

What is Complex Trauma?

Michael Guiding

Abstract

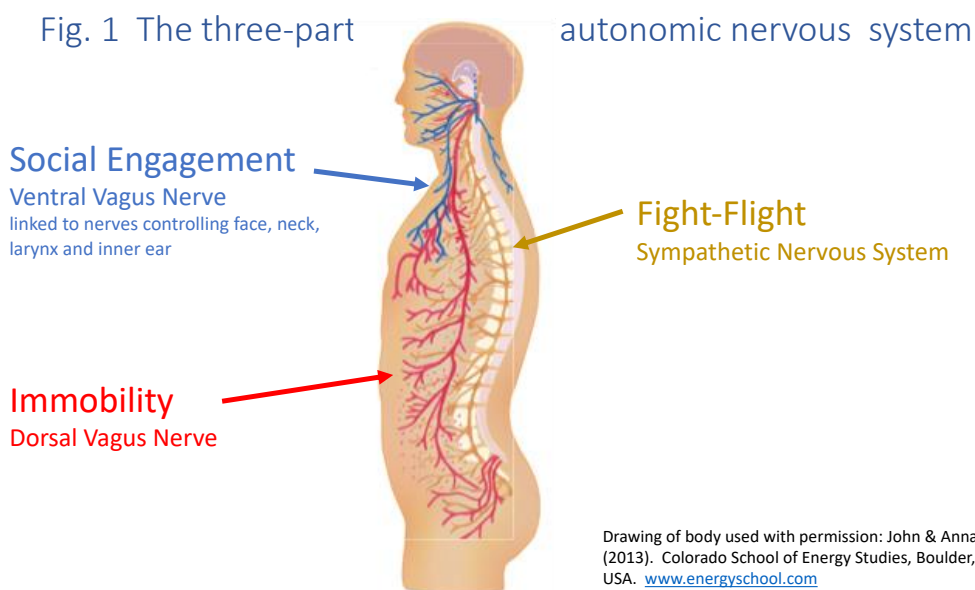
Diagnostic criteria relating to trauma, in ICD-11 and DSM 5, are presented as lists of symptoms with no attempt at understanding the mechanisms of trauma, or at seeing them in the context of human biological and social systems. This seriously limits their usefulness to the psychological therapist. This paper is an attempt at such an understanding, starting from the perspective of the biological fear system. It argues that trauma is an autonomic nervous system dysfunction in which fear responses cannot de-activate, and that complex trauma is the chronic failure of fear system de-activation and the impact of this failure on a wide range of other systems with detrimental consequences for physical and mental health and social integration.

Introduction

In this paper I want to put forward the argument that Trauma is a condition in which the biological fear system has failed to switch off in the aftermath of the threat that triggered its activation, and that Complex Trauma is the result of a *chronic* failure of fear system de-activation and of the dysfunctional impact of this failure, over time, on a complex network of biological, behavioural, cognitive, relational and social systems. I want to explain in some detail what I am referring to as the “fear system”, and why it might in certain circumstances fail to deactivate, before looking at the devastating consequences of this failure on the highly complex and finely balanced organism of the human person.

Defining the Fear System

My definition of the “fear system” covers the oldest aspects, in evolutionary terms, of our biological responses to threat. Stephen Porges (2011) describes three separate stages in the evolution of our response to threat. The earliest developing response, immobility, is a metabolic shutdown evolving around 500 million years ago, which allows a prey animal to appear dead to a predator. The next stage,



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evolving around 300 million years ago, is quite the opposite response, a heightened mobilisation which delivers a sudden burst of energy to allow a prey animal to run from or fight off a predator. This second stage has long been popularised under the term “fight-flight”, while an understanding of immobility is a more recent development. The third stage, evolving only in mammals around 80 million years ago, is the development of a social engagement system in which the primary response to threat is to seek care from others. These three threat-response systems have their own control networks in the brain and are activated in the body by three different branches of the autonomic nervous system (see fig. 1).

Immobility is brought about by the activation of the dorsal vagus nerve (coloured red) which goes from the brainstem to all the major organs of the body. Fight-flight is triggered by the sympathetic nervous system (coloured gold) which goes via the spine to all the major organs, and the social engagement system (coloured blue) is activated by the metabolic calming mediated by the ventral vagus nerve, linking the brainstem to the heart and the lungs, and connected at the brainstem with the cranial nerves controlling the muscles of the face, neck, larynx and inner ear (Porges 2011).

I am using the term “fear system” to cover the two older non-social threat-response systems, immobility, which I will also refer to as fear-collapse¹, and heightened mobilisation, which I will also refer to as fear-arousal. I am going to bring one further biological response under the general umbrella of the fear system, and that is the orienting response, the reaction to the first indications of danger, as this plays an important part in the biology of complex trauma. The orienting response confusingly goes under many names, but I am going to refer to it as fear-alert. I want to look at these three components of the fear system, first of all noting biological changes and relating this to predation among animals, hence the illustrations, but then putting this into the context of human experience since we share these biological systems with the other mammals².



Fear Alert – orienting to threat

Fear-alert is the response to a potential danger, just sensed, when there is a need to find out more about what and where the danger is. When this response is activated the body becomes tense and motionless, with a raised heart rate³, and hearing, eyesight and sense of smell become more acute. The startle response is heightened including a stronger signal in the nerves controlling the limb muscles, and the withdrawal of signal in the ventral vagus nerve which effectively removes the “calming brake” on the heart (Kolacz & Porges, 2018, Baldwin, 2013). This potentiates the body for immediate violent activity if the threat is perceived to be imminent.

When humans experience fear-alert, the obvious thing we notice is our tension as we hold ourselves still, and the fact that we often hold our breath, as the noise of our breathing can interfere with hearing sounds of danger. We

¹ The body’s immobility response can be triggered by a number of threats such as fluid loss, infection, toxins or exhaustion (Levine, 2010). When using the term “fear-collapse” I am purely referring to the immobility response *when it is triggered by the activation of the fear system*, whether this relates to a real physical threat, traumatic memories, disturbing attachment emotions or negative thoughts.

² While there are undoubtedly huge differences within mammalian species in the development of a social engagement system, the older threat-response systems are common to all mammals, including humans. As Peter Levine notes (2010, p.27), “...since we share the same survival parts of the brain with other mammals, it only makes sense that we share their reactions to threat”.

³ This can follow an initial drop (Schauer & Elbert, 2010). Might this perhaps be an evolutionary throwback to the more primitive immobility response?

may rarely notice enhanced perception but are more likely to be aware of our thoughts. These are entirely focused on potential danger, and our general sense is of being wary and on-edge.

