Neurocognitive Mechanisms of Unconscious Fear in Adolescent Anxiety and Intervention Studies

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Date: 2024-04-09T00:00:00+00:00

Abstract

The prevalence of anxiety disorders peaks during adolescence, exerting profound impacts on adolescent behavior and psychological health. Current understanding of the onset and development of adolescent anxiety primarily relies on research concerning conscious fear processing, neglecting the critical fact that prefrontal function and top-down cortical control functions remain immature during adolescence. Consequently, applying such top-down intervention mechanisms to clinical treatment of adolescent anxiety may entail certain limitations. Examining automatic fear processing in adolescents may help elucidate the pathological mechanisms underlying adolescent anxiety. This study takes healthy adolescents, anxiety-vulnerable adolescents, and adolescents with anxiety disorders as research subjects to investigate: 1) the cognitive neural mechanisms governing the developmental trajectory of unconscious fear processing in adolescents and the modulatory effects of chronic stress hormones; 2) the role of unconscious fear processing in the development of adolescent anxiety; and 3) non-invasive intervention approaches for adolescent unconscious fear. This research will provide scientific support for the prevention, identification, and intervention of adolescent anxiety, thereby promoting comprehensive physical and mental health development in adolescents.

Full Text

Preamble

Identifying the Impact of Unconscious Fear on Adolescent Anxiety: Neural Mechanisms and Interventions

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Abstract: Anxiety disorders reach their peak prevalence during adolescence, profoundly affecting young people's behavior and mental well-being. Current understanding of adolescent anxiety development relies primarily on research into conscious fear processing, overlooking the critical fact that prefrontal cortical function and top-down control are not yet fully mature in adolescents. Therefore, applying such top-down intervention mechanisms to clinical treatment of adolescent anxiety may have inherent limitations. Examining automatic fear processing in adolescents may help clarify the pathological mechanisms underlying adolescent anxiety. This study investigates healthy adolescents, anxiety-vulnerable youth, and those with anxiety disorders to explore: (1) the developmental cognitive neural mechanisms of unconscious fear processing in adolescents and the influence of chronic stress hormones; (2) the role of unconscious fear processing in adolescent anxiety development; and (3) non-invasive intervention methods for unconscious fear in adolescents. This research will provide scientific support for the prevention, identification, and intervention of adolescent anxiety, promoting comprehensive healthy development.

Keywords: fear, anxiety disorders, adolescent, cognitive neural mechanism, fear extinction

1. Research Significance

Adolescence is a period of rapid development in physical, emotional, cognitive, and socio-psychological domains, during which mood disorders such as depression and anxiety become common health concerns. Anxiety ranks as the sixth leading cause of disease and disability among children aged 10–14 globally, severely impairing learning, memory, and social functioning while representing a significant risk factor for mental disorders during adolescence and adulthood (Bittner et al., 2007). Cognitive Behavioral Therapy (CBT) is the most effective psychotherapeutic approach for anxiety disorders with evidence-based support (Craske & Stein, 2016), yet it shows limited efficacy in treating adolescent anxiety, with low success rates (James et al., 2013) and high relapse rates (Ginsburg et al., 2014). Therefore, elucidating the pathological mechanisms underlying the onset, development, and maintenance of anxiety in youth and optimizing intervention strategies is crucial.

Abnormal fear processing, particularly in fear conditioning, generalization, and extinction, is considered a key mechanism in anxiety-related disorders (Treanor et al., 2021). Understanding these mechanisms can guide interventions for anxiety disorders associated with fear processing deficits. Neuroimaging evidence indicates that hyperactivity in the amygdala and insufficient prefrontal cortex (PFC) regulation contribute to abnormal fear processing in adolescents (Zimmermann et al., 2019). These structural and functional changes relate to adolescent developmental trajectories. Subcortical structures such as the amygdala and

striatum develop earlier than the PFC, with amygdala volume peaking during puberty (Caballero et al., 2016). Consequently, even healthy adolescents show stronger amygdala activation and fear learning, along with weaker fear extinction, compared to adults when processing fearful faces (Pattwell & Casey, 2013). In healthy adults, the PFC can effectively regulate defensive responses generated by the amygdala. However, exposure therapy, a common behavioral technique in CBT, primarily derives from research on conscious fear learning and extinction in adults (McNally, 2007), assuming mature PFC development. This may explain its suboptimal effectiveness in treating adolescent anxiety.

