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Social anxiety disorder: looking back and moving forward

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Abstract

Fifty years have passed since social anxiety disorder (SAD) was first differentiated from other phobias. In the years since research has largely aligned with the zeitgeist of categorical classificatory frameworks, and has spanned identifying causes, maintenance factors and innovative interventions. Despite significant advances in the field, the capacity to conceptualise SAD as an independent entity is limited given the heterogeneity and dimensionality of diagnostic criteria, high rates of comorbidity, and non-specificity of aetiological mechanisms, maintaining factors and approaches to treatment. The Research Domain Criteria (RDoC) initiative was developed in an effort to overcome the inherent limitations posed by descriptive diagnostic systems – particularly in terms of reliability and validity – and in doing so seeks to facilitate research into underlying pathophysiological and behavioural mechanisms that cut across traditional diagnostic boundaries. The RDoC framework is furnished with a 'matrix', which in essence corresponds to a set of research principles that attempt to reconcile neuroscience and psychopathology. This review outlines a rationale for integrating SAD research with the RDoC approach, and offers examples of how future studies may wish to frame hypotheses and design experiments as the field moves towards classifying dimensions of psychopathology through a mechanistic understanding of underlying neurobiological and behavioural processes.

Overview

A half century has passed since social phobia entered the lexicon of psychiatry as an isolated disorder separable from specific phobias and agoraphobia, with Marks & Gelder (1966) first reporting on such differentiation. Since then, the concept of social phobia transitioned from being a relatively neglected condition (Liebowitz *et al.* 1985) to being identified as a highly prevalent disorder across the globe (Kessler *et al.* 2005*a*; Stein *et al.* 2017) – and is now referred to as social anxiety disorder or SAD (APA, 2013). As with all psychiatric conditions, however, diagnosis is made on the basis of presenting signs and symptoms, and no objective tests are available to support clinical judgements. In an attempt to tackle inherent problems of diagnostic subjectivity in psychiatry, the National Institute of Mental Health (NIMH) formed the Research Domain Criteria (RDoC) initiative, which aims to identify 'new ways of classifying mental disorders based on dimensions of observable behaviour and neurobiological measures' (NIMH, 2008). The RDoC seeks to organise research efforts across differing units of analysis for a range of domains/constructs, whilst acknowledging individual differences in environmental and developmental influences – and is by design a flexible framework insofar as it seeks to accommodate changes to its conceptualisation through integration of emerging knowledge. The RDoC has been heralded as welcome departure from existing diagnostic systems, which arguably lack validity and have hindered efforts in identifying causal mechanisms (Keshavan & Ongur, 2014), but has also been criticised on grounds that it has the potential to de-emphasise psychological, social and contextual factors (Lilienfeld, 2014). This review will briefly describe the history of SAD classification before discussing limitations of such categorical approaches. We then offer an approach for positioning SAD research within the emerging RDoC framework, highlighting the utility of leveraging specific research methods within this initiative towards developing novel classificatory systems.

The evolution of SAD

Historical descriptions of social anxiety – or related constructs (e.g. speech anxiety) – date back to Hippocrates, with Burton (1621) noting that Cicero '… trembled still at the beginning of his speech' (p. 261). Both Dugas (1898) and Hartenberg (1901) wrote about such anxieties, with the latter characterising *timideté* as 'a combination of fear, shame, and embarrassment felt in social situations, and which affected psychosocial competence through attacks ('*accès*') of fear' (Berrios, 1996, p. 273). Janet's (1903) concept of '*psychasthenia*' incorporated the features

