The Role of Different Sensory Modalities in Stress Contagion and Its Neural Mechanisms

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Abstract

Stress contagion refers to the phenomenon where an individual, when observing or coming into contact with another individual in an acute stress state, is unconsciously influenced by the other's negative emotions and matches their own physiological and psychological state to that of the other party. Experimental paradigms for stress contagion are divided into two types: vicarious stress and cross stress. In the vicarious stress paradigm, observers receive stress information transmitted from demonstrators through single or multiple sensory modalities. In cross stress, observers directly interact with demonstrators after the demonstrators have experienced stress, receiving stress information transmitted through multiple sensory modalities. Behavioral responses triggered by different sensory information in stress contagion share similarities, all accompanied by reduced spontaneous activity, increased anxiety behaviors, and elevated cortisol levels, although the underlying neural circuits and key brain regions are not entirely consistent. Compared with single sensory modalities (visual, auditory, and olfactory), stress contagion effects induced by multiple sensory modalities are stronger. The amygdala is a hotspot brain region for stress contagion, with significant activation observed across different experimental paradigms. Future research needs to identify brain regions of interest based on the neural mechanisms through which different sensory modalities influence stress contagion, while emphasizing the importance of experimental paradigms for stress contagion.

Full Text

Preamble

The Role of Different Sensory Channels in Stress Contagion and Its Neural Mechanisms

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Abstract: Stress contagion refers to the phenomenon where an individual unconsciously absorbs the negative emotional state of another person in acute stress through observation or direct contact, matching their own physiological and psychological state to that of the stressed individual. Experimental paradigms for studying stress contagion fall into two categories: vicarious stress and stress crossover. In vicarious stress paradigms, observers receive stress information transmitted through one or multiple sensory channels from a demonstrator. In stress crossover paradigms, observers come into direct contact with the demonstrator after the latter has experienced stress, receiving stress information through multiple sensory channels. While the behavioral responses triggered by different sensory modalities show similarities—such as reduced autonomous activity, increased anxiety-like behavior, and elevated cortisol levels—the underlying neural circuits and key brain regions are not entirely consistent across modalities. Compared to single sensory channels (visual, auditory, or olfactory), stress contagion effects are stronger when multiple sensory channels are involved. The amygdala has emerged as a hotspot brain region in stress contagion, showing significant activation across various experimental paradigms. Future research should pay careful attention to experimental paradigms and identify brain regions of interest based on the neural mechanisms through which different sensory channels influence stress contagion.

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Stress is typically defined as a series of cognitive, physiological, and behavioral changes that occur when an individual confronts threatening events (Habib et al., 2001). In social life, people frequently encounter stress from economic, interpersonal, and psychological sources, making the management of stress responses a critical issue for individual survival and social development (Pfeifer et al., 2021). Stress contagion—also referred to as stress transmission, vicarious stress, or social transfer of fear—describes the process by which individuals unconsciously absorb the negative emotions of others in acute stress through observation or contact, matching their own physiological and psychological state to that of the stressed individual and experiencing the other's stress (Engert & Grant, 2019). This phenomenon holds important adaptive value for environmental survival and shows cross-species consistency (Perez-Manrique & Gomila,

2022). Park et al. (2021) proposed that observing others experience stress promotes the formation of psychological models in observers, enabling adaptive responses when encountering similar stimuli in the future. This perspective aligns with simulation theory, which posits that understanding others' behaviors and intentions requires mental simulation of observed actions (Gallese & Goldman, 1998). However, stress contagion can also trigger stress-related disorders such as pain sensitivity, depression, and anxiety (Carnevali et al., 2017; Ueda & Neyama, 2017). Therefore, understanding the behavioral responses and neural mechanisms underlying stress contagion has significant implications for modern life.

Current research on stress contagion overlaps with studies on empathy and emotional contagion, yet stress contagion is not equivalent to these concepts. Research on empathy and emotional contagion encompasses both positive and negative emotions (Panksepp & Lahvis, 2011), with negative emotions including various non-stress states such as anger and disgust (Hess & Blairy, 2001; Vermeulen & Mermillod, 2010). In contrast, stress contagion specifically refers to an individual perceiving another's stress state and consequently triggering corresponding physiological responses. This review focuses exclusively on studies that induce stress states in observers.