Fear Arousal – active defence (Fight-Flight)



Once the danger is obvious and close the brain's fear centre, the amygdala, triggers a huge number of changes in the body, via the sympathetic nervous system, to enable survival through fight or flight (Sapolsky 2004).

An immediate release of adrenaline causes the heart rate to shoot up, and the chambers of the heart expand to increase the volume of blood pumped with each heartbeat. The interlinked systems of the hypothalamus, the pituitary gland and the adrenal glands are activated to produce a series of cortisol-based hormones which trigger the release of glucose and other nutrients into the bloodstream from storage cells throughout the body.

The blood stream is now nutrient-rich, and blood is being pumped at higher speed and greater volume to the muscles of the limbs, hugely boosting strength and speed. The blood supply to the gut and other areas non-essential in crisis is shut down by a narrowing of blood vessels, and sweating enables the body to prevent overheating. The small muscles at the base of hair follicles contract, so hair stands on end, making the prey look bigger in an attempt to scare off a predator.

The hormone vasopressin is released to retain water in case of injury and blood loss, and non-opioid painkillers⁴ are released so the pain of injury is numbed and does not detract from the effort to survive. The amygdala also inhibits the activity of the cortex⁵ eliminating all focus on anything apart from the danger at hand.

When fight-flight is activated in humans, the onset may be experienced as a sudden wave of upwards-moving energy with the heart pounding in the chest. We may also experience nausea and other abdominal discomfort as the gut shuts down, sweating as the body's cooling system operates, dry mouth as vasopressin prevents water loss and goosebumps as muscles contract at the base of hair follicles. If we

⁴ Painkillers which don't cause drowsiness, and therefore don't interfere with fight or flight actions.

⁵ The most recently evolved part of the brain which, among many other functions, governs thinking, willed actions and much social behaviour including language.

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are hurt or injured, we may not feel any pain in the moment, and our ability to think is hugely limited; all our thinking mind will do in these circumstances is focus on danger and how to avoid or eliminate it.

Fear Arousal – “locked” defence and “tipping point” (Freeze and Fright)

However, when we cannot run or fight, we go into freeze⁶, like a “rabbit in the headlights”. The fear-arousal response is still fully active with pounding heart and stress hormones in the bloodstream (Le Doux 1998), but the muscles lock and are held in rigid tension which can result in trembling. This state of “*flight or fight response put on hold*” (Kozłowska et al. 2015, p.267) can switch back instantly to flight if an escape route presents itself. If there is no escape, it can switch to fight as a last resort, hence the expression “fighting like a cornered rat”, but it also easily slips into fright.



Fright is the peak of fear arousal when there is no escape and no perceived chance of survival. This is the tipping point where fear-arousal switches into fear-collapse (Schauer & Elbert 2010). Peter Levine notes that this is most likely to happen where a sense of terror and a sense of being trapped are combined, hence the frequency of the experience of fear-collapse during rape (Levine 2010).

For humans the state of freeze is aptly described as being “petrified”, in other words “turned into stone”. Externally we are rigid, though we might “shake with fear”, as our tense muscles start trembling, but internally our heart is pounding. Alongside sweating, nausea and dry mouth we might feel lightheaded

⁶ There is much confusion around the use of the term “freeze”. Some writers (e.g. Schauer & Elbert 2010) use it to describe the orienting response, which I prefer to call “fear-alert”, and others (e.g. Levine, 2010 and Van der Kolk 2014) use it to describe an immobility collapse. Kozłowska et al. in my view muddy the waters by applying the word “freeze” to three different biological responses within one page of text (2015, p. 270). Fear-alert, freeze and immobility do all have a common element of being motionless for a time, but they belong to different biological states. I understand the stillness of fear-alert as being underpinned by a *moderate* muscular tension which could switch into powerful action, but also quickly relax into rest. However, I see the freeze state as involving a stronger, more rigid muscular tension which is holding in check the powerful energy of fear-arousal which cannot calm quickly. As for using the term freeze to describe fear-collapse, this makes no sense to me at all. Immobility is often referred to as “tonic” immobility owing to the fact that in this state muscles have no “tone” or tension – they are floppy or flaccid. The word freeze is associated in our minds with the rigid state of water, so it perfectly fits the “locked” or rigid state of fear-arousal and creates confusion if applied to fear-collapse. Joseph LeDoux provides an account of the freeze state which makes it clear that it belongs to fear-arousal. He describes a rat, subjected to a fear conditioning experiment which “...stops dead in its tracks and adopts the characteristic freezing position – crouching down and remaining motionless, except for the rhythmic chest movements required for breathing. In addition, the rat’s fur stands on end, its blood pressure and heart rate rise, and stress hormones are released into its bloodstream” (Le Doux 1998, pp. 141-142). I think that the definition of “freeze” is only one part of a wider confusion in the literature of threat-response which may not have fully digested the Polyvagal Theory of Stephen Porges (2011) and still relies on an understanding of tonic immobility formulated decades previously (Gallup 1977).

or dizzy and our mind can blank. This may be combined with a sense of our legs going from under us as we reach the tipping point into fear-collapse.

Fear Collapse – passive defence (Immobility)



When fear-arousal has failed to save us and death seems imminent, the body goes into metabolic shutdown – fear-collapse. There are two mechanisms for this. In the first and possibly the earliest form of shutdown (Kolacz & Porges 2018) the brain releases opioid painkillers⁷ into the bloodstream. In the second the

dorsal vagus nerve triggers a sudden drop in heart rate and blood pressure by widening blood vessels as in a faint (Porges 2017). This means that the cells of our brain, muscles and major organs are starved of oxygen and nutrients, and the cell respiration cycle that provides the energy driving each cell slows or stops. In the brain, the cortex shuts down first, followed by the limbic system⁸. The activation of fear-collapse means that the prey suddenly collapses, and appears to be dead, lying limp and unresponsive on the ground, often with eyes open and showing little sign of breathing. This metabolic collapse serves as a key survival strategy as it triggers a shutdown of the predator’s attack instinct, and this can open up a window of opportunity for escape, particularly if a predator has to fight off competition.

