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ANXIETY DISORDERS (L BROWN, SECTION EDITOR)

Executive Functioning Constructs in Anxiety, Obsessive–Compulsive, Post-Traumatic Stress, and Related Disorders

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Abstract

Purpose of Review We synthesize theories proposing complex relations between cognitive functioning and anxiety-related concepts. We evaluate vulnerability theories suggesting that deficits in various cognitive functioning domains predict future anxiety-associated concepts. We examine scar theories asserting the opposite direction of effects (i.e., anxiety predicting cognitive dysfunction). Furthermore, we examine more novel frameworks on this topic.

Recent Findings Reliable evidence exists for the scar and vulnerability theories. This includes mounting data on diverse anxiety symptoms predicting cognitive dysfunction (and conversely) unfolding at between- and within-person levels (dynamic mutualism theory). It also includes data on the stronger effects or central influence of anxiety (versus non-anxiety) symptoms on executive functioning (EF; i.e., higher-order cognitive control governing myriad thinking and action repertoires) versus non-EF domains and vice versa (network theory). In addition, it reviews emerging evidence that *enhanced* cognitive control can correlate with *higher* anxiety among children (overgeneralized control theory).

Summary The generally inverse relations between anxiety symptoms and cognitive dysfunction are bidirectional and complex within and between persons. Plausible mediators and moderators merit more attention, including immune, metabolism, and neural markers and the social determinants of health.

Keywords Anxiety disorders · Executive functioning · Scar theory · Vulnerability model · Network analysis · Overgeneralized control model

Introduction

Anxiety disorders are widespread, with an estimated lifetime prevalence rate of about 3.2 to 28.8% of the general adult population in the US [1] and other countries in Europe, Asia, Africa, the Middle East, and South America [2]. Common anxiety symptoms include chronic and intense worries, fears, panic attacks, and avoidance of social and performance contexts that may or may not be evaluative. Anxiety and related disorders include generalized anxiety disorder (GAD), panic disorder, agoraphobia, social anxiety disorder, obsessive–compulsive disorder (OCD), and posttraumatic stress disorder (PSTD) [3]. Economically, these disorders incur substantial costs (e.g., $42.3 billion a year in the US, 451€ in Europe) [4, 5] and, in 2010, contributed to 26.8 million disability-adjusted life years (i.e., total years of life lost due to premature death, suboptimal health, or disability) globally [6]. Furthermore, heightened anxiety symptoms were reliably associated with poorer school functioning, social relationships, job performance, physical health problems, and other quality-of-life indicators [7]. Therefore, understanding the risk factors of elevated anxiety disorder symptoms is essential.

Executive functioning (EF) deficits are potential modifiable proximal and distal risk factors for anxiety and related disorders. EF refers to an array of multifaceted higher-order cognitive control processes needed to efficiently appraise benefits and risks, prioritize, solve problems, implement ideas, and plan with good foresight [8]. Given how EF governs the regulation of numerous cognitive and behavioral processes, EF issues correlate with myriad anxiety and related disorders [9]. The *attentional control theory* (ACT)

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[10] proposes that high trait anxiety and pathological worry coincide with weaker *inhibition* (refraining from autopilot reactions), *shifting* (developing new rules, shifting between unique thinking modes), and *working memory* (WM; tracking and updating data simultaneously) [11]. The earlier iteration of ACT posited that high trait anxiety and worry were associated with shifting and inhibition deficits and that WM was relatively intact except in stressful or threatening contexts. However, recent empirical studies have challenged this proposition, given emerging evidence for more significant associations between trait anxiety and worry and compromised WM (vs. inhibition and shifting) [12]. Thus, attention control problems could arguably correlate with difficulties suppressing or disengaging from repetitive negative thinking [13].

