Current research on cognitive aspects of anxiety disorders

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Purpose of review

Cognitive dysfunction is frequently reported in anxiety disorders. Our aim is to describe recent advances concerning these cognitive aspects.

Recent findings

Cognitive dysfunction in anxiety disorders can be classified into four domains. The first concerns executive functions, mainly attentional processes. The second concerns memory, including deficits in working, episodic, and autobiographical memory. The third encompasses maladaptive cognitions, or thoughts and beliefs. Finally, a burgeoning area of research (mainly in obsessive–compulsive disorder and posttraumatic stress disorder) concerns metacognitions, or thoughts and beliefs about one's own thoughts and beliefs. All of these dysfunctions may contribute to maintain or aggravate anxiety disorders. When developing and implementing interventions, researchers and clinicians alike must consider these cognitive aspects, and may need to tailor their approaches accordingly.

Summary

Advances have clearly been made in the elucidation of the cognitive functioning associated with anxiety disorders. It remains unclear if particular cognitive profiles can help to distinguish anxiety disorders from one another, although emerging evidence suggests this may be the case. Further clarification will add to our understanding of the development and maintenance of these disorders, and may provide targets for future therapy and endophenotypes.

Keywords

anxiety, attention, cognition, memory, metacognition

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Introduction

For decades, theoretical models and clinical practice have described the presence of cognitive impairment in anxiety disorders (e.g. [1,2]). Indeed, cognitive impairment *per se* is a symptom of anxiety disorders, representing both a cause and a consequence. Cognitive deficits need to be examined regarding their meaning vis-à-vis the illness as a state-dependent variable, a trait abnormality or as vulnerability markers of specific disorders [1].

Much empirical work has been conducted with the aims of elucidating these cognitive aspects. Generally, the cognitive deficits can be grouped into attentional biases, memory dysfunction, and cognitive and metacognitive vulnerabilities. Despite substantial advances, however, the literature remains heterogeneous and inconsistent, with methodological shortcomings often blamed for the ambiguity. In addition, most research has been focused on obsessive–compulsive disorder (OCD) and posttraumatic stress disorder (PTSD), with much less work on the phobic disorders. Still, recent evidence suggests that particular cognitive profiles may distinguish specific anxiety disorders.

Herein, this article will review recent research on the cognitive deficits in anxiety disorders. First we will provide a brief introduction to anxiety disorders and their neural correlates. Next, we will turn to the empirical literature to describe the profiles of the cognitive impairment that has been reported, highlighting particular findings with respect to the individual disorders. Finally, we will describe how the pharmacological treatments used to mitigate pathological anxiety may have a role to play with respect to cognitive function. We acknowledge the significance of neuropsychological tests in this discussion but we will not examine them *per se* in this text.

Anxiety disorders

The anxiety disorders form a category of maladies in the Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV that are characterized by pathological and

abnormal fear and anxiety [3]. They represent the most prevalent psychiatric disorders, with epidemiological research revealing a lifetime prevalence rate of 28.8% [4]. The subtypes include generalized anxiety disorder (GAD), panic disorder, phobic disorders, agoraphobia, social anxiety disorder (SAD; also known as social phobia), OCD, and PTSD [3]. The anxiety disorders are frequently comorbid with other psychiatric disorders, especially major depressive disorder [5]. They are often chronic or recurring in nature, and are associated with a significant decrease in quality of life, increased personal distress, sexual dysfunction, addiction, greater mortality and suicide risk, and negative economic impact [5].

Neuroanatomical correlates

The findings are not invariable, but particular regions and neural circuits have been implicated in anxiety disorders and may underlie many of the associated cognitive features (Table 1) [6,7].

Attentional biases and executive dysfunction

Attention allocation to threatening stimuli is regarded as a necessary adaptive mechanism but anxious individuals seem to disproportionately direct their attention to such stimuli [8]. In a recent review, Cisler and Koster [9••] identified the three main components of attentional bias: facilitated attention (threat-related stimuli are detected faster than unrelated stimuli), attentional disengagement (difficulty disengaging attention away from threatening stimuli), and attentional avoidance (directing attention away from threatening locations). A different balance of these components may operate at any one instant depending on the stage of information processing observed, the task utilized, and the particular anxiety disorder [9••,10].