The dual-system theory posits that fear generation involves two distinct neural circuits. The first involves cortical regions responsible for conscious perception, while the second involves subcortical regions like the amygdala for threat detection and defensive responses—operating largely at an unconscious level (LeDoux & Pine, 2016). These unconscious mechanisms initiate brain and bodily reactions that subsequently produce conscious fear. In unconscious fear processing, brain regions generating fear experience, such as the PFC, anterior cingulate cortex, and insula, may remain unaffected. Given that unconscious fear processing involves minimal cortical participation, this characteristic may offer new avenues for developing effective interventions for adolescent anxiety. However, most research on adolescent fear conditioning has focused exclusively on conscious fear learning (Zhou et al., 2022), neglecting automatic unconscious fear processing. Therefore, uncovering the brain and cognitive mechanisms underlying unconscious fear processing during adolescence, its role in anxiety development, and related intervention mechanisms represents a critical component in advancing adolescent anxiety treatment.

This study investigates unconscious fear learning in both healthy adolescents and those with anxiety disorders, aiming to: (1) systematically reveal the developmental and cognitive neural mechanisms of unconscious fear processing in adolescents; (2) explore the role of unconscious fear processing in adolescent anxiety development; and (3) investigate intervention mechanisms for unconscious fear in anxious adolescents using neurofeedback technology. Understanding the developmental characteristics of unconscious fear acquisition, generalization, and extinction provides a theoretical foundation for the pathogenesis of fear-related mental disorders in adolescents and offers alternative perspectives for basic research and clinical intervention. This study also aims to reduce abnormal unconscious fear responses, thereby decreasing the incidence and disability rates of anxiety disorders during adolescence.

2.1 Adolescent Developmental Trajectories of the Prefrontal Cortex and Amygdala in Relation to Anxiety

The higher incidence of anxiety disorders in adolescents compared to other age groups may relate to unique brain development trajectories. Research indicates that anxiety-related brain functional systems do not develop synchronously during adolescence, and these developmental asynchronies may trigger anxiety onset

and progression. Neuroimaging studies of fear learning and extinction in adults demonstrate that the PFC, particularly the medial prefrontal cortex (mPFC), and the amygdala play crucial roles in associative memory, fear learning, and extinction (Orsini & Maren, 2012; Sevenster et al., 2018). Both regions undergo significant structural and functional changes during development. Compared to children and adults, adolescents show increased amygdala activation when viewing emotional faces and performing emotional expression tasks (Scherf et al., 2013). Longitudinal studies reveal that amygdala activity during emotional face processing gradually increases from late childhood (age 10) to early adolescence (age 13) (Pfeifer et al., 2011). Additionally, adolescents exhibit higher amygdala activation rates than adults and children during fear learning and emotional go/no-go tasks (Hare et al., 2008; Lau et al., 2011). Anatomically, amygdala volume peaks during mid-adolescence in individuals aged 4–18 (Hu et al., 2013). Notably, larger amygdala volume during adolescence correlates with lower emotional regulation success (Pagliaccio et al., 2014). The mPFC exerts top-down control over subcortical regions to regulate fear and anxiety expression. However, mPFC maturation lags behind the amygdala in adolescents, resulting in immature and weak top-down regulation.

Compared to healthy peers, anxious adolescents show altered connectivity between the medial, dorsolateral (dlPFC), and ventrolateral (vlPFC) prefrontal cortices and the amygdala, with diminished PFC control over amygdala responses. These activation and connectivity differences may cause anxious adolescents to exhibit more intense negative emotions (Weems & Pina, 2010) and interpret relevant information as more negative or threatening. Thus, insufficient inhibition of amygdala neurons may underlie the neural basis of adolescent anxiety disorders.

2.2 The Role of Fear Conditioning, Generalization, and Extinction Paradigms in Adolescent Anxiety Research

Fear acquisition, generalization, and extinction paradigms are commonly used to investigate abnormal behavioral, cognitive, and neural processes in anxiety disorders. During fear acquisition, a neutral stimulus repeatedly paired with an aversive unconditioned stimulus (US) forms a conditioned response (CR) (Wang, E, et al., 2021). One neutral stimulus serves as the conditioned stimulus (CS+) paired with the US, while another (CS-) is never paired. Even without the US, the CS+ elicits fear responses. The amygdala, part of the temporal lobe limbic system, is closely associated with fear and anxiety and forms the neural basis of fear acquisition (LeDoux, 2000). During fear conditioning, sensory inputs from cortical and thalamic nuclei project simultaneously to the lateral amygdala (LA) (Collins & Pare, 2000). The basolateral nucleus (BLA) integrates these sensory signals and serves as the convergence point for CS-US associations, forming persistent synaptic transmission. The BLA then transmits signals to the central amygdala (CeA), which projects to the hypothalamus, brainstem, and other regions related to CR expression (Yehuda & LeDoux, 2007). Anxious adolescents