Stress contagion is influenced by multiple factors, including prior experience, familiarity, and the sensory channels through which stress signals are received. Studies on acute stress in rodents have found that mice previously exposed to electric shocks exhibit stronger stress responses when witnessing conspecifics receiving shocks compared to naïve mice (Carrillo et al., 2015). Stress contagion occurs more readily between individuals with close social relationships. Compared to individually housed rats, pair-housed rats show increased freezing behavior after observing companions undergo drug or social stimulation (Langford et al., 2006; Lidhar et al., 2017). Recent research indicates that sensory channels constitute an important factor affecting stress contagion (Perez-Manrique & Gomila, 2022), as different sensory modalities transmit social information through distinct pathways (Sliwa et al., 2022).

Stress contagion typically occurs within the same species, though in some cases, presenting only odor signals (Kiyokawa et al., 2009), auditory signals (Bussey et al., 2007), or visual signals (Nakashima et al., 2015) from a stressed companion is sufficient to trigger the phenomenon. While animals readily capture visually transmitted information, this modality is limited by object occlusion and restricted sightlines. Olfactory receptors can detect low-concentration chemical molecules lingering in the air, and social information molecules persist over time (Pause, 2012). Moreover, both humans and animals use vocalizations to convey emotional states (Concina et al., 2019). Importantly, interactions between different sensory modalities facilitate social information processing (Damon et al., 2021). For instance, in human studies, simultaneously presenting olfactory information with semantically consistent visual information enhances odor recognition (Gottfried & Dolan, 2003), fear-related olfactory cues acceler-

ate the categorization of emotional faces (Kamiloglu et al., 2018), and auditory cues facilitate the recognition of emotional faces (Vesker et al., 2018). These findings are consistent with animal research (Hernandez-Lallement et al., 2022).

This review categorizes experimental paradigms of stress contagion and synthesizes research on how visual, auditory, olfactory, and multisensory channels influence stress contagion, exploring the neural mechanisms underlying different sensory modalities. The aim is to clarify the mediating effects of sensory channels on stress contagion and further understand how different sensory signals trigger this phenomenon.

2 Experimental Paradigms for Stress Contagion

In stress contagion research, subjects are tested in pairs. The individual experiencing the stress event is termed the "demonstrator," while the individual observing the demonstrator's stress experience is called the "observer." When observers witness demonstrators undergoing stress events (e.g., electric shock, pain, social defeat), their stress systems activate, producing physiological and emotional responses similar to those elicited by direct stress exposure (White & Buchanan, 2016). Based on whether observers directly witness the demonstrator's stress experience or interact with the demonstrator after the stressful event, stress contagion paradigms can be divided into two types: vicarious stress and stress crossover (Carnevali et al., 2020; Peen et al., 2021). Both paradigms are used in human and animal studies, though animal models offer greater experimental control and visualization, making them ideal for illustrating these approaches.

The witness stress paradigm is a classic vicarious stress model in which demonstrators and observers are separated within the same cage by a transparent partition, allowing demonstrators to transmit information to observers (Warren et al., 2020). In the classic witness stress paradigm, a perforated transparent barrier permits the transmission of visual, auditory, and olfactory information [Figure 1: see original paper]. To investigate the role of single sensory channels, researchers present observers with isolated sensory information (visual, auditory, or olfactory) or selectively block specific modalities. In visual channel studies, researchers use black opaque partitions to block visual information transmission, demonstrating visual information's crucial role in stress contagion [Figure 1b: see original paper] (Jeon et al., 2010). Langford et al. (2006) confirmed the modulatory role of sensory channels by individually disrupting visual, auditory, or olfactory function. In olfactory and auditory channel studies, researchers present observers with only the demonstrator's vocalizations or pheromones (olfactory information) to directly examine these modalities' contributions [FIGURE:1c, d] (Chen et al., 2009; Lee et al., 2021).

