Neural Mechanisms of Emotional Processing and Their Impact on Cognitive Control: A Focus on Anxiety and Positive Affect

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Abstract. This review focuses on two commonly opposing moods—anxiety and positive affect. Both emotions rely on the same neural systems but shift their balance in different directions. Anxiety leads the system towards threat detection. Increased sensitivity to threat-related signals strengthens activity in limbic regions while weakening functional coordination with prefrontal control systems. As a result, cognitive control becomes more nervous, with reduced processing efficiency and less flexible executive regulation. In contrast, positive affect tends to promote a more balanced pattern of network activity. Dopaminergic modulation and stronger fronto-striatal and fronto-parietal connectivity support broader attentional scope and more flexible goal-directed behaviour. This state facilitates cognitive flexibility, working memory updating and adaptive control. Accordingly, anxiety and positive affect can be generally defined as two different configurations of the same large-scale networks, particularly the salience network and executive control systems in neural and neuromodulatory levels. This network-based approach offers an integrated explanation of the dynamic balance between negative and positive emotions regulating cognition across normal function and mental illness. This integrative perspective has important implications for understanding affective disorders, ageing-related cognitive decline, and interventions aimed at enhancing executive resilience.

1 Introduction

How does the emotion relate to cognition? This issue has a long research history in biology, philosophy, and psychology. Early statements tend to conceive the emotion as an antagonist of rational cognition, suggesting that efficient cognitive control demands the inhibition of affect. Today's cognitive neuroscience is far closer to the functional contribution of affect to directing attention and exerting executive functions. In real life, exercising cognitive control does not occur very frequently on emotionless tasks. Decision making in uncertain or social evaluation conditions tends to be affected by affect; instead, it is nearly always dominated by emotions, especially when faced with uncertainty, danger or a prize. The investigation of

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emotion–cognition interaction is critical for understanding not only how cognition adapts, but also why it fails in emotional disorders such as generalised anxiety disorder and social anxiety disorder, where the dysfunction of fronto-limbic circuits leads to deficits in executive functioning.

Traditionally, emotion and cognition were considered as two largely separate processes. Emotion is often treated as an interfering signal to rational thinking and cognitive control. However, recent evidence from cognitive neuroscience suggests a different view: emotion and cognition are deeply related. As argued by Pessoa, emotional and cognitive functions rely on highly overlapping neural systems instead of completely separate brain modules, implying that emotional states can fundamentally shape attention and executive control [1]. This perspective has switched from a modular explanation towards a more comprehensive view based on networks, which conceive of emotion and cognition to arise through interaction between distributed large-scale neural systems. In this model, the affective and the cognitive components are interacting with each other based on what is needed in a given situation or for a specific task to be performed.

The influences of specific emotions on cognitive control are crucial for clarifying the neural mechanisms underlying both adaptive and maladaptive behaviour. This review will initially focus on two representative emotional states—anxiety and positive affect, exploring their underlying neural mechanisms and their differential effects on cognitive control.

2 Neural mechanisms of emotion

Emotional processing is not localised to a single brain region; it is supported by a distributed and highly interactive neural network. Modern cognitive neuroscience showed that emotion and cognition are products of overlapping systems instead of functionally separated modules. This network view revealed the ability of emotional states to affect perception, attention and executive control via dynamic interaction across a number of cortical and subcortical areas.

Consequently, emotion processing interacts with cognitive control closely, especially when rapid adjustments were required in the environment. The point for emotion processing is an interconnected set of limbic and paralimbic structures which attribute affective values to internally and externally stimuli. In this region, activity is modulated by various transmitter systems, enabling plasticity in response to the environment. Additionally, limbic and paralimbic structures are central components of this circuit. The amygdala plays an important role in the rapid detection of emotionally relevant stimuli. This detection is especially sensitive to signals of threat or ambiguity and can occur prior to conscious perception. The insular cortex supports this task through combining with the integration of interoceptive signals such as heart rate, thus contributing to the subjective feeling of emotion. By comparison, positive affective state and motivation have strong relationships with activation of the ventral striatum. Ventral stiatum is a region important for reward processing and reinforcement learning. Together with the ventral striatum, the core circuitry through which emotional value is ascribed to internal and external stimuli was formed.

