well. For example, looking at receptors and neural communication at hormone level.

5.3 Limitations, Future Research, and Methodological Issues

The quality is in many regards questionable when looking at the methods and research published. The attempts made herein uses new methods that are not well established. Further, the longitudinal attempts use mathematical models on cross-sectional samples, which may interfere with quality and generalizability. The research on PTG need to be more thoughtful and requires some guidelines and standards for measurement.

Several questions remain in four different aspects: Methodological issues, definition issues, treatment and buffering questions and questions about multidisciplinary issues. The following section will discuss each of these aspects.

5.3.1 Methodological Issues: In many instances, there is a search for regions of interest. However, the studies report results in line with what they were looking for. To find what one wants to see is problematic, and point to biases in study design and thoughts. This review questions the old fear-related thoughts and suggesting new directions for further research. The most important question in future research would be: how can we limit biases in neuroscientific research?

5.3.2 Issues Regarding Definitions: The definition of PTSD may seem clearcut as stated in the DSM. However, when looking critically at this definition, it is not. What kind

of trauma is traumatic enough? Is it only the traumas stated in the definition that is ground for PTSD, or is it depending on individual response rather than the severity of trauma? In the definition of PTG we find several controversies as well. How many subfactors are involved in PTG? As the state of research looks today, there is no answer. Of course, there will be continuous research and new studies that test the factor structure of PTG. In this regard looking at a multidisciplinary construct need a multidisciplinary definition. The most prominent explanation from this review is that both PTSD and PTG is based in emotion regulation, and this line of research needs to be entwined into future research. The most critical issue for further research would be to find a definition that is suitable and widely accepted, both for PTSD and PTG.

5.3.3 Treatment and Buffering Issues: Should we treat people affected or raise strong individuals? It is of relevance to answer this question to continue the research on trauma-related outcomes. The definitive relation between PTSD and PTG is not clear. One might say that PTSD is one end of a continuum and PTG is on the other end, but the research presented suggest otherwise. The state of today does not tell us which mechanisms decide if we get PTSD or PTG, even if several factors are found. If the research presented in this review is a base for answering the question about treating or building, it would suggest that we should build strong individuals from the beginning. Treatment needs to be a target when PTSD has emerged, and we should focus more on good quality environments building strong people in prevention of PTSD. The most important question, if we decide that building strong individuals is the best option, is how we can integrate interventions and practices into school structures, workplaces, and other mandatory environments.

5.3.4 Multidisciplinary Issues: Regarding this intersection between PTSD and PTG the research is limited, questioning what we do know about the formation of trauma-related outcomes. This intersection must be studied in multidisciplinary teams, and use questions that overlap between areas of research. As this multidisciplinary area of human processing evolves, definitions and research variables need to be put together. Research from neuroscience paints one picture, whereas the behavioral aspects paint another picture. When these two areas work in harmony, the answer to the most fundamental questions regarding emotions can be answered. The most important question in this regard is: what can we learn from other disciplines about our area, before putting our research design together.

5.3.5 Limitations of this review: The two theories chosen for comparison in this review are still under development, requiring further empirical support before they can be fully acknowledged as accurate representations of reality. The aim was to give a new venue in

the search for plausible mechanisms and common characteristics of the process of PTSD and PTG. However, to extend the findings in this review caution needs to be addressed, and a more systematic search for more theories that elaborate on PTSD and PTG.

5.4 Conclusion

The aim of the current paper was to look at neural aspects of why some people develop PTSD and others PTG after a traumatic event. This was attempted by answering three questions, below is the answers to each question as found in the review.

1. Can we determine what type of individuals that will develop PTSD and PTG based on current research?

Based on the research presented we can to some extent see who will develop PTSD and PTG. However, it is not defined how far after the event PTG can develop. The research supporting a positive answer to this question is that initial PTSS leads to higher rate of PTG (Lowe, Manove, & Rhodes, 2013). That social support, both before and after trauma leads to PTG (Kirkpatrick & Heller, 2014; Prati & Pietrantonio, 2009). It is also suggested that PTSD and PTG can co-occur in people suggesting that they are not the opposite side of the same continuum (Zhou, Wu, & Chen, 2015), but rather distinct constructs that evolve from different brain-processes. At a biological level measuring cortisol is an effective way of determining if PTSD will emerge. However, these studies did not look at PTG in any timeframe after measurement. Even if more research is needed, the reviewed articles point in a positive direction.

2. Can we by looking at both constructs find a neural base for the development of PTSD and PTG?

This question has no clear answer in the current review. On the positive side we have studies suggesting that these constructs co-occur (Du, et al., 2018; Schubert, Schmidt, & Rosner, 2016), thus point to the importance of addressing these constructs at the same time. On the negative side we have the fact that there are different constructs (Ryff, et al., 2006; Schubert, Schmidt, & Rosner, 2016), thus different ways of examination. My suggestion is to follow both ways. Looking at population-studies to find prevalence and patterns of trauma-related outcomes and to look at experimental studies investigating specific questions related to one construct.

3. Are there any survival benefits of developing PTSD and PTG?

All the characteristics of PTSD can be explained by evolutionary theories (Bracha, 2006), and the development of PTG can be explained by well-being research (Garland, Goldin, & Fredrickson, 2015; Fredrickson, 2001). It is important to avoid danger and it is important to grow and learn for our survival, and thus it seems likely these properties evolved for survival purposes.

In sum, the state of trauma-related outcomes is in its infancy and need more research. A broader scope of focus is essential to capture the full state of trauma-related outcomes. Looking at PTSD and PTG in the same participants is one way, but these attempts need to be grounded in definitions not yet established. The same yields for emotion regulation and PTSD, as for mindfulness as a process of both relieving PTSD and building PTG. When this research collects more whole-person data about how we handle traumatic events, the individuals affected by these events can be supported and relieved of the after-effects.

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