The stress crossover paradigm can also trigger stress contagion. Demonstrators and observers are placed in separate, non-interfering cages, preventing observers from directly receiving information during the demonstrator's stress exposure.

After the stress task concludes, experimenters co-house the observer with the demonstrator [Figure 1e: see original paper], allowing the observer to experience stress contagion through direct contact (Carnevali et al., 2017). This paradigm enables investigation of multisensory channel effects on stress contagion. Both paradigms assess stress contagion effects through indicators such as observer corticosterone levels, freezing behavior, and anxiety- or depression-like behaviors.

3.1 Behavioral and Physiological Changes Induced by Visual Information in Stress Contagion

Animal studies demonstrate that blocking visual information reduces or even eliminates stress contagion effects. Researchers use black opaque partitions to prevent visual information transmission from demonstrators to observers [Figure 1b: see original paper]. Compared to transparent partitions, opaque barriers significantly reduce observers' freezing behavior when witnessing demonstrators receive electric shocks (Ueno et al., 2020) and decrease social avoidance behavior after observing demonstrators experience social defeat (Iniguez et al., 2018). Guzman et al. (2009) found that blocking visual information prevented activation of observers' stress systems during demonstrators' shock exposure. Interestingly, when demonstrators exhibit resistance to social stress, observers exposed to the same stressor also show stress resistance, but only when visual information is available (Iniguez et al., 2018). To establish visual information's unique contribution, researchers systematically removed visual, auditory, and olfactory cues, finding that only visual blockage significantly reduced stress levels (Langford et al., 2006).

Human studies similarly demonstrate that visual information can trigger stress contagion. Researchers commonly use heart rate variability and cortisol levels from the HPA axis as acute stress indicators (von Dawans et al., 2021). As bystanders, others' suffering can evoke personal stress experiences, including elevated cortisol and increased heart rate (Davis, 1980; Eisenberg, 2000; Engert et al., 2014). Researchers have successfully induced stress responses in observers by presenting videos (2-second silent clips) or images of demonstrators' pain expressions (Benuzzi et al., 2018; Botvinick et al., 2005; Lamm et al., 2007; Lamm et al., 2010; Olsson & Phelps, 2007). One study showed that merely watching training videos where demonstrators formed associations between neutral and stressful stimuli was sufficient for observers to develop aversive responses to neutral stimuli (Pärnamets et al., 2020). Compared to direct stress exposure, stress stimuli observed after watching others experience stress show generalization effects (Dou et al., 2023). Additionally, researchers have asked subjects to observe demonstrators undergoing social or physiological stress through glass partitions or computer screens. Results indicate that the relationship between observer and demonstrator significantly influences stress contagion: approximately 40% of observers showed stress responses when watching intimate partners undergo social stress (Engert et al., 2014), whereas only 17% exhibited contagion when

observing strangers (Erkens et al., 2019). Stress modality may also be an important factor. In physiological stress studies, observers' subjective pain ratings increased when viewing images of demonstrators in pain, though the proportion of observers showing stress responses was not reported (Saarela et al., 2006). Consequently, direct comparisons between physiological and social stress paradigms in visual stress contagion remain difficult. Previous research demonstrates that close relationships increase the proportion of observers showing visual stress contagion, but the relationship between social and physiological stress effects remains unclear.

3.2 Brain Regions Involved in Visual Information-Induced Stress Contagion

According to the "dual-pathway model," visual emotional information can be transmitted to the anterior affective system (including the amygdala, temporal pole, and orbitofrontal cortex) before reaching the occipital cortex, enabling top-down modulation of visual attention (Rudrauf et al., 2008). The anterior affective system then projects visual emotional information to the anterior cingulate cortex (ACC) and insular cortex (IC) for further cognitive processing and regulation (Markovic et al., 2014; Rabinak et al., 2011; Seo et al., 2014).

