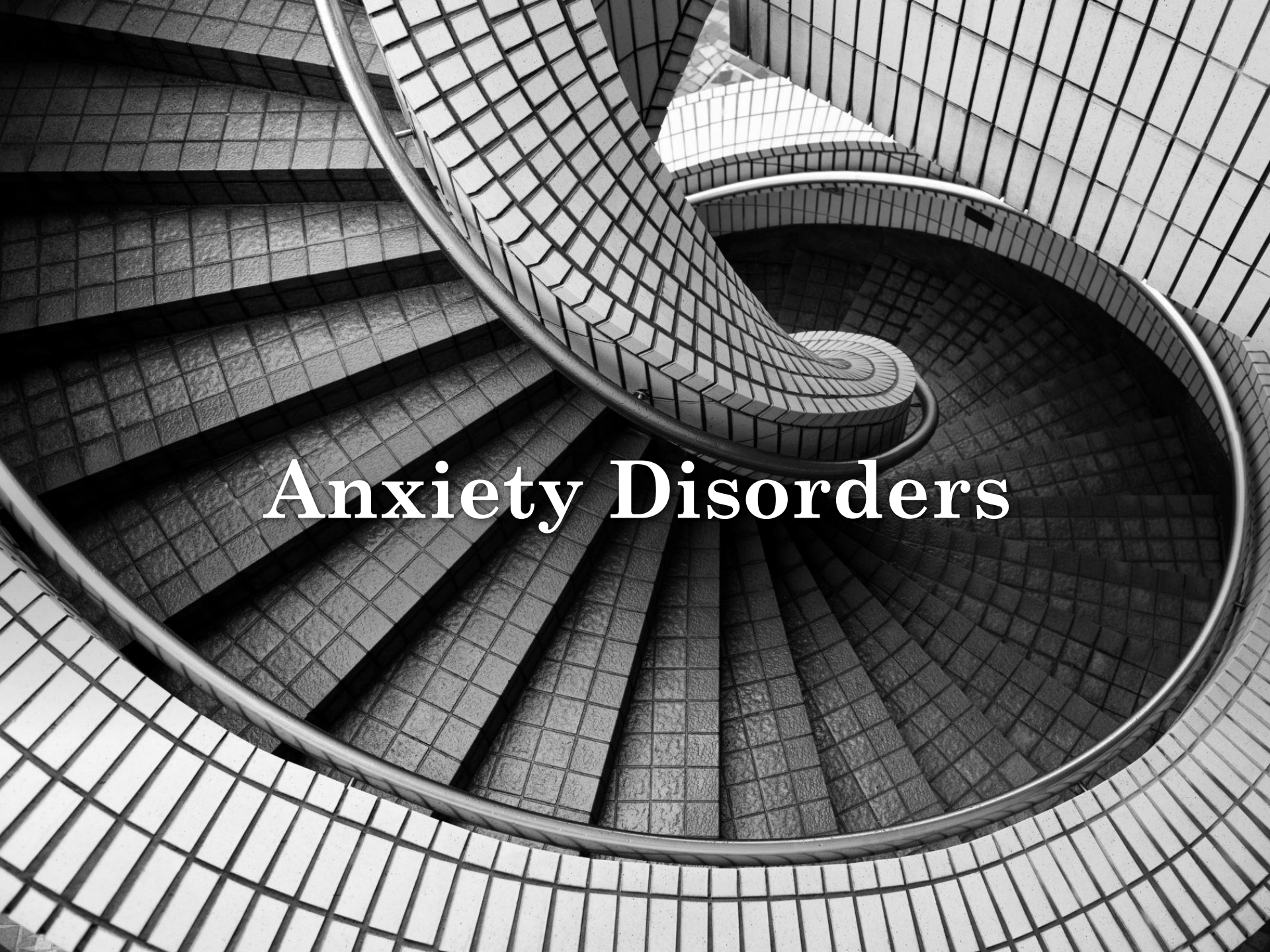


A black and white photograph of a spiral staircase. The staircase is viewed from above, showing the circular pattern of the steps and the railing. The floor is covered in a grid of small, square tiles. The railing is made of metal and follows the curve of the stairs. The lighting creates strong shadows, emphasizing the depth and curvature of the staircase.

Anxiety Disorders

• Overview of Lecture-

- What is anxiety?
 - Cognitive processes
 - Neurobiology of anxiety
 - Developmental aspects
- Types of anxiety
 - DSM classifications
 - GAD
 - SAD
 - Agoraphobia
 - Phobia
 - Panic disorder

When does anxiety become a disorder?

Anxiety is a normal human response to objects, situations or events that are threatening

Anxiety is different from fear due to its cognitive component (i.e. fear of the future)

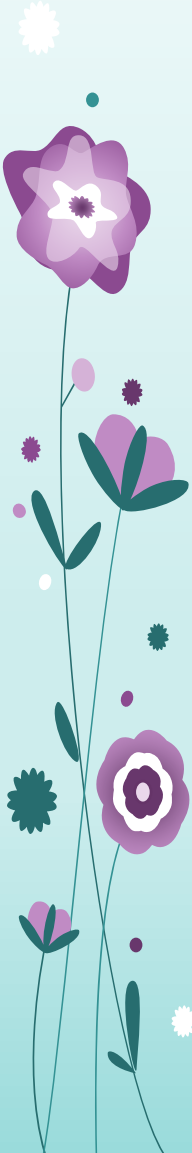
Anxiety can be helpful and adaptive (e.g. anxiety about giving lectures!)

Anxiety becomes a disorder when out of proportion or when it significantly interferes with life.

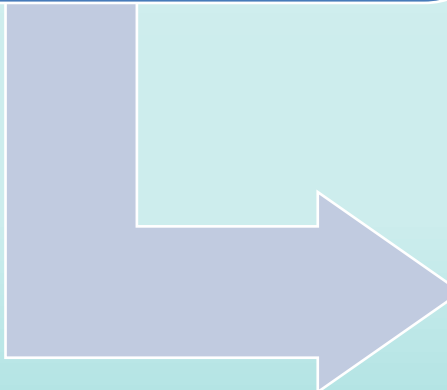
Fear VS. Anxiety

Fear and anxiety serve important evolutionary functions to keep us safe - fear allows us to combat/avoid immediate threats or danger, whereas anxiety increases vigilance and improves our ability to identify uncertain or potential threats.

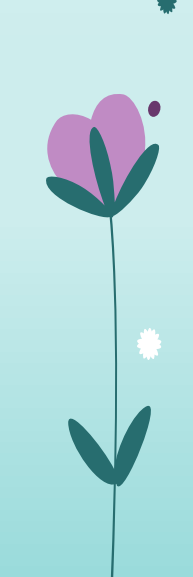
But, anxiety disorders can develop when anxiety or fear is excessive or occurs in the absence of true threat; immediate or future.