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of social anxiety, and he is credited with coining the term 'social phobia' (Berrios, 1996). Whilst social anxiety symptoms were judged as part of the anxiety neuroses throughout the twentieth century (Berrios, 1996), the Japanese viewed the fear of facing other people ('*taijin kyofu-sho*') as separate from other phobias (Prince, 1993). The first and second editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM) (APA, 1952, 1968) allowed diagnosis of phobic reactions/phobic neurosis, but these were not specific to social fears. Social phobia was formally recognised as a separate disorder in DSM-III (APA, 1980), but this definition was viewed as too narrow to adequately capture all individuals with the disorder. For instance, a DSM-III diagnosis of social phobia could not be made if avoidant personality disorder (APD) was also present (Heimberg *et al.* 2014). This hierarchical rule was challenged (Liebowitz *et al.* 1985) prior to the release of DSM-III-R (APA, 1987), which subsequently included a 'generalised social phobia' category, to account for non-specific social fears. Social anxiety disorder became the primary naming convention in DSM-5 (APA, 2013). The International Classification of Diseases (ICD-10) (WHO, 1992) classifies social phobias alongside phobic anxiety disorders. Common to both nosologies is fear and avoidance of situations (e.g. giving a speech, attending social events) in which individuals believe they will be scrutinised or humiliated. A 'performance-only' specifier can be applied in DSM-5, but the utility of this subtype remains unclear (D'Avanzato & Dalrymple, 2016) given most individuals with diagnosable SAD report fearing more than one situation (Stein *et al.* 2000).

It is evident that the desire for meaningful classification continues in psychiatry, particularly given its utility in guiding treatment selection, or indeed whether to treat at all (Craddock & Mynors-Wallis, 2014). However, there are significant limitations to pursuing SAD as a categorical entity on the basis of phenomenology alone. There is mounting evidence that (i) diagnostic criteria for SAD are polythetic, contributing to diagnostic heterogeneity, (ii) its core features exhibit significant dimensionality (i.e. symptoms are also present, albeit to varying degrees, in non-clinical individuals), (iii) there are high rates of comorbidity in SAD (which is likely related to high correlations between phenotypes), and (iv) whilst aetiological, maintenance and treatment models have been proposed as specific to SAD, they can often be applied across diagnoses (i.e. exhibit transdiagnosticity).

Limitations of the categorical approach to SAD

Polythetic categorical diagnostic frameworks and within-category heterogeneity

The DSM and ICD frameworks are polythetic, referring to the fact that diagnostic categories are defined by multiple symptoms, and not all symptoms are required for a diagnosis. Accordingly, different combinations of symptoms can lead to the same diagnosis (Krueger & Bezdjian, 2009). Whilst all symptoms are required for a SAD diagnosis in DSM-5 (APA, 2013), within-item criteria are polythetic. Criterion A for SAD in DSM-5 requires marked fear or anxiety about *one or more* social situations, which can vary widely in context. Criterion B relates to fear of negative evaluation, yet the core fear can also vary across individuals. As an example, one individual may have significant fear of being observed eating, believing it will lead to negative evaluation and embarrassment, whilst another may fear conversing in groups as it may lead to rejection, yet both may be diagnosed with SAD (assuming other criteria are also met). Hence, whilst SAD is positioned in DSM (and ICD) as a distinct categorical entity, there is clear heterogeneity in symptom presentations across individuals. As described above, a common distinction historically has been between 'generalised' and 'non-generalised' SAD. The only subtype specifier in DSM-5 (APA, 2013), namely performance fear, is said to be qualitatively distinct from generalised SAD given its later age-of-onset, less shyness and behavioural inhibition, no familial relationship, and stronger psychophysiological responses (Bogels *et al.* 2010). Yet the core fear – of (negative or positive) evaluation – is observed across subtypes (Bogels *et al.* 2010; Weeks *et al.* 2009), suggesting that similar cognitive processes may be operating.