can learn CS-US associations similarly to healthy adolescents (Michalska et al., 2017). However, they exhibit stronger fear responses to the CS+ (Britton et al., 2013). Furthermore, healthy adolescents show stronger amygdala activation to danger cues (CS+ > CS-), whereas anxious adolescents show higher activation to safety cues (CS- > CS+), indicating abnormal fear cognitive evaluation and neural circuits for fear learning.

Fear generalization refers to CR extending to stimuli similar to the CS, known as generalized stimuli (GS). Generalization is clinically observed in fear-related mental disorders; for example, adolescents who have experienced car accidents may fear other vehicles with similar features (e.g., color, appearance, category) (Lei et al., 2019; Wang, Wang, et al., 2021). As stimulus similarity to the CS+ decreases, fear response intensity shows a gradually decreasing gradient, termed the generalization gradient (Lei et al., 2018; 2017). The amygdala and hippocampus in the limbic system play important roles in fear generalization: the CeA modulates fear generalization, while the hippocampus is involved in discriminative processing (Ciocchi et al., 2010; Kumaran & McClelland, 2012). Lissek et al. (2012) proposed a neurobiological model of conditioned fear generalization. When a current stimulus closely matches the threat stimulus (CS+), the hippocampus initiates "pattern completion," activating the CA3 region and projecting signals to fear expression circuits (e.g., amygdala, insula), generating fear responses (Lissek et al., 2014). When similarity is low, the dentate gyrus (DG) initiates "pattern separation," projecting signals to fear inhibition-related regions (e.g., vmPFC) to suppress fear responses (Greenberg et al., 2013). Thus, fear generalization represents a balance between fear excitation and inhibition. El-Bar et al. (2017) studied fear generalization in clinically anxious adolescents aged 9–18 using auditory stimuli, finding that anxious individuals had lower perceptual discrimination thresholds and broader generalization curves, with generalization intensity increasing with anxiety severity. These adolescent findings align with adult research (Dou et al., 2020; Lopresto et al., 2016). Healthy individuals show decreased vmPFC activation as GS approach CS-, while anxious individuals show insufficient vmPFC activation to GS similar to CS+, failing to inhibit fear responses and resulting in higher fear ratings. These findings indicate that excessive fear generalization begins during adolescence.

Fear extinction involves repeated presentation of a previously US-paired CS without the US, leading to decreased fear responses over time. The extinction neural circuit primarily includes the amygdala, mPFC, and hippocampus. Animal studies confirm that the mPFC, particularly the infralimbic cortex (IL, homologous to human vmPFC), may regulate amygdala output through the BLA to inhibit fear via intercalated cells (Cho et al., 2013; Strobel et al., 2015). Compared to adults, even healthy adolescents show impaired fear extinction. Negative functional connectivity between the PFC and hippocampus, and between the PFC and amygdala, correlates with extinction recall deficits and state anxiety in adolescents. Additionally, the ventral hippocampus fails to effectively inhibit functional connectivity between the BLA and mPFC in adolescents, affecting fear extinction (Xie et al., 2021).

2.3 Unconscious Fear Research Paradigms and Their Cognitive Neural Mechanisms

Fear-related stimuli can be processed at both conscious and unconscious levels. Unconscious fear refers to pre-attentive, automatic fear responses to fear-related stimuli without conscious awareness. Sensory unconsciousness occurs when stimuli cannot be consciously perceived due to sensory limitations (Tamietto & Gelder, 2010). For example, when stimulus intensity is too weak or presentation duration is below detection threshold, conscious perception typically fails even with attention. Common paradigms include backward/forward masking, where a mask presented after brief stimulus presentation prevents conscious perception, and continuous flash suppression, where changing patterns projected to one eye prevent perception of target images projected to the other eye. Backward masking is particularly common in unconscious fear research. Studies using these paradigms show that participants cannot verbally report fear stimuli presence, yet unconsciously perceived emotional stimuli elicit physiological responses, specific electrophysiological components, spontaneous facial muscle activity, and activation in brain regions including the amygdala, superior colliculus, basal ganglia, and occipital lobe.

