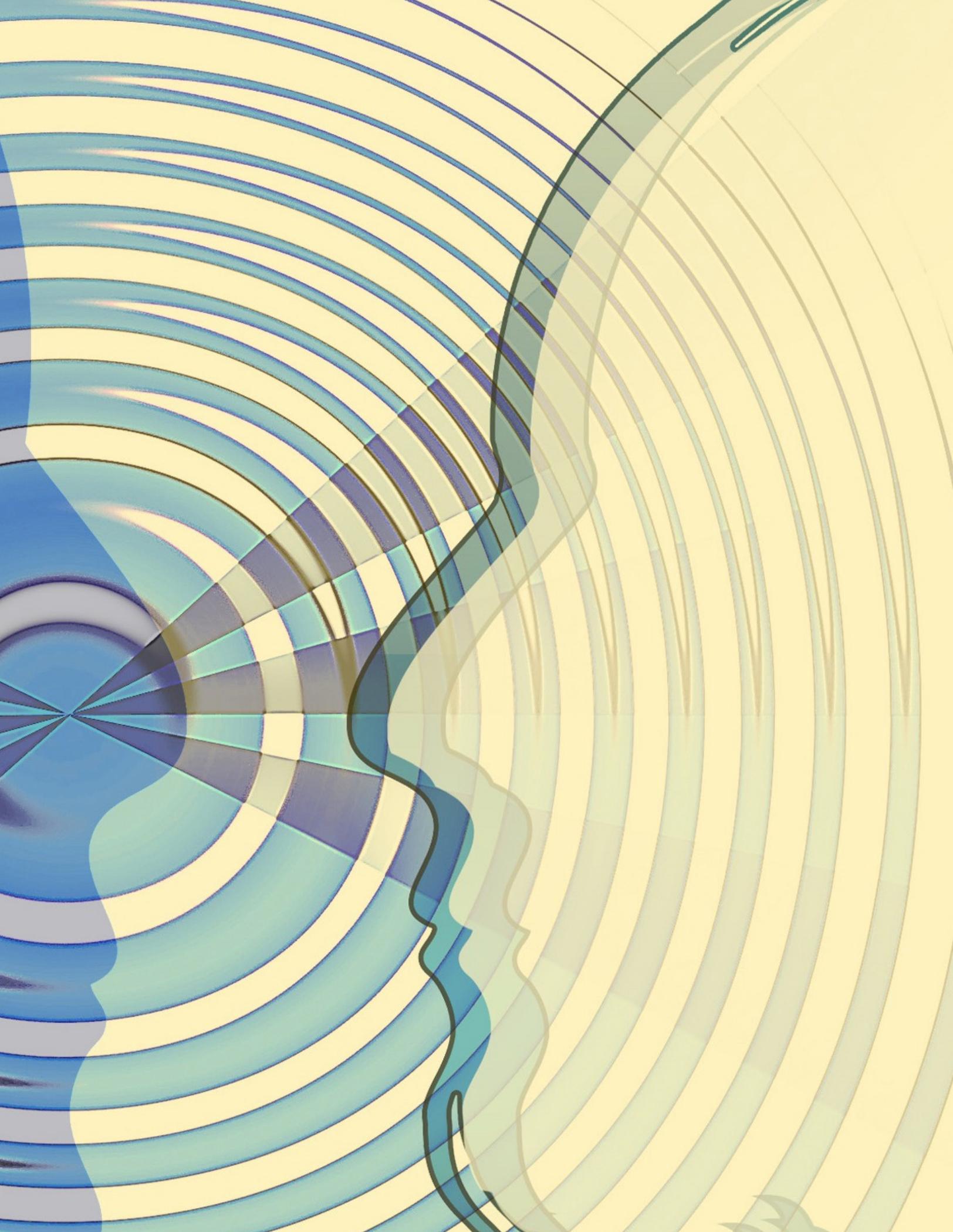




I Feel Your Pain, Do You Feel Mine?