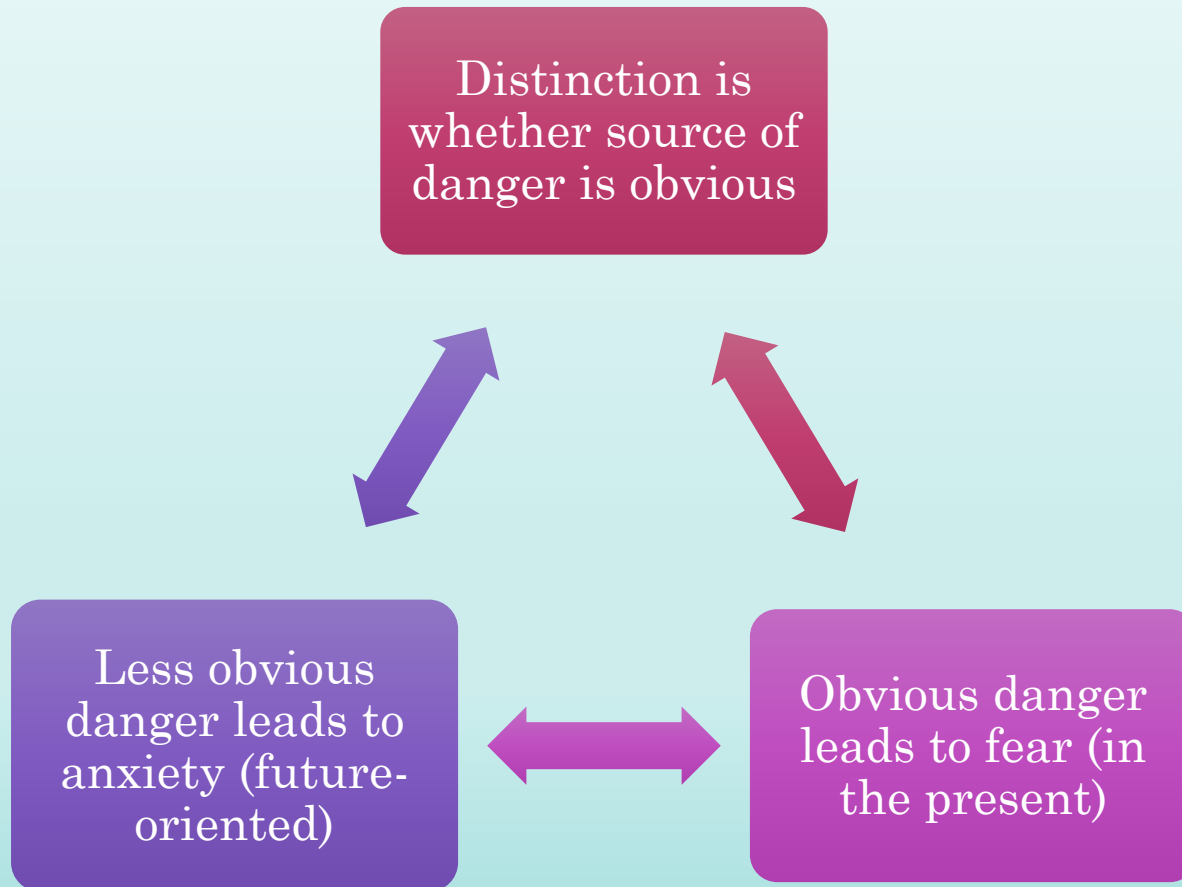
Fear is typically defined as a phasic and abrupt fight-or-flight response accompanied by intense arousal in response to an immediate and identifiable threat.



Alternatively, anxiety is often defined by a more prolonged state of tension, worry, and apprehension regarding uncertain, and potentially negative, future events. (Barlow, 2000)



Fear and Anxiety - Response Patterns



Anxiety in DSM - a group of anxiety disorders—that share obvious symptoms of clinically significant fear or anxiety.

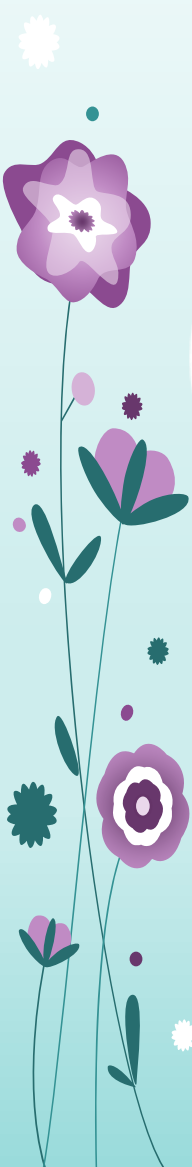


Anxiety is part of the normal human experience, we all experience it in varying degrees.

In 2013, there were 8.2 million cases of anxiety in the UK (Fineberg et al., 2013).

It can have a positive or negative impact and is referred to as Adaptive anxiety

Anxiety Disorders

A decorative illustration on the left side of the slide features a vertical stem with several stylized flowers. The flowers are in shades of purple, pink, and white, with green leaves. Some flowers have multiple layers of petals, while others are simple daisies. The stem is thin and green, with small green dots and white dots scattered along it.

They affect approximately 25 to 29 % of the U.S. population -most common category of disorders for women and the second most common for men (Berglund, Delmar, et al., 2005).

Although there is no activation of the fight-or-flight response as there is with fear, anxiety does prepare or prime a person for the fight-or-flight response should the anticipated danger occur.

Anxiety – Emotions, cognitions and behaviours

Cognitive/subjective level - negative mood, worry about possible future threats or danger, self-preoccupation, and a sense of being unable to predict the future threat or to control it if it occurs.



Physiological level - creates a state of tension and chronic over-arousal, which may reflect risk assessment and readiness for dealing with danger should it occur.



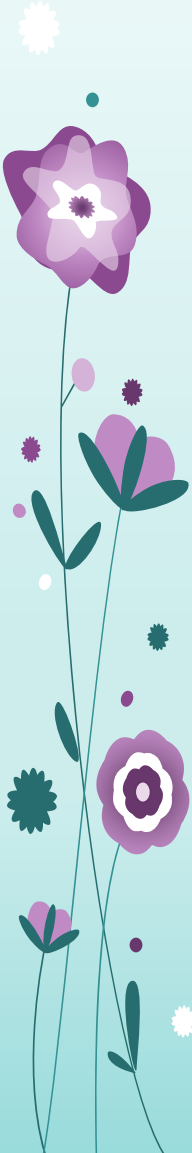
Behavioural level - create a strong tendency to avoid situations where danger might be encountered, but without the immediate behavioural urge to flee, as there is with fear. (Barlow, 1998, 2002).

DSM 5 Anxiety Disorders

- Separation Anxiety Disorder
- Panic Disorder
- Agoraphobia
- Specific Phobia
- Social Anxiety Disorder (Social Phobia)
- Generalised Anxiety Disorder
- Substance Induced Anxiety Disorder
- Anxiety Disorder Attributable to Another Medical Condition
- Anxiety Disorder Not Elsewhere Classified



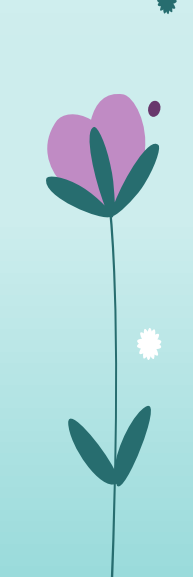
Anxiety



Chronic anxiety becomes maladaptive if it interferes with everyday functioning.

The most common form of anxiety disorder is GAD- excessive worrying about the future.

In addition there are specific anxiety disorders; social anxiety disorder (SAD), phobias and panic disorder.



Where in the brain...?