Threat-related visual stimuli, such as survival-relevant (snakes, spiders) and socially relevant (fearful, angry faces) stimuli, can elicit fear responses without conscious perception (Esteves et al., 1994; Öhman & Soares, 1994). Masked fearful faces have been most extensively studied, eliciting stronger amygdala activity than other expressions (Suslow et al., 2006). Research indicates that adolescents and adults process unconscious facial emotions via different pathways. Healthy adults typically activate a right-lateralized pathway comprising the superior colliculus, occipital lobe, and amygdala. In contrast, adolescents show left-lateralized amygdala activation, lateral striatum, and temporal pathways during masked facial emotion processing (Killgore & Yurgelun-Todd, 2010). This left amygdala response pattern to unconscious fearful faces has been validated in children, suggesting that amygdala responses may become right-lateralized with age. However, the developmental turning point and whether lateralization differs between children and adolescents remain unclear. Future research should employ longitudinal or cross-sectional designs to compare neural differences in unconscious fear face processing across early, middle, and late adolescence.

Unconscious fear can be established not only through prepared threat stimuli but also via Pavlovian conditioning (Mertens & Engelhard, 2020). However, research using associative learning to study unconscious fear remains in its infancy. Given the overlap between anxiety-related brain regions and those involved in associative learning, integrating these approaches is essential. Unconscious fear conditioning studies typically employ masking procedures to prevent stimulus awareness or use distraction tasks and instructional manipulations to divert attention from CS-US contingencies without interfering with CS perception. Functional imaging studies show significant amygdala activation during both conscious and unconscious CS+ presentation (Bertini et al., 2013; Knight et

al., 2005). A recent fMRI study found that unconscious extinction (CS+ presented below awareness threshold) significantly activated fear regulation-related brain regions (amygdala, hippocampus, orbitofrontal cortex), emotion regulation regions (vmPFC, frontostriatal, dlPFC), and visual pattern recognition regions (fusiform gyrus), without inducing distress (Siegel et al., 2018). The neural mechanism of unconscious extinction may align with memory reconsolidation. Subliminal CS+ presentation reactivates conditioned fear memory, which, without physiological fear arousal, is updated and reconsolidated as a new inhibitory memory no longer associated with fear. When CS+ reappears, the reconsolidated memory no longer generates high fear levels, gradually eliminating conditioned fear responses. As Pavlovian conditioning paradigms are important for predicting adolescent anxiety disorders, future research should combine them with unconscious paradigms to investigate adolescent anxiety development mechanisms.

2.4 Abnormal Activation of Unconscious Defense Circuits in Multiple Anxiety Disorders

Rapid threat detection is crucial for defensive responses, but uncontrolled activation of this automatic defense system causes significant distress. Many panic disorder (PD) patients experience panic attacks without conscious threat awareness, characterized by brief, intense fear or discomfort with physical symptoms (palpitations, nausea, sweating, trembling, choking) and mental states (fear of death, loss of consciousness). Because the fear trigger is unclear, this response is often considered irrational. Notably, abnormal activation of unconscious defense circuits is found across various anxiety-related disorders (Taschereau-Dumouchel et al., 2018), including specific phobias, PTSD, panic disorder, trait anxiety, autism, maltreated children, callous-unemotional traits, and violent offenders. These abnormal circuits center on the amygdala and its functional connectivity with the PFC, though patterns differ across symptoms. For example, Killgore et al. (2014) found that compared to healthy controls, PTSD patients showed greater left amygdala activation during masked fearful face processing, while panic and specific animal phobia showed reduced vmPFC activation. In maltreated children, autism, and callous-unemotional individuals, viewing subliminally presented threat faces elicited stronger right amygdala activation than happy or neutral faces (McCrory et al., 2013; Vizueta et al., 2012). Given the relevance of unconscious defense circuits to adolescent anxiety, future research should apply unconscious fear paradigms to test normal and different types of anxious adolescents to better understand their role in anxiety development.