Literature supports the assumptions of ACT and the cognitive model of pathological worry. Data aggregated across 82 meta-analyses showed notable cross-sectional links between constructs of anxiety (fear, worry, panic attacks, obsessions, posttraumatic stress) and cognitive functioning (attention, EF, memory, processing speed, visuospatial abilities), with small-to-large effect sizes [14]. These findings led to the development of the *c*-factor of psychopathology theory [14], which postulates that neurocognitive problems cut across all mental disorders. Also, anxiety disorders, OCD, and PTSD uniformly corresponded with stronger electroencephalography (EEG)-indexed beta wave frequency (vs. theta and delta brain wave oscillations) and suboptimal performance on EF and verbal memory tests [15]. Excessive beta wave frequencies typically indicate behavioral and cognitive inflexibility [16], features consistently observed in anxiety and associated disorders [14]. However, our understanding of their links must go beyond correlations at a single time point. Prospective studies are essential to establish temporal precedence and covariation, prerequisites for weak causal inferences [17]. Prospective analyses allow us to determine if (a) cognitive dysfunction serves as a risk factor for anxiety-related psychopathology, (b) cognitive dysfunction emerges from untreated anxiety-related disorder(s) or symptoms, or (c) complex bidirectional relations exist between anxiety and cognitive dysfunction constructs.

The current review evaluates theories proposing complex longitudinal relations among EF, other cognitive functioning, and anxiety-related constructs. We examine vulnerability models that argue that EF issues and associated cognitive functioning predict anxiety-linked constructs and scar theories that assert the reverse effects. Furthermore, we discuss more novel frameworks on this topic, including the dynamic mutualism, network, and overgeneralized control theories of EF deficits and anxiety constructs. The present review concludes with clinical implications and suggestions for future directions on this topic.

Vulnerability Models: EF-Related Deficits Predict Anxiety Constructs

Figure 1 summarizes the tenets of vulnerability models and scar theories. Several theories have proposed that EF and related deficits might contribute to the development of future anxiety disorders, OCD, and PTSD across the lifespan. *Vulnerability models*, such as the *cognitive model of pathological worry*, theorize that attentional control deficits predict and lead to the initiation and maintenance of excessive and uncontrollable worry [18]. Relatedly, the *iterative reprocessing model* postulates that contexts with novelty, ambiguity, uncertainty, or contradictory information necessitate people to pause and reflect before decision-making [19]. This pause requires harnessing EF-linked abilities such as inhibition, set-shifting, WM, and processing speed. Persistent failure to pause and deliberate on novel information to optimize choice-making using EF capacities predicts lower versatility to adapt to unexpected or new events, conflict resolution

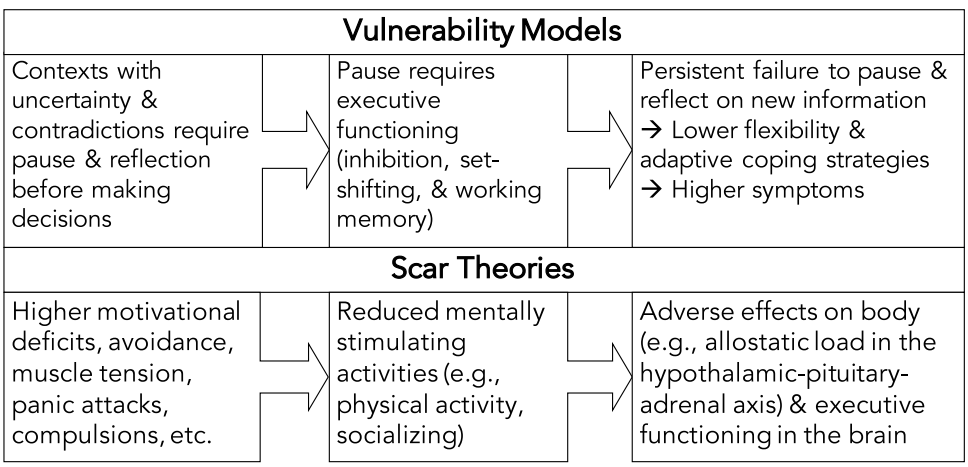

Fig. 1 Summary of vulnerability models and scar theories

deficits, and unhelpful action repertoires (e.g., avoidance, reassurance-seeking) in the long run [20]. Each of these is a typical attribute of anxiety and related disorders. Other theories posit that executive dysfunction could correlate with anxiety, OCD, and PTSD by hindering WM, inhibition, and various EF strategies to optimally respond to stressful events and disengage from social and non-social threats over lengthy periods [21].