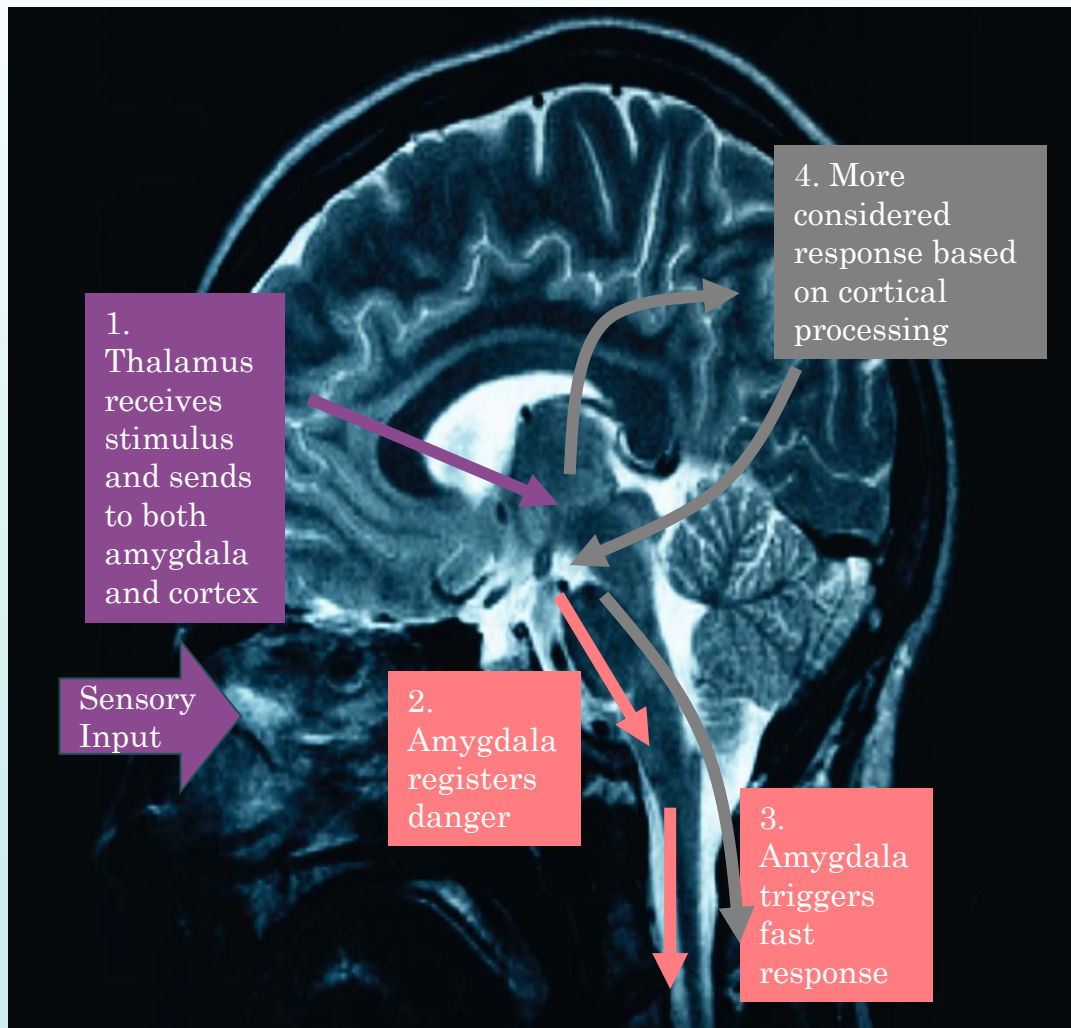
To identify neural circuits underlying fear and anxiety symptomatology, specific experimental paradigms have been developed.

Fear conditioning paradigm

- Pairs an aversive stimulus (eg, electric shock) with a neutral stimulus (eg, a blue light), resulting in the neutral stimulus becoming a signal of imminent threat.
- This permits separation of brain circuits that instill “fear” from those involved in the processing of the same or identical stimuli without fear content.
- A number of key structures have been identified that generate and modulate fear responses to the conditioned threat signal. Specifically, the thalamus integrates sensory input from the primary sensory cortices and sends output to the amygdala.

The Amygdala and the Limbic System

- The role of the Amygdala is to keep us safe – it sometimes thinks we are in danger when we are not, sending oxygen out to our arms and legs to prepare us to fight, flight or freeze.
- This equates to less oxygen for our thinking brain (or prefrontal cortex) – making it difficult for us to think clearly when experiencing anxiety.
- Parts of the brain involved in fear response - Thalamus, amygdala and hypothalamus, which then instruct the endocrine glands and autonomic nervous system.
- Two systems: `evolved fear` module versus `considered response` = “fight or flight” versus “feel the fear and do it anyway (or do it differently)”



Two systems/mechanisms of response:

Interestingly, evidence of differential patterns is also emerging, such that within a spectrum of disorders ranging from more fear-based to more anxiety-based, greater involvement of emotion-generating regions is reported in panic disorder and specific phobia, and greater involvement of prefrontal regions is reported in generalized anxiety disorder and posttraumatic stress disorder.

Anxieties are built into our system; most people feel anxious when looking down from a high building and infants around 9 months will cry when their mother leaves them.

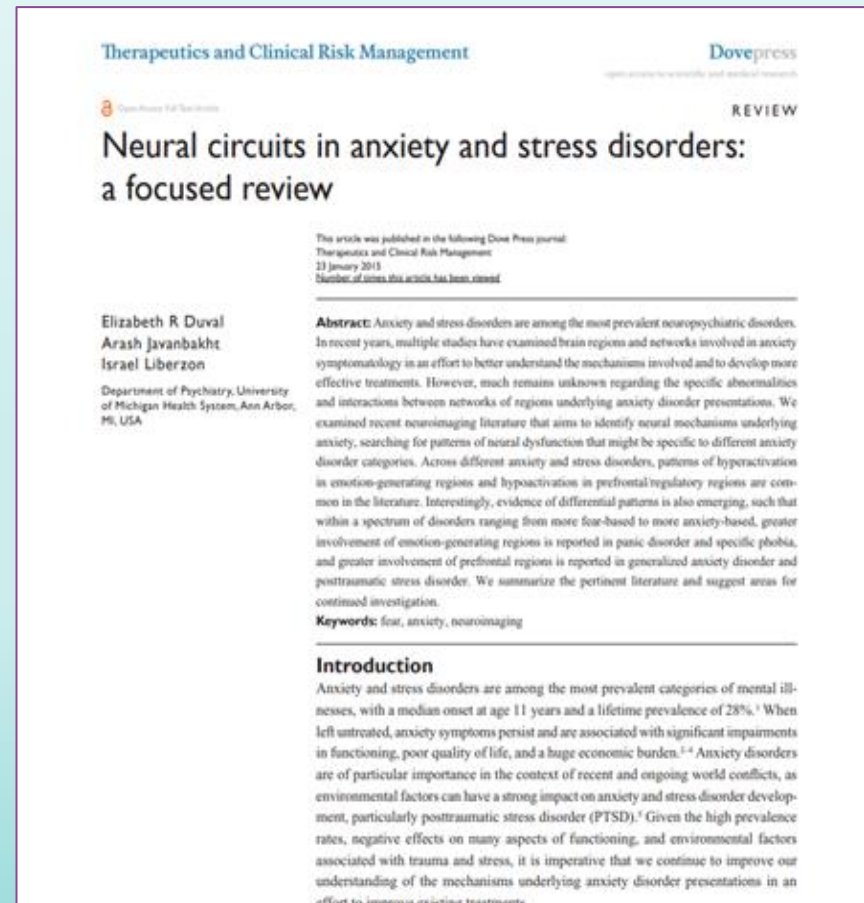
To date, we do not fully understand the fear and anxiety systems; one example of this is the different cortical networks used within PTSD and panic disorder, they are believed to use these two separate systems.