2.5 Application of Neurofeedback Technology in Regulating Anxiety-Related Circuits

Fear and anxiety involve measurable physiological arousal changes, including increased electrodermal activity (EDA), heart rate (HR), and pupil dilation, which are abnormally elevated in anxiety patients. Anxiety patients also show

reduced vagal tone and poor parasympathetic control over sympathetic arousal, making relaxation after stress more difficult. Additionally, anxiety-related neural arousal is detectable via EEG, with evidence linking reduced alpha activity to anxiety. Given these close associations, researchers have used neurofeedback interventions (monitoring neural/physiological activity via EEG, fNIRS, fMRI, and providing real-time visual/auditory feedback for conscious self-regulation) to improve social anxiety, relieve fibromyalgia (Kimmig et al., 2019), increase pain thresholds (Peng et al., 2020), and treat substance addiction (Martz et al., 2020). fMRI-based neurofeedback can guide participants to reduce limbic activity by activating frontal regions to treat anxiety disorders (Mennella et al., 2017). In a spider phobia study, participants received cognitive reappraisal training while viewing spider images (e.g., considering spider aesthetics, humanizing spiders, imagining safe approaches) and regulated feedback stimulus size to learn methods for inhibiting or activating specific brain regions. Neurofeedback guided participants to increase prefrontal activity (related to emotion regulation) and reduce insula activity (related to fear), effectively reducing spider fear (Zilverstand et al., 2015). Since conscious CS+ presentation induces fear and tension, researchers have introduced neurofeedback during fear extinction to reduce CS+ awareness, such as using multivariate fMRI signals to decode neural representations of fear stimuli in real-time and associating them with reward to reduce fear-related visual cortex activation. Thus, neurofeedback technology shows strong operability for intervening in fear-related disorders through unconscious processes.

3. Problem Statement

Adolescent anxiety relates to brain developmental trajectories, with even healthy adolescents being highly vulnerable to anxiety. However, current understanding of adolescent anxiety primarily references adult research findings, which do not align with adolescent developmental patterns, limiting clinical intervention efficacy. Unconscious fear processing circuits offer unique advantages for revealing anxiety-related bottom-up processing symptoms and better match adolescent emotional processing characteristics. Yet research on adolescent unconscious fear processing and its neural mechanisms remains scarce. Applying unconscious fear research methods may help reveal adolescent anxiety development mechanisms. Additionally, few studies have examined relationships between adolescent stress hormones and anxiety, though abnormal unconscious fear processing in individuals with high chronic stress may represent a critical component in the pathological trajectory of anxiety-related mental disorders. Traditional cognitive therapies emphasize top-down emotion regulation, which is particularly difficult for adolescents. For emotionally distressed youth with immature PFC function, negative emotions lead to negative cognitions, reducing treatment efficacy. Some non-invasive neurofeedback approaches may be more suitable. Therefore, based on revealed mechanisms of unconscious fear brain processing, using neurofeedback technology for intervention may improve clinical treatment outcomes.

Based on the above, this study aims to investigate adolescent unconscious fear development and its cognitive neural mechanisms, reveal the role of unconscious fear learning in adolescent anxiety disorder development, and explore intervention mechanisms using neurofeedback. This research holds important scientific significance and application value for understanding the relationship between unconscious fear development and anxiety, potentially opening new avenues for clinical intervention in adolescent anxiety disorders.

4. Research Design

Given the importance of perceptual unconscious fear research in explaining human unconscious fear mechanisms, this study primarily uses perceptual unconscious methods to investigate adolescent unconscious fear processing. The research consists of three main components: Study 1 examines developmental trajectories of neural circuits related to adolescent unconscious fear processing and explores cognitive neural mechanisms using Pavlovian conditioning paradigms, while investigating the regulatory effects of chronic stress on conditioned fear acquisition, extinction, and generalization. Study 1 provides new explanations for why adolescence is a period of high anxiety vulnerability and why anxiety disorders are more prevalent in adolescents than other age groups. Study 2 deepens understanding of unconscious fear processing by comparing different anxiety disorder types, highlighting potential differences in brain activation patterns. These two studies complement each other, providing a comprehensive perspective for understanding and addressing adolescent anxiety. We expect both studies to point to underdeveloped PFC activation in healthy adolescents or insufficient PFC activation in anxious adolescents. Study 3 uses the neural mechanism foundation from earlier work to target the vmPFC as the intervention region, exploring neurofeedback effects on adolescent unconscious fear processing to provide new perspectives for clinical intervention. The specific design is as follows [Figure 1: see original paper].

4.1 Development and Cognitive Neural Mechanisms of Adolescent Unconscious Fear Processing

fMRI technology offers high spatial resolution, accurately revealing specific brain regions and neural network dynamics involved in unconscious fear processing. Using a longitudinal design, Experiment 1 recruits healthy early adolescents (ages 8–13), mid-adolescents (14–17), and late adolescents (18–22) to investigate neural representations of unconscious fear development using fMRI combined with face masking paradigms (Killgore & Yurgelun-Todd, 2010), deepening understanding of brain region functions and their interactions in fear processing. The task includes two conditions: unconscious fear and unconscious neutral. In the unconscious fear condition, target stimuli are fearful faces; in the control condition, they are neutral faces. For optimal masking, target and mask stimuli feature the same person within a trial. Participants are unaware of target stimuli presence, only knowing that faces appear briefly on screen, and their task is

to judge face gender.