Congruent with *vulnerability models*, evidence exists that EF and related cognitive dysfunction could be risk factors for subsequent anxiety-linked problems. Parent-reported behavioral inhibition tendencies in toddlers predicted more socially anxious behaviors (e.g., avoidance, reassurance-seeking) in middle childhood. Also, reduced set-shifting abilities mediated behavioral inhibition predicting future social anxiety [22]. In preadolescents (9–12 years), more executive dysfunction and processing speed problems notably predicted more behavioral and emotional issues across 2 years, above and beyond the baseline and demographic variables [23]. Similarly, after 9 years, community adults' performance-based difficulties with inhibition, set-shifting, WM, and inductive reasoning predicted more GAD symptoms and diagnosis [24]. Also, a recent qualitative review showed that baseline lower EF, WM, attention, verbal learning, and processing predicted more PTSD arousal and intrusion symptoms after 10 days to 6 months in victims of motor vehicle accidents and bushfires [25]. All of these studies controlled for outcome scores at baseline. Few published studies on vulnerability theories to date reported null effects [e.g., 26], and future quantitative syntheses should clarify whether this is true by determining if "file drawer" effects exist. Thus, there is ample evidence for the cognitive model of pathological worry and the iterative reprocessing model.

Scar Theories: Anxiety Constructs Predict EF-Related Deficits

Simultaneously, it is plausible that the experience of chronic heightened anxiety and related symptoms leads to future EF issues. *Scar theories* posit that elevated anxiety in the form of chronic avoidance, tension, panic attacks, compulsions, and posttraumatic stress could impede the practice, development, and maintenance of EF strategies across time [27]. For instance, long-term anxiety-driven excessive avoidance could decrease the opportunities to hone EF skills by reducing mentally stimulating activities, such as physical activity, and disengaging from socially rewarding hobbies and projects [28]. Moreover, the *resource allocation theory* asserts that attention issues, unhelpful thinking patterns (e.g., worry, social anxiety-related post-event brooding, obsessions, posttraumatic flashbacks), and higher anxiety symptom-linked distress could reduce finite EF resources across time [29].

Consistent with *scar theories*, much data indicates that between persons, anxiety and related issues could precede and predict future EF deficits across persons and diverse developmental stages, even after adjusting for outcomes at baseline. Parent- and self-rated anxiety, irritability, and hyperactivity predicted later attenuated neural responses in cognitive control-related brain regions (e.g., left cuneus) and issues with error monitoring, WM, and attention following 4 [30] to 14 years [31], over and above baseline EF indices. Furthermore, heightened anxiety, frequent abuse, and neglect predicted worse attention and EF deficits more potently among persons with low socioeconomic status in children and adolescents 7 to 33 years later [32]. In socioeconomically diverse adults, higher trait- and state-anxiety and posttraumatic stress symptoms independently predicted reduced EF and episodic memory across 6 months [33] to 18 years [34]. Similarly, using structural equation modeling (SEM), which reduces measurement error, more self- and parent-rated anxiety symptoms in early adolescence predicted WM issues after a few years in late adolescence, but not vice versa [35]. Moreover, subjective and performance-based EF deficits predicted future acute, potential, and sustained perceived threat characteristic of anxiety, OCD, and PTSD from adolescence to adulthood [36]. Likewise, among adults, within-person 9-year rise in pathological worry predicted a subsequent 9-year reduction in inhibition and set-shifting scores at the next time lag from midlife to older adulthood [37].

Furthermore, consistent with scar theories, recent narrative reviews and meta-analyses of adults with and without mild cognitive impairment consistently evidenced that all anxiety disorders functioned as independent distal risk factors for major neurocognitive disorders during midlife and late life [38], sometimes across ten or more years [39]. Data aggregated across four longitudinal studies showed that cognitively intact older adults with (vs. without) heightened anxiety symptoms had a 24% higher risk of all-cause dementia at follow-up above and beyond initial symptoms [40]. Also, baseline anxiety (vs. depression) symptoms independently predicted a 4- to 12-year decline in global cognition, processing speed, delayed verbal memory, and verbal fluency among Brazilian [41] and German [42] community older adults.