Animal studies indicate that the ACC is involved in pain affect, emotional behavior, and pain sensation (Xiao et al., 2019), while the IC is associated with observing pain (Zhang et al., 2022). Jeon et al. (2010) inactivated observers' ACC by injecting lidocaine (a local anesthetic and antiarrhythmic agent), finding that treated mice failed to show physiological or behavioral responses matching their demonstrators. Using Cacna1c conditional knockout mice with impaired Cav1.2 Ca2+ channels, which disrupt synaptic transmission and neuronal excitability in the ACC, they found these observers were resistant to stress contagion. To verify the ACC's specificity, Jeon inhibited the ventral posterolateral and posteromedial thalamic nuclei and lateral amygdala—regions known to be important for fear learning (Keum & Shin, 2019)—but found that disrupting these areas did not produce similar effects. Furthermore, the ACC can regulate negative emotion transmission through the basolateral amygdala (BLA). Inhibiting the ACC-BLA circuit alters real-time amygdala representations of aversive cues, and selectively suppressing ACC-to-BLA projections impairs stress contagion (Hernandez-Lallement et al., 2022). Additionally, glutamatergic projections from the IC to the BLA are essential for observational pain formation. Selective activation or inhibition of the IC-BLA circuit respectively enhances or reduces observational pain intensity in mice, revealing this neural circuit's role in modulating observational pain (Zhang et al., 2022).

Human neuroimaging studies show that ACC neuronal activity changes when observing others' fear or fearful facial expressions (Fallon et al., 2020). Functional magnetic resonance imaging (fMRI) studies reveal significant ACC activation when observers watch their partners experience painful stress (Singer et al., 2004). Compared to viewing neutral expression videos, painful expres-

sion videos elicit greater activation in the IC and ACC (Benuzzi et al., 2018). Similarly, viewing static images of potentially painful situations (e.g., a hand being cut, a cheek being punctured) activates the ACC and IC more than neutral images (Jackson et al., 2005; Akitsuki & Decety, 2009; Cao et al., 2019; Christov-Moore et al., 2019; Lassalle et al., 2019). Saarela et al. (2006) further found that when subjects viewed demonstrators' pain expressions, the ACC encoded not only the observers' stress emotions but also the intensity of expressed pain. A meta-analysis of 32 human fMRI studies confirmed the important roles of ACC and IC in pain-induced stress contagion (Lamm et al., 2011). However, numerous studies have found that viewing images or videos of oneself under stress also activates the ACC and IC, sometimes even more strongly (Benuzzi et al., 2018; Singer et al., 2004; Zaki et al., 2016). These results suggest that the ACC may not be specific to visually induced stress contagion but rather encodes and responds to all pain-related stimuli.

In summary, animal and human studies demonstrate that visual channel information can trigger stress contagion effects, with visual emotional information from the external environment transmitted through the ACC to the BLA to regulate stress contagion processes. Current evidence remains limited, and whether other brain regions and neural circuits participate in visual stress contagion requires further investigation.

4.1 Auditory Information-Induced Stress Contagion

Sound effectively transmits emotional information, with both humans and animals expressing different emotions through vocal intensity, frequency, and pitch (Laukka et al., 2008; Swain et al., 2018). Animal studies reveal that rodents emit ultrasonic vocalizations at different frequencies to express positive and negative emotions, with 22 kHz ultrasonic calls signaling stress and fear (Wöhr, 2018). These vocalizations are produced specifically to warn conspecifics of danger (Wöhr & Schwarting, 2008).

In animal models, researchers investigate auditory information's role in stress contagion by recording demonstrators' vocalizations during stress and playing these recordings to observers [Figure 1c: see original paper]. In electric shock-induced stress experiments, observers exhibited typical stress responses simply by hearing demonstrators' 22 kHz calls (Chen et al., 2009), with this auditory contagion modulated by familiarity between observer and demonstrator (Kim et al., 2010). When researchers compared recordings of demonstrators' stress vocalizations with artificially generated sounds of similar frequency, observers showed specific stress responses only to authentic calls, demonstrating their ability to discriminate between real and artificial stress signals (Ouda et al., 2016). Beyond 22 kHz calls, rodents also perceive the cessation of activity sounds as danger information and the resumption of activity sounds as safety cues. When demonstrators exhibit freezing behavior after shock exposure, the resulting silence effectively triggers observers' freezing responses, producing stress contagion. Playing pre-recorded rat activity sounds eliminates observers'

stress responses (Pereira et al., 2012). However, studies using chemical stressors found that auditory impairment alone was insufficient to block stress contagion (Langford et al., 2006), suggesting that auditory information is not a necessary condition for stress contagion.