As humans we experience fear-collapse as a downward drop in energy, or a “sinking feeling”. We feel faint, our posture slumps, and our muscles feel heavy as lead. We might feel nauseous as the gut shuts down, and may lose control of our bladder or bowels. We can experience numbness which dampens our emotional responses along with a sense of unreality or being “out of body”. As we descend into fear-collapse, it becomes increasingly difficult to think; the phrase “thinking through treacle” catches a sense of cognition slowing down, and eventually thinking ceases. Our ability to be sociable, with its control system in the cortex, is also gradually paralysed. We lose the ability to make sense of social cues and can no longer hear the human voice properly (Porges 2011). As the limbic system is shut down, we lose all emotional responses including our sense of fear (Levine 2010). While the victims of serious accidents, violent assault or rape may experience a full fear-collapse including physical prostration, I would argue that most people are familiar with fear-collapse in less extreme forms, as I hope will be clarified below in my comments on low mood, depression and dissociation.

The Fear System and Trauma

⁷ Painkillers which induce drowsiness, unlike those released in fear-arousal.

⁸ The limbic system is a part of the brain that is older in evolutionary terms than the cortex and controls our emotional responses and social instincts such as affectional bonds.

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If we list the ways we experience the activation of the fear system (fig. 2 below) and the behaviours they trigger (fighting, fleeing, avoiding, freezing, collapsing) it is clear that trauma symptoms cover the whole range of fear system responses.

When these biological changes happen in the body in response to a real physical threat, they are potentially life-saving and are not thought of as trauma. What we describe as trauma are the same responses triggered in the aftermath of the original threat by experiences that carry any sort of echo or reminder of the original threat, or responses triggered by less significant or imagined threats.

Fig. 2 Fear System Responses and Trauma Responses

Fear-alert

- Body held still and tense
- Holding breath
- Raised heartrate
- Hearing, eyesight and smell more acute
- Thoughts focus on potential threat
- Feeling on edge and wary

Fear-arousal (Fight and Flight)

- Upward rush of energy
- Heart pounding in chest
- Nausea
- Sweating
- Dry mouth
- Hair standing on end (goosebumps)
- Don't notice being hurt
- Thinking hijacked to focus only on danger

Fear-arousal (Freeze and Fright)

- Muscles locked and rigid
- Trembling
- Heart pounding
- Dizzy, lightheaded, mind blanking
- Heart rate peaks
- Tipping into collapse

Fear-collapse (Immobility)

- Downward drop of energy, "sinking feeling"
- Muscles heavy as lead
- Posture slumps, eyelids and facial muscles droop
- Feeling faint
- Numbness
- Nausea, loss of bladder or bowel control
- "Thinking through treacle", brain fade
- Loss of desire and ability to socialize
- Unreal "out of body" sense
- Physical collapse and loss of sense of fear

How the Fear System goal-corrects

Why is it that some experiences of major threat can be recovered from quickly, while others leave a long-lasting traumatic legacy? This happens because in some cases the fear system is unable to switch off after a threat and remains chronically activated, which can be seen as a problem of "goal-correction" (Heard & Lake 1997). Biological systems are goal-corrected. This means that they are triggered by a threat or a need, for example hunger, and switch off when their goal is met, for example by eating until full. The Fear System is triggered by threat. It switches off when the goal of survival is met. If it fails to switch off, it remains chronically active (unregulated) and we have a type of "fear disorder", just as, when the biological systems that tell us we have eaten enough fail to work properly, we can have an eating disorder.

Why does the fear system fail to switch off? To answer this we need to look back at the context of the evolution of the fear system – the struggle between prey and predator. If an animal is attacked by a predator, but manages to escape by the activation of its fear system in fight or flight, it then burns off the energy generated by the fight-flight response in its escape, reaches a place of safety and rests. The

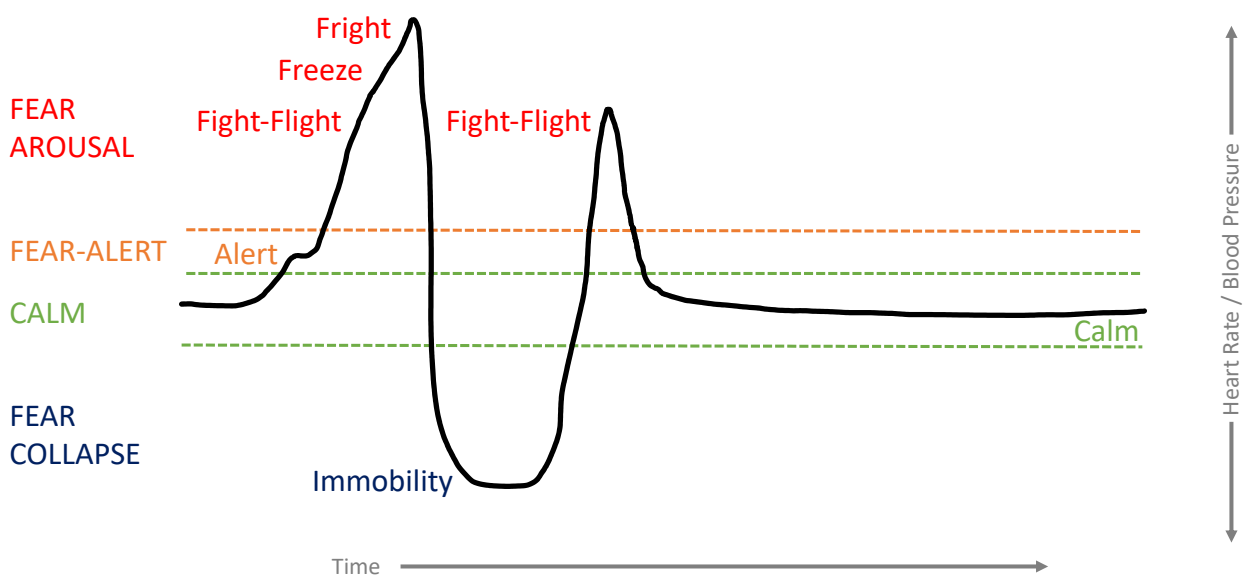
muscles relax, heart rate returns to calm, stress hormone production ceases, and the fear system switches off until it is next needed⁹.

However, if the fight-flight response does not result in escape, and the animal is caught by a predator and goes into fear-collapse, there are two further ways the fear system can switch off. The first way is that a “window of opportunity” for escape can arise, in which case fear-collapse suddenly switches back to fear-arousal, and the animal gets away, reaches a place of safety and rests¹⁰.

The second way the fear system can switch off following a fear-collapse, is when the immediate context becomes a safe place, as in the example in footnote 9 below when the predator is chased off by other animals who do not constitute a threat to the prey. What happens then is that the animal comes out of fear-collapse with deep, spontaneous abdominal breathing, followed by a period of shaking or trembling, which is very different from the shaking of the freeze state¹¹. These mechanisms “reset” the nervous system, and the fear system switches off (Levine 2010, Chapter 1).