By Lucy Friedberg



The experience of emotion and pain stem from our interactions with our environment, peers, and selves, capturing the essence of the complete human experience. Our ability to verbally communicate our “feelings” aids in fostering close bonds with each other. The action of feeling holds a double meaning. On one hand, you can physically touch something, experiencing a tactile sensation. While on the other hand, you can also inwardly ‘feel’ intangible sentiments like emotion, encapsulating our introspective experience. Without being able to communicate either physical or internal feelings, our relationships to each other would be completely different and unsustainable. Throughout time, our frontal lobes have evolved to reflect the developing complex social structures and higher cognitive functioning required to uphold it [1]. Expressing an emotion enacts both meanings of “feeling.” A physical stimulus triggers a cascading effect in the brain to produce the conscious thought that you feel “sad.” This sequence inherently suggests that there is an overlap in the emotion and pain signaling networks of our brain [2].

What is Emotion?

Movies, pictures, conversations, and almost every interaction we have with our environment elicit an “emotion.” Emotion is applicable to almost every individual. Our emotional schema develops throughout our entire lives, teaching us how to respond to certain situations [2,3]. In a technical sense, emotions are central brain states that create a cause-and-effect interaction with other brain structures stemming from stimuli and behaviors. These brain states arise from neurochemical interac-

tions such as the release of neurotransmitters. Neurotransmitters serve as chemical messengers between neurons, the brain’s foundation for transmitting sensory input from the external world. Electrical signals travel between neurons along the axon and trigger the release of the neurotransmitter. One commonly known neurotransmitter is dopamine, which plays a crucial role in the modulation of emotion [4,5].

When defining emotion, rather than thinking about neural connections or the release of neurotransmitters, people utilize adjectives such as happy, sad, or mad. Emotions are states of being produced by activation of certain areas in the brain such as the amygdala or thalamus. A common misconception is that one brain area corresponds to one emotion. Instead, the brain is a complex network with communications between different structures. These structures ultimately distribute signals to the rest of our body to produce behavior. The behavior or actions produced is what people think of when imagining emotion. The amygdala isn’t where “fear” is located, but rather, it is involved in the evaluation of emotional importance of a stimulus, and is especially active in response to fear [6].

Earlier theories state that the basic emotion and dimensional models are two separate measures of emotion. The discrete emotion perspective states that we have a propensity for certain emotions such as happiness, anger, and disgust [7]. These exist as categorical, basic, and specific emotions. The dimensional perspective suggests that emotions exist along a range of dimensions based on valence (positive vs. negative) and arousal (activated vs. deactivated) [7]. The dimensional spectrum of emotional experience is determined by where valence and arousal land

within the range. Building on the theory of an emotional spectrum, recent models of emotions state that emotions are learned from previous experiences [7]. This suggests that emotions are derived from the ability to recall and apply conceptual knowledge to a situation and produce the appropriate behavior (or emotion) [7]. These models are important to understand because they establish the basis of our emotional experience. They also begin to distinguish between states of being, and feeling, and provide theories for their origin.

Where do we Feel Emotion?

Attempting to decipher emotions has been a prevalent study across philosophical and scientific disciplines throughout all of history. Since the late 19th century, there have been several different theories as to how the brain produces emotional states that ultimately influenced the modern idea of the limbic system [8]. Dr. William James and Dr. Carl Lange answered the question of “After seeing a bear in the woods, does our heart race because we are afraid? Or are we afraid because our heart is racing?” by stating that we are afraid due to the physiological act of our hearts racing [8]. The James-Lange Feedback Theory requires a prior set of changes in the body to produce emotion [8]. What these changes are determine the following experience of emotion [2,8].

In the 1920s, Dr. Walter B. Cannon and Phillip Bard disagreed with James-Lange. They claimed that because humans experience such a wide variety of specific emotions, physiological responses for a single emotion are indistinguishable [8]. For example,

