Anxiety Disorders

As a Disorder of the Attachment Archetype

Bowlby considered care-eliciting, care-giving, competitive power-seeking and cooperating as derivatives and developmental expressions of the affiliation & bonding archetype. **Neurotic illness** may occur due to deficient parental care frustrating ***archetypal anticipations***, as maturation proceeds through a sequence of *innate expectations* which the environment either fulfils or fails to meet.

Schore (2002) argues that the predisposition for **arousal dysregulation** under stress in those with anxiety disorders, results from dysregulation of both central and autonomic nervous system circuits as a result of **failure of the attachment & affiliation archetype** causing maldevelopment of the right frontal cortex.

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As a Disorder of the Hierarchical Ranking Archetype (Agonistic Anxiety)

Stevens & Price (2000) suggest this may occur when an exaggeration of the normal inner sanction against inappropriately assertive behaviour causes excessive fear that one will suffer defeat in an agonistic encounter.

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As a Disorder of the Courtship & Mating Archetype (Hedonic Anxiety)

Stevens & Price (2000) suggest this may be due to an exaggeration of the normal inner sanction against making oneself unattractive.

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As a Disorder of the Threat Response Archetype

Failure to Switch off the Fear Mental Sub-Module

Nesse (1990) argues that the syndromes of pathological fear of DSM-IV correspond to different exaggerations of normal subtypes of fear responding to specific situations:

Fear Subtype	Corresponding Situation (Danger)
Panic	Imminent attack by predator
Agoraphobia	Environment in which attack is likely
General anxiety	Environment that is unsafe in general
Conflictual anxiety	Socially unaccepted impulses
Social anxiety	Threats to status or group membership
Small animal phobias	Dangerous small animals
Hypochondriasis	Disease
Separation anxiety	Separation from protective parent
Stranger anxiety	Harm from strange humans
Personal inadequacy	Rejection by allies or group
Obsessive cleanliness	Infectious disease
Obsessive hoarding	Lack of food or other resources
Blood/injury	Wound
From Table 2. Subtypes of Fear & Their Corresponding Situations (Nesse, 1990)	


Joseph LeDoux conceptualises the arousal dysregulation that occurs in anxiety disorders to result from dysfunction of the ***fear module***, leading to "phobias, panic attacks & PTSD emerging out of the depths of the unconscious workings of the fear system" (LeDoux 1996).

LeDoux, in his book The Emotional Brain, defines psychotherapy as "a process through which our neocortex learns to exercise control over evolutionarily old emotional systems". He appears to conceptualise PTSD as an over-learnt survival response, with the neural correlate being inadequately opposed activation of the limbic lobe, in particular the amygdala, by the neocortex, following exposure to a trauma. He proposes that the way to overcome PTSD is to introduce an **active coping response** i.e. activation of the neocortex.


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As a Disorder in the Triune Brain


Stevens & Price (2000) suggest the following "neuro-evolutionary" perspective, utilising the **triune brain model** by MacLean (1985):

Brain Level	*Substrate*	*Treatment*
1. CORTEX (Reason)	Frontal areas Mediate the learned associations responsible for release of panic & avoidance behaviour	Psychotherapy
	GABA receptors	

2. LIMBIC (Emotion)	Anticipatory anxiety	Benzodiazepines
3. REPTILIAN (Instinct)	Brainstem nuclei (locus coeruleus) Mediate panic attack symptoms	Antidepressants

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Generalised Anxiety Disorder

Akiskal 2002 suggests the following subtypes of the anxious temperament :

- the anxious phobic style reflects protection against physical dangers
- the anxious sensitive avoidant style reflects protection against threats to the self
- shy phobic individuals stay at the periphery of the group, gaining the advantages of tl group but away from the critical eye of the leader.
- generalised anxious harm avoidant type they have an exaggerated sense of protection against danger to kin. They worry about harm befalling their family
- phobic anxious temperament are hypersensitive to separation & leaving familiar surroundings creates a dependence that is necessary for the development of the fami

Akiskal 2002 argues that **General Anxiety Disorder** represents an exaggeration of normal personality disposition. He considers GAD "**altruistic** anxiety" subserving the survival of one's extended phenotype in a kin selection paradigm. The function of worrying is to protect your kin, to resist relaxing, to avoid being prey to danger. He argues that dependency is important for human evolution & warns against considering it pathological.