The heterogeneity of SAD is further apparent when considering closely related constructs. For instance, distinguishing 'shyness' from SAD can be challenging given shyness is frequently an antecedent and central feature of the clinical syndrome (Bruch, 1989). Whilst SAD and shyness have been differentiated in terms of symptoms experienced, functional impairment and quality of life (Heiser *et al.* 2009), others emphasise that they exist on a continuum (Chavira & Stein, 1999), with SAD being a form of extreme shyness (Stein, 1999). Of course not all shy individuals meet criteria for SAD, and hence this trait may more be a vulnerability factor for the disorder and indeed other forms of psychopathology (Heiser *et al.* 2003). Thus, whilst shyness can exist independently of SAD, is also closely intertwined with the phenomenology of the disorder. There is also substantial diagnostic overlap between SAD and APD (APA, 2013). Turner *et al.* (1991) found that 75% of those with SAD also met criteria for APD. Despite their similarities, Turner *et al.* (1992) demonstrated that those with APD and generalised SAD had more severe social anxiety and impaired social functioning compared with those with 'pure' generalised SAD, consistent with the view that APD may be a more severe expression of SAD (Herbert *et al.* 1992). Differentiating shyness from SAD, or SAD from APD, thus often creates a diagnostic dilemma, namely in how to categorise SAD from 'normative' but perhaps inhibited levels of social functioning (shyness), and more pervasive and 'severe' expressions of social avoidance and interpersonal sensitivity to negative evaluation.

Dimensionality of core features

Social anxiety has also been conceptualised within a dimensional framework (Merikangas *et al.* 2002; Ruscio, 2010), which acknowledges the importance of differences amongst those who fall at either side of some – often arbitrarily set – categorical diagnostic threshold (Helzer *et al.* 2006). Weeks *et al.* (2009) revealed that the core cognitive features of SAD – fear of negative and positive evaluation – more reflect a dimensional, rather than taxonic, latent structure. That is, these features are not specific to SAD and also manifest in non-clinical individuals. Moreover, such symptoms are suggested to be underpinned by low approach and high avoidance temperamental variables (Rodebaugh *et al.* 2017), which themselves exhibit dimensionality (Roth & Cohen, 1986). Huppert *et al.* (2003) also revealed that negative social interpretation biases are positively correlated with social anxiety symptoms, suggesting that biased information processing may also be dimensional in nature. A caveat of such studies is that they were conducted with non-clinical samples, limiting generalisation to clinical populations. However, Ruscio (2010) indicated that DSM-IV diagnostic features of SAD appear to exist on a

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continuum with less severe social anxiety symptoms, and thus shifting from categorical to dimensional classification models may improve the predictive validity of the diagnosis. Conversely, Weeks *et al.* (2010) identified a SAD taxon using symptomatic indicators in a community sample, providing support for the utility of categorical diagnosis. Given the contrasting findings, it is difficult to draw conclusions as to whether SAD is best represented dimensionally or categorically, or indeed a combination of both. In spite of the lack of consistency across studies, it has been suggested that the dimensional approach may afford greater utility over categorical models for refining understanding of the mechanisms of disorders such as SAD (Sanislow, 2016).

The main function of the dimensionally defined behaviours and cognitions noted above is (perceived) harm avoidance, in line with Beck's original formulation (Beck *et al.* 1985). Continued engagement with avoidance behaviours is believed to maintain threat appraisals in SAD (Clarke & Wells, 1995; Bögels & Mansell, 2004), but also panic disorder (Salkovskis, 1991), and generalised anxiety disorder (GAD) (Borkovec *et al.* 2004). Of importance, the type of threat is somewhat specific to differing disorders (e.g. attentional bias towards social cues in SAD *v.* physiological sensations in panic disorder – the caveat here being that biases seemingly isolated to a specific disorder may also occur in other conditions). Hence, a useful distinction for future research may be to investigate dimensional aspects of avoidance and threat appraisal across disorders, giving rise to transdiagnostic 'functions', whilst acknowledging the importance of the 'form' (content) of differing processes for specific disorders. This dimensional, transdiagnostic approach to SAD is particularly relevant given it is frequently comorbid with other disorders.

Comorbidity and correlated phenotypes

SAD is frequently comorbid with other anxiety disorders including GAD, panic disorder, agoraphobia, as well as major depression, dysthymia, and substance and alcohol abuse in both epidemiological (Schneier *et al.* 1992; Kessler *et al.* 2005*b*) and treatment-seeking samples (Brown *et al.* 2001). It has been argued, however, that 'comorbidity' is an artefact of categorical diagnostic nosologies that do not accurately account for symptom heterogeneity (Maj, 2005). In line with this, it has been suggested that disorders such as SAD may be better conceptualised as part of a broader structure, where correlated features amongst disorders is emphasised (Eaton *et al.* 2010).