Experiment 1 Hypothesis: We hypothesize that amygdala responses to masked fearful faces will weaken with age, while prefrontal activation will strengthen.

ERP technology offers high temporal resolution, providing immediate brain responses to stimuli and revealing rapid cognitive processing of unconscious fear signals. Eye-tracking data reveals visual attention allocation during unconscious fear processing. Experiment 2 uses ERP combined with eye-tracking to investigate cognitive processing of unconscious fear, aiming to reveal temporal dynamics and immediate mechanisms. All participants complete a 2 (awareness: supraliminal, subliminal) × 2 (emotion: neutral, fearful) face masking task. A fixation point appears for 500 ms, followed by a 30 ms target stimulus. In the conscious condition, a 100 ms blank interval precedes a 900 ms mask. In the unconscious condition, the mask appears immediately for 900 ms followed by a 100 ms blank. Participants then judge face gender within 2 s. Pupil diameter data, fixation time on masked faces, and ERP data during target presentation are recorded.

Experiment 2 Hypotheses: (1) ERP: Adult research shows subliminal fearful faces elicit more N2/P3 components than supraliminal faces, and occipital P2 amplitude is sensitive to unconscious fearful versus neutral stimuli. We hypothesize N2, P3, and P2 amplitudes will decrease with age. (2) Pupil diameter: Pupil responses reflect cognitive and emotional processing of fear independent of conscious perception. We hypothesize pupil dilation during fear processing will decrease with age. (3) Gaze duration: Adolescence is a sensitive period for socio-emotional development. We predict younger adolescents will show more gaze avoidance (shorter fixation on eyes and lips), with longer fixation on fearful face eyes and lips as age increases.

Experiments 3, 4, and 5 use Bayesian consciousness classification procedures combined with Pavlovian conditioning to investigate adolescent unconscious fear learning, extinction, and generalization mechanisms. Since stress hormone receptors are widely distributed in fear learning-related brain regions and play key regulatory roles in fear memory (Liu et al., 2021), Experiments 3–5 collect hair samples to measure chronic stress hormone levels, integrating cortisol data with behavioral and physiological data to investigate chronic stress regulation of adolescent conditioned fear.

Experiment 3 investigates adolescent unconscious fear extinction in healthy adolescents. Participants complete unconscious fear acquisition (Öhman & Soares, 1998) and standard fear extinction tasks. During fear acquisition, a 500 ms fixation is followed by a 30 ms CS, then masked by a 100 ms mosaic image. The US appears 500 ms after CS onset and lasts 500 ms. One face (CS+) always follows the US, while another (CS-) never does. Participants rate US expectancy (1 = impossible, 9 = certain) and response confidence (1 = complete guess, 3 = somewhat certain, 5 = completely certain).

Skin conductance and heart rate responses are recorded during CS presentation. During extinction, CS+ and CS- each appear 16 times for 500 ms without US. Bayesian classification converts fear expectancy ratings to binary variables ($ 4 = *highprobability*, < 4 = *lowprobability*)*andconfidenceratings*($2 = "unconfident," >2 = "confident"). Signal detection theory calculates Type I indices: hits (high probability on CS+), correct rejections (low probability on CS-), misses (low probability on CS+), and false alarms (high probability on CS-). Type II indices are also calculated: hits (confident on Type I hits/correct rejections), correct rejections (unconfident on Type I misses/false alarms), misses (unconfident on Type I hits/correct rejections), and false alarms (confident on Type I misses/false alarms). Logistic d1' and d2' are computed for each participant; those with B values <1/3 are classified as metacognitive unconscious, >3 as metacognitive conscious, and others as insensitive. Behavioral and physiological data from fear acquisition and extinction are analyzed across groups.

Experiment 4 investigates neural circuits of adolescent unconscious fear extinction in healthy adolescents and adults. The experiment spans two days. Day 1 includes habituation, standard fear acquisition, and unconscious fear extinction tasks. Day 2 includes an extinction retention test. During habituation, participants view all face stimuli. During acquisition, CS appears for 6 s with 16–20 s inter-trial intervals, including 12 CS+ and 12 CS- trials with 9 CS+ US pairings (75% reinforcement). During extinction, CS appears for 30 ms and is immediately masked by a 170 ms mosaic pattern for 16 CS+ and 16 CS- trials without US. The retention test presents 12 CS+ and 12 CS- trials (6 s each) without shock. Participants rate fear face expectancy during acquisition, extinction, and retention. BOLD, skin conductance, and heart rate responses are recorded.