Social anxiety disorder

There exist a limited number of neuropsychological studies in SAD, but they suggest possible impairment in executive function and verbal processing of phobia-related words [11,12]. Face paradigms are often used in the study of attentional allocation in SAD since these individuals experience their difficulties in interpersonal situations. High SAD seems to be associated with abnormalities in processing emotionally charged faces [13], notably fear [14]. Buckner *et al.* found that those with higher SAD had more difficulty disengaging from negative social cues (disgust faces) but not from the positive social cues (happy faces) [15]. Gamble and Rapee [16•] showed that attentional bias was only evident in SAD subjects during the early processing of social cues, with subsequent processing patterns matching those of the healthy controls.

Generalized anxiety disorder

Regarding GAD, MacNamara and Hajcak [17] used the event-related potential (ERP) method to reveal reduced processing efficiency of aversive stimuli, in addition to behavioural evidence for attentional bias. Interestingly, several recent studies showed that this attentional bias can be redirected with explicit instruction and training, which in turn may reduce excessive worry [18,19].

Obsessive–compulsive disorder

Investigation into cognitive dysfunction in OCD is relatively developed but is fraught with inconsistencies. For instance, executive dysfunction is commonly claimed to be the primary cognitive deficit in OCD [20], and yet OCD is often associated with impaired performance on the Object Alternation Task (OAT), but not the Wisconsin Card Sorting Task (WCST), despite both being established tests of executive function [20], but see [21]. Further, many designs confound the components of facilitation and inhibition [22]. More recent paradigms have thus been designed with the aim of tapping the different components of attentional biases. In a modified alternation task, those with OCD committed more perseveration errors following prior correct response but did not differ from controls regarding shift errors, or perseveration errors after prior incorrect responses [23]. Bannon *et al.* [22] measured facilitation

Table 1 Brain structures implicated in anxiety disorders and the associated cognitive dysfunction

Disorder	Clinical features	Structures implicated in aetiology and cognitive dysfunction
Panic disorder	Chronic, unexpected panic attacks, worry about future panic attacks, strong somatic symptoms and sympathetic nervous system arousal	Amygdala, hippocampus, thalamus, brain stem, frontal cortex
Social anxiety disorder	Chronic fear of social interactions or performance, of negative evaluation	Amygdala, PFC
Generalized anxiety disorder	Chronic worry and anxiety, somatic symptoms	Amygdala, medial PFC (literature is limited)
Obsessive–compulsive disorder	Intrusive thoughts and behavioural compulsions	Thalamo-cortico-striatal loops, notably OFC, ACC, possibly parietal and occipital regions
Posttraumatic stress disorder	Repeated re-experiencing of the traumatic experience, hyperarousal, numbing or avoidance of trauma-related stimuli	Amygdala, medial PFC, hippocampus

ACC, anterior cingulate cortex; OFC, orbitofrontal cortex; PFC, prefrontal cortex. Data from [3,6,7].

and inhibition independently and showed OCD to be associated with stronger facilitation and weaker inhibition than controls in a word processing task, whereas no evidence for inhibition was found in a recent negative priming task [24]. Amir *et al.* [25•] found that those with OCD evinced an attentional bias towards threatening words only in the first portion of the task, and the bias was correlated with symptom severity. The results suggest that the bias may not be stagnant over time, perhaps reflecting habituation to threat [25•].

Posttraumatic stress disorder

Similarly to studies in OCD, investigators are beginning to tease apart the facilitation and inhibition components of attentional biases in PTSD. In a visual search task, those characterized with high PTSD showed increased interference to trauma-related words relative to low PTSD individuals, suggesting that those with high PTSD do not have difficulty disengaging from threat in general but rather from trauma-related stimuli [10]. Nevertheless, the evidence for attentional bias is not unequivocal. A review of the modified Stroop task in PTSD showed that, contrary to the general assumption, the majority of dissertations reviewed did not find evidence for bias [26].

Memory dysfunction

Memory dysfunction has been recognized as a key feature of anxiety disorders for some time (e.g. [27]).

Social anxiety disorder

In an ecological paradigm, individuals with social anxiety and confederates gave speeches and received standardized feedback on theirs and the confederate's performance [28]. Those with SAD remembered the confederate's feedback as more positive than their own, indicating a recognition bias [28]. Autobiographical memory deficits have been recently reviewed in SAD and it seems that significant biases may exist with respect to social-threat memories and related imagery that may serve to maintain SAD [29].