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Post Traumatic Stress Disorder

As a Disorder of the Attachment Archetype

Schore (2002) argues that PTSD occurs due to ***Trauma-Induced Excessive Pruning of Right Brain Circuits*** in childhood, causing **unopposed amygdala excitability** (or over-activation of the fear sub-module).

Schore delineates developmental precursors predisposing to post-traumatic stress disorder. He suggests that **disorganised-disoriented insecure attachment**, a pattern common in infants abused in the first two years of life, negatively impacts on the developmental trajectory of the right brain, which is dominant for attachment, affect regulation, and stress modulation, thereby setting a template for the coping deficits of both mind and body that characterise PTSD symptomatology.

Schore argues that Bowlby's **neurophysiological control system** involved in regulating instinctive attachment behaviour is located in the **right orbitofrontal area** and its cortical and subcortical connections. This system is specialised to show a flexible response in stressful contexts of uncertainty. He goes on to argue that early childhood trauma massively dysregulates and alters the developmental trajectory of the right hemisphere and the orbitofrontal system of the frontal lobes.

The higher regulatory systems of the right hemisphere form extensive reciprocal connections with the limbic and autonomic nervous systems. Both the autonomic and central nervous systems continue to develop postnatally, and the assembly of these limbic-autonomic circuits is directly influenced by the attachment relationship. In this manner, the internalised regulatory capacities of the infant develop in relation to the mother, and thus, as Bowlby suggested, the mother shapes the infant's stress coping systems.

Schore (2002) suggests that the **infant** post-traumatic stress episodes of hyperarousal and dissociation **imprint the template** for later childhood, adolescent, and adult post-traumatic stress disorders, all of which show disturbances of autonomic arousal, abnormal catecholaminergic function, neurologic soft signs and dissociation. This would be symptomatically expressed as a cycling between intrusive hypersympathetically driven terrifying **flashbacks** and traumatic images and parasympathetically driven **dissociation, avoidance, and numbing**. Recent models of PTSD refer to stressor-induced oscillations between traumatic and avoidant states, and cycling between the bi-directional symptoms of emotional re-experiencing and emotional constrictedness **(Antelman et al 1997)**.

Supporting this model is a growing body of research demonstrates orbitofrontal dysfunction in PTSD. The right orbitofrontal system is thought to act as the neural basis by which humans control their instinctive emotional responses through cognitive processes, and the emotional disturbances of PTSD are proposed to have their origins in the inability of the right prefrontal cortex to modulate amygdala functions.


Also supporting this model is the emerging evidence for considering those who develop PTSD as those who are most likely to develop the disorder as a result of **prior risk factors.** **Yehuda** argues that the development of PTSD represents an alternative trajectory to the normative response (Yehuda 2003). She suggests that the challenge, in the aftermath of a major trauma, is to determine who is at risk for failing to recover.

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As a Disorder of the Threat Response Archetype


It is possible that humans differ in the degree to which stress induces neurobiological perturbations of their threat response systems, which may result in a differential capacity to cope with aversive experiences. Morgan et al (2001) argued, based on their research, that individual variations in neuroendocrine responses may explain some psychological and behavioural responses to acute stress. These individual differences exist before trauma exposure and may be used to test constructs of stress hardiness and stress vulnerability in humans. This type of distinction may promote the exploration of a "selective fitness" hypothesis in the development of PTSD.


Schore (2002) argues that attachment experiences experienced early in life may be particularly important in shaping the individual's pattern of stress responses in later stages of life. Just as ***disorders of affect regulation*** may result from maternal deprivation or abuse, & contribute to many mental disorders, including anxiety disorders, mood disorders, borderline personality and antisocial personality disorders, PTSD may be a ***disorder of fear regulation*** in individuals made vulnerable by maternal deprivation or abuse.