Latent variable modelling has been used to examine underlying structural relationships between differing conditions. Such analyses indicate that many disorders (e.g. anxiety disorders, unipolar mood disorders, substance use disorders) can be grouped together under two or three dimensions reflecting either internalising or externalising psychopathology. For example, Krueger (1999) demonstrated that SAD was part of a broad internalising factor along with simple phobia, agoraphobia and panic disorder (also constituting a 'fear' subfactor in a three-factor model), as well as major depression, dysthymia and GAD (these latter conditions comprising an 'anxious-misery' subfactor in a three-factor model). The internalising construct has recently been incorporated into a new hierarchical classification model (Kotov *et al.* 2017) – the Hierarchical Taxonomy Of Psychopathology (HiTOP) – as a 'spectra' (consisting of constellations of syndromes). These spectra are said to be influenced by a higher order general psychopathology dimension (e.g. *p* factor; Caspi *et al.* 2014). The constructs of fear and distress are subfactors of the internalising spectra, which then relate to the expression of syndromes and disorders. Fear components within the internalising spectra are said to include features such as interactive anxiety, performance anxiety, situational phobia and trait anxiety. Such structural models highlight the non-orthogonality of conditions such as SAD, and position the disorder as just one form of maladjustment where distress is internally focussed. Together, such findings stress the need for refining how we study disorders such as SAD, particularly in terms of whether we continue to conceptualise and treat the condition as if it were a distinct entity.

Non-specificity of aetiological and maintaining factors and treatment approaches

Cognitive models of SAD (Clarke & Wells, 1995; Rapee & Heimberg, 1997; Hofmann, 2007; Moscovitch, 2009; Heimberg *et al.* 2010) state that those with the condition engage in maladaptive cognitive and behavioural strategies (e.g. dysfunctional anticipatory processing, negative social-evaluative cognitions, avoidance and escape behaviour) prior to, during, and following perceived social evaluative situations, which in turn maintains the disorder. Whilst these models have been invaluable in providing an elemental account of the disorder's maintaining factors, it is beneficial to detail their antecedents. Beck & Clark (1988) proposed an information processing (*cf.* cognitive) theory of anxiety and depression, following Beck's (1967) original cognitive–behavioural formulation of depression. In essence, the maintaining factors of SAD correspond to three cognitive–behavioural factors, namely, (i) products, (ii) processes and (iii) schemas/core beliefs. The central element, 'processes', refers to cognitive processes (e.g. attentional/memory biases) that influence the appraisal of situations (e.g. threat misappraisal). The 'products' of such processes may manifest as negative automatic thoughts and/or avoidance behaviours. These products can also influence cognitive processes, given their bidirectional relationship. At the bottom of the hierarchy lie schemas/core beliefs, which influence an individual's view of self, others and the world. Such models have also been applied to GAD, obsessive–compulsive disorder and psychosis (Beck *et al.* 1985; Beck & Rector, 2005), and hence represent a generic framework for understanding mechanisms of emotional disorders more broadly.

The search for aetiological mechanisms has largely proceeded in alignment with categorical diagnostic frameworks (Spence & Rapee, 2016). Different risk factor combinations (e.g. genetics, neurobiology) may result in development of SAD ('equifinality'), but importantly any given risk factor may be associated with other disorders ('multifinality'). This latter concept is at the heart of transdiagnostic models of psychopathology (Nolen-Hoeksema & Watkins, 2011). For instance, Spence & Rapee (2016) highlight overly controlling/intrusive parenting as contributing to inhibited temperament in children – and thus risk for the disorder – but note these parenting styles are not unique for increasing risk of SAD. Indeed, such parenting may contribute (or be a response) to a more general trait anxiety early in life (Negreiros & Miller, 2014). Spence & Rapee (2016) further note that adverse/stressful life events and trauma increase risk for developing SAD. However, these factors again lack specificity to the condition. It is hence increasingly apparent that the field should embrace novel approaches to elucidate the differing influences of transdiagnostic *v.* disorder-specific aetiological factors (Spence & Rapee, 2016). In keeping with this, Lahey *et al.* (2017) proposed an evidence-based taxonomy of psychopathology based on shared and unique