In addition, beyond emotion detection, the regulation and cognitive consequences of emotion depend on prefrontal and cingulate regions. The prefrontal cortex(PFC) supports executive functions such as working memory, attention control and goal-directed behavior, while also exerting top-down regulation over subcortical emotional regions [2]. According to Rolls, the anterior cingulate cortex(ACC) serves as an interface between emotion and cognition by monitoring conflict and signaling the need for cognitive control by changing the efficiency of prefrontal regulation or by increasing conflict demands, particularly under conditions of stress or heightened affective arousal [3].

Looking at it from a neuro-signalling point of view, emotion affects cognition via alterations to neuronal activity and neurotransmitters. Emotion can increase firing rate or

change the relative amount of excitatory (mainly glutamatergic) and inhibitory (mostly GABAergic) Neurotransmitters (e.g., dopamine [DA], serotonin [5-HT] and norepinephrine), which are known to have strong effects on the activity of limbic networks, which influence attentional orienting, cognitive flexibility and control strategies.

Generally, through limbic evaluation of emotional salience, top-down prefrontal regulation and neuromodulatory signalling, emotion can bias cognitive processing in a context-dependent manner. Thereby providing a neural framework for understanding how specific emotional states modulate cognitive control.

3 Neural mechanisms of anxiety

Based on the network-based framework above, anxiety can be understood as a specific emotional state. It is characterised by a sustained tendency toward threat detection and uncertainty processing. Rather than activating entirely distinct neural structures, anxiety reflects a functional shift within the broader emotion–cognition network. In this case, threat-related circuits become increasingly influential.

Firstly, at the subcortical level, anxiety is strongly associated with increasing amygdala responsivity to threatening stimuli. Transient fear develops in response to an immediate threat. In comparison, anxiety incorporates anticipatory processing of uncertain threat. As for anxiety, a heightened state of vigilance will exist, facilitates bottom-up signalling from the amygdala to the cortex [4]. Consequently, even task-irrelevant emotional information may receive preferential processing, leading to heightened amygdala activation and altered amygdala–prefrontal coupling in individuals with anxiety [4].

Secondly, anxiety can influence prefrontal regulatory mechanisms. Under normal conditions, the prefrontal cortex exerts modulation from top to bottom over limbic structures, allowing flexible adjustment of attention and behaviour. However, under anxiety, this regulatory becomes less efficient. Excessive limbic activation may weaken fronto-limbic functional connectivity, particularly between the amygdala and the dorsolateral prefrontal cortex (dlPFC). This disruption compromises top-down control over threat-related processing [5]. Similarly, the anterior cingulate cortex (ACC) may result in heightened activation when increased conflict arises between internally generated threat representations and externally imposed task demands [5].

Thirdly, neurotransmitter systems maintain the network balance. Increased noradrenergic and related signalling increases arousal and vigilance. At the same time, serotonergic shifts are likely to influence both the emotional sensitivity and stability. These modulatory effects may promote a system that is more reactive but less adaptive at the behavioural level.

In summary, there are no entirely new neural mechanisms in anxiety. Instead, it is an indication of a reorganisation of the existing cognitive networks. Subcortical vigilance circuits take on greater prominence, while prefrontal regulatory control becomes relatively less effective. This change offers an explanation in terms of neural systems as to why anxiety frequently impairs executive functioning, especially in challenging and/or unpredictable situations.

4 Neural mechanisms of positive affect

Depending on the same network-based framework, positive affect can be understood as an emotional state influenced by enhanced reward sensitivity and motivational engagement. Similar to anxiety, positive affect does not activate an entirely separate neural system. Instead, it reflects a functional reorganisation of the existing cognition network, in which reward-related and goal-oriented circuits become more prominent.

Firstly, at the subcortical level, positive affect is strongly associated with activation in the ventral striatum, particularly the nucleus accumbens. This region plays a central role in reward processing and reinforcement learning. Unlike anxiety, which tends to amplify threat-related signals from the amygdala, positive affect enhances sensitivity to rewarding and goal-relevant stimuli [6]. Although the amygdala is often linked to threat detection, it also participates in encoding emotionally salient information more broadly, including positive stimuli [6]. Therefore, in positive affective states, amygdala activity may contribute to evaluating the motivational significance of rewarding cues rather than signalling danger.

Secondly, positive affect also has an effect on the regulation-related functions of the prefrontal cortex. In states of positive emotion, prefrontal areas, including dlPFC, might be engaged in a less restricted way. Instead of being restricted to threat-based vigilance, executive networks are likely to be more exploratory or open in their operation; this would support cognitive flexibility. While ACC remains monitoring for conflict, the perceived level of conflict might be reduced during positive emotion states, leading to more optimal use of mental effort.