Obsessive–compulsive disorder

While memory dysfunction was purported to be integral to OCD in theoretical accounts (e.g. [30]), recent reviews have failed to delineate a clear profile. Still, the impairment that has been found is generally attributed to disorganization during encoding [20,31]. Overall, visual memory impairment is reported more consistently than verbal memory impairment [32]. In a recent functional MRI study, Nakao *et al.* [33] found behavioural and functional evidence for working memory impairment. The most reliable finding across studies is reduced confidence in one's own memory, a feature of metamemory ([32], but these are discussed in the following section). Nevertheless, Moritz *et al.* [34] did not find support for any of the memory theories of OCD and suggest that the 'occasional instances' of memory dysfunction may be due to cognitive biases such as perfectionism and inflated sense of responsibility (both discussed below). Cuttler and Graf [35] reviewed the literature and noted that perhaps the ambiguous evidence for memory dysfunction may be because most studies concern retrospective memory while hardly any have examined prospective memory.

Posttraumatic stress disorder

There is evidence that disorganization of trauma-related memories is significant in the maintenance of PTSD, but this conception is not without criticism (see [36], as discussed below). Jelinek *et al.* [37] thus decided to see if perhaps only particular portions of the trauma memory were disorganized. When those with PTSD were asked to describe the worst moments of their traumatic experience, they showed more unfinished thoughts, used fewer words, and more of the present tense as compared with the rest of the narrative. The authors suggest that these worst moments be preferentially targeted in treatment [37].

Regarding memory dysfunction *per se*, the most consistent cognitive impairment found in PTSD is verbal memory impairment of medium effect size [38]. Some have suggested that the verbal memory impairments might be due to impaired executive control that in turn impairs learning and encoding strategies [39]. Preliminary evidence also exists for an implicit memory bias for trauma relevant and negative stimuli [40]. The domain of autobiographical memory, a specific type of declarative memory concerned with personal events and facts, has been the centre of much investigation in PTSD [41]. As in depression, PTSD is associated with an overgeneral memory bias [41–43]. The literature also suggests that the verbal and autobiographical memory deficits most likely represent preexisting risk factors [42]. The presence of working, verbal, nonverbal, and autobiographical memory deficits has recently been found in acute PTSD, indicating that they are apparent from the inception of the disorder [44•].

Cognitive and metacognitive deficits and vulnerabilities

Recently, there has been a shift in focus from 'cold' cognitive dysfunction (e.g. attentional bias, memory deficits) to 'hot cognition' (e.g. cognitive styles, personality, metacognition) [45]. Cognitions refer to thoughts and beliefs, while metacognition refers to thoughts and beliefs about these thoughts and beliefs [46]. Determining which cognitive styles – consistent heuristics one uses to process information, including ways of perceiving, thinking, and problem solving – are associated with anxiety disorders has also received attention [47].

Cognition

Intolerance of uncertainty is one cognitive process that may represent a unifying factor across anxiety disorders [48]. Intolerance of uncertainty is the inability to tolerate ambiguity, and those with high intolerance of uncertainty are likely to interpret ambiguous stimuli as threatening [49]. Intolerance of uncertainty may represent a unifying factor across anxiety disorders [48]. Fergus and Wu found that GAD and OCD were similarly associated with intolerance of uncertainty, and that intolerance of uncertainty was the one cognitive process that predicted GAD and OCD when the other cognitive processes and distress levels were controlled for [50]. In a study comparing intolerance of uncertainty levels across symptom groups, those with GAD or SAD had comparable levels of intolerance of uncertainty, and those with both GAD and SAD had the highest [48]. It has been suggested that intolerance of uncertainty may mediate other noted cognitive vulnerabilities such as anxiety sensitivity – tendency to fear anxiety-related feelings [48], and feelings of inflated responsibility, which is typical of OCD [51].

Fear of negative evaluation (FNE) has often been associated with SAD. In a study of SAD, Haikal and Hong demonstrated that FNE and looming cognitive style (LCS – the feeling of rapidly intensifying threat) interact with socially demanding situations to give rise to anxiety symptoms [52]. SAD also seems to be associated with the negative interpretation of positive social events, more so than panic disorder with or without agoraphobia, and GAD, but comparable to OCD [53].

Cognitions and belief domains consistently identified in OCD include perfectionism, intolerance of uncertainty, overestimation of threat (OET), and inflated responsibility (in that harm will come to oneself or others if they do not undertake preventive action) (e.g. [45,54–56]). Regarding cognitive styles, Harkin and Kessler suggest that checking may in fact represent a detrimental cognitive style [57].

In a review of the cognitive vulnerabilities associated with PTSD, negative attributional style (tendency to attribute negative events to oneself), rumination (tendency to think repetitively about negative emotions and distress and to worry about the meaning of these feelings), anxiety sensitivity, and LCS emerged as significant, although the authors emphasize that few studies have been conducted and have mostly been cross-sectional and retrospective [58].