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causal factors across differing first-order dimensions (i.e. latent constructs defined by correlations amongst symptoms). Their hierarchical model states that, (i) some genetic and environmental variables increase risk across all first-order dimensions, (ii) there are non-specific causal influences across higher order dimensions (e.g. internalising psychopathology) and (iii) some causal influences are specific to different first-order dimensions, and possibly even subsets of symptoms. This highlights that both specific and non-specific genetic and environmental factors contribute to psychopathology, which has important implications for whether SAD can continue to be conceptualised as discrete condition.

There has been an active search for neurobiological correlates of SAD, but as with all psychiatric conditions findings have been largely non-specific. Although the field has not yet identified specific neuronal biomarkers with prognostic value in isolated disorders including SAD (Linden, 2012), it is possible that methodological limitations have obscured identification of casual mechanisms (Poldrack & Farah, 2015). Advances to neuroimaging technologies are likely to allow unprecedented insight into multilevel systems (e.g. molecular, cellular, neural populations and circuits), which may assist with identification of neural substrates of psychopathology. For example, whilst the amygdala was broadly identified in early imaging studies as a canonical brain region involved in the pathophysiology of SAD, it has also been implicated in the expression of other anxiety disorders (Etkin & Wager, 2007), mood disorders (Drevets, 2003), psychosis (Velakoulis *et al.* 2006) and borderline personality disorder (Herpertz *et al.* 2001). The field has subsequently narrowed its focus, and recent work suggests that the 'extended amygdala' – the bed nucleus of the stria terminalis (BNST) – may be more pertinent to anxiety disorders (Lebow & Chen, 2016). The BNST is believed to process less specific, long duration threat information and hence may contribute to sustained anxiety rather than fear (i.e. phasic anxiety), aligning with the view that anxiety disorders be recast as disorders of vigilance (Davis & Whalen, 2001). In contrast, the central amygdala appears more implicated in fear responding than sustained anxiety. It may hence be plausible that amygdala reactivity corresponds to a latent vigilance factor across anxiety disorders, and possibly even across the spectrum of psychiatric conditions. In line with this, grey matter loss in anterior cingulate and insula cortices has been identified as a common factor across diagnostic groups including SAD (Goodkind *et al.* 2015). Furthermore, whilst genetics influence SAD (Hettema *et al.* 2001), it has been difficult to identify genetic markers at both the disorder and endophenotype level given the heterogeneity of diagnostic features and multiple genetic variants involved (Flint & Munafo, 2007).

In terms of treatment, cognitive–behavioural therapy (CBT), particularly individual-based, has the strongest evidence base, compared with other psychotherapies (Mayo-Wilson *et al.* 2014). CBT for SAD will usually involve a client and therapist working together to target and modify maladaptive cognitive processes (e.g. cognitive restructuring), and dysfunctional behavioural patterns (Rodebaugh *et al.* 2004). Whilst CBT is effective for SAD in helping to modify disorder-specific beliefs around social threat – suggesting a degree of specificity – very similar principles and strategies are used with good effect across psychiatric conditions (Hofmann *et al.* 2012). Consequently, transdiagnostic CBT approaches have been developed towards the ultimate goal of a 'unified treatment for emotional disorders' (Barlow *et al.* 2004). Studies have shown that unified treatments perform better than waitlist across comorbid disorders, and may be comparable to disorder-specific treatments (McEvoy *et al.* 2009; Norton & Barrera, 2012). Although consensus has not been reached on whether unified CBT approaches can be offered in replacement of disorder-specific treatments, evidence points towards the former being complementary to the latter (Rector *et al.* 2014). A similar story emerges for the pharmacological treatment of SAD. Both phenelzine and tranylcypromine are effective agents for SAD despite being developed for depression (Sareen & Stein, 2000). A meta-analysis of placebo-controlled studies revealed that phenelzine had the largest effect size for reducing SAD symptoms (Blanco *et al.* 2003). The same meta-analysis reported a very large effect size for clonazepam (used for numerous other psychiatric disorders), and found selective serotonin reuptake inhibitors (SSRIs) to be effective in treating SAD (Blanco *et al.* 2003). The SSRIs were of course developed initially as 'antidepressants' but are broadly effective for anxiety disorders (Baldwin *et al.* 2005). Hence, despite their effectiveness in SAD, the psychotherapeutic and drug treatments offered are not disorder specific, which likely reflects the heterogeneity of conditions being treated (Cuthbert & Insel, 2013).