The key factor is *safety*. If there is no place of safety in the aftermath of a serious threat, the fear system cannot switch off. In trying to understand what this means, let’s start by looking at what happens when there is a place of safety and the fear system *can* switch off.

Fig. 3 When the Fear System is able to switch off



In fig. 3 we see a chart illustrating metabolic arousal and collapse in the example just given where an animal tries to escape a predator, is caught and goes into collapse, and then a window of opportunity presents itself and there is a successful escape. The animal would start in a place of calm, go into fear-

⁹ I am using the term “switches off” very loosely. A more correct description is that it goes into “vigilant quiescence” (Heard & Lake 1997, p. 41)

¹⁰ For a video illustration see <https://michaelguilding.com/2019/02/27/prey-and-predator-illustrations-of-the-fear-system-in-action/>, third example.

¹¹For a video illustration see <https://michaelguilding.com/2019/02/27/prey-and-predator-illustrations-of-the-fear-system-in-action/>, second example].

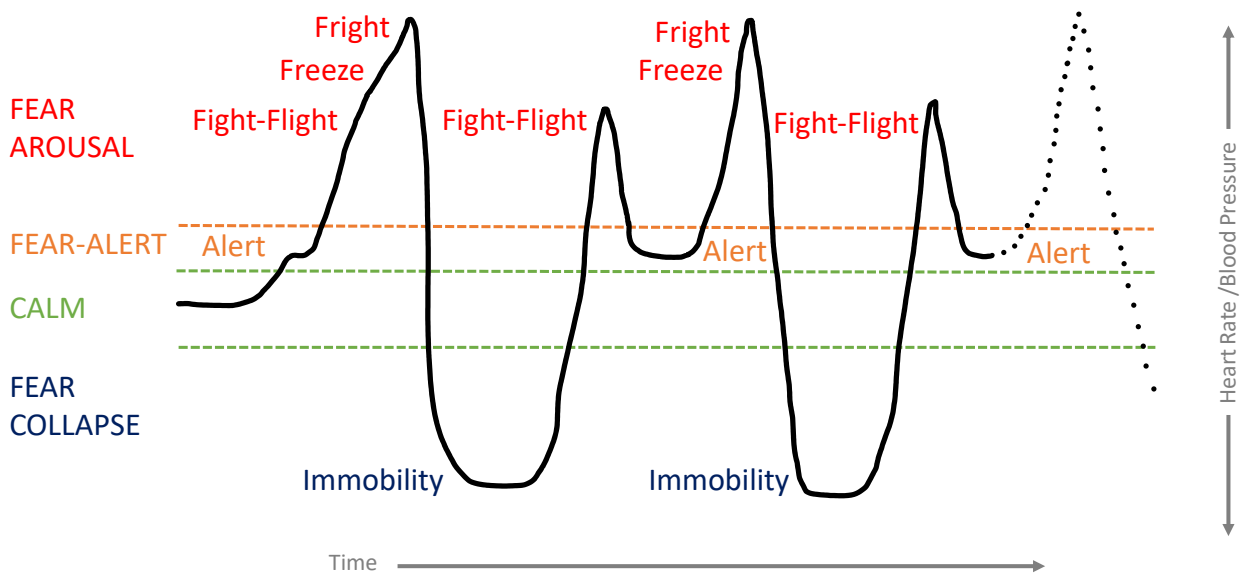
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alert when danger was sensed, and then into fight-flight or freeze when the danger was imminent. At the moment of capture the heart rate would peak in fright triggering an immobility collapse. Then once an opportunity for escape presented it would switch back into fight-flight and escape to a place of safety and rest until a state of calm was reached¹².

When the Fear System fails to goal-correct

However, where there is no place of safety that can be reached after the original threat, the body cannot calm. This is illustrated in fig. 4. The first part of this graph is identical to fig. 3, showing arousal,

Fig. 4 When the Fear System *fails* to switch off



collapse and the switch back into arousal of the original threat. However, in the absence of a place of safety and calming the animal remains in fear-alert. In this state the nervous system is much more volatile and less serious threats can trigger further states of arousal and collapse that again only resolve into fear-alert, rather than relaxing into calm. In this way an ongoing cycle can be established which can repeat continuously at any indication of threat.

If we translate this into our experience as humans, there are additional factors which contribute to continued fear activation. As highly social beings, social rejection is experienced as a major threat which triggers the physicality of the fear system response, but physically acting out our responses to social rejection may be impossible or inappropriate. An additional complication comes from the fact that our cognitive development as humans has given us the ability to think about the future and thus to terrify ourselves by imagining a huge variety of future threats. However, the physical activity of fighting and running that can reset the fear system is not available in response to future imagined threats. In the case of either social rejection, or future imagined fears, the creation of a place or a sense of safety may be

¹² It is important to note that we do not go through all the stages of fear arousal illustrated in fig. 3 in a set order, nor do we choose which state will be activated, as these are automatic processes acting far faster than thinking. Fight or flight may be triggered without freeze, and freeze without fight or flight. Certain threats may activate fright immediately without any intermediate steps, and fright can switch into a fear-collapse so suddenly that it is only the collapse which is noticed.

impossible, and with no ability to relax we get stuck in a continuing cycle of chronic fear activation as our fear system cannot reset through intense physical activity, or through deep breathing and shaking.

When the fear system fails to switch off, systems which were part of its goal-correction become dysfunctional, so effectively the longer the fear system remains active, the harder it becomes to deactivate. At a physiological level, tension that is not released following a threat builds into a chronic state of tension in the body. This prevents both the deep breathing and post-threat shaking that reset the fear system, and contributes to chronic shallow breathing which favours sympathetic nervous system dominance and the continuance of fear system activation (Tabor et al. 2019). At the level of hormone production, if the state of fear activation continues for too long the cortisol receptors in the brain, which shut down production when cortisol is no longer required, become desensitized. This disables a important feedback loop, making it more difficult for the fear system to deactivate (Sapolsky 2004).

