Why Do We Fear?
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The Biological Basis of Fear

Fear is perhaps the most evolutionarily conserved emotion because, without it, life would be short. It is fear that enables organisms to detect and respond to threats ranging from poisonous snakes to natural disasters. But what actually happens in the brain when we feel fear? And why does this critical emotion sometimes go so badly awry?

The Neurobiology of Fear

The response to fear starts with the detection of threat – the sight of a predator or the sound of a scream. As those signals pass to the brain they trigger the amygdala, a pair of almond-shaped regions of the brain within the medial temporal lobe that are critical for processing emotional stimuli, especially those related to danger. The amygdala processes a fear signal before it has reached the area of the brain dealing with higher functions, which is why you may jump or scream at a sudden surprise before rapidly realising that it doesn't present a threat.

Much of what we know about the amygdala's role in fear comes from studies of individuals with damage to this structure. The most famous case is that of Patient S.M., a woman with Urbach-Wiethe disease, a rare genetic disorder that resulted in degeneration of her amygdalae. Remarkably, S.M. shows a profound lack of fear in situations that would normally be terrifying—such as being held at knife-point or walking through a haunted house. She also fails to recognize fear in others' facial expressions and does not experience the typical bodily sensations associated with fear. Importantly, while S.M. lacks fear, she is capable of experiencing other emotions, which suggests that the amygdala is specifically tuned to fear processing, not emotionality in general. Her case has been pivotal in demonstrating the amygdala's centrality in initiating and interpreting fear responses.

Fight or Flight

Once activated, the amygdala communicates with multiple brain regions, but its interaction with the hypothalamus is particularly important. This region, directly next to the amygdala, is responsible for initiating the autonomic and endocrine responses to fear. It triggers the release of stress hormones via the hypothalamic-pituitary-adrenal (HPA) axis and activates the sympathetic nervous system, leading to the characteristic physiological symptoms of fear: increased heart rate, pupil dilation, and rapid breathing.

Key amongst the hormonal responses to fear is adrenaline. This molecule, which is also known as epinephrine, is released from the adrenal medulla (situated above the kidneys) during the "fight or flight" response. Adrenaline is responsible for most of the changes that we associated with being afraid: it increases heart rate, dilates air passages, mobilizes energy stores, and redirects blood flow to muscles.

Interestingly, the brain/adrenaline pathway is bidirectional. At the same time as stimulating physiological effects, adrenaline signals back to the brain leading to the release of the hormone norepinephrine, which stimulates memory consolidation, thereby ensuring that organisms remember dangerous situations more vividly than less emotionally-charged ones. At the same time, the physical symptoms of fear also influence our emotional perception of it. Volunteers injected with adrenaline prior to watching a horror movie responded more dramatically to frightening scenes and rated the film itself as more terrifying than those exposed to a placebo.

When Fear Goes Wrong

Although fear is critical to protect us from threats, as with any biological system it can sometimes go wrong, often with catastrophic consequences. Panic disorder is a clinical condition characterized by recurrent, unexpected panic attacks - intense episodes of fear accompanied by physical symptoms such as heart palpitations, dizziness, and shortness of breath – without an underlying cause.

Twin and family studies indicate a strong heritable component to panic disorder, with heritability estimates around 40–60%. Several genes have been implicated, though no single gene has been definitively identified as causative. One of the most studied candidates is the COMT gene, which influences the degradation of neurotransmitters like dopamine and norepinephrine. People suffering from panic disorder frequently have a version of this gene which degrades these molecules more slowly, suggesting that excessive residual levels of fear-associated neurotransmitters may be one contributor to the condition.

Other genetic studies have suggested a link with the serotonin system. One particular polymorphism in the serotonin transporter gene SLC6A4 is associated with heightened amygdala responses to fearful stimuli and a greater risk for anxiety disorders, including panic disorder.

The Role of the Microbiome in Fear

Although we think of fear as a deeply personal response, in recent years an increasing body of data is emerging suggesting that our individual fear behaviours may be strongly influenced by the microbes that live in and on us. Perhaps the most striking example is *Toxoplasma gondii*, a parasitic protozoan that infects warm-blooded animals. In rodents, *T. gondii* infection leads to a loss of innate fear of cat odour, making them more likely to be eaten by cats—the parasite's definitive host. This behavioural manipulation is associated with cysts in the amygdala and alterations in dopamine signalling. *T. gondii* is not a specific parasite of humans, but we nonetheless often end up infected due to our association with both cats and rodents. In humans, the parasite produces dormant cysts in muscle or neural tissue that, for decades, were assumed to be asymptomatic. However, recent evidence suggests that infected individuals may exhibit changes in risk-taking behaviours, such as driving at excessive speed, suggesting that in humans, as in rodents, the parasite may be dampening fear responses.

Equally fascinating is evidence from mouse studies that the gut microbiome—the trillions of microbes residing in the digestive tract—has a significant influence on brain function via the

so-called gut-brain axis. Mice raised without exposure to microbes show heightened amygdala activity and elevated anxiety levels, which can be normalised following introduction of a normal microbiome. As yet it remains unclear whether similar effects may occur in humans, but since many bacterial species within the gut microbiome can either produce, or degrade, human neurotransmitter molecules such as gamma-amino butyric acid, it seems highly plausible that our personal fear responses may be modulated in a similar way – something worth thinking about next time you are deciding what to eat before heading to the cinema for a horror movie!

Further Reading

The amygdala and fear response: What Is The Amygdala: Function & Brain Location

Adrenaline and 'fight or flight' behaviours: Understanding the stress response - Harvard Health

The biology of panic disorders: Neurochemical and genetic factors in panic disorder: a systematic review | Translational Psychiatry

Behavioural manipulation by *Toxoplasma:* Toxoplasma gondii infection and behaviour – location, location, location? - PMC

The role of the microbiome in emotional responses: Gut feelings: associations of emotions and emotion regulation with the gut microbiome in women - PMC