Fear and anxiety use both higher and more primitive brain processing. The higher level relates to cognition and can be induced- think of flying, whereas the primitive processes tend to be automatic. The limbic system can respond to stimuli on its own- the Amygdala responds to an angry face which triggers the ANS reaction.

LeDoux (2002, 2003) shows that there are two pathways for processing fear- cortical and sub-cortical. The cortical pathway is slower and more deliberate.

Supporting evidence:

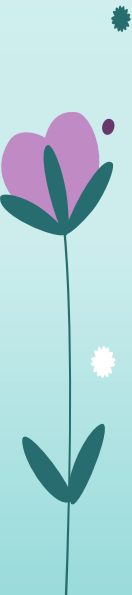
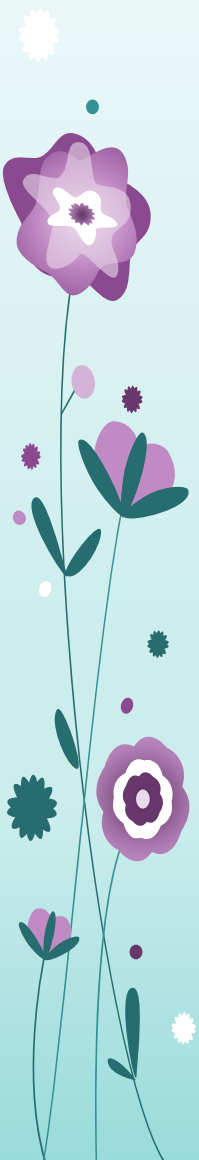
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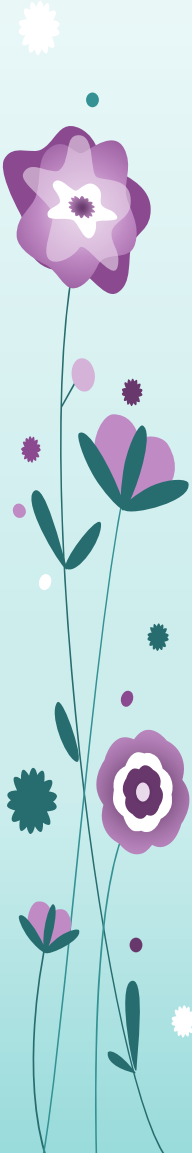


How `Plastic` or `Fixed` are these systems?

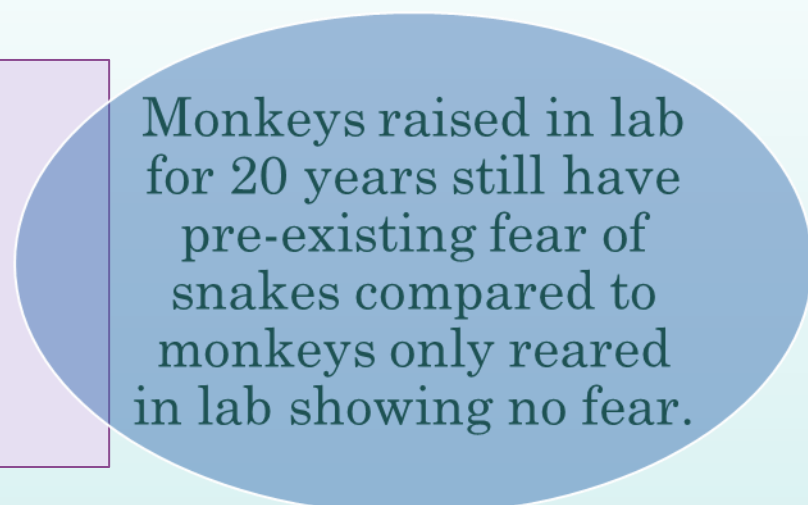
Certain phobias run in families but we do not all have the same fears, suggesting a degree of learning.

Classic studies by Ohman & Mineka (2001) demonstrate this-





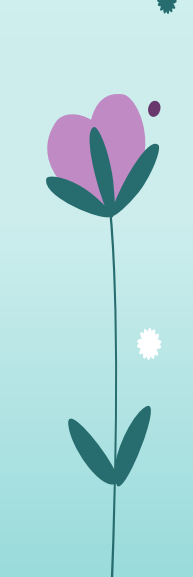
Fears, Phobias, and Preparedness: Toward an Evolved Module of Fear and Fear Learning (Ohman and Mineka, 2001)



Monkeys raised in lab for 20 years still have pre-existing fear of snakes compared to monkeys only reared in lab showing no fear.

Mineka paired the monkeys together and the young lab raised monkeys learned via observation. They also learned to fear snakes via watching a video of a monkey demonstrating fear to a snake.

Next she edited the video to show fear of a flower. This time the lab reared young monkeys did not learn the fear – so the fear is innate (evolutionary pre-determined to fear snakes).



Panksepp (2004) Fear can be evoked in four ways:

- By painful stimuli-shock can induce fear
- Pairing with aversive stimuli
- If evolutionary cues are present (rat smells cat)
- Creating frustrating situations (waiting for a reward that doesn't come)

- Therefore, anxiety occurs if worrying that negative events will happen or alternatively anxiety occurs if positive events do not!
- This suggests that our brains are trying to predict and anticipate the future, based on the occurrence of previous events. If unexpected events happen our EEG changes.

Testing the Anxiety response – individual differences

`Dot probe task`

Individuals are asked to focus on an X on a screen with a word each side.

They have to press a key each time the word is replaced by a dot.

Threatening words slow speed down and increase the rate of errors.

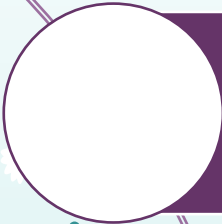
This even occurs if the words are masked!

These findings suggest that the primitive amygdala is responsible for this (McLeod & Mathews, 2012).

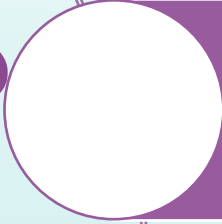
Finally, anxious individuals focus on the negative, expect the negative to happen and interpret ambiguous events as negative - this bias leads to both the development and maintenance of anxiety.

<https://www.youtube.com/watch?v=KVkPGkG7vZQ>

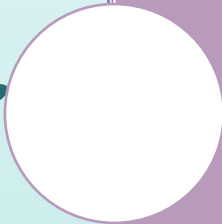
Types of anxiety



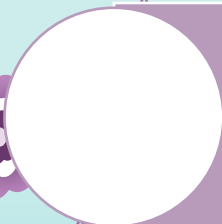
A) Subjective Anxiety- why? Fear in consciousness is difficult to describe and is measured via scales.



B) Panic Anxiety- rushes of intense anxiety but not necessarily accompanied by autonomic arousal. (Panic feeling is subjective).



C) Trait Anxiety- an enduring personality trait/disposition. E.g. internalising, neuroticism, negative affectivity, anxiety sensitivity (ASI). However, whilst we have some predictability, it only accounts for 15% of the variance (Naragon-Gainey 2010).



D) Physiological Anxiety- Autonomic arousal without reference to a persons subjective state. Our body reactions not always in synch with our consciousness.



E) Anxiety as a ***perception*** of physiological arousal- social cognition proposes that our physiological arousal is less important than our perception of it!