Auditory information is similarly important for humans, who transmit emotional information through changes in vocal intensity. Individuals show increased vocal intensity after experiencing stressful events (Feldman, 2007). Research indicates that anxious mothers exhibit elevated vocal intensity during stress, which increases infants' arousal levels, representing a transmission of maternal stress to infants (Smith et al., 2021).

4.2 Brain Regions Involved in Auditory Information-Induced Stress Contagion

During auditory information-induced stress contagion, the BLA and periaqueductal gray matter (PAG) play important roles. Previous research demonstrates strong connections between auditory cortex and both BLA and PAG (Ouda et al., 2016). Amygdala damage prevents freezing behavior induced by auditory information (Choi & Brown, 2003), while the BLA facilitates plasticity in auditory cortex encoding of negative vocalizations (Concina et al., 2019). The neural circuit from auditory cortex to PAG directly controls sound-driven defensive behaviors (Wang et al., 2019).

Demonstrators' 22 kHz calls effectively activate brain regions associated with negative emotions in observers, including the BLA, lateral amygdala, and hypothalamus, whereas 50 kHz calls that induce positive emotions activate frontal association cortex, nucleus accumbens, and parafascicular thalamus (Sadananda et al., 2008). Studies show that when observers are exposed to demonstrators' 22 kHz calls, their auditory cortex, PAG, and BLA become activated. Approximately 37% of neurons in the BLA and lateral amygdala respond to 22 kHz sounds, with increased firing frequency (Parsana et al., 2012). To precisely identify key brain regions for rats' auditory stress responses, researchers designed three conditions: observers receiving live demonstrator stress vocalizations, observers receiving recordings of these vocalizations, and observers receiving artificially synthesized 22 kHz sounds. Comparisons between the first two groups revealed no differences in activation patterns or levels across all brain regions, indicating rats cannot distinguish between live and recorded stress contagion vocalizations. However, comparisons between these groups and the artificial sound group showed distinct activation patterns and levels in auditory cortex, BLA, and PAG, demonstrating rats' ability to differentiate between artificial 22 kHz sounds and genuine stress-transmitting vocalizations. In summary, the BLA and PAG are critical brain regions involved in discriminating stress-related auditory cues.

5.1 Behavioral and Physiological Changes Induced by Olfactory Information in Stress Contagion

Rodents possess a highly developed olfactory system capable of detecting low-concentration pheromone molecules in the air from considerable distances, enabling early danger avoidance. In conspecific recognition, olfactory information is even more important than visual cues (Corridi et al., 1993). Transmitting stress-related information represents a primary function of the olfactory system (Stevenson, 2010), with stressed animals releasing alarm pheromones (APs) from their vibrissal pads and anal glands to warn or alert nearby conspecifics (Kiyokawa et al., 2018; Kiyokawa et al., 2013).

Presenting olfactory information alone can induce stress contagion effects. Researchers found that when male rats receive electric shocks, testosterone secreted from the vibrissal region elicits active behavioral responses, while anal gland secretions trigger autonomic nervous system responses (Kiyokawa et al., 2004). Swabs from the anal region of socially defeated demonstrators, when placed in observers' cages, cause elevated corticosterone levels and stress-related synaptic plasticity changes in observers, with contagion severity positively correlating with time spent sniffing the demonstrator's anal region (Lee et al., 2021) [Figure 1d: see original paper]. When mice or prairie voles are housed with stressed conspecifics, both species show enhanced pain sensitivity to chemical, thermal, and mechanical stimuli (Smith et al., 2017; Walcott et al., 2018). In these experiments, pain sensitivity was induced solely through olfactory cues, as observers exhibited increased pain sensitivity simply by contacting bedding used by demonstrators.