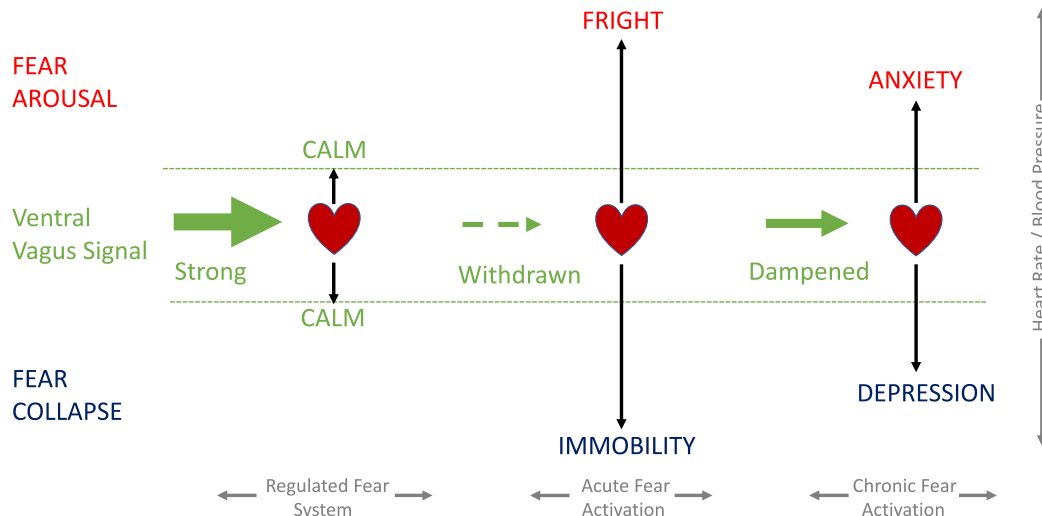
At the level of the autonomic nervous system, chronic fear activation disables what is perhaps the most important mechanism in switching off the fear response. This is the ventral vagus nerve, described by Porges as the “calming brake” on the heart, which connects directly to the heart’s sino-atrial node, its inbuilt pacemaker. The sino-atrial node prompts the heart to beat at c. 90-100 beats per minute, perfect for the fear-alert stage and for a sudden switch into fight-flight. However, when the ventral vagus nerve is activated, it overrides this default setting and slows the heartrate down to around 60 beats per minute¹³ bringing us into a state of calm.

In the face of a threat, the activating signal on the ventral vagus nerve is withdrawn in order to enable the heartrate to rise in fear-alert and fear-arousal. However Kolacz & Porges (2018) note that when the fear system is *chronically* active, in other words when the ventral vagal signal is chronically withdrawn, this signal becomes “dampened” or weakened. So, to use an analogy, just as a leg muscle will atrophy if not used for walking for a time, so the strength of signal on the ventral vagus will weaken if it is withdrawn for too long in fear activation.

The impact of the weakened ventral vagus

¹³ This is highly variable in individual cases.

Fig. 5 Fear regulation and the weakened ventral vagus



It should be noted, however, that the “calming brake” description given to the ventral vagus is only one half of the story. The ventral vagus nerve does not just slow down the fast heartbeat of fear-arousal, but also speeds up the slow heartbeat of fear-collapse, acting as a regulator of the volatility of the autonomic nervous system under threat (Porges 2017).

Fig. 5 is an attempt to illustrate the implications of a weakened ventral vagal signal. The left side of the diagram represents the regulated fear system with a strong signal on the ventral vagus. In the face of less serious or imagined threats this restrains both the rise of heart rate in fear arousal and its fall in fear collapse resulting in the maintenance of relative calm under threat. In the middle of the diagram we see acute fear system activation where the ventral vagal signal is withdrawn. Here, in the face of a real and present threat, there is no restraining force to prevent the peak of fear arousal in fright or the full extent of fear-collapse in immobility. On the right side of the diagram we see the result of chronic fear system activation. There is a signal on the ventral vagus so there is some restraining power but in the face of less serious or imagined threats this is insufficient for regulation, and the dampened signal results in “intermediate” states of fear-arousal or fear collapse .

Making sense of anxiety and depression

I have categorised these intermediate states of arousal and collapse as anxiety and depression. I think that many could accept a definition of anxiety as a chronic state of fear-alert with frequent switches into fight-flight, but depression has always been less well understood.

Seeing depression as an immobility response that can only be *partially* regulated by a weakened ventral vagus signal makes complete sense of the loss of energy in depression as this is a real metabolic shutdown, not something “just in the mind”. It also makes sense of the loss of the desire and the ability to socialise due to a shutdown of areas in the cortex controlling the social engagement system, and also the slowing and failure of thinking as cognitive systems in the cortex are disabled¹⁴.

¹⁴ See <https://michaelgilding.com/rethinking-depression/> and <https://michaelgilding.com/2019/02/18/depression-a-biological-response-to-threat/> for the development of my thoughts on this topic.

Mapping the psychological impact of complex trauma against fear system responses

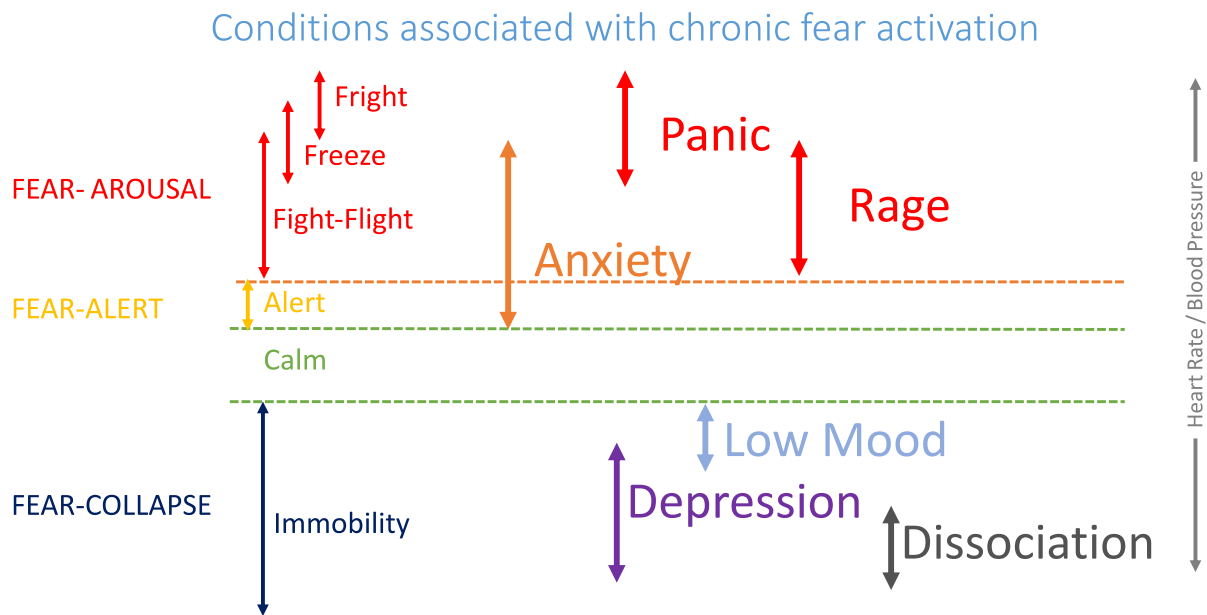


Fig. 6 is an attempt to map the common terms we use to describe our experience of fear system activation alongside the biological responses of fear-alert, fear-arousal and fear-collapse. Anxiety, as noted above, is a persistent state of fear-activation varying from fear-alert to fight-flight, while Rage (or uncontrolled anger) corresponds to the fight stage of arousal. The symptoms of panic place it at the higher end of fear arousal covering freeze and fright and possibly also the initial sensations of tipping into a fear-collapse, e.g. “feeling dizzy, *unsteady*, lightheaded or *faint*” (American Psychiatric Association 2000, p.432).