Consistent with scar theories, the pattern of results observed in community epidemiological studies generalizes to clinical and other distressed populations. This is the case even after adjusting for baseline individual differences. Anxiety-linked constructs predicted cognitive dysfunction in adult patients with traumatic brain injury [43], early multiple sclerosis [44], systemic lupus erythematosus [45], Parkinson's disease [28], and patients consuming medical cannabis [46]. Also, worse

PTSD symptoms predicted less efficient neurocognition among military veterans and Holocaust survivors, and alleviating PTSD reverses compromised EF and related cognitive dysfunction across lengthy periods (e.g., 1 to 7.6 years) [25, 47]. Likewise, Taiwanese patients with OCD (vs. healthy controls) were more likely to develop cognitive impairment, vascular dementia, and Alzheimer's disease over 18 years [48]. Thus, evidence for scar theories exists in the general population and clinical samples.

However, there is a dearth of studies investigating scar theories' *mediators* (or proxy mechanisms). Mediators refer to variables that intervene in the temporal pathway of anxiety-associated constructs predicting later EF issues. *Scar theories*, such as the *perseverative cognition hypothesis* [49], argue that prolonged heightened anxiety, worry, and related symptoms could adversely affect bodily systems by raising, accumulating, and sustaining high-stress hormones (e.g., cortisol, epinephrine), inflammation (e.g., proinflammatory cytokines), and related biomarkers over prolonged durations [50]. The buildup of allostatic load could negatively impact the hypothalamic–pituitary–adrenal axis (HPA) and EF-related brain networks and regions (e.g., dorsal prefrontal and anterior cingulate cortices, striatum, thalamus) [51, 52]. Allostatic load refers to the persistent buildup of chronic stress-induced wear-and-tear of the HPA and connected bodily systems [53]. Also, excessive worry and other repetitive thinking could dovetail with weakened right amygdala-ventromedial prefrontal cortex (vmPFC) connectivity and enhanced amygdala-ventral tegmental area (VTA)/striatum coupling in the long-term [49]. Notably, the VTA/striatum releases dopamine, a neuromodulator that correlates with motivational appetitive actions and processes underpinning anxiety and fear states and inhibitory control deficits over time [27]. Plausibly, inhibitory control and other EF issues coincide with aberrant dopamine signaling, and amygdala-VTA connectivity heightened anxiety symptoms across long durations [27, 49].

Some data consistent with the perseverative cognition hypothesis and related scar theories exist. Among community-dwelling young, midlife, and older adults, heightened plasma levels of proinflammatory markers (interleukin-6, C-reactive protein, fibrinogen) mediated the relation between excessive worry (but not panic symptoms) and reduced EF 2 to 18 years later in two separate samples [54, 55]. Dopaminergic brain activity mediated social anxiety and related symptoms predicting attention, set-shifting, and associated problems, particularly in infants and children [27]. However, elevated diurnal cortisol did not mediate the association between anxiety symptoms in young adulthood and EF, memory, and processing speed deficits in midlife among community adults [56]. An enhanced understanding of the *specific* neural, immune, and endocrine markers implicated in the relations among EF and anxiety-linked constructs can offer more rigorous tests of the perseverative cognition hypothesis and guide the design of empirically supported therapies.

Dynamic Mutualism Theory of EF- and Anxiety-Related Constructs

Furthermore, the *dynamic mutualism theory* argues that the associations between anxiety constructs and EF problems are *complex*, *multilevel*, and *bidirectional* [57]. Collectively, EF and related cognitive dysfunction could be risk markers that could compound one another long term to increase the severity of anxiety-associated constructs and vice versa throughout life. Testing the dynamic mutualism theory requires understanding between- and *within-person changes* across time in the relationship between cognitive dysfunction and anxiety-linked constructs. However, most prior studies used traditional statistics (e.g., ordinary least squares regression; OLS), latent growth curve, and cross-lagged panel models to understand the prospective relations between anxiety- and EF-related constructs. These methods inform us of *between*-person, but not *within*-person, bidirectional links over time [58]. OLS also violates the independence or error assumption as it does not adjust for the nesting of repeated assessments within persons over time [59]. Understanding within-person changes is necessary because, beyond individual differences (between-person level of analyses), within-person inferences constitute the heart of clinical science to develop *personalized* assessments and therapies [60]. Also, findings at the between- (vs. within-) person level could sometimes differ in magnitude and direction [61]. The dynamic mutualism theory posits that the inverse relations between anxiety and cognitive dysfunction constructs occur between *and* within persons over time. Moreover, data reliably showed diversity in the trajectories of anxiety and EF constructs [62], underscoring the importance of studying within- *and* between-person levels and changes in these variables over time.