Theories of Avoidance

- **Dysfunctional Avoidance** (Barlow et al., 2011) - particularly for explaining phobias. Some phobias can be understood in terms of trauma but others not.
- For many years the two factor model dominated the literature, but was flawed on many accounts = focus on the experience of fear as the instigator and motivator of avoidance. However many people fear objects that they have never encountered (Wittchen et al., 2010) - Eg agoraphobics do not go out, but still fear it!
- `The Bio-informational Theory of Avoidance` (Lang 1985). Emotions are understood as behavioural acts which rely upon unconscious associative networks and memory nodes linked to diverse neurophysiological substrates. But highly criticised as being over complicated (Barlow 2002).

Social Cognitive Theories of Avoidance

Phobias, anxieties and panic attacks occur due to conscious appraisal of the self and our circumstances (Amundsson & Beck 2013).

They explain people's beliefs, expectations and judgements about things as representable to themselves in conscious awareness and allow them to communicate them.

Context specific thoughts that are underlined by cognitive consciousness

Measuring perceived danger is central in a causal role- anticipatory anxiety is measured as the level of anxiety people THINK they would reach IF they did a specific task. Anticipatory panic is the likelihood (0-100%) that doing the task would induce a PA. likewise the same for perceived efficacy and perceived control.

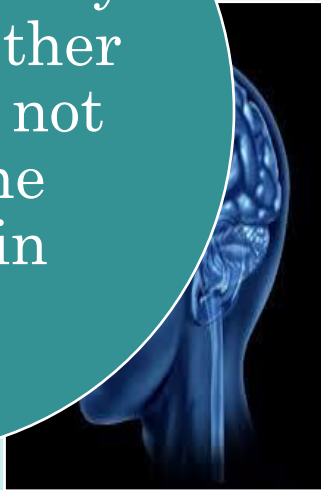
Anticipatory anxiety and panic were strong predictors of avoidance, implying that people's self-perceptions affect their behaviour.

Neurobiology of anxiety

Anxiety involves: Prefrontal cortex, amygdala (hyperactive), and hippocampus.

- These areas involved in cognition, emotion processing, memory, vigilance, attention to threat and the REGULATION of fear

Fear and anxiety typically studied together but they do not involve the same brain areas.



Neurotransmitters involved: Primarily GABA - the major inhibitor in the brain (stops neurons from firing – stops thinking over and over the same thoughts/stops the anxiety when it reaches the amygdala). The less GABA the more anxiety you feel. GABA receptors are dense in the PFC, amygdala and hippocampus.

Animal studies show that using serotonin to increase GABA in the amygdala reduces fear. An adult rat nurtured and licked in infancy has a greater expression of GABA activity in the amygdala and fewer signs of fearfulness and stress (Fries et al., 2004).

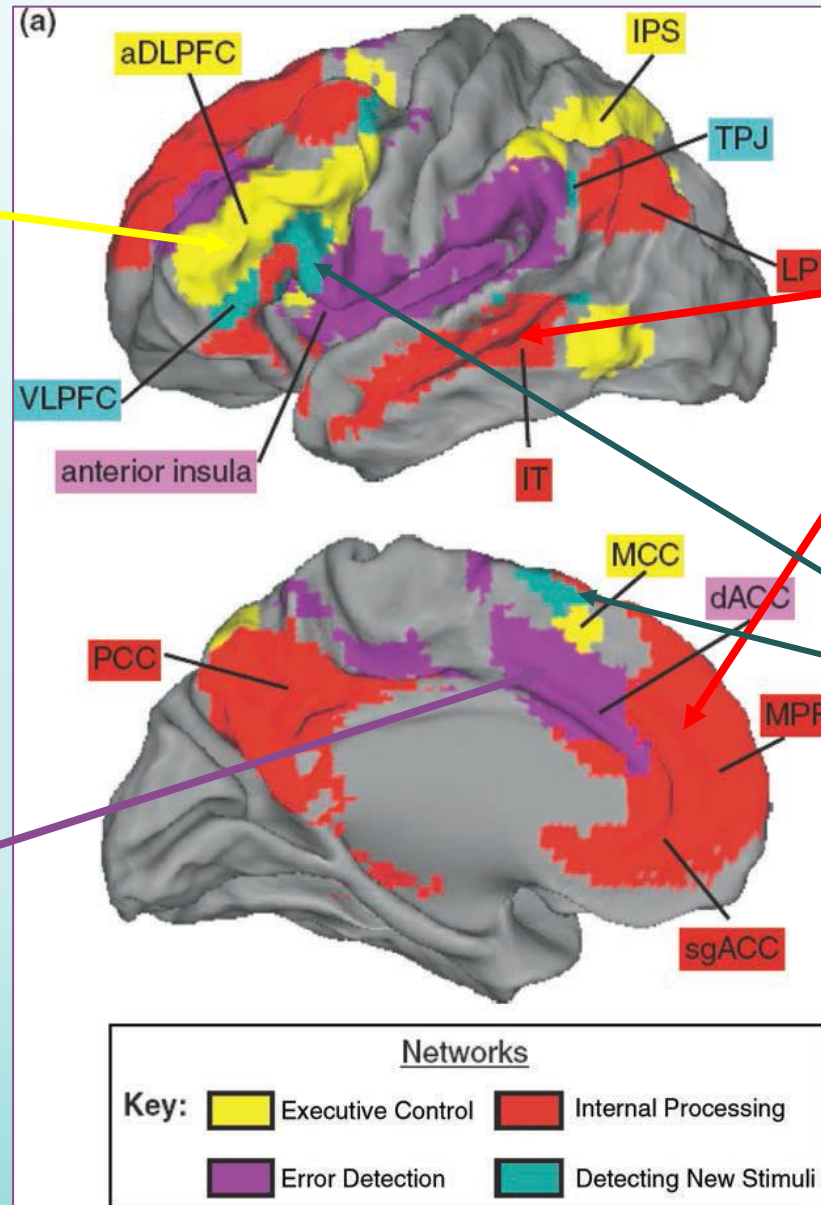
Chad et al., (2012) Four functional areas

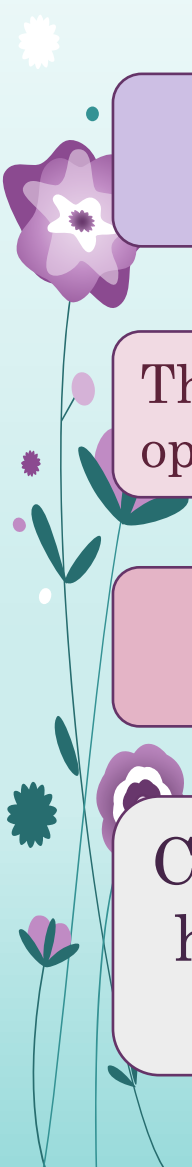
Executive control

Default network

Ventral attentional

Salience network





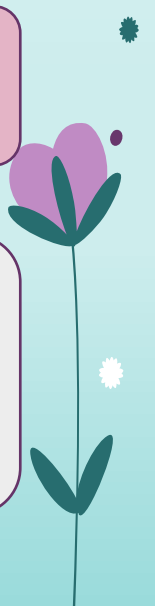
The SALIENCE network- (cingulo-opercular) for detecting errors or conflicts

The EXECUTIVE CONTROL- (fronto-parietal) to implement increased control

The VENTRAL ATTENTIONAL- to detect new stimuli as opposed to the task in hand

The DEFAULT- (internal processing) for self-inspection, planning and emotional regulation

Chad et al., (2012) propose that individuals with AD's have specific patterns that are overactive in salience and underactive in executive and default regions.