Impairing olfactory function reduces stress contagion effects. During observer-demonstrator interactions, observers can discriminate stress states through olfactory information, avoiding the odor of stressed rats when given a choice (Mackay-Sim & Laing, 1981). Interestingly, this pattern reverses when observers are co-housed with demonstrators, with observers spending more time sniffing the demonstrator's anal region and typically showing long-term depressive phenotypes. Chemically ablating observers' olfactory epithelium before social interaction with stressed demonstrators eliminates both avoidance and approach effects (Lee et al., 2021).

Human studies have collected tears from subjects watching fear-inducing films and presented them to neutral-state subjects for sniffing. Compared to control groups, experimental groups showed faster reaction times and stronger facial responses to negative faces (Kamiloglu et al., 2018), demonstrating that humans can also transmit emotional states through olfactory information.

5.2 Receptors for Detecting Stress Pheromones

The olfactory system comprises two pathways: the main olfactory system (MOS) and the vomeronasal/accessory olfactory system (Tirindelli et al., 2009), with

pheromones processed by both pathways. Research has identified three neuronal targets for social pheromones: Grueneberg ganglion (GG) cells, vomeronasal epithelial cells, and main olfactory epithelial cells, with GG cell activation proving necessary for pheromone-induced stress contagion (Brechbuhl et al., 2008). Located at the nasal tip, GG cells express multiple MS4A receptor types activated by 2,6-dimethylpyrazine (a mouse aversion-signaling pheromone) (Greer et al., 2016). Studies show that 2-tert-butyl-4,5-dihydrothiazole is an alarm pheromone that activates GG cells and triggers avoidance behavior in rodents (Brechbuhl et al., 2013). Predator odors, TMT (2,4,5-trimethylthiazoline) from fox feces, and 2-PT from ferret anal glands also activate GG cells, producing effects similar to APs. Thus, the sensory pathways for conspecific danger signals and predator threat information highly overlap (Brechbuhl et al., 2013). In addition to these compounds, 4-methylpentanal and hexanal are alarm pheromones secreted from the perianal region, with receptors distributed in both vomeronasal and main olfactory epithelia. When both pheromones are secreted simultaneously, they trigger defensive behaviors (Inagaki et al., 2014). APs and predator odors induce c-Fos expression in brain regions involved in aversive responses, including the BLA and paraventricular nucleus (PVN) (Kiyokawa et al., 2005). The amygdala encodes both positive and negative odors (Jin, 2015) but responds more strongly to negative odors (Gottfried, 2002). In summary, GG cells serve as primary receptors for stress pheromones in the olfactory system, while the BLA and PVN represent important brain regions in this process.

6.1 Behavioral and Physiological Changes Induced by Multisensory Information in Stress Contagion

While single sensory channel information can trigger stress contagion, real-world information is typically transmitted through multiple sensory channels. Investigating the role of multisensory channels in stress contagion is crucial for understanding this phenomenon in naturalistic contexts. Different sensory channels influence each other during social information transmission. For example, maternal olfactory cues affect infants' processing of facial expressions at both behavioral and neural levels, increasing observation time for familiar faces (Durand et al., 2020) and reducing neural responses to fearful faces (Jessen, 2020). In a human study presenting observers with videos of demonstrators undergoing social stress, the combination of visual and auditory signals successfully induced heart rate acceleration in observers (Dimitroff et al., 2017). In animal models, multisensory stress contagion can be studied using either the witness stress paradigm [Figure 1a: see original paper] or the stress crossover paradigm [Figure 1e: see original paper].

Previous research shows that multisensory information produces stronger and more stable stress contagion effects than unisensory information. The integration of information from different sensory channels enhances danger perception, leading to more robust stress contagion. A meta-analysis on emotional contagion found that olfactory or visual impairments reduce fear and pain contagion