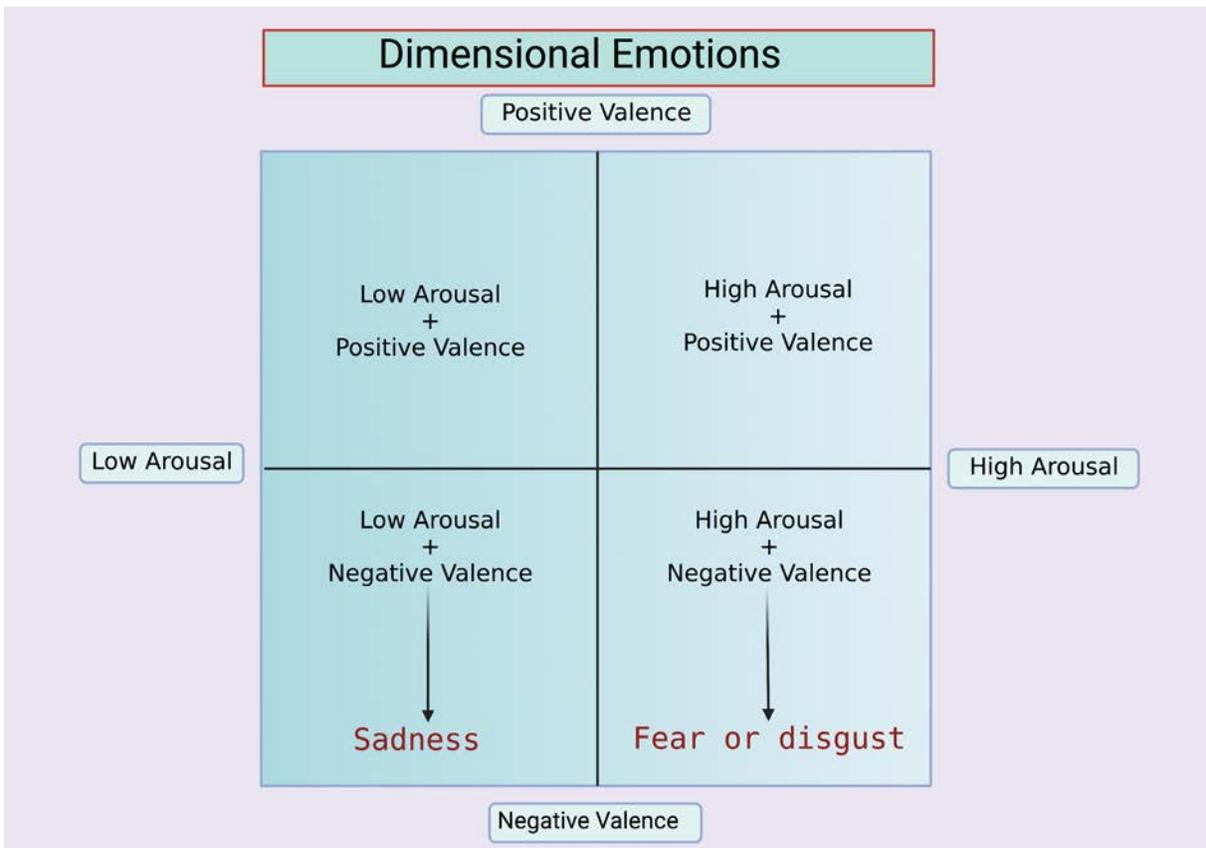


Image created by Lucy Friedberg with BioRender.com

a multitude of scenarios can cause a person's skin to flush, such as sexual arousal, anger, and embarrassment. In addition, Cannon and Bard pointed to the fact that you can encounter emotion prior to any associated physiological change [8]. Instead, Cannon and Bard suggested that in the brain, the thalamus directs information to the cortex for the generation of feelings (the emotional experience), and to the hypothalamus for the generation of a bodily response (expression of behavior resulting from the emotional experience) [2].

A decade later, Dr. James Papez proposed the Papez Circuit. This circuit defined emotional processing as occurring in the medial walls of the forebrain. Specifically, the structures directly connected to the hypothalamus [2,31]. Building on this theory, Dr. Paul Maclean's developed the concept of the Limbic System. From 1940-