Metacognition

Metacognitive research is most progressed in OCD. As mentioned above, the most consistent finding regarding memory in OCD (notably with 'checkers') is diminished confidence (e.g. [32,59,60]). Paradoxically, repeated checking seems to leave memory accuracy intact but further reduces confidence and vividness, driving more checking and leading to a vicious cycle (e.g. [59,61]). Nedeljkovic and Kyrios proposed a four-factor model of metamemory consisting of general confidence in memory, decision making, concentration, and perfectionism about cognitive functioning [62]. They subsequently found that general confidence in memory uniquely predicted symptom severity [63]. In an electroencephalography (EEG) study, it was shown that source memory may be associated with memory dysfunction *per se* while item memory accuracy correlated with cognitive confidence, suggesting that perhaps different aspects of memory are affected more than others by reduced confidence [64].

A particular metacognitive style associated with OCD is cognitive self-consciousness. This is the tendency to be overfocused on one's own mental processes regardless of

Table 2 Brief summary of recent cognitive deficits associated with anxiety disorders

Disorder	Attentional and executive dysfunction	Memory dysfunction	Abnormal cognitions and metacognition	References
Social anxiety disorder	Impaired processing of phobia-related words, emotionally charged faces	Recognition bias, autobiographical memory deficits	Intolerance of uncertainty, fear of negative evaluation, looming cognitive style, negative interpretation of positive social events	[11,12,28,29,48,52,53]
Generalized anxiety disorder	Impaired processing of aversive stimuli		Intolerance of uncertainty	[17–19,48,50]
Obsessive–compulsive disorder	Executive dysfunction, attentional bias, impaired word processing	Abnormal encoding, visual memory, working memory impairment	Intolerance of uncertainty, perfectionism, overestimation of threat, inflated responsibility, cognitive self-consciousness, reduced confidence in memory ability	[20–24,25•,31–35, 45,51,54–57,59–65]
Posttraumatic stress disorder	Executive dysfunction, attentional bias	Disorganization of portions of trauma memory, verbal memory, working memory, autobiographical memory impairment	Negative attributional style, rumination, anxiety sensitivity, looming cognitive style	[10,26,36–43,44•, 58,66,67]

content, and may explain why those with OCD assign such important to their intrusive thoughts [65]. Solem *et al.* [54] identified three metacognitive knowledge domains in OCD: thought-fusion beliefs, beliefs about rituals (the need to perform them), and the stop signals or criteria indicating when to stop the ritual.

Concerning PTSD, Bennett and Wells [36] have provided support for the metacognitive model [36]. Results showed that beliefs about the trauma memory predicted PTSD symptoms while memory disorganization, often believed to be significant in PTSD, did not.

Table 2 provides a compilation of the different domains of cognitive functions affected in anxiety disorders, as described in the text above [66,67].

Cognitive modulation by pharmacological treatments

Anxiety disorders are commonly treated with benzodiazepines, antidepressants, and other antianxiety compounds. While these drugs have proven efficacious in the alleviation of anxiety, they have also been shown to impair cognition to various degrees. In Hindmarch's [68] recent literature review of SAD, serotonin reuptake inhibitors (SSRIs) affected cognition the least while benzodiazepines (especially lorazepam) did the most. Further studies of this nature are required so that clinicians may make informed decisions about which treatments to prescribe.

Conclusion

Cognitive impairment may exacerbate existing symptoms, may disturb coping mechanisms, and may cause other difficulties in daily functioning. It remains unclear to what extent the aforementioned cognitive deficits either precede or are a result of a particular anxiety disorder [1]. Longitudinal studies will be required to elucidate this issue. Such studies may also reveal if certain states of an anxiety disorder are associated with specific types or severity of cognitive impairment. Additionally, these deficits may be indicative of prognosis: memory specificity in PTSD was predictive of symptom severity 6 months later [69]. Profiling of cognitive dysfunction may also be useful for identifying potential endophenotypes. A recent study of individuals with OCD, healthy controls, and their respective relatives suggests that aspects of executive dysfunction (decision making, planning, and mental flexibility) represent viable endophenotypes [70]. Finally, there is evidence that memory performance may be used to measure the success of treatment [71]. Vogele *et al.* [72] showed that cognitive changes play a clear role in mediating the success of exposure therapy in phobia and agoraphobia. Overall, many advances have been made in understanding the cognitive impairments in anxiety disorders, but the literature remains heterogeneous. There seems to be a trend towards the use of more ecological and sophisticated paradigms. Such tendencies will serve to further our understanding of this subject.

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