Expected Results: Research shows adolescents are impaired in fear extinction compared to adults (Ganella et al., 2018). Specifically, adolescents show comparable fear acquisition and extinction but fail to retrieve extinction memories during recall tests on Day 2, exhibiting higher skin conductance responses than adults with reduced dlPFC activity. Additionally, adolescents show reduced activity in vmPFC, dlPFC, temporoparietal junction, and posterior cingulate cortex during extinction recall versus late extinction. Given these neural correlates, we hypothesize adolescent unconscious fear extinction remains impaired, manifesting as greater fear responses during extinction and recall phases and reduced prefrontal region activation.

Experiment 5 investigates neural mechanisms of adolescent unconscious fear generalization in the same participants as Experiment 4. The experiment includes habituation, standard fear acquisition, and unconscious fear generalization phases. Habituation involves passive viewing of all stimuli. Acquisition includes 12 CS+ and 12 CS- trials (6 s each) with 9 CS+ US pairings (75% reinforcement), with US appearing during the final 500 ms of CS+ and lasting 500 ms (16–20 s inter-trial intervals). Generalization includes six conditions: CS+ (0%), GS1 (20%), GS2 (40%), GS3 (60%), GS4 (80%), and CS- (100%),

with 12 trials each (72 total). All stimuli appear for 30 ms and are masked by a 170 ms mosaic pattern without US. Participants rate fear face expectancy while BOLD and skin conductance responses are recorded.

Expected Results: Conscious fear generalization research shows stimulus discrimination improves with age, with older adolescents showing less overgeneralization (Reinhard et al., 2022). Adult studies implicate the hippocampus and vmPFC in perceptual fear generalization. We hypothesize adolescents will show higher unconscious fear generalization than adults, accompanied by weaker vmPFC and hippocampus activation.

4.2 The Role of Unconscious Fear Processing in Adolescent Anxiety Development

This section compares unconscious fear processing across adolescents with social anxiety, panic disorder, generalized anxiety disorder, and PTSD against healthy controls to explore its role in anxiety development.

Experiment 6 uses fMRI to investigate unconscious fearful face processing mechanisms in socially anxious adolescents. Twenty adolescents with social anxiety disorder, 20 with panic disorder, 20 with generalized anxiety disorder, 20 with PTSD, and 20 healthy adolescents are recruited, matched on gender and age. We expect all four anxiety disorder groups to show hypervigilance to unconscious fear stimuli compared to controls, but with distinct neural patterns across disorder types.

Experiment 7 uses fMRI to compare unconscious fear conditioning mechanisms across these groups. The same participant groups complete unconscious fear acquisition and generalization tasks (as in Experiment 5) plus an unconscious extinction phase (CS appears for 30 ms, immediately masked by 170 ms mosaic pattern for 16 CS+ and 16 CS- trials without US) and an extinction retention test (12 CS+ and 12 CS- trials, 6 s each, without shock).

Expected Results: Panic disorder features relate to deficits in unconscious threat processing. Compared to controls, panic disorder patients show reduced vmPFC activation during unconscious fearful face processing. We hypothesize panic adolescents will show abnormal unconscious fear learning, manifesting as hypervigilant responses and difficulty extinguishing fear. Excessive fear generalization characterizes social anxiety, generalized anxiety, and PTSD. During fear generalization tasks, generalized anxiety and PTSD patients show broader generalization gradients and abnormal brain activity, including anterior insula, dorsomedial PFC, dlPFC, and ventral hippocampus. We hypothesize that at the unconscious level, generalized anxiety and PTSD adolescents will show excessive fear generalization with broader behavioral and neural generalization gradients than controls, exhibiting heightened activation in fear-related regions and reduced activation in fear inhibition regions.

4.3 Intervention Mechanisms for Adolescent Unconscious Fear

This section recruits healthy adolescents to explore intervention mechanisms for unconscious fear using neurofeedback technology.