Thirdly, the involved neurotransmitter systems can be responsible for such changes. Dopaminergic signalling associated with positive affect occurs along mesolimbic and mesocortical pathways. Increased dopamine availability increases the reward prediction, motivation and working memory updating. Unlike the noradrenergic hyperarousal of anxiety, dopaminergic regulation is more likely to facilitate flexibility than vigilance. Additionally, serotonin is involved with the maintenance of stable moods and the regulation of mood fluctuations. Thus, network dynamics shift towards approach behaviour and adaptive exploration.

Importantly, these neural adjustments may have particular relevance in ageing populations by mitigating age-related declines in fronto-striatal functional connectivity. Anthony et al. demonstrated that positive affect can decrease the impact of neurodegenerative processes on cognitive training plasticity in older adults [7]. Their findings suggest that positive emotional states may support functional connectivity and neural adaptability, even in the context of age-related decline. These effects go beyond simple mood enhancement. Positive affect appears to interact with large-scale brain networks in ways that help preserve executive potential under challenging conditions [7]. Positive affect appears to modulate large-scale brain networks, particularly fronto-striatal and fronto-parietal connectivity. This modulation may help maintain executive control even as cognitive flexibility declines with increasing age.

Overall, positive affect does not eliminate the influence of limbic structures. Instead, it reflects a rearrangement of the emotion–cognition network. In this state, reward sensitivity and motivational engagement become more influential than threat monitoring. Subcortical reward systems interact more fluidly with prefrontal regulatory regions, creating neural conditions that support flexible cognitive control and adaptive goal-directed behaviour.

5 Effects of anxiety on cognitive control

The impact of anxiety on cognition is not limited to its emotional component. It becomes particularly dominant in higher-order cognitive processes that involve executive control. This process requires an individual to ignore task-irrelevant stimuli, maintain task goals and flexibly allocate attention. However, anxiety creates a bias toward threat processing. This bias can interfere with ongoing executive control processes.

Firstly, anxiety is associated with damage to inhibitory control. In paradigms such as the Go/No-Go and Stroop tasks, anxious individuals often show slower reaction times and higher error rates. These effects are particularly apparent when task stimuli carry emotional salience, suggesting that anxiety does not fully destroy cognition but selectively disrupts processes

requiring suppression of prepotent responses. Anxiety distracts attentional resources toward potential threats and reduces the availability of resources for goal-directed behaviour.

Secondly, empirical neural evidence supports this behavioural pattern. Wang et al. used task-based functional near-infrared spectroscopy (fNIRS) to examine the neural correlates of anxiety during inhibitory tasks [8]. It revealed altered activation patterns in prefrontal regions among individuals with higher anxiety levels. Specifically, reduced or dysregulated activation in the dorsolateral prefrontal cortex (dlPFC) was observed during response inhibition. The dlPFC plays an essential role in maintaining task goals and controlling competing impulses [8]. Therefore, decreased recruitment of this region compromises the neural efficiency underlying inhibitory control in anxiety.

Thirdly, anxiety increases conflict monitoring demands in the ACC. When emotionally salient information is present, conflict between task-relevant goals and internally generated threat signals may increase. The anterior cingulate cortex (ACC) is thought to detect this heightened conflict. This heightened conflict signal places additional demands on the executive network. Therefore, cognitive control is reduced under stress. Anxiety is not equal to a breakdown of cognition. Rather, it reflects an overactive vigilance system characterised by heightened threat detection and reduced prefrontal regulation. In addition, frameworks such as Attentional Control Theory provide a useful account of these findings. According to this model, anxiety primarily reduces processing efficiency rather than overall performance [9]. More specifically, anxious individuals may still perform tasks successfully, but they require greater cognitive effort. During this stage, the brain may exhibit altered patterns of neural activation in order to achieve the same outcome [9]. This helps explain why neuroimaging studies often reveal compensatory activation in executive regions, even when behavioral impairments are modest.

Neurochemically, increased noradrenergic activity and stress-related physiological activation may further exacerbate this imbalance. While this state heightens vigilance and sensory sensitivity, it also narrows the focus of attention. In this situation, neural processing tends to prioritise rapid threat evaluation over cognitive control. When this mode is repeatedly activated, it can result in chronic deficits in executive function among those predisposed toward anxiety.