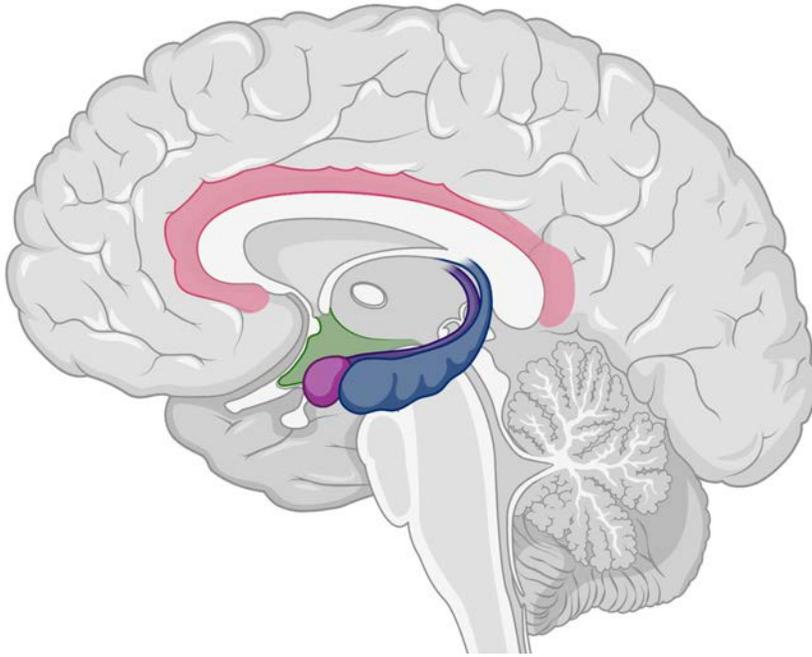
1970, the control of emotional behavior was thought to require recognition of a significant stimulus triggering subsequent activation of a specific emotional response [9]. Maclean believed it was the hippocampus that identified significant stimuli [2]. After 1970, the 'modern' limbic system was established [9]. Here, the amygdala is cited as the key proponent of the limbic system. The amygdala determines what a stimulus is, and whether something needs to be done about it. While not exclusive to fear processing, the amygdala plays a significant role in influencing approach or avoidance behavior in response to a frightening or threatening stimulus [9].

Another part directly linked to the limbic system is the insula [34]. The insula filters all bodily information and integrates it to form a representation of the state of our body to our consciousness [34]. Alongside the insula during the active experience of feelings is the

anterior cingulate cortex (ACC). These two brain areas function to project the experience of maternal and romantic love, anger, fear, sadness, happiness, disgust, and trust [1,2,21].

Why do we Experience Physical Pain?

Have you ever placed your hand on a hot surface and swiftly pulled it back? Evolutionarily, being able to perceive pain is critical to human survival. Fight or flight is a common adaptation that is referred to when understanding instinct and fitness. It is what tells us to remove our hand from a hot surface or to defend our territory. Being able to distinguish these signals is the job of the central nervous system and pain receptors called nociceptors. Nociception is the process of detecting, experi-



Highlight of the limbic system. Image created by Lucy Friedberg BioRender.com

encing and encoding harmful or very unpleasant stimuli [11]. The three types of stimuli nociceptors are activated by are temperature (thermal), mechanical (stretch/strain) and chemical (inflammatory) [11].

What Hurts?

Physical pain is typically described as a part of the body feeling discomfort. This can range from slight, to extreme discomfort. The direct pain matrix is made up of the cingulate, insular, and certain somatosensory areas. The anterior cingulate cortex, parts of the prefrontal cortex, and insula have rich nociceptive and motor receptor regions [32]. The interconnected nature of these brain areas allow output of ACC pain to influence immediate behavioral reactions [32]. So, if you put your hand on something hot, the nociceptors signal that there is nociceptive pain, or acute tissue pain, signaling the brain to remove your hand from the hot surface to prevent further injury. The involvement of the secondary somatosensory cortex and posterior insula

processes are crucial to experiencing physical pain [6,11]. The ability to process tactile stimuli is essential in understanding our relationship to the physical world.