Experiment 8 investigates self-regulation mechanisms of unconscious fear using neurofeedback. Forty healthy adolescents participate in neurofeedback training (NFT) and unconscious fear testing phases. The NFT phase includes two sessions, each with ten 30 s baseline blocks and 40 s self-regulation blocks. The vmPFC is targeted using TurboBrainVoyager software for real-time fMRI data analysis, with feedback displayed on screen. During baseline blocks, a grass icon appears, and participants are instructed to think of nothing. During self-regulation blocks, a flower bud and thermometer appear, and participants use strategies (e.g., thinking happy thoughts) to fully open the flower and increase thermometer readings. Participants then complete an unconscious fear test where spider, snake, and mushroom images are rapidly presented (30 ms) and masked by mosaic patterns (1800–2800 ms inter-trial intervals). The test includes two sub-phases: one requiring use of NFT self-regulation strategies during viewing, the other passive viewing only.

Expected Results: Neurofeedback' s advantage lies in high spatial resolution, enabling detection of deep cortical structures like vmPFC. We hypothesize that through vmPFC neurofeedback training, adolescents will learn appropriate strategies to control brain activity and regulate emotions. Consequently, adolescents will show enhanced vmPFC-amygdala functional connectivity and reduced fear responses during unconscious fear image viewing.

Experiment 9 uses neurofeedback to regulate adolescent unconscious conditioned fear. Forty healthy adolescents are randomly assigned to neurofeedback training or sham feedback groups. The neurofeedback procedure matches Experiment 8; the sham group receives real but task-unrelated feedback targeting the motor cortex. Participants attend five sessions spaced at least five days apart. Sessions 1 and 5 are pre/post-tests measuring unconscious conditioned fear acquisition, extinction, and generalization (as in Experiment 7) and neurofeedback mastery. The middle three sessions are neurofeedback training.

Hypothesis: Participants receiving real feedback training will learn to autonomously regulate brain activity related to conditioned fear, reducing excessive stimulus responses.

5. Theoretical Construction

Anxiety disorders are prevalent mental health problems in adolescents with high morbidity and disability rates. Basic neuroscience research on adolescent anxiety represents a frontier in youth mental health. Fear plays a crucial role in anxiety disorders, as illustrated by the proverb "once bitten by a snake, ten years afraid of a rope," depicting how excessive fear responses disrupt normal life and lead

to trauma- and anxiety-related disorders. Fear-related stimuli are processed at both conscious and unconscious levels. While conscious fear research provides key mechanisms and potential translational models for clinical treatment, it has not yielded significant clinical benefits, partly due to neglecting unconscious fear processing and the potential interactions between conscious and unconscious processes.

Most adolescent anxiety research applies adult fear learning methods, ignoring adolescent brain developmental trajectories and their impact on emotion and behavior. This study focuses on adolescent anxiety, using unconscious perception principles and neuroscience techniques to investigate cognitive neural mechanisms of unconscious fear processing, its pathological role in adolescent anxiety, and related intervention mechanisms. Study 1 uses face masking paradigms combined with fMRI, ERP, and eye-tracking to examine developmental and cognitive neural mechanisms of unconscious fear processing, learning, extinction, and generalization across early, middle, and late adolescence. Study 1 aims to investigate physiological and cognitive neural mechanisms of innate and acquired unconscious fear processing and chronic stress hormone regulation. Study 2 builds on Study 1 by exploring unconscious fear processing mechanisms in social anxiety, panic disorder, generalized anxiety, and PTSD adolescents, addressing the role of unconscious fear processing in anxiety development. Study 2 compares unconscious fear processing across anxiety types to reveal its role in pathological anxiety development. Study 3 uses neurofeedback technology to intervene in adolescent unconscious fear. Study 3 employs novel non-invasive brain modulation techniques to preliminarily explore intervention mechanisms. This research will provide scientific support for adolescent anxiety prevention, identification, and intervention, promoting comprehensive healthy development.

Theoretically, this study will significantly contribute to understanding adolescent anxiety psychopathology. Although unconscious processes operate outside awareness, they critically influence emotion and behavior, with various mental disorders involving unconscious influences. Given that the amygdala matures early while PFC regions mature late in adolescence, unconscious fear processing may dominate during this period. While many studies have explored anxiety from a fear conditioning perspective, none have investigated cognitive neural mechanisms of unconscious fear' s influence on adolescent anxiety. Practically, this study is the first to introduce neurofeedback technology into unconscious fear processing research. Anxiety disorder treatment should not rely solely on medication but should emphasize psychological approaches; improving cognitive-emotion regulation through psychotherapy may be more reasonable and effective than medication. Providing adolescents with online feedback about neural activation patterns can help train and improve self-regulation abilities. This new method application can fundamentally solve emotional problems in youth and has important applied value for optimizing adolescent anxiety treatment.

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