I see low mood as a state where the immobility system has been triggered but there is still reasonably good ventral vagal regulation. Where there is less effective regulation I am suggesting that the immobility response is experienced as varying degrees of depression on a continuum from low mood through to severe depression¹⁵.

Alongside this in more severe states of depression and immobility is the phenomenon of dissociation¹⁶. This is a very poorly defined concept, but I see the sense of unreality and being “out of body” that it describes as being our experience of the numbing opiates released by the brain in immobility, or our

¹⁵ I struggled with this hypothesis initially because while it was compatible with a pattern of cycling between anxiety and depression, it did not explain anxiety experienced *at the same time as* depression. However, I now think that when the immobility response activates in *intermediate* states of fear-collapse, the sympathetic nervous system can still function, which makes sense of highly distressing symptoms in which anxiety can be experienced alongside debilitating depression. This sympathetic nervous system co-activation ceases in a full fear-collapse as the limbic system shuts down and our sense of fear disappears. The interrelation of these biological systems is complex and fig. 6 deliberately oversimplifies both in representing anxiety and depression purely as opposing states of arousal and collapse, and also in combining heartrate and blood pressure on the y-axis as if they always worked in tandem.

¹⁶ Porges (2017 p.12) speculates on the basis of Polyvagal Theory that “there may be gradations in reactions to life threat from total shutdown and collapse mimicking the death feigning responses of small mammals to an immobilization of the body during which the muscles lose tension and the mind dissociates from the physical event.”

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experience of perceptual parts of the cortex being disabled by oxygen starvation as a result of the metabolic shutdown, or both.¹⁷

The complexity of complex trauma

At this stage I want to go back to my opening definition of complex trauma as a chronic failure of the body's fear system to deactivate once the threat that triggered it has passed, and the dysfunctional impact of this failure over time on a complex interconnected network of biological, behavioural, cognitive, relational and social systems. We have just looked at the impact of this failure on the autonomic nervous system leaving it over-responsive to threat, stuck in hypervigilant fear-alert, and cycling between fear-arousal and fear-collapse.

In the simplest form of trauma which might meet the criteria for a PTSD diagnosis, with a single threat of relatively short duration experienced by an adult, the impact, in terms of hypervigilance accompanied by the triggering of terror, aggression or depressive collapse, can have a debilitating effect on wellbeing and functioning. However, this can often be alleviated in a fairly straightforward way within a few sessions of EMDR or one of the body-based psychotherapies (e.g. Ogden et al. 2006).

In complex trauma however where there were perhaps multiple threats of long duration and the impact on the autonomic nervous system was not alleviated over time, the failure of the fear system to goal-correct has wide-reaching consequences. The continuing volatility of the autonomic nervous system which results from this failure affects multiple systems which become dysfunctional and create feedback loops in which this dysfunction becomes more and more entrenched. This is particularly the case when threats were suffered in infancy and childhood, and even more so where the parent or parents who are the child's fundamental source of safety are themselves the threat.

In these circumstances the task of training the autonomic nervous system out of hypervigilance and hyper-reactivity into calm becomes highly complex, no simple short-term intervention is effective, and painfully won progress in one area can easily be wiped out by a threat in another area. I have already noted the psychological impact of an unregulated fear system in terms of anxiety, panic, low mood, depression and dissociation, but will now look at the other areas or systems that become dysfunctional as a result of complex trauma, and, in their turn, contribute to the chronic triggering of fear responses.

The impact of the unregulated Fear System on physical health, pain and energy

The unregulated fear system, as it cycles between extreme metabolic activity and metabolic shutdown, hugely stresses the whole organism causing cardiovascular disease, gastrointestinal disorders and, dependent on whether stress is intermittent or continuous, either over-stimulating the immune system,

¹⁷ This view of dissociation as belonging to fear-collapse has been challenged by colleagues who use the term more broadly to also encompass the "dizzy brain" and disconnect from reality experienced in the freeze and fright stages of fear-arousal. It makes sense to me that the way we perceive our own body could be profoundly affected by the non-opioid painkillers released in fear-arousal which are powerful enough for soldiers not to notice major injury in battle. The narrow focus on danger and survival in fear-arousal clearly also affects our perception of our surrounding reality. My sense is that these interior and exterior perceptions generated by fear-arousal are different from those experienced in fear-collapse, and I wonder if we need another word for the fear-arousal sensations. However, I am aware that there is real difficulty involved in observing and describing fear reactions which are not static phenomena but switch rapidly between different biological responses, and I wonder whether some experiences of dissociation in the context of panic may be generated by the sudden tipping into immobility that can be part of the "fright" response.

causing autoimmune illnesses such as rheumatoid arthritis, or lowering immune function, opening the body up to a whole range of viral or bacterial infections (Sapolsky 2004).

Unregulated fear activation can also cause dysfunction in our pain-signalling systems. The ventral vagus nerve that is the key factor in regulating our fear system responses also has an important role in inhibiting pain signalling throughout the nervous system. When the signal on the ventral vagus is weakened through chronic fear activation, its ability to inhibit pain signals can fail, resulting in chronic pain conditions (Kolacz & Porges 2018).

Additionally, unregulated fear activation can cause dysfunction in our systems of energy production. Sapolsky (2004) writes very clearly about the huge energy drain involved in repeated activations of fear-arousal. This is not just the considerable amount of energy required to fight or run for your life, but also the amount of energy taken up in the process of continually releasing nutrients into the bloodstream from storage cells, and then putting them back into storage if they are not burnt up in fight-flight, resulting in ongoing energy deficit. Fear-collapse also creates an energy deficit as the metabolic shutdown disables aerobic (oxygen-based) energy production, and chronic anaerobic energy production involving the production of lactic acid and then its subsequent reprocessing uses up more energy than is actually produced (Myhill 2014). The cycling between fear-arousal and fear-collapse created when the fear system cannot deactivate may be a key contributor to energy disorders such as Chronic Fatigue Syndrome (Gupta 2002).