Don't Get Your Wires Crossed

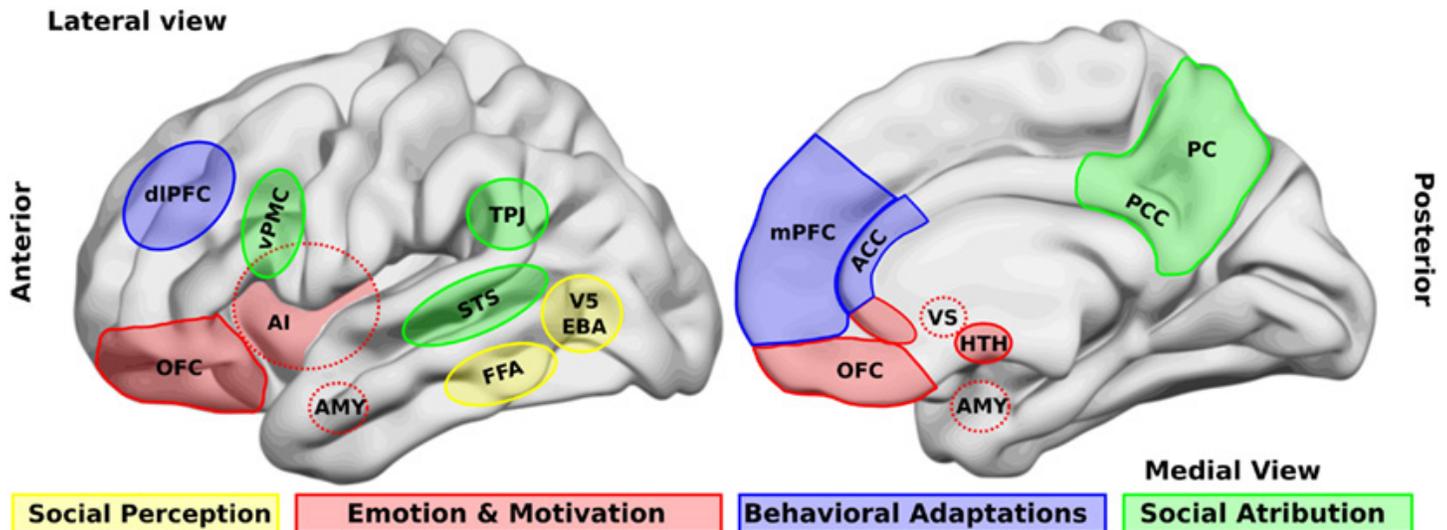
When people say as a figure of speech, "love hurts" or you have your "feelings hurt", they actually mean it. After examining both emotional and pain processing, recent research supports the idea that the two networks are not separate [13]. While everyone experiences emotional pain, it can be harder to articulate since it seems more abstract than physical pain. With physical pain, you can usually point to a spot on the body and where and why it hurts. With emotional pain, if you say your body aches because you're sad, people may sympathize, but do not understand the extent that your physiological state changes in response to being sad.

If you have ever grabbed your knee when seeing a basketball player fall hard on their

own knee, you are not alone. The anterior cingulate cortex (ACC), parts of the prefrontal cortex, and insula are regions of the brain that signal physical pain, but they are also responsible for behaviors stemming from our emotions as well [13]. Part of the foundation of this research is the finding that the anterior insula (AI_n) is activated not only when experiencing displeasing stimuli that activate nociceptors, but also when viewing someone else experiencing a displeasing stimuli (or even making a displeased face) [13,26]. The cingulate and insular regions are activated when viewing someone in pain, supporting the evidence of emotional pain activating the same brain regions as physical pain.

Particularly, researchers have been able to link the nociception areas and neural recruitment to insular and cingulate regions, supporting the theory of the same regions for two types of pain [10,12,13,26].

Most people relate to the common physical pain of scraping your knee or stubbing your toe. Remembering your own painful experience may elicit a twinge of pain. Empathy is the ability to share and understand another's feelings. Being empathetic, for example, requires recalling how you felt at a time when you scraped your knee and relating to a person in an appropriate emotional way to comfort them. One study linked the common painkiller Acetaminophen (Tylenol) to a reduction in empathy [14]. As mentioned earlier, the ACC is activated when an individual is experiencing distress or when viewing someone else in distress. The reduction of ability to feel pain and consequently the ability to empathize indicates a certain degree of overlap between areas in the brain that express physical and emotional pain. Tylenol reduced both personal distress when experiencing social pain



Billeke P and Aboitiz F (2013) Social cognition in schizophrenia: from social stimuli processing to social engagement. *Front. Psychiatry* 4:4. doi: 10.3389/fpsy.2013.00004 <http://journal.frontiersin.org/article/10.3389/fpsy.2013.00004/full>

(exclusion), but also when viewing others in distress [14].