The arguments favour a model of **excessive activation of the threat response neural circuitry in infancy** due to environmental failure to provide a safe & secure caretaker environment, when the relevant circuits are being sculpted and pruned by experience. In the last decade, a growing body of neurobiological research on PTSD has uncovered dysfunctional frontal-subcortical systems, and altered functional activity of the orbitofrontal cortex, anterior cingulate, and amygdala (Schore 2002).


Yehuda (2003) perceives exposure to traumatic stress as resulting in a **fear response**, which involves the initiation of concurrent and instantaneous biological responses that help assess the level of danger and then organise an appropriate behavioural response. She indicates that it is not possible to diagnose PTSD in the immediate aftermath of a trauma, according to DSM-IV, because of the diagnostic stipulation that symptoms occur for at least one month. Most (94%) trauma survivors have some type (or degree) of PTSD response (Rothbaum et

al 1993), which gradually recedes over time in most people (Kessler et al 1995).

Thus, PTSD may represent the **failure to recover** from a universal set of reactions (Yehuda 2003). Failure to contain or control the initial biologic response to stress appears to lead to a cascade of events resulting in symptoms of hyperarousal, recollection of intrusive events and avoidance of reminders. This perspective is then consistent with the view of PTSD as a disorder of ***failure to switch off activation of the threat response archetype***.


Derrick Silove (1998) also views PTSD as an ***'Over-learnt Survival Response'***. He hypothesises that a primitive learning centre in the limbic system rehearses traumatic memories immediately following exposure to trauma, inducing involuntary memories of the novel threat. He postulates that the mechanism developed during an evolutionary phase when there was an absence of sophisticated cognitive mechanisms and automatic learning following a single exposure to a potentially fatal novel threat would have conferred a survival value in the species. Further evolution led to the development of the neocortical pathway to process trauma memories. He suggests that PTSD results from **asynchrony between the two phylogenetically distinct pathways** in vulnerable individuals under conditions of extreme stress resulting in failure of cortical inhibition to limit the trauma rehearsal generated by the limbic lobe. **Silove** also suggests that a **mismatch between archaic biological mechanisms & novel cues in the modern environment** may play a role.


LeDoux (2001) argues that a **defective orbitofrontal system** operates in PTSD. He postulates that there is **a lack of orbital prefrontal feedback** regarding the level of threat, so that the organism remains in an amygdala-driven defensive response state longer than necessary. The **emotional learning** persists in the form of memories that seize control of mental life and behaviour. He showed that pathways leading from the central nucleus of the amygdala to the brainstem initiate defensive freezing and associated autonomic and endocrine reactions **(**LeDoux 2001**).**


The right orbitofrontal system is thought to act as the neural basis by which humans control their instinctive emotional responses through cognitive processes, and the emotional disturbances of PTSD are proposed to have their origins in the inability of the right prefrontal cortex to modulate amygdala functions (Hariri 2000).


LeDoux suggests that the introduction of an **active coping response** may reroute processing from the pathway inducing **dysfunctional passivity** to one controlling successful engagement with the environment. Rather than going to the central nucleus and engaging the passive fear response, the information is sent from the lateral nucleus to the basal nucleus of the amygdala, which does not project to the brainstem but instead to motor circuits in the ventral striatum. By engaging these alternative pathways, passive fear responding is replaced with an active coping strategy. This diversion of information flow away from the central nucleus to the basal nucleus, and the learning that takes place, does not occur if the rat remains passive. It requires that the rat take action. It is **"learning by doing,"** a process in which the success in terminating the conditioned stimulus reinforces the action taken. When

the rat shifts from passive to active coping, it is performing the neurological equivalent of "getting on with life" (LeDoux 2001).