Neurobiology of anxiety: Heritability

Gross & Hen (2004) - anxiety is a developmental disorder affected by both genes and the environment - twin studies - heritability is around 30-40%.

Higher concordance for monozygotic (vs. dizygotic) twin pairs

- Panic disorder: Heritability of .48
- Phobia heritability: Around 50%. Varies for specific phobias-
- Social phobia: Heritability of 51%, Situational phobia: 46%, Animal 47%, blood injury 59%.
- GAD: Heritability ranging from 15-30%.

Anxiety: Developmental aspects

Children and adolescents across all cultures show similar profiles of anxiety and fear.

- Example: Infants (human and non-human) show distress vocalizations when separated from their mothers.
- By adolescence, the fear turns to anxiety in that the object of concern is not present. Social anxiety about a future situation is common among adolescence.

Normal expressions of fear and anxiety becomes pathological when it interferes with daily life or causes distress.

2.5% to 5% of children and adolescents meet criteria for anxiety disorders (Rapee, 2009).

Specific phobias develop earliest:
Early to middle childhood.

Social phobia: Early to middle adolescence.

Obsessive-compulsive disorder:
Mid to later adolescence.

Panic disorder: Early adulthood.

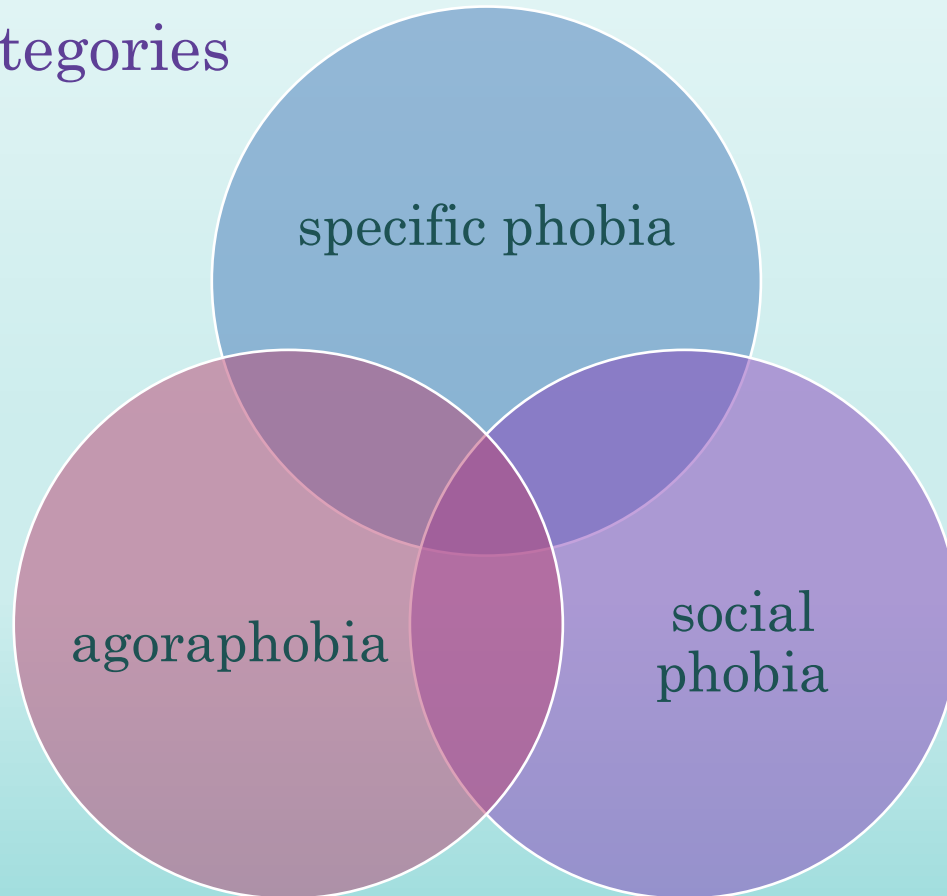
The Disorders



Phobias...the most common anxiety disorders

A phobia is a persistent and disproportionate fear of some specific object or situation that presents little or no actual danger and yet leads to a great deal of avoidance of these feared situations (*DSM-5*)

Three main categories
of phobias:



Top ten phobias...

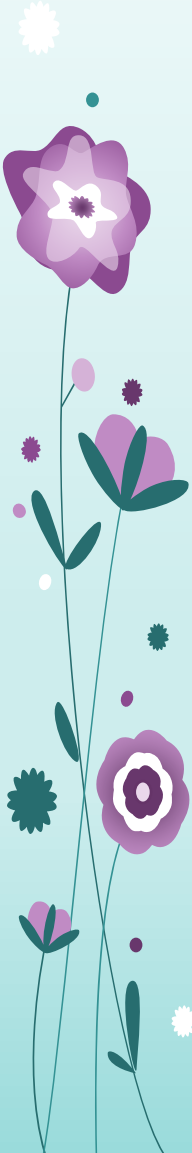
And recently...
Tryptophobia

THE NATION'S TOP TEN PHOBIAS

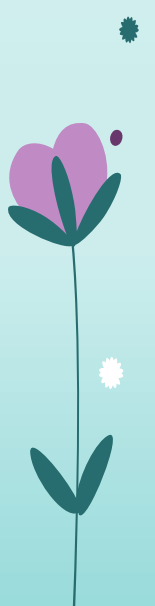
- 1 Arachnophobia — spiders
- 2 Social phobia — social or public situations
- 3 Aerophobia — flying
- 4 Agoraphobia — open or public spaces
- 5 Claustrophobia — enclosed spaces
- 6 Emetophobia — vomiting
- 7 Acrophobia (vertigo) — heights
- 8 Cancerphobia — developing cancer
- 9 Brontophobia — thunderstorms
- 10 Necrophobia — death (your own and others')



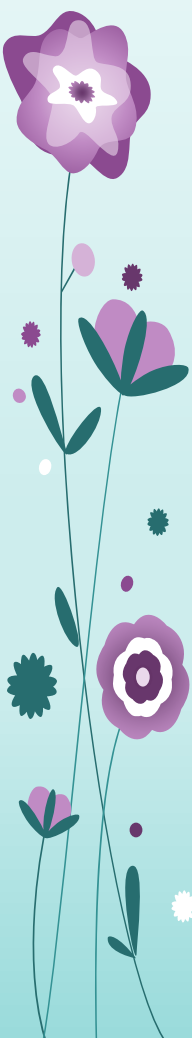
Separation Anxiety Disorder

A decorative floral illustration on the left side of the slide, featuring a tall stem with several purple and white flowers and small green leaves.

As the infant develops, they recognise the concept of object permanence, particularly when they interact with other children. However, for some individuals they continue to experience distress- they worry that something will happen to their caregiver and have nightmares which can continue into adulthood.

- Symptoms are more often found in girls
 - 4% incidence in childhood
 - 1.6% in adolescence
 - 0.9-1.9% in adulthood
- 
- A decorative floral illustration on the right side of the slide, featuring a tall stem with several purple and white flowers and small green leaves.

Separation Anxiety Disorder



Diagnosis is made when suffering is longer than 4 weeks, with 3 out of 8 symptoms present-

Distress without attachment figure

Worries about welfare of caregiver

Worries about getting lost

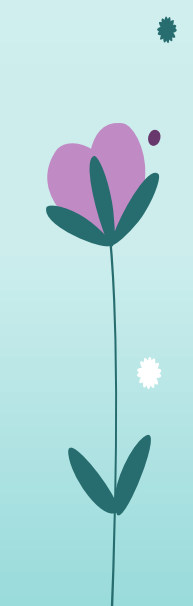
Unwilling to leave the home

Fear of being alone

Unwilling to sleep alone outside the home

Nightmares of separation

Physical complaints



Generalized anxiety disorder (GAD)

Characterized by excessive anxiety and worry for more than 3 months.