Emotional Pain

Being empathetic is crucial to our social relationships because it allows us to connect to, and understand what other people are expressing on a deeper level. It helps us understand both the negative and positive affects [15]. Negative affects include emotional or psychological pain. Psychological pain is defined as an unpleasant feeling resulting from a negative experience that is caused by an inability or deficiency of the self [12,16]. This feeling is persistent, but unsustainable. The need to love, to be loved, and connect with others are core psychological needs that if not met can lead to feeling unhappy or dissatisfied [12,16]. Across several experiments assessing psychological pain, the ACC proved to be the most active [10,12]. This extends the ACCs involvement to the experience of sadness, while the insular functions to a greater degree for both negative affect and physical pain [10,12].

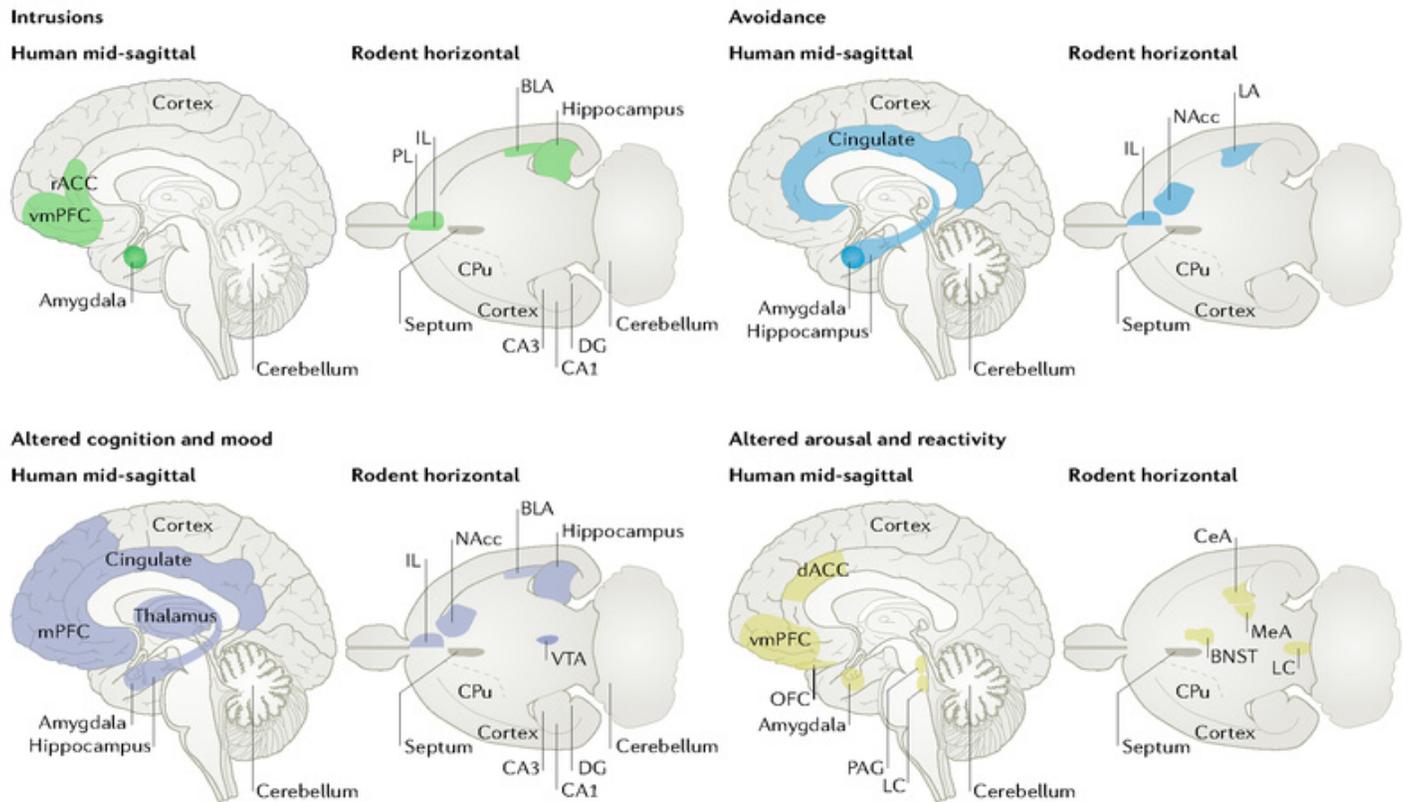
Examining Our Social Selves

Being able to perceive social exclusion historically meant a higher chance of survival since in early times, those in groups were more likely to live longer [17]. Feeling emotional and physical pain similarly could be a function of evolution, given that an emotional or social threat (being alone) could result in a physical one (dying). When examining our social selves, the development of self-esteem is frequently fixated on, especially in adolescent aged individuals. Self-esteem is feeling good or bad about oneself which subsequently affects our emotional state. It is an internal gauge of social inclusion or exclusion in relation to others [17,18]

A theory developed by Leary et al. (1995) draws parallels between social happiness and self-esteem. Sociometer theory indicates that self-esteem fluctuates, and is influenced by social experiences. Those who are socially rejected or deemed an outcast have lower self-esteem than those who perceive themselves as included or

well-liked. This theory involves our "self" in a social context, providing a psychological framework for our outward behaviors. In a study done on social exclusion, researchers found that regardless of whether a person knew the other people, they exhibited distress at being excluded from a game [27]. Since a sense of belonging is at the forefront of satisfying our penchant for inclusion, studying social exclusion in the context of pain provides an objective perspective.

Theories such as sociometer theory and attachment style provide social implications for the connectivity of our brain structures. In a study done looking at which brain areas are active during social exclusion, researchers found the ACC to be more active during exclusion than inclusion. This activation parallels studies done looking at physical pain [27]. Understanding attachment is important in the context of social pain because it provides an emotional structure that forms a foundation for baseline social interactions. For example, it cues sensitivity to exclusion which may correlate to the activity or reactivity of the ACC [10,12,13,22].