Taken together, anxiety influences cognitive control through both behavioural and neural mechanisms. It takes attention toward threat, increases conflict monitoring demands and reduces the efficiency of prefrontal regulatory systems. These alterations do not eliminate executive function altogether. Instead, they shift the balance of the emotion–cognition network toward vigilance and away from flexible goal-directed control.

6 Effects of positive affect on cognitive control

In contrast with anxiety, when the task requires flexibility, positive affect appears beneficial for cognitive control to update and adapt regulation. Instead of directing the attentional focus towards threat-related stimuli, positive emotions seem to expand cognitive excitation and encourage explorative thinking. This prolonged processing mode enables a more efficient distribution of attentional resources. Additionally, it supports improved behavioural adaptation to shifting task demands. These effects may be facilitated by enhanced fronto-parietal coupling.

Firstly, positive affect has been shown to enhance cognitive flexibility and improve working memory performance. Behaviourally, it is reflected in higher accuracy and reduced switch costs in task-switching and n-back paradigms. People experiencing happiness often perform more efficiently when switching between tasks and tend to do better on specific tasks that require multiple sources of information. Lautenbach demonstrated that positive emotional states were linked to enhanced executive functioning, especially in measures of

cognitive flexibility and information updating [10]. Importantly, these improvements were not merely the result of feeling happier, they reflected genuine changes in executive performance, including lower switching costs and more accurate working memory updating. Unlike anxiety, which selectively impairs inhibitory control under emotional interference, positive affect appears to strengthen adaptive ability and flexibility with goals.

Secondly, in neuroscience, these behavioural effects can be demonstrated in terms of large-scale fronto-striatal and fronto-parietal network dynamics. These networks involve interaction between reward-related signals from lower brain regions and regulatory control from the prefrontal cortex. Positive affect is closely related to a more balanced interplay between subcortical reward structures and prefrontal regulatory mechanisms. This balance may reduce excessive conflict signalling within the anterior cingulate cortex (ACC). Higher efficiency in mesocortical dopaminergic pathways is associated with stronger activation of the dorsolateral prefrontal cortex (dlPFC). This supports goal maintenance and working memory updating. In contrast to the processes often observed in anxiety like hypervigilant and conflict-driven, positive affect promotes a more stable and flexible pattern of connectivity within executive control networks.

Thirdly, neuromodulatory processes also contribute to this change in network balance through brief increases in dopamine activity. Dopaminergic signalling within mesolimbic and mesocortical circuits enhances reward sensitivity and cognitive flexibility. When dopaminergic modulation reach the optimal level, the signal-to-noise ratio within executive networks is improved. In contrast to the excessive noradrenergic activity of anxious states, dopaminergic modulation promotes motivation for solutions and adaptive exploration. Additionally, balanced serotonergic activity may help stabilise regulatory processes, preventing impulsive responding while maintaining flexible control. Through these mechanisms, positive affect supports a neural configuration that facilitates efficient executive functioning.

In conclusion, positive affect influences cognitive control in several important ways. Dopaminergic modulation helps executive control systems work more efficiently, supporting both goal maintenance and flexible updating of information. Balanced serotonergic activity helps maintain regulatory stability without suppressing adaptability. Compared with anxiety, positive affect promotes a more flexible and adaptive mode of cognitive control.

7 Comparative perspective: anxiety vs. positive affect

Despite there are really different through distinct emotional states, anxiety and positive affect operate in the same large-scale emotion–cognition structure. The key difference does not lie in the use of entirely different brain regions. Rather, it lies in how distributed brain networks interact and its function under different emotional states. From a network perspective, these contrasting behavioural patterns can be understood as shifts in the balance among systems. These include the salience network, prefrontal executive systems and reward-related fronto-striatal circuits.

Firstly, at the behavioural level, anxiety and positive affect represents opposing effects on executive functioning. Anxiety tends to prioritize threat monitoring and impair performance on tasks requiring sustained inhibitory control and the maintenance of goal stability under uncertainty. In contrast, positive affect is linked to a broader attentional scope and greater cognitive flexibility. These changes support more efficient task switching, improved working memory updating, and adaptive exploration. In everyday situations, anxiety often leads to rigid and cautious control, whereas positive affect supports more flexible and adaptive responses. For example, during an exam, an anxious student may repeatedly check for potential mistakes and focus excessively on minor uncertainties, which reflects vigilance-dominated control. In contrast, a student experiencing positive affect may

more easily change strategies when encountering a difficult question, flexibly allocating attention and updating their approach.