LeDoux speculates, in his book The Emotional Brain, the hypothesis that "the struggle between thought and emotion nay ultimately be resolved, not simply by the dominance of neocortical cognitions over emotional systems, but by a more harmonious integration of reason and passion in the brain" (LeDoux 1996).

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As a Disorder of Imbalance Within the Triune Brain

This model was inspired by Conflict Within the Triune Brain Model of psychopathology proposed by MacLean (1985) and Stevens & Price (2000), & the PTSD models proposed by Schore (2002) & LeDoux (2001) above.

Imbalance between the right frontal cortex & the subcortical limbic lobes may lead to excessive excitation of the amygdala (limbic lobe), which is inadequately opposed by the cortex:

Brain Level	
1. CORTEX (Reason)	Defective prefrontal inhibition
2. LIMBIC (Emotion)	Activation of neural circuits inducing trauma rehearsal
3. REPTILIAN (Instinct)	

The Blocked Middle Level Losing Model of Anger-Induced Depression suggests that persistence of this situation may lead to development of a depressive disorder.

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As a Mental Module Disorder

As a Disorder of "Flashbulb" Memory

The term "**flashbulb memory**" was coined by Brown & Kulik (1977) to refer to the vivid recollections that humans may have of events considered to be of particular significance to the individual or group.▫They argued that the **flashbulb** effect is marked by extremely vivid and durable images containing a common informational structure and evoked by instances of high surprise and consequentiality, representing a unique type of memory.▫These memories are described as having a photographic quality and as being accompanied by a detail-perfect apparel of contextual information (weather, background music, clothes worn, etc.) pertaining to the time and place where the event was first known.▫They may also evoke emotions similar to the ones felt upon hearing the news.▫The **emotional arousal** serves to enhance the scope of detail of the memory and the **source of arousal** is an important determinant of an event's memorability (Libkuman et al 1999).▫There is evidence that exposure to flashbulb events evokes recall of similar events (Mahmood 1999).

It has been suggested that **flashbulb memories** are formed by the activity of an ancient brain mechanism evolved to capture emotional and cognitive information relevant to the survival of the individual or group.▫▫

Sierra & Berrios (1999) suggest that **flashbulb memories** should be considered as phenomena occurring in **drug flashbacks, palinopsia** (perseveration or recurrence of a visual image after the stimulus has been removed)**, palinacusis** (repetition of sounds after the stimulus has been removed), **post-traumatic memories** as well as▫**phobias, panic attacks, obsessional disorder, phantom-limb phenomena and depressive melancholia**.▫▫All of these experiences share clinical features such as paroxysmal repetition, sensory vividness, a capacity to trigger emotions, dysphoria, and a tendency for the rememberer to shift from the role of actor to that of observer and for the reminiscence to become organised in a stereotyped narrative.▫▫

Kandel proposes that the underlying molecular mechanism whereby this occurs may be the rapid removal of the family repressors that inhibit the conversion of short to long-term memory storage (Kandel, 2001).▫▫

The underlying biology of excessive activation of the threat response archetype might be **sensitisation**, a form of learned fear in which a person or an experimental animal learns to respond strongly to an otherwise neutral stimulus.▫▫An excessive activation of the threat response neural circuitry leading to **hyperarousal & hypervigilence** may result from an individual being **sensitised** by an aversive stimulus.▫▫Kandel provides the example of a person being suddenly exposed to an aversive stimulus, such as a gunshot going off nearby, to explain how a person will be sensitised by the unexpected noise.▫As a result, that person will be frightened and will now startle to an otherwise innocuous stimulus like a tap on the shoulder.▫ He details the biological underpinnings of this process in the sea slug *Aplysia,* an animal that

can learn to enhance its defensive reflex responses to a variety of subsequent stimuli, even innocuous stimuli, following application of an aversive stimulus to it. The simple nature of its nervous system permitted researchers to explore the molecular biology of learning & memory **(Kandel, 2001)**.

This process supports the proposal by **Schore (2002)** that attachment experiences experienced early in life may be particularly important in shaping the individual's pattern of stress responses in later stages of life.

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