Fenster, R. J., Lebois, L., Ressler, K. J., & Suh, J. (2018). Brain circuit dysfunction in post-traumatic stress disorder: from mouse to man. *Nature reviews. Neuroscience*, 19(9), 535–551. <https://doi.org/10.1038/s41583-018-0039-7>

Brain areas such as the insular regions, medial prefrontal cortex, and anterior cingulate cortex that have been identified as being activated by emotional pain such as social exclusion. In the context of empathy and experiencing pain, their activation provides support that these areas are also activated by physical pain [12,13,21,26]. These studies have greater implications for biopsychological interventions and general unification of understanding individual experiences. It also deepens our understanding of empathy, which is fundamental to our social relationships and subsequently, well-being [15,27,28].

Autism Spectrum Disorder

Autism spectrum disorder (ASD) presents several emotional and sensory processing variances

including both hypo and hypersensitivity to certain stimuli. Studying the emotional and physical pain processing in participants with ASD presents the opportunity to examine divergent neurological functions. This would allow for the development of a more comprehensive understanding of the individual experience of pain [15]. Difficulty with perception and processing the emotions of others is commonly observed in ASD. However, whether this difficulty arises from a general struggle to relate to emotional states, or from struggling to perceive affective expressions is not fully understood. A recent study used pain evaluation in the self and others to link emotional and sensory perception in individuals with ASD. The goal was to distinguish whether these individuals have difficulty recognizing other emotional states due to discrepancies in relating to emotional experience (own or other) or if it is

specific to the perception of others [15,19]. They found that participants with ASD demonstrated no difference in evaluating intensity of their own pain to participants without ASD. However, they also found that participants with ASD exhibited more difficulty estimating the pain intensity of others, specifically when evaluating pain level based on facial expressions. These results contrast earlier findings of neurotypical brains showing activation in insular and ACC regions when viewing photographs of people in pain. To support this, the researchers recorded a difference in neural activation between ASD participants identifying pain based on context rather than bodily expressions. Those with ASD had reduced neural activation in response to higher social complexity [19].

Not only did individuals with ASD demonstrate decreased neural activation in response to

pain identification of others (reduced empathic ability), they also had weaker brain activation in the anterior insula and anterior cingulate cortex. This provides strong evidence for the role of insular and cingulate cortices in pain-related empathy, or the overlapping of emotional and physical pain processing [12,20,21,26]. Through outlining how both neurodivergent and neurotypical individuals experience pain, interpret social stimuli, and process affect, a clear relationship between the signaling of physical and emotional pain develops.