Supporting the dynamic mutualism theory, greater anxiety symptom severity predicted future decreased parent-rated EF, and vice versa, at between- and within-person levels in a sample of 6- to 14-year-old children [63]. Such findings generalize to adults. Within-person higher-than-usual anxiety symptoms and COVID-19-related worries correlated with worse performance-based cognitive function across five time points in community adults [64]. Additionally, low (vs. high) social support accentuated the reciprocal links between EF deficits and posttraumatic stress symptoms [25]. Also, a bivariate dual latent change score (BLCS) analysis showed that an increase in sleep-related worries predicted declines in EF, planning, inhibition, and shifting scores among kindergarten children and vice versa [65]. Similarly, using BLCS, more previous worry symptoms forecasted sharper declines in WM and vice versa among adolescents [66]. Likewise, growth in worry tendency across a time lag coincided with decreases in verbal WM, processing speed, and spatial cognition at the adjacent time lag, and vice versa, in older adults aged 40 to 84 [61]. However, another BLCS analysis in a

distinct older adult sample aged 70 to 110 failed to observe within-person change-to-future change relations between anxiety and global EF [67].

Taken together, the dynamic mutualism theory may apply to unique anxiety constructs (pathological worry vs. non-specific anxiety symptoms) and specific samples (e.g., younger vs. older adults). These tenets of dynamic mutualism theory across human development are testable by harnessing BLCS and related techniques (e.g., random-intercept cross-lagged panel models; RI-CLPM) [68]. BLCS informs *change-to-future change within-person relations*, and RI-CLPM tells us *level-to-future level within-person connections*. They optimally separate between- and within-person variances at the population level, reduce measurement error, and adjust for regression to the mean and baseline scores, unlike widely utilized difference scores and residualized change score methods [69]. To this end, hierarchical linear models, BLCS, RI-CLPM, and related techniques are suitable for continually evaluating the above-stated tenets of scar, vulnerability, and dynamic mutualism theories of EF- and anxiety-related constructs.

Network Theory of EF- and Anxiety-Related Constructs

Recently, a network theory connecting anxiety and EF constructs emerged. This network theory asserts that EF- (vs. non-EF-associated) components would centrally and more potently relate to anxiety-linked constructs (e.g., fear, social anxiety) compared to other mental disorder symptoms within and across time [70]. As EF (vs. non-EF) deficits are more entwined with problems in meta-cognition and disengaging from threats, they more robustly interact with and reinforce specific anxiety (vs. non-anxiety related) constructs such as worry, avoidance, and intolerance of uncertainty [71]. Plausibly, theorized patterns of EF issues centrally connecting with anxiety constructs could occur within and outside of socially evaluative and performance-based contexts. Collectively, this *centrality hypothesis* guides treatment because prioritizing central components (e.g., alleviating anxiety constructs, enhancing EF) helps to alter factors maintaining chronic heightened anxiety [72].

To evaluate the network theory of EF- and anxiety-related constructs, regularized partial associations via cross-lagged panel network models (CLPN) [73] identify the components (or *nodes*) with the *highest centrality* vital to affecting and interacting with other EF- and anxiety-related nodes in the system. Network theory provides a more all-inclusive picture of anxiety disorders' risk factors and consequences. CLPN is almost identical to conducting various multiple regression analyses at the same time. However, CLPN has the comparative advantage of harnessing regularization methods to enhance the likelihood of removing weak or trivial node-to-node relations (*edges*) in the network (i.e., minimizing false positive edges). Also, CLPN (vs. SEM and other traditional statistics) reduces collinearity issues as regularization shrinks all coefficients toward each other and eliminates non-informative edges. Thus, CLPN enables testing scar and vulnerability theories simultaneously (e.g., iterative processing theory vs. perseverative cognition hypothesis) by involving more anxiety disorder and cognitive functioning nodes in a comprehensive network and detecting nodes with the strongest centrality. CLPN (vs. typical SEM) approaches do not assume local independence, so they are more informative of different node-to-node links across constructs within a web of nodes [74].