in rats compared to multisensory conditions. While olfactory or visual cues alone can transmit negative emotional responses, they are weaker than multichannel information (Hernandez-Lallement et al., 2022). Warren et al. (2013) combined the witness stress paradigm with social defeat stress, separating observers from demonstrators with a perforated partition that allowed visual, olfactory, and auditory information transmission. After observing demonstrators undergo social defeat, observers were placed in a cage with an aggressive mouse separated by a transparent barrier. Results showed observers exhibited typical depression- and anxiety-like behaviors, weight loss, elevated plasma corticosterone, and social avoidance at both 24 hours and one month post-stress. Using an opaque, non-perforated partition to block olfactory and visual information (and reduce auditory transmission) successfully prevented stress contagion. In social defeat studies using multisensory channels, observers showed elevated heart rate and sympathetic activation after initial contact with demonstrators, along with social avoidance behavior (Carnevali et al., 2017). This indicates that in multisensory stress contagion, observers not only receive demonstrators' stress signals but can also identify the stressor. In Allsop et al.'s (2018) experiment, demonstrators underwent training to associate a neutral stimulus with stress, which observers witnessed through a transparent perforated partition. When demonstrators subsequently showed stress responses to the neutral stimulus, observers exhibited similar responses, demonstrating that the results of demonstrators' stress association learning can be transmitted to observers through multisensory channels.

6.2 Brain Regions Involved in Multisensory Information-Induced Stress Contagion

In multisensory stress contagion research, the PVN in the hypothalamus secretes corticotropin-releasing hormone (CRH) and oxytocin (OT), both crucial for stress contagion. CRH secretion represents the initial hormonal response to acute external stress, transmitting chemical signals to the pituitary to promote adrenocorticotropic hormone release, which subsequently stimulates corticosterone secretion (Tsigos & Chrousosb, 2002). OT participates not only in female lactation and maternal behavior but also in promoting social comforting behavior (Chun et al., 2022). Research shows that glutamatergic synaptic remodeling on CRH neurons in the demonstrator's PVN can be transmitted to observers, inducing metaplasticity of glutamate synapses on CRH neurons in the observer's PVN (Sterley et al., 2018). Meanwhile, activating OT neurons in the PVN enhances observers' stress responses (Pisansky et al., 2017). In multisensory studies using electric shock as a stressor, observers' PVN CRH neurons activated upon detecting demonstrators' alarm pheromones (Sterley et al., 2018).

Additionally, Allsop et al. (2018) used electric shock as an unconditioned stimulus and auditory stimulation as a conditioned stimulus. After repeated pairings, demonstrators showed stress responses to the auditory stimulus alone.

Observers witnessed this learning process through a transparent perforated partition and subsequently exhibited stress responses to the paired auditory stimulus. In this process, the neural circuit from BLA to ACC played a crucial role; inhibiting this circuit blocked stress contagion, with information flowing from ACC to BLA (Allsop et al., 2018). Knapska et al. (2006) found that when observers approached and sniffed demonstrators that had received electric shocks, the BLA was activated. A study using electric shock stress and direct observer-demonstrator contact found that both stress contagion and direct stress activated the lateral amygdala (Jones & Monfils, 2016). Another electric shock study showed that stress contagion induced synaptic changes in the observer's hippocampus, a neural alteration considered a precursor to depression (Lee et al., 2021). A stress crossover study using monogamous prairie voles found that when male demonstrators underwent restraint stress, female observers who contacted them showed lower anxiety behaviors and elevated PVN OT levels compared to isolated observers (Chun et al., 2022). This may reflect OT's role in increasing female prosocial behavior, triggering an alternative stress response—"friendliness." However, this interpretation remains controversial, as Pisansky et al. (2017) found opposite results: intranasal OT administration enhanced freezing responses during stress contagion. This enhancement resulted from OT projections to the ACC that increased cellular activity, with chronic administration downregulating amygdala OT receptor expression. The researchers also noted that intranasal OT specifically affected stress contagion without influencing direct fear responses (Pisansky et al., 2017). These inconsistent findings may stem from methodological differences: Pisansky et al. used male observers with unfamiliar demonstrators, whereas Chun et al. used female observers with their male partners. Previous research shows that sex is an important factor affecting stress responses (von Dawans et al., 2021), and familiarity significantly influences stress contagion effects (Hernandez-Lallement et al., 2022).