So, the unregulated fear system disrupts the normal functioning of many other biological systems¹⁸ creating chronic conditions of physical illness, pain and fatigue. These in their turn negatively impact a person's mood, mindset, ability to relate to others, to work and to earn a living and thus their sense of personal autonomy and their ability to make changes to, or move out of, external circumstances that may be keeping them trapped in continuous fear activation.

The impact of the unregulated Fear System on patterns of behaviour

Our fear response is essentially a physical reaction to threat so it shows itself most clearly in our behaviours. We are born with our fear system fully developed but without the ability to regulate it. This regulation has to be provided at first by our parents responding in an attuned way to our needs and soothing our fears. Our biological systems for regulating fear reactions develop in response to this good early nurture (Hart 2011). However, if our parents are unable to regulate their own fear responses, and as a result are anxious, depressed, angry or preoccupied, these biological systems don't fully develop and we can potentially live our whole lives with a fear system that cannot properly deactivate.

In more serious cases where the parents not only fail to create a context of safety for the child, but are themselves the source of danger, as in the case of physical, sexual or emotional abuse and neglect, the child lives in a permanent state of fear system activation, driving behaviours in adult life such as self-neglect, self-harm, avoidance, violence, or addiction. Each of these bring practical consequences that make breaking out of the cycle of fear more and more difficult.

Self-neglect repeats patterns of early mistreatment, and can perpetuate fear system activation. Self-harm can be a risky attempt at self-regulation, activating opioid painkillers to dampen emotional distress, where shame can compound isolation and inhibit careseeking. Avoidant behaviours, driven by chronic states of

¹⁸ Even affecting cells at a genetic level by shortening the telomeres which protect DNA during cell reproduction (Tyrka et al. 2010).

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either fear-arousal in flight mode, or of fear-collapse, progressively shut down opportunities for careseeking and self-development and can lead to an intensification of anxieties. Violent and offending behaviours, driven by chronic states of fear-arousal in flight mode, prompt hostility from others, further traumatisation within the prison system, and a progressive dislocation from the wider society. Addictions such as the use of drugs, alcohol or gambling, which are an attempt to self-soothe or self-vitalise in the context of the emotional turbulence of an unregulated fear system (Maté 2012), can destructively alter neurological functioning, put an end to supportive relationships and impair the ability to work.

The impact of the unregulated Fear System on patterns of thinking and education

In their different ways both fear-arousal and fear-collapse profoundly affect our ability to think. In fear-arousal the amygdala inhibits the cortex and focuses our thinking purely on detecting and surviving danger. Any other information may be heard, but it cannot be processed at the time. A chronic state of fear-arousal ensures that, over time, this focus on danger becomes habitual, negatively affecting the way we think about others and the world we are living in. This habitual outlook keeps re-triggering our fear system responses and closing down any openness to potential help.

In fear-collapse, oxygen starvation disables the cells of the cortex. As the body is de-energised, what thinking remains becomes hopeless. At a certain stage of collapse we are unable to process anything we may hear, and we then become unable to hear clearly what is said to us¹⁹, and the mechanisms for memory encoding and recall fail.

Putting all this together, it is clear that fear system activation severely hampers effective learning and education. A child from a home where there has been no emotional regulation, cycling between fear-arousal and fear-collapse, may not be able to take in and process information and may not be able to cope with school work. At the time this will be a severe blow to the child's self-esteem, but in the longer term it can limit life-options and income potential and thus can become an obstacle to escaping from a fear-dominated environment.

The impact of the unregulated Fear System on relationships

Fear system activation affects our social engagement system in two key ways. In fear-arousal, where we focus only on danger, we automatically start seeing those around us as a source of danger. In fear-collapse our entire social engagement system is disabled as the cortex and then the limbic brain shut down and we lose both the desire and the ability to relate to others. Chronic fear system activation thus has a disastrous impact on our ability to make and sustain relationships. As the social engagement system is our primary means of responding to threat and enabling "survival with wellbeing" (Heard, Lake & McCluskey 2009) by maintaining connectedness with others, this locks us into a feedback loop of reliance purely on the isolating responses of the fear system.

As our relationships with our primary caregivers form a template for future relationships, those with a history of good fear regulation are able to trust others and maintain relationships of equality which are supportive and companionable, and which then contribute hugely to the ongoing regulation of fear responses. However, children who have been unable to seek care from parents often struggle to be able to trust or seek care from others in later life, and trauma suffered in infancy and early childhood in

¹⁹ As the muscles of the inner ear, that control the tension of the eardrum which enables us to distinguish the human voice from background noises, start to fail (Porges 2017).

relational contexts can create deeply defensive patterns of relating to others such as are seen in personality disorders (Zhang et al. 2012). Where the fear system is chronically active, relationships tend to be unequal. The relationship pattern can become “one-up, one-down”, with one party dominant and the other submissive (Heard & Lake 1997), either fixed in these roles, or switching between them in highly volatile exchanges. Where the “other” is viewed as a threat, the fear response is to control them, either directly through force, or intimidation, or indirectly through various forms of manipulation. Such relationships cannot be safe places, though they may be familiar places, and so they contribute to the ongoing failure of the fear system to deactivate.

The impact of the unregulated Fear System on our social context

I have focused up to this point on the impact on individuals of an unregulated fear system, but individuals are part of societies, and fear-driven reactions of individuals and groups over time profoundly affect the culture and workings of human societies and can turn them into unsafe contexts for many, creating feedback loops which further dysregulate the fear systems of individuals. In fear-arousal, both individually and as societies, we focus on danger, and we then find danger in any sort of *difference*, be it race, nationality, tribe, class, gender, religion or sexuality²⁰. When societies react to difference with fear responses, the process of “othering” (Brons 2015) can result in oppression, persecution, war and genocide. In this way complex trauma begets behaviours in groups, nations and cultures which inflict profound harm, endlessly perpetuating the problem of complex trauma throughout the generations.

Conclusion

In this article I have tried to describe the mechanism behind the experience of trauma as a failure in goal-correction of the fear system and to show how, if this is not alleviated in a timely fashion, this can become a chronic condition, a “fear system disorder” which, becoming more engrained over time and affecting an ever wider number of related systems, becomes progressively harder to rectify. This chronic condition of continuing fear system activation is what we mean by Complex Trauma.