Despite efficacious treatments for SAD, it remains unclear why a significant number of individuals do not respond to first-line psychotherapeutic or pharmacological interventions. Inaccurate diagnosis, and subsequent inappropriate treatment, has been suggested as one reason for suboptimal outcomes (Stein & Stein, 2008). New approaches to classification, such as that put forward by the RDoC framework, may hence be of benefit in eventually optimising treatment for SAD. Should the field be successful in validating novel domains through integration with neuroscience, it may be possible to develop and select more precise treatments – in essence, 'precision medicine' for psychiatry (Insel, 2014).

Overcoming limitations posed by categorical psychiatry: embracing RDoC

Despite the limitations detailed above, the last 50 years of research into SAD has dramatically increased understanding of its aetiology, maintenance and treatment. However, there is a need for further clarifying such factors, and it is apparent that the field may benefit by aligning with emerging frameworks that seek to link observable aspects of brain and behaviour with symptom expression (Insel, 2014). This constitutes a significant change for psychiatry, with the initial litmus test being whether the field is willing to abandon the status quo of polythetic-categorical models. Even still, embracing RDoC does not necessarily spell the end for SAD as a clinically useful construct. Instead, utilising the enormous body of research that has identified common and shared factors for SAD, in conjunction with novel methodological approaches into pathophysiological mechanisms, may aid discovery of more precise models for optimal treatment selection and prediction. For instance, computational models (e.g. generative embedding) applied to neuroimaging data have offered insight into schizophrenia subtyping (Brodersen *et al.* 2014). Such approaches allow for pathophysiologically informed separation of psychiatric disorders (Stephan *et al.* 2009), and may ultimately help inform diagnosis and treatment. Psychiatry is hence equipped with a rich set of methods (in terms of acquisition and analysis of physiological parameters), affording it with a unique opportunity to examine the clinical utility of defining disorders by underlying pathophysiological mechanisms.

The RDoC comprises a two-dimensional matrix spanning domains/constructs and units of analysis – so designed as adaptable constructs, thus enabling incorporation of evolving

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developments across fields – which are intended to guide research endeavours towards developing a more mechanistic understanding of mental illnesses (Cuthbert, 2014). A central tenet is in identifying the normal distribution of functioning, with relative deviations to this perhaps assigned disorder or disease status. This overcomes issues faced by DSM/ICD regarding whether disorders are categorically different from normal or whether they vary on a continuum. As Cuthbert (2014) highlights, '… many paradigms have been developed that can provide measures of both behavioural performance and of related functional brain activity in a large population, thus providing some sense of the normal distribution' (p. 31). As psychiatry moves to integrate with the RDoC framework, it has been recommended that studies recruit as broadly as possible (spanning normative to impaired) across the constructs of interest. It is conceded, however, that the RDoC will first be tested in convenience samples of individuals who will likely meet criteria for disorders under current diagnostic systems (Cuthbert, 2014). For a true representation of dimensionality, it will be important to study individuals with a variety of other psychopathologies, from mild to severe, as well as incorporate those with normative functioning. For example, healthy individuals (with no frank psychopathology) should be recruited alongside those with other anxiety disorders (e.g. GAD, panic disorder and agoraphobia), mood disorders, personality disorders and psychotic disorders. Less 'severe', yet still mildly impairing, psychopathologies (including, but not limited to, adjustment disorder) should also be included in the search for aetiological and maintaining factors across the RDoC matrix.