PTSD/Chronic Pain

Being able to understand emotional processing has great implications for fields studying post-traumatic stress disorder (PTSD) and chronic pain. Both PTSD and chronic pain emanate from a biological, psychological, and social field. Chronic pain and PTSD are extremely prevalent across the US and affect people's day to day lives. An incongruence between emotional processing and the experience of physical pain can lead to chronic pain or even PTSD. Chronic pain is the presence or sensation of physical pain in the absence of a clear source of pain over an extended period [33].

People experiencing PTSD often display intense negative emotional reactions, higher reactivity to certain stimuli that may trigger the traumatic experience, avoidance of that stimuli, and chronic overstimulation [24]. Generally, PTSD is classified as an anxiety disorder, and chronic pain is often comorbid with PTSD. Using brain imaging, researchers have been able to establish a relationship between PTSD and increased blood flow in the insular region [23,24]. There is a distinction in pain perception between people

with PTSD and those who experience isolated trauma. Those who do not display PTSD symptoms following a traumatic event tend to have higher pain perception than those with PTSD. Mickleborough et al. (2014), associated this with the changes to signaling between the cortical and subcortical structures in the two different groups.

A common self-preservation response to trauma is dissociation. This entails both psychological and physiological responses. People often report feeling "emotionally numb" to most stimuli. Research shows that there are neurological changes that occur that can account for someone dissociating. In the same study done by Mickleborough et al. (2014), they associated induced dissociative states with more activation in the insula, and a simultaneous reduced sensitivity to pain. The occurrence of dissociation can account for the increased pain threshold in those experiencing PTSD compared to those without PTSD symptoms

[23,24].

At rest, the brain demonstrates spatially and temporally distributed or synchronous blood-oxygen level-dependent (BOLD) variation [25]. In the brain of chronic pain patients, there is constant processing of "background" pain signaling. Spontaneous pain stemming from the background pain conflicts with both conscious and subconscious processes [25]. This occurrence contributes to fundamental neuronal plasticity in response to chronic pain. The existence of background pain is attributed to episodic memory resulting in a constant recall of the original pain or injury. The persistent recall resulting in the background pain is evident by examining structural changes to certain pain areas in the brain such as insula, ACC, and Thalamus regions [1,25]. PTSD emotional responses and correlating them to structural changes in the brain draws a stronger connection between the experience and process-



ing of emotional and physical pain.

Chronic perceived pain conditions are often correlated with response activation in the thalamus. Thalamic activation is connected to the time since onset of the chronic pain, with differing responses short-term to long-term [29]. Hyper-perfusion occurs in short-term pain, and hypoperfusion in long-term chronic pain, demonstrating adaptive changes during the development of chronic pain [29]. Operant theory of chronic pain is the idea that there are two distinct aspects of pain, sensory and behavioral. Sensory aspects of pain are essentially unobservable while behavioral aspects are observable and measurable. These are categorized under pain behaviors which are subject to the influences of learning and experience. Under this theory, pain

can be reinforced via negative and positive reinforcement causing the occurrence of pain to linger [11,29].

Similar to PTSD symptoms, chronic pain creates neurological changes in the brain that demonstrate and support the interconnected nature between emotional and pain processing [29,30]. Several studies have shown a link between chronic pain and mood disorders such as depression and anxiety leading researchers to claim that psychosocial factors affect pain perception [29]. This model is referred to as the biopsychosocial model of pain. These altered states of somatic awareness and pain catastrophizing indicate changes to the peripheral or central nervous system that are crucial to signaling pain. This model has been extremely influential in

shaping new behavioral, psychosocial, and physical interventions to pain management. It allows for researchers and doctors to gain a fuller image of individual differences in the experience of pain. It also highlights the multidimensionality to pain processing, both emotional and physical [29,30].

Understanding why humans feel emotions and pain separately allows for a deeper understanding of how the two sensations are related. People often assume physical pain is the only relevant pain to our survival, but research consistently demonstrates that our social needs and subsequent social pain if these needs are not met can be as painful. Comparative research establishes the brain as an active, interconnected entity with a strong network to produce behavior.







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