Complex trauma impacts every aspect of human experience from the volatility of the autonomic nervous system and the resultant anxiety and mood disorders, to chronic states of physical illness, pain and fatigue. It can establish patterns of self-neglect and self-harm, and avoidant, violent or addictive behaviours, disable cognition and inhibit educational opportunity. It lies at the root of personality disorders and can destroy trust and supportive relationships. It can undermine the ability to earn a living, trapping people in hostile environments, with a loss of status, autonomy and power, and leaving them with little sense of belonging to society. It can overwhelm whole societies resulting in the worst crimes against humanity, and have an impact across generations.

In a following article I will look at a number of implications for psychological therapies that arise from this understanding of complex trauma, and give examples of the application of this perspective in clinical practice. In a further related article I will examine the difficult but crucial task we have as therapists of regulating our own fear system responses. Without this self-regulation there can be no place of safety for our clients, and only from this place of safety can the work to alleviate complex trauma begin.

²⁰ See Nussbaum (2018) for a contemporary account of fear system activation in society.

References

- American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders*. Fourth Edition, Text Revision. Washington, DC: APA.
- Baldwin, D. V. (2013). Primitive mechanisms of trauma response: An evolutionary perspective on trauma-related disorders. *Neuroscience and Biobehavioural Review* [Online], 37, pp. 1549-1566. Available at: <https://doi.org/10.1016/j.neubiorev.2013.06.004> [Accessed 1 April 2018]
- Brons, L. (2015). Othering, an Analysis. *Transcience. A Journal of Global Studies* [Online]. 6(1), pp.69-90. Available at: <https://hcommons.org/deposits/item/hc:13619/> [Accessed 12 Nov. 2020]
- Gallup, G.G. (1977). Tonic Immobility: The role of Fear and Predation. *The Psychological Record* [Online], 1, pp. 41-61. Available at: <https://www.researchgate.net/publication/332735513> [Accessed August 2020]
- Gupta, A. (2002). Unconscious amygdalar fear conditioning in a subset of chronic fatigue syndrome patients. *Medical Hypotheses* [Online], 59(6), pp. 727–735. Available at: [https://doi.org/10.1016/S0306-9877\(02\)00321-3](https://doi.org/10.1016/S0306-9877(02)00321-3) [Accessed 17 October 2020]
- Hart, S. (2011). *The Impact of Attachment: Developmental Neuroaffective Psychology*. New York: Norton.
- Heard, D. and Lake, B. (1997). *The Challenge of Attachment for Caregiving*. London: Routledge.
- Heard, D., Lake, B. and McCluskey, U. (2009). *Attachment Therapy with Adolescents and Adults. Theory and Practice Post Bowlby*. London: Karnac.
- Kolacz, J. and Porges, S.W. (2018). Chronic Diffuse Pain and Functional Gastrointestinal Disorders after Traumatic Stress: Pathophysiology through a Polyvagal Perspective, *Frontiers in Medicine* [Online], 5:145. Available at: <https://doi.org/10.3389/fmed.2018.00145> [Accessed June 2018]
- Kozłowska, K., Walker, P., McLean, L. and Carrive, P. (2015) Fear and the Defence Cascade: Clinical Implications and Management. *Harvard Review of Psychiatry* [Online], 23(4), pp. 263-287. Available as: <https://doi.org/10.1097/HRP.000000000000065> [Accessed June 2017]
- Le Doux, J. (1998). *The Emotional Brain. The mysterious underpinnings of emotional life*. New York: Touchstone.
- Levine, P. (2010). *In an Unspoken Voice: How the Body Releases Trauma and Restores Goodness*. Berkeley, Calif: North Atlantic Books.
- Maté, G. (2012). Addiction: Childhood Trauma, Stress and the Biology of Addiction. *Journal of Restorative Medicine* [Online], 1 (1). Available at: <https://doi.org/10.14200/jrm.2012.1.1005> [Accessed November 2020]
- Myhill, S. (2014). *Diagnosis and Treatment of Chronic Fatigue Syndrome*. London: Hammersmith Health Books.
- Nussbaum, M.C. (2018). *The Monarchy of Fear*. New York: Simon & Schuster.
- Ogden, P., Minton, K. and Pain, C. (2006). *Trauma and the Body: A Sensorimotor Approach to Psychotherapy*. New York: Norton.
- Porges, S. W. (2011). *The Polyvagal Theory: Neurophysiological Foundations of Emotions, Attachment, Communication, and Self-Regulation*. New York: Norton.

- Porges, S. W. (2017). *The Pocket Guide to the Polyvagal Theory; The Transformative Power of Feeling Safe*. New York: Norton.
- Sapolsky, R.M. (2004). *Why Zebras don't get Ulcers*. 3rd ed. New York: Holt Paperbacks.
- Schauer, M. and Elbert, T. (2010). Dissociation following Traumatic Stress: Etiology and Treatment. *Journal of Psychology* [Online], 218(2), pp. 109-127. Available at: <https://doi.org/10.1027/0044-3409/a000018> [Accessed June 2017]
- Tabor, A., Bateman, S., Scheme, E. and Schraefel, M.C. (2019). *Breathing Physiology and Guided Breathing Exercise: A Primer* [Online]. University of New Brunswick Internal Technical Report (#TR199-241). October 2019. Available at: <https://www.cs.unb.ca/tech-reports/documents/TR19-241.pdf> [Accessed 17 October 2020]
- Tyrka, A.R., Price, L.H., Kao, H-T., Porton, B., Marsella, S.A. and Carpenter, L.L. (2010). Childhood Maltreatment and Telomere Shortening: Preliminary Support for an Effect of Early Stress on Cellular Aging. *Biol Psychiatry* [Online], 67(6), pp. 531–534. Available at: <https://doi.org/10.1016/j.biopsych.2009.08.014>. [Accessed 18 October 2020]
- Van der Kolk, B. (2014). *The Body Keeps the Score*. London: Allen Lane.
- Zhang, T., Chow, A., Wang L., Dai, Y. and Xiao, Z. (2012). Role of childhood traumatic experience in personality disorders in China. *Comprehensive Psychiatry*, 53, pp. 829-836. Available at: <https://www.sciencedirect.com/science/article/pii/S0010440X11002008> [Accessed 11 December 2020]

Photos

- p.2 Meerkat. Indianapolis Zoo. Available at: <https://www.indianapoliszoo.com/exhibits/deserts/meerkat/>. Permission requested
- p.3 Lion hunting. Online image – unable to trace.
- p.4 Fox and Marmot. “The Moment” by Yongqing-Bao; Winner of Wildlife photographer of the year 2019. Available at: <https://www.nhm.ac.uk/wpy/gallery/2019-the-moment> Permission requested
- p.5 Leopard. From You Tube video, available at: <https://www.youtube.com/watch?v=IAtW7nJUcRA>