How might existing SAD research inform the RDoC matrix?

Two constructs under the RDoC negative valence systems domain, namely 'acute threat ('fear')' and 'potential threat ('anxiety')', parallel a range of SAD features. The former, 'acute threat' is defined as 'activation of the brain's defensive motivational system to promote behaviours that protect the organism from perceived danger … [involving responses to conditioned or unconditioned threat stimuli]'. The latter, 'potential threat', is defined as 'activation of a brain system in which harm may potentially occur but is distant, ambiguous, or low/uncertain in probability … [characterised by high vigilance to low imminence threat]' (NIMH, 2017). A key feature of SAD, fear of negative evaluation, may hence be conceptualised across the acute threat ('fear') and potential threat ('anxiety') constructs. The former is particularly salient in light of evidence linking fear of negative evaluation in SAD with conditioned fear (startle) responses (Lissek *et al.* 2008). Fear of evaluation may also be studied under the sustained threat construct (also under negative valence systems), as well as within the cognitive systems domain (e.g. particularly attentional biases). The behavioural avoidance and safety behaviours commonly seen in SAD have previously been formulated as reflecting efforts to circumvent feared outcomes (Hofmann, 2007), and may be best operationalised across both the acute threat and potential threat domains in RDoC. It may also be useful to examine features of SAD under RDoC domains other than negative valence systems, for example, across the social processes and positive valence systems domains. The 'affiliation and attachment' construct under the former encompasses social information processing (e.g. of social cues), and the behavioural and physiological consequences of disrupted social relationships. This is particularly relevant given cognitive models suggest that those with SAD misinterpret ambiguous social cues (Clark & Wells, 1995). There is likely to be utility in examining constructs such as 'approach motivation' within the positive valence systems domain, reflecting mechanisms and processes that modulate maintenance of approach behaviour, which often manifests as disrupted in SAD via behavioural avoidance (Rapee & Heimberg, 1997).

An essential component of the RDoC is to extend established cognitive and behavioural features, including fear of evaluation, avoidance and safety behaviours, to other conditions. Specifically, the field may seek to further understand these features through the lens of RDoC 'perceived threat' across those who may typically be diagnosed with mood and psychotic spectrum conditions and even eating disorders – the latter two also being associated with fear of negative evaluation (Gilbert & Meyer, 2005; Kinoshita *et al.* 2011). Within any of the RDoC constructs, it will be critical to move beyond descriptive (e.g. self-report) features, and examine the role of other units of analysis, such as predisposing environmental factors, potential cellular/molecular/brain circuitry contributions, as well as physiology (e.g. psychophysiology) and observable behaviour (e.g. neuropsychology). Fear of negative evaluation, and avoidance/safety behaviours, are just a few examples of what is likely to be many (especially when transdiagnostic factors are examined), but nonetheless offer a useful starting point for reframing our thinking as RDoC-inspired programmes of research gain traction.