Supporting the network theory of EF- and anxiety-related constructs, issues with inhibition and WM (vs. other non-EF nodes) were central nodes that bridged across externalizing symptoms (e.g., impulsivity, irritability) and internalizing symptoms (e.g., anxiety) [75]. However, the cross-sectional design of this study precluded causal inferences. Only three prospective studies to date have examined EF-anxiety links using CLPN. First, among youths, decreased WM (vs. other nodes such as inhibition) and elevated irritability centrally predicted future internalizing symptoms (e.g., avoidance, anxiety) in attention-deficit hyperactivity disorder after one year [76]. Also, consistent with scar (vs. vulnerability) theories, higher somatic and anxiety symptoms (vs. depressed mood) more strongly predicted lower reduced WM and processing speed scores a year later, but not vice versa [77]. Somatic symptoms were the most central nodes impacting other future nodes (vs. vice versa) in this study which recruited an all-women middle-aged adult sample [77]. Likewise, among older adult men and women, prior anxiety and depression nodes (vs. other nodes such as agitation, apathy, and disinhibition) centrally negatively impacted subsequent EF (vs. non-EF) nodes [78]. However, a limitation of these studies is that none separated within- and between-person effects, which is necessary for the reasons stated above. Future studies with three or more time points could advance the network theory of EF and anxiety-related constructs by using CLPN methods that separate between- and within-person EF and anxiety processes [79].

Overgeneralized Control Model of EF- and Anxiety-Related Constructs

The literature is replete with evidence of substantial *inverse* links between anxiety- and EF-related constructs. However, it is also conceivable that higher anxiety levels can dovetail with *better* inhibition and associated EF domains for some subgroups and under specific settings. The *overgeneralized control model* of anxiety and EF postulates that trait anxiety and social anxiety symptoms correspond with *enhanced* reactive (vs. proactive) cognitive control capacities, particularly

for highly behaviorally inhibited children [80]. Relatedly, the *dual-mechanisms theory of cognitive control* distinguishes between two temporally unique and interrelated yet hugely independent cognitive control skills: reactive control and proactive control [81]. Whereas reactive control is the late-stage momentary enlisting of cognitive resources, frequently in response to conflict, proactive control refers to early-stage (preparatory) choice-making and persistence of goal-relevant information across time. Disproportionate inhibitory and reactive control can lead to anxiety-related freezing behaviors in social, evaluative, and non-social situations, especially for children with an anxious temperament [82].

Concordant with the overgeneralized control model, higher inhibition in toddlerhood predicted more anxiety symptoms 10 years later, particularly for children with worse proactive cognitive control [83]. Likewise, a recent study identified a subgroup of children for whom enhanced inhibition ability coincided with higher anxiety symptoms after accounting for prefrontal cortex-amygdala connectivity [84]. Similarly, greater trait and social anxiety corresponded with enhanced cognitive control capacities among youths after adjusting for their electroencephalography (EEG) brainwave patterns and temperament [85]. Also, the overgeneralized control model hypothesizes that some subgroups of individuals might show negative or non-linear (e.g., inverted U-shaped) connections between EF and anxiety constructs across time [27, 86]. Future studies should continue to test the assumptions of the overgeneralized control model by using appropriate person-centered advanced statistics. An example includes elucidating heterogeneous growth trajectories with group iterative multiple model estimation (GIMME) [87]. GIMME can determine group-, subgroup-, and within-person associations among cognitive functioning and anxiety constructs over time and provide more formal tests of the propositions of the overgeneralized control model.