Bodily symptoms: Feeling on edge and muscle tension. In fact muscle tension is one of the most consistent symptoms.

Anxiety must lead to one of more of the following:

- Avoiding activities that can have negative outcomes
- Over preparation for activities that can have negative outcome
- Procrastination due to worries
- Repeatedly seeking reassurance due to worries

GAD often precedes depression by up to 7 years.

- Along with depression, GAD is one of the most frequently diagnosed mental disorders in the US.
- It is seen in twice as many females as males.
- The prevalence in one year is 3.1%
- The lifetime prevalence is around 5.7% (Kessler et al., 2005).
- GAD peaks in middle age then begins to decline.
- Most individuals with GAD (80%) also meet the criteria for other disorders such as MDD, PD or Social phobia (Brown et al., 2001).



Treatment: Medications



Since the 1970's BZ's have been prescribed which have shown effects within one week in a reduction of anxiety by 65-70% with some individuals showing full recovery. However, if stopped the symptoms reoccur.

Benzodiazepines are thought to influence GABA activity.

- Individuals with anxiety have reduced GABA activity which in turn results in less inhibition of those brain structures that are involved in threat responses.

Azapirones are also used.

- Buspirone is a common azapirone
- Influences serotonin receptors in the brain and influences more of the cognitive components of GAD.
 - Has fewer side effects than benzodiazepines.
- Antidepressants are also common.
 - Both tricyclic antidepressants such as imipramine and SSRIs such as paroxetine have been shown to be effective with GAD.

GAD- Treatment



CBT is the approach that has received the most attention from researchers.

- Behavioral approaches: Relaxation training and other such techniques.
 - Cognitive behavioral approaches: Automatic thinking.
 - Train clients to detect internal and external anxiety cues and to apply new coping skills that focus on both psychic and somatic symptoms.
 - More recently, in RCT mindfulness treatment more effective than stress related strategies (Hoye et al., 2013)
- 

GAD: Treatment



- Identifying the anxiety associated thoughts, images, beliefs, etc.
- Discussing these to bring out their causal role.
- Leading clients to question the validity of thoughts or beliefs, and to search for evidence.
- Helping clients to develop alternative less anxiety arousing assumptions or interpretations.
- Testing alternative viewpoints in homework assignments or experiments.
- Teaching the above methods as self-helping coping devices to be used in real life.

Social anxiety disorder (SAD)

Concerns over meeting new people or public speaking. Fears of humiliation, embarrassment or rejection. These are common concerns, however, when these feelings are severe and last for over six months, then it would be considered a social anxiety disorder (SAD).

SAD is characterized by marked fear or anxiety about one or more social situations in which the individual is exposed to possible scrutiny by others.

Lifetime prevalence: 8%.

- More common in women than men.
- Associated with mood disorders and substance abuse.

Role or prior experiences: 92% of adults with social anxiety report to having experienced negative social events in childhood prior to becoming socially anxious.

Diagnosis

- The persistent and severe fear of social situations which is manifested in one of three types-
- Social interactions
- When observed
- When performing

- They are afraid of offending people, they fear humiliation and rejection, believing that the outcome of social interaction will be negative.
- What is the difference between this and avoidant personality disorder?

SAD: Neuroscience perspective

The social brain regions are involved in social anxiety-

Amygdala, PFC, ACC and insula areas. Important for social and emotional processing.

The insula areas may be overactive.

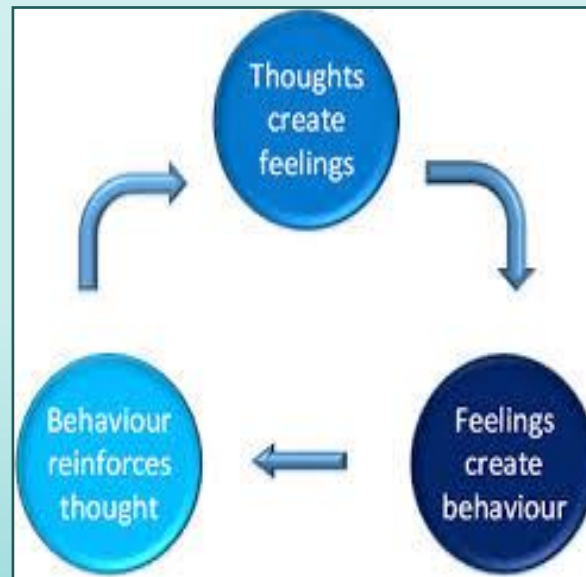
Cognitive processes may not inhibit the amygdala, leading to greater emotional responses than required by the situation.

Evolutionary explanation: Individuals with social anxiety process social situations with evolutionarily older alarms systems such as the amygdala whereas non-anxious individuals process the same situation with newer cognitive/analytic processes using the prefrontal cortex. Evidence for this comes from Ohman (2009)- SAD individuals have enhanced amygdala responses when shown hostile face.

SAD: Treatment

Therapies include: CBT, exposure therapy, social skills training, group CBT, and medications.

CBT assumes that social anxiety is produced by the person's automatic thoughts in the social situation or the expectation of a social situation. The task is to help the person detect and restructure these thoughts and expectations.



SAD: Treatment

Psychopharmacological approaches: MAO inhibitor, (phenelzine sulfate).

More recent medications have included SSRIs (paroxetine and sertraline).

The norepinephrine-serotonin reuptake inhibitor venlafaxine has also been used.



Good success with exposure therapy, creating a hierarchy of situations and the fear/anxiety the person expects to feel. The individual then moves through scenarios or real situations (Abramowitz et al., 2011).

Agoraphobia



Agoraphobia is the condition in which a person experiences fear or anxiety when being in public.

Panic disorder and agoraphobia can co-occur.

Both CBT approaches and antidepressants are effective in treating agoraphobia.

Can involve public transportation, open spaces, or places that are crowded. Can also be a fear of leaving the house.

One characteristic of agoraphobia is the concern that escape from the situation would be difficult.

Age of onset: early adulthood.

More common in women than men.

May be caused by early-childhood trauma.

Specific Phobias

A specific phobia is the condition in which an individual experiences fear or anxiety to a particular condition or object.

Common phobias are fear of snakes, spiders, flying, height, blood, injections, and the dark.

Lifetime prevalence natural phobias: 8.9-11.6% collectively.

Except for height phobia, large gender differences have been found with more females than males displaying phobias.

Situational phobias also quite common (5.2-8.4% prevalence rates).

Fear or heights is most common (3.1-5.3% prevalence rates).

Over 50% of people with one phobia were found to also have 3 or more additional phobias during their lifetime.