The basic elements of RDoC-inspired research

The RDoC was proposed as a framework to better understand how basic dimensions of functioning (from genetics through neurobiology to self-report) underlie the range of human behaviour from normal to abnormal (Insel, 2014). The operationalisation of these criteria across differing units of analysis will provide a unique opportunity to understand dimensions of differing psychological constructs (e.g. acute threat, potential threat) using a range of methods from neuroscience (molecular, neurocircuitry, behaviour) (Cuthbert & Insel, 2013). The RDoC matrix details five domains, each with a range of subconstructs, which reference specific psychological processes. The negative valence systems domain has five sub-constructs across seven units of analysis, giving rise to a 5×7 matrix. Specific research methods, or 'elements', are noted within cells across differing matrices. For instance, the RDoC details a range of elements or factors as potentially contributing to the acute threat ('fear') subconstruct. These include differing molecular (e.g. dopamine, glutamate, vasopressin) and cellular (e.g. neuronal, glial) targets, brain circuitry (e.g. ventromedial prefrontal cortex, medial/lateral/basal amygdala), physiology (e.g. skin conductance, heart rate), behaviour (e.g. response inhibition, social approach) and self-report (e.g. quantifying distress) variables, which may be measured during experimental manipulations such as the Trier Social Stress Test (Kirschbaum *et al.* 1993). In some cases, these elements are also implicated in other sub-constructs or even differing higher level systems; for example, vasopressin is also included in the 'loss' and 'frustrative non-reward' sub-constructs of negative valence systems, and in the 'affiliation and attachment' sub-construct of the social processes domain. In contrast, the (medial/lateral/basal) amygdala is proposed as specific to the acute threat ('fear') construct. Ultimately, the specificity of differing elements to various domains will be borne out by future research, albeit each with a finite level of resources through which to test various translational hypotheses. Importantly, the RDoC matrix does not reflect an exhaustive list of domains, constructs and elements, but is rather a dynamic blueprint allowing integration of

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methodological and conceptual advances, and hence offers an innovative framework through which to reconsider disorders such as SAD (Cuthbert, 2014).

Whilst the RDoC may assist in contributing to a reconceptualisation of psychiatric illnesses, it has been criticised for being overly 'neurocentric' by principally regarding mental symptoms as emergent phenomena of disrupted (neuro)biological processes (Lilienfeld, 2014). However, such a view – that maladaptive psychological states emerge from aberrant neurobiological systems – aligns with scientific evidence that indeed all subjective experience is dependent on brain functioning (Kendler, 2005). Notwithstanding this, it has been noted that RDoC-inspired investigations should seek to differentiate between neurobiological aetiology and mediation, in essence acknowledging environmental influences on brain function. Additionally, researchers should seek to minimise specific situational factors (e.g. time of day of testing, nature of laboratory setting) that contribute to low cross-situational and temporal consistency (Lilienfeld, 2014). Further, it will be important to clarify the relative contribution (e.g. in terms of heritability) of endophenotypes and intermediate phenotypes over traditionally studied exophenotypes (comprising signs and symptoms). Indeed, whilst some have argued that the RDoC oversimplifies the complexity of psychopathology (Wakefield, 2014) by ignoring the richness of disorder or descriptive psychopathology (rather than of *dis*ease – by definition, that arising from pathology), the approach arguably offers a tenable alternative, and perhaps a solution, to the limitations inherent in the DSM/ICD approaches to classification. Spence & Rapee (2016) note – in overviewing aetiological factors in SAD – the difficulties associated with any identified risk factors being transactional and reciprocal, such that there is a dynamic interplay between expression/emergence of symptoms/vulnerability and eventual disorder onset. Providing answers to these questions will take time, but it is hoped that current and future research endeavours will lead to further advances in identifying susceptible individuals, and classifying, treating and even preventing SAD, if such a category remains over the ensuing years.

Conclusions

In the 50 years that have passed since Marks & Gelder's (1966) formative paper the social anxiety literature has proliferated and now spans classification and diagnosis, cognitive and neurobiological mechanisms underlying maintenance, aetiological processes, and both psychological and psychopharmacological treatments. SAD has since emerged as a highly prevalent condition, and whilst the theoretical models that have been described offer hope towards improving and enhancing treatments for the condition, it remains difficult to achieve full symptom resolution, and indeed longer term remission, in such patients. The future of SAD will largely be dependent on research efforts of psychiatry as a whole, and may hinge on what the RDoC framework delivers. Will future treatment interventions be focused on first identifying and targeting transdiagnostic pathophysiological aberrations, followed by specific focus on symptoms unique to the condition? Let us hope the next 50 years are as fruitful as the last.

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