Secondly, at the neural network level, these opposing profiles can be distinguished in terms of the coordination between limbic evaluation systems and prefrontal control circuits. In anxiety, potential threats are assigned exaggerated salience. This bias strengthens bottom-up signalling from limbic regions such as the amygdala and the insula. Additionally, the insula contributes to heightened interoceptive awareness of arousal. This imbalance increases conflict demands and amplifies the recruitment of monitoring systems, particularly the anterior cingulate cortex (ACC). Moreover, the effect becomes most evident when internally generated threat signals compete with external task goals. Instead, under positive affect, salience is more likely to be allocated to rewarding or goal-relevant information. This bias supports approach-oriented learning and adaptive behavioural updatin. The view is consistent with network-based frameworks, which emphasise the emergent integration of neural circuits and the context-dependent shaping of behaviour. The salience network can be viewed as a key coordinating system. In addition, it links limbic signals with executive control resources. Through this integration, it influences whether attention and control are directed toward threat monitoring or adaptive goal pursuit [11].

Thirdly, neuromodulatory mechanisms further differentiate these network configurations. Anxiety is commonly associated with heightened noradrenergic arousal and related stress signalling, which amplifies vigilance and sensory responsiveness but can narrow attentional scope and reduce cognitive flexibility. In contrast, for anxiety, positive affect is more closely associated with dopaminergic modulation within mesocortical and mesolimbic pathways. This modulation enhances reward sensitivity and supports flexible engagement of prefrontal regions such as the dorsolateral prefrontal cortex (dlPFC). In functional terms, noradrenergic-dominant states support rapid detection and defensive prioritisation, whereas dopaminergic-dominant states support approach-oriented motivation and adaptive exploration.

Taken together, anxiety and positive affect can be understood as two contrasting configurations of the same emotion–cognition network. Anxiety predisposes the system toward hypervigilance and conflict sensitivity at the expense of efficient executive control. In contrast, positive affect reorients network dynamics toward reward-related salience, balanced connectivity and flexible goal-directed regulation. A network-level perspective provides a coherent framework for understanding these effects. By highlighting the coordinated interaction of systems such as the salience network, it helps explain how emotional states generate distinct patterns of cognitive control across real-world contexts [11].

8 Conclusion

The review has initially examined how emotional states shape cognitive control through the dynamic reconfiguration of large-scale neural networks. Beyond traditional modular statements, the evidence reviewed here supports a network-based framework in which emotion and cognition emerge from reciprocal interactions among limbic, prefrontal and neuromodulatory systems. Within this shared structure, anxiety and positive affect do not activate entirely distinct neural structures. Instead, they keep a different balance of the same emotion–cognition network in different directions.

Anxiety can be characterised by increased weighting of threat salience and heightened limbic activation. It is also associated with reduced fronto-limbic coupling and greater conflict-monitoring demands. Together, these conditions are typically linked to lower processing efficiency and diminished executive flexibility. Conversely, positive emotions are associated with increased dopamine transmission, greater prefrontal cortex striatal coupling and a wider range of attention. A reward-oriented configuration supports flexible task switching and effective recruitment of executive networks. Together, these opposing patterns

suggest that similar brain networks are differentially regulated by affective states. Depending on different situation, this regulation can promote either defensive vigilance or approach-oriented behaviour.

Importantly, adopting a perspective of network offers a more integrative understanding of both typical functioning and psychopathology. This way emphasises the importance of targeting. Cognitive control deficits are not simply caused by dysfunction in a single brain region. Instead, they are better understood as imbalances in large-scale brain connectivity and neuromodulatory systems. This framework takes advantage of affective disorders, ageing-related cognitive decline and interventions aimed at enhancing executive functioning.

Future research should further clarify how dynamic functional connectivity varies across emotional states in real different situation. Particularly, more precise use of neuroimaging may reveal how large-scale brain network activity interacts with underlying neurochemical processes. Long-term studies should examine the effects of repeated exposure to specific emotional states, especially for high-risk populations. More research may clarify whether sustained exposure leads to persistent changes in executive network efficiency. Additionally, positive affect could be explored as potential modulators of cognitive resilience, especially in vulnerable populations such as older adults or individuals at risk for affective disorders.

In conclusion, the character of anxiety and positive affect on cognitive control cannot be fully explained by region-specific theory. It requires a broader view of how brain networks interact and reorganise. Emotional states do not merely depend on cognition. They dynamically regulate the neural conditions under which cognitive control needs. Recognizing this relationship offers a critical framework for both theoretical models and applied research in affective and cognitive neuroscience.

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