7 Common Neural Mechanisms of Stress Contagion Across Sensory Channels

Synthesizing previous research using different stressors and sensory channels reveals that whether through visual, auditory, or olfactory information (Benuzzi et al., 2018; Dou et al., 2023; Kiyokawa et al., 2005; Parsana et al., 2012; Sadananda et al., 2008), or through direct observer-demonstrator contact (Allsop et al., 2018; Jones & Monfils, 2016), the amygdala is consistently activated. In both rodents and humans, the amygdala is involved in observational fear learning and social cognition (Allsop et al., 2018; Olsson et al., 2007; Olsson & Phelps, 2007). Research shows that social threat signals can reach the amygdala through different sensory channels, highlighting its crucial role in stress contagion (Debiec & Sullivan, 2014; Knapska et al., 2006; Lidhar et al., 2017; Olsson et al., 2007).

Functionally, the amygdala can be subdivided into the central amygdala (CeA), cortical amygdala (CoA), and BLA (Knapska et al., 2007; Spampanato et al.,

2011). The BLA is considered a fundamental neural structure for encoding both negative and positive emotions, showing activation during social information transmission (Debiec & Sullivan, 2014; Knapska et al., 2006) and participating in behavioral and physiological stress response regulation (Bhatnagar et al., 2004). The CeA plays an important role in physiological responses to stressors and serves as an integration hub for different stimuli (Gilpin et al., 2015; Kong & Zweifel, 2021).

Previous research demonstrates that 20 kHz ultrasonic stimulation activates both the CeA and BLA during stress contagion (Beckett et al., 1997; Parsana et al., 2012; Sadananda et al., 2008). Visual stimulation activates the lateral amygdala during stress contagion (Allsop et al., 2018; Jeon et al., 2010), though no evidence currently shows CeA activation. In olfactory research using restraint stress, studies report amygdala activation without strictly distinguishing subregions (Chun et al., 2022). In studies permitting post-stress direct contact, demonstrators' CeA is activated (Knapska et al., 2006). Regardless of whether electric shock, pain, or social defeat paradigms are used, observers' amygdalae are activated (Finnell et al., 2018; Ouda et al., 2016; Smith et al., 2017). Social defeat stress activates observers' CeA (Finnell et al., 2018), while electric shock or pain stress activates both BLA and CeA (Knapska et al., 2006; Ouda et al., 2016; Smith et al., 2017). In conclusion, different stress contagion paradigms consistently activate the amygdala, though different paradigms may activate different amygdala subregions.

[Figure 2: see original paper] Key brain regions in stress contagion: BLA (basolateral amygdala); CeA (central amygdala); PVN (paraventricular nucleus); ACC (anterior cingulate cortex); IC (insular cortex); PAG (periaqueductal gray matter).

8 Summary and Future Directions

Visual, auditory, or olfactory information alone can trigger stress contagion effects, but multisensory channels produce stronger effects. Behavioral responses to different sensory information show similarities, typically involving reduced exploratory activity and increased freezing time, though the underlying neural pathways and key brain regions differ. The amygdala is a critical brain region for stress contagion, activated by all sensory channels and stressors during this process. Stress contagion carries both adaptive survival value and negative consequences for individuals. Although substantial evidence has accumulated, many questions remain unanswered. Based on the current state of research, future studies should focus on the following areas:

First, touch plays an important role in emotional transmission, yet research on touch-induced stress contagion is scarce, and current studies cannot clarify the behavioral responses and neural mechanisms involved. Waters et al. (2017) attempted to study touch's role in human stress contagion using a mother-infant separation paradigm, but this research did not present touch information in

isolation, as maternal touching was accompanied by olfactory cues and visual facial expressions. These results require cautious interpretation, as mother-infant stress contagion may arise from interactions between touch and other sensory channels. Future research should present touch information in isolation to explore its behavioral and neural mechanisms in stress contagion.

Second, how individuals integrate stress information across different sensory channels remains a critical question for future research. Although this review summarizes existing studies on single and multisensory channels and compares them in the multisensory section, current evidence cannot directly compare single-channel, dual-channel, and multisensory information effects. Moreover, different sensory combinations may have different effects—for example, visual-olfactory versus visual-auditory integration may differ and require further exploration.

Note: Figure translations are in progress. See original paper for figures.