Phobia

TABLE 1. Clinical features of SP types

Phobic features	Types			
	Animal phobia	Natural environment	Situational phobia	B-I-I phobia
Prevalence	3.3–5.7%	4.9–11.6%	5.2–8.4%	3.2–4.5%
Onset	6.3–9.2 years	6.5–13.6 years	13.4–21.8 years	5.5–9.4 years
Gender ratio	Female > male	Female > male, most common type among males	Female > male	Mixed findings
Impairment			Seeking professional help, medication, interference with daily and social life	
Focus of fear	Disgust, revulsion	Danger of harm	Danger of harm	Physical symptoms (fainting), disgust, revulsion
Physiological fear response	Activation of dorsal anterior cingulate cortex, anterior insula			Vasovagal fainting, activation of bilateral occipito-parietal cortex and thalamus
Comorbidity	Depression	Depression, heights phobia in women → anxiety disorders	Affective disorders, childhood-onset disorders, substance use disorders, panic attacks	Marijuana abuse, depression, panic disorder, OCD, AG, SAD, Among diabetics → peripheral vascular disease, cardiovascular disease
Risk factors	Experiential, genetic			Women, low education

Phobia: Neuroscience perspective

Genetic relationships exist, but not very strong, so whilst situational and animal phobias do have links it is also likely to be learned.

Areas involved in phobia: Anterior cingulate cortex, the insula, the medial prefrontal cortex, the orbitofrontal cortex, and the thalamus

- Example: When fear objects are shown to individuals with phobias, exaggerated activity is seen in the amygdala, the insula, and the cingulate cortex.
- fMRI's were obtained for a specific spider phobia, where neutral pictures were paired with 3 pictures; spider, aversive scenes such as mutilation and neutral scenes. These were compared to a non phobic group with resulting differences in enhanced activity in PFC, ACC, Amygdala, Thalamus, with also greater activity in response to phobia related condition stimuli.

Phobia: Treatments

Exposure therapy is the typical treatment approach (Antony & Barlow 2002).

Debate has ensued regarding length of treatment. Either 1 three hour treatment or shorter treatments 2-3 times. Ost (1996) either is sufficient to bring about change.

Using these techniques, Straube et al., (2006) measured brain changes, in which the goals of therapy were to

1) hold a tarantula for 10 min

2) catch a moving/non moving spider 10 times

3) catch spiders in the basement of a building 3 times

4) touch a rapidly moving house spider

Doing this showed reduction in activity in the insula, ACC and related structures.

Panic disorder

It happens without an actual situation which would suggest danger.

Panic attacks come quickly and carry with them an intense feeling of apprehension, anxiety or fear (Craske et al., 2010; Fava & Morton, 2010).

Physiological symptoms can include shortness of breath, trembling, heart palpitations, dizziness, faintness, and hot or cold flashes. The person can also experience the world as if it were not real and be concerned about dying.

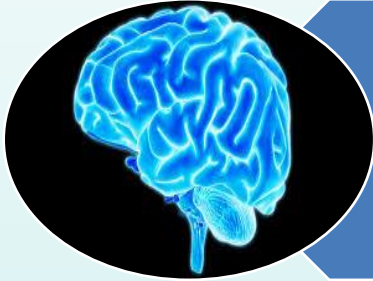
Symptoms usually peak within the first 10 minutes of the attack. The experience of one's heart pounding (97%) and dizziness (96%) are reported by almost all individuals who experience panic attacks.

Age of onset: Early adulthood, although panic disorder can be seen in children and adolescents (21-23%).

More common in women than men.

Lifetime prevalence rate: 4.7% .

Neuroscience perspective: Panic disorder

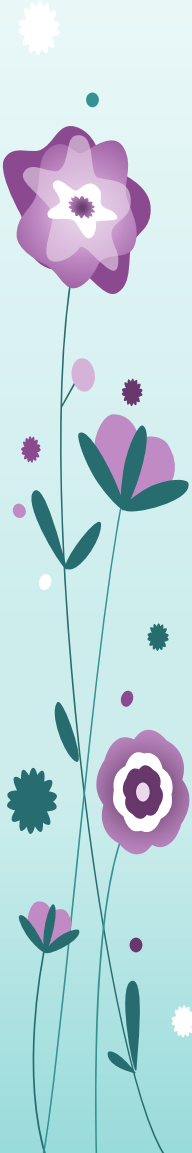


Panic and anxiety involve different areas of the brain (Graeff & Del-Ben 2008).

Anxiety is integrated in the forebrain whereas panic is organized in the midbrain, especially the basal ganglia and limbic structures.

- Decreased gray matter in individuals with panic disorder has also been reported in these areas (Lai, 2011).

Treating Panic disorder



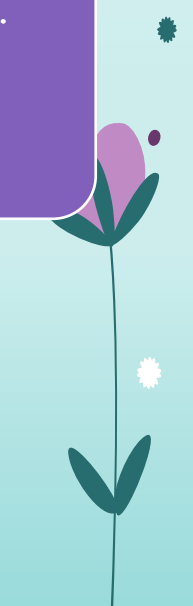
Medication is given- mainly SSRI's and BZ's.



CBT can be given with the purpose of educating the person regarding the nature of panic disorder symptoms, to allow for cognitive restructuring to reduce the catastrophic thoughts.

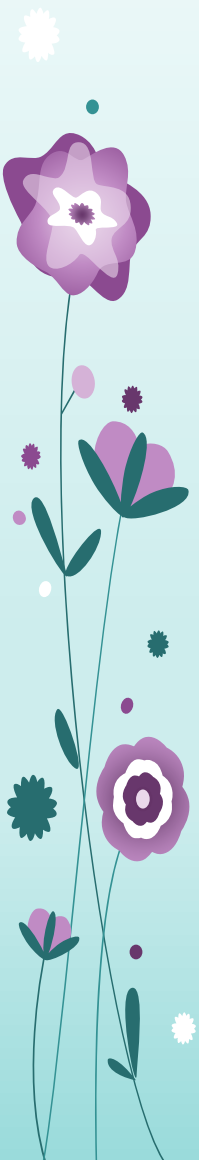


CBT and medication combined in ultimately the best (Mitte 2005).

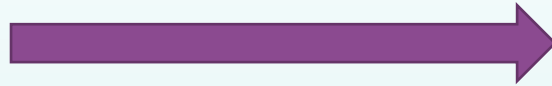


To conclude...

- The neuropsychology of anxiety related disorders is complex and not yet fully understood.
- Fear and anxiety have overlap with subjective, behavioural and neurological factors. However, there are key differences.
- We know that there are at least two different ways of acquiring phobias, depending on what type of phobia they are. These pathways are based on disgust-avoidance responses and also fear conditioning acquisition.
- Another acquisition mechanism may be through anxiety 'expectancies' and the interpretation of bodily sensations as threatening.
- Many researchers argue that anxiety disorders lie on a continuum, with fear at one end and anxiety at the other, each modulated by overlapping as well as distinct brain regions.



FEAR



ANXIETY

- Hippocampus, Amygdala and ACC implicated across all anxiety disorders.
- PD & SP deficits in emotion generating areas,
- SAD deficits in emotion generating areas and those for sensory control.
- GAD deficits in emotional regulation (frontal region).
- PTSD deficits in both regions.

Panic disorder

- Physiological symptoms and avoidance.
- Thalamus, Amygdala, Insula, dACC.

Specific phobia

- Intense fear/distress, avoidance, hypervigilance.
- Thalamus, Amygdala, Insula.

SAD

- Cognitive distortion, fear, avoidance and vigilance.
- Less connectivity with Amygdala and vIPFC, and between Insula and dACC.

PTSD

- Reliving of experiences, negative mood, hypervigilance.
- mPFC, rACC.

GAD

- Excessive worry over ambiguous cues, cognitive errors.
- mPFC, rACC.

It is evident that both fear and anxiety are involved, but the difference is the **DISPERSION** of **FUNCTIONAL** changes in the brain.