Contextualizing EF- and Anxiety-Related Constructs Using EMA

Thus far, most studies testing vulnerability, scar, dynamic mutualism, network, and overgeneralized control theories of EF- and anxiety-related constructs used cross-panel data sets with time intervals spanning months and years. In addition to long durations, these theories propose that the relations among EF, anxiety, and related constructs can unfold across minutes, hours, and days, and shifts based on contextual factors (e.g., distractions, task-at-hand) [88]. They posit that anxiety and cognitive functioning constructs are trait-like *and* state-dependent [89]. A more granular test of these theories and understanding their state-dependent nature requires adopting ecological momentary assessment (EMA) and passive sensing strategies [90]. With high precision, these methods can delineate the contextual relations between specific events (e.g., social exchanges), location, avoidance, anxiety, worry, and cognitive dysfunction and refine existing theories. EMA and passive sensors can fine-tune our understanding of how cognitive dysfunction, worry episodes, panic attacks, avoidance, compulsions, and other anxiety-linked constructs relate to one another in daily life.

To date, only three studies have investigated the links among anxiety, cognitive function, and related constructs using EMA. Substantiating scar (vs. vulnerability) models, higher anxiety predicted a more significant emotional-Stroop inhibition effect in the next few hours, but not vice versa, among young and midlife adult smokers and non-smokers [91]. Likewise, increased EMA-based self-reported everyday stress correlated with less efficient WM task performance during the next moment, and older (vs. younger) age magnified this effect [92]. Relatedly, data collection using an EMA inhibition task on children and adolescents is underway and will shed light on the distinct day-to-day relations among anxiety, irritability, inhibition, and accompanying constructs in this population [93]. No studies thus far have used passive sensing data capturing technologies to understand the relations between EF- and anxiety constructs, rendering this a fruitful area to explore in clinical science. Future studies could also use cutting-edge techniques with EMA data, such as multilevel vector autoregressive regression (MLVAR), which can offer a fine-grained test of the abovementioned theories in everyday life [94].

Limitations and Future Directions

Two overarching limitations of the current literature merit attention. The present literature on this topic is saturated with cross-sectional studies, as reflected by a recent meta-analysis [14]. Cross-sectional studies are problematic as they preclude causal inferences without temporal precedence. Advancing our understanding of vulnerability, scar, dynamic mutualism, network, and overgeneralized control models and facilitating causal inferences necessitate the field to conduct more naturalistic longitudinal cohort studies. Ideally, longitudinal observational studies should include three or more time points to test the mediators (i.e., proxy mechanisms) of the complex associations between EF- and anxiety-related constructs. Second, there remains ample room to enhance understanding of *within-person trajectories* of EF- and anxiety-linked constructs and their associations to better elucidate idiographic (vs. nomothetic) change relations. Causality operates at the between-person *and* within-person level: Patterns that apply across persons in the population (i.e., between-person) do not necessarily contribute to anxiety-related symptoms and EF in a unique

person; changes that include that particular person do (i.e., shifts at the within-person level) [95]. Such efforts require developing neurocognitive tests with strong reliability at between- and within-person levels and good construct validity with scores on other ambulatory EF assessments [96]. They also call for using advanced statistics discussed throughout this review.

Conclusions

The current review highlights some important clinical implications. First, clinical science can benefit from testing how and which patients with anxiety and related disorders cognitive-behavioral therapy (CBT) could enhance neurocognition. Recent data showed that although 40 sessions of CBT did not alter cognitive function, the inconsistency between subjective and performance-based cognitive functioning observed in pre-treatment disappeared post-treatment for patients with major depressive disorder [97]. Second, alternative approaches to standard CBT may remediate cognitive functioning. An up-to-date meta-analysis showed the promise of mindfulness-based interventions to improve global EF [98], albeit in primarily healthy controls. Cognitive control training could be another option. Recent studies showed that adaptive WM training laden with neutral (vs. negative) materials could decrease perseverative thinking (e.g., worry, ruminative brooding) and enhance WM in young, healthy adults, and habitual worriers [99]. Also, randomized trial-based evidence is mounting that regular physical activity (e.g., 20–120 min 1–5 times a week), even in a non-structured way, could, over time, enhance EF- and memory-linked neural correlates and behavioral EF test scores for community and clinic populations [100]. Clinical science can profit from more gold standard prospective-observational treatment effectiveness studies and randomized controlled trials targeting EF and related cognitive functioning in anxiety disorders.

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Declarations

Conflict of Interest The authors declare no competing interests.

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