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A Place for Sexual Dysfunctions in an Empirical Taxonomy of Psychopathology

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Abstract

Sexual dysfunctions commonly co-occur with various depressive and anxiety disorders. An emerging framework for understanding the classification of mental disorders suggests that such comorbidity is a manifestation of underlying dimensions of psychopathology (or “spectra”). In this review, we synthesize the evidence that sexual dysfunctions should be included in the empirical taxonomy of psychopathology as part of the internalizing spectrum, which accounts for comorbidity among the depressive and anxiety disorders. The review has four parts. Part 1 summarizes the empirical basis and utility of the empirical taxonomy of psychopathology. Part 2 reviews the *prima facie* evidence for the hypothesis that sexual dysfunctions are part of the internalizing spectrum (i.e., high rates of comorbidity; shared cognitive, affective, and temperament characteristics; common neural substrates and biomarkers; shared course and treatment response; and the lack of causal relationships between them). Part 3 critically analyzes and integrates the results of the eight studies that have addressed this hypothesis. Finally, Part 4 examines the implications of reconceptualizing sexual dysfunctions as part of the internalizing spectrum, and explores avenues for future research.

Keywords

Sexual dysfunctions; depression; anxiety; internalizing; nosology; hierarchical taxonomy of psychopathology; female sexual interest/arousal disorder; female orgasmic disorder; pelvic pain disorder; hypoactive sexual desire disorder; erectile dysfunction; premature ejaculation

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Introduction

Symptoms of sexual dysfunctions[1]—or low sexual function— broadly include low or absent sexual desire, difficulty becoming and/or staying sexually aroused, difficulty attaining orgasm, or experiencing rapid orgasm, and sexual pain (American Psychiatric Association [APA], 2013). Low sexual function is not only common —with an estimated prevalence of about 40–45% for women and 20–30% for men (Lewis et al., 2010)— it is often associated with marked personal distress (Hayes et al., 2008; Lewis et al., 2010), relationship distress (e.g., Bossini et al., 2014), low self-esteem (e.g., McCabe & Althof, 2014), and decreased quality of life (e.g., Bossini et al., 2014; Laurent & Simons, 2009). Despite this, low sexual function is rarely considered as an indicator of psychological functioning beyond the specialized field of sex research.

While sexual dysfunctions are independently associated with negative outcomes, they also commonly co-occur with emotional disorders, such as depressive and anxiety disorders (Laurent & Simons, 2009) and this comorbidity can have a particularly detrimental impact. For example, comorbidity between sexual dysfunctions and emotional disorders is associated with higher rates of suicidality, as well as increased chronicity and severity of the disorders, relative to either cluster of disorders alone (e.g., Dell'Osso et al., 2009; Michael & O'Keane, 2000; Rajkumar & Kumaran, 2015). When the disorders are not treated together, this comorbidity is also associated with worse long-term treatment outcomes, such as higher dropout, poorer response, and poorer adherence (e.g., Bossini et al., 2014; Dobkin, Leiblum, Rosen, Menza & Marin, 2006; Laurent & Simons, 2009). Given their strong negative impact when combined, more research is needed to understand the nature of the relationships between sexual dysfunctions, emotional disorders, and psychopathology more broadly. Characterizing the nature of these relationships is key to gaining insight into low sexual function, and is a prerequisite for understanding the mechanisms and processes that underlie comorbidity, which is necessary for effective diagnosis and treatment.

In this review, we propose that while there has been limited research that has explicitly addressed the question of how and where sexual dysfunctions fit into the bigger picture of psychopathology, a synthesis of the extant literature suggests we may already have an answer. Some readers may be skeptical about integrating sexual dysfunctions into a model of psychopathology because variations in sexual response are not necessarily indications of pathology within a person; rather, they can be normative or adaptive reactions to current circumstances, such as a particularly stressful period or relationship difficulties (Bancroft et al., 2003b). However, when low sexual functioning persists without a clear cause (e.g., not due to a non-sexual mental disorder, substance misuse, a medical condition, inadequate sexual stimulation, or severe partner distress; APA, 2013), it can become problematic. For example, when these experiences cause distress in the form of worry, anxiety, or disruption to nonsexual aspects of a relationship, there is a need for support and/or intervention (Balon,

[1]Symptoms of sexual dysfunctions —as delineated in the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*)— are collectively referred to as low sexual function throughout this review. This label is used to differentiate between diagnosed sexual dysfunctions with associated clinically significant distress, and the measurement of symptoms of these disorders without a formal diagnosis (cf. Bancroft, Loftus & Long, 2003b; Lutfey, Link, Rosen, Wiegel & McKinlay, 2009). Where diagnosed disorders (i.e., with accompanying distress) are referred to, the term "sexual dysfunctions" is used to correspond with the *DSM*.

Segraves & Clayton, 2007). Indeed, this is how the *Diagnostic and Statistical Manual for Mental Disorders* (*DSM*) defines clinically significant distress, and delineates the threshold on the range of sexual experience that distinguishes between 'normal' and 'disordered' sexual function (Stein et al., 2010)[2].

Rather than stigmatizing experiences of low sexual function as 'mental disorders,' or endorsing the DSM's putatively narrow, categorical, and prescriptive definitions of sexual experience (cf. Moynihan, 2003; Tiefer, 2002), our review aims to elucidate an *empirically supported* understanding of the psychological aspects of sexual function in a descriptive psychopathology framework. That is, we aim to gain new insight by synthesizing current knowledge of the empirical relationships between the features of experiences like depression, anxiety, and low sexual function as they co-occur, which can clarify the path to understanding their etiologies. This approach does not rely on a reductive view of sexuality by defining it as a purely psychological experience; rather, it allows for the roles of many other processes involved in the profoundly complex experience of sex and sexuality (e.g., biological and medical vulnerabilities, social context, and environmental factors), which are often conceptualized in a biopsychosocial framework (e.g., Black, 2005; Tiefer, 2002; Wincze & Weisberg, 2015).

Our review has four parts: In Part 1, we briefly explore current thinking about the structure of psychopathology, and describe the current structural model that has been derived from the systematic patterns of comorbidity among mental disorders. In Part 2, we review the *prima facie* evidence supporting the formation of the hypothesis that sexual dysfunctions should be included in this structure as part of the internalizing spectrum. In Part 3 we review the preliminary studies that have addressed this hypothesis. Finally, in Part 4 we discuss the implications of this idea and avenues for future research.

Part 1: Current thinking about the structure of psychopathology

Common mental disorders are significantly correlated and have consistently been found to co-occur at higher rates than would be expected by chance (Kessler, Chiu, Demler & Walters, 2005; Kessler et al., 1994). These patterns of comorbidity between mental disorders have been an ongoing difficulty for psychopathology research since the release of the third edition of the DSM because they highlight the lack of distinction of disorders as diagnostic categories (Kessler et al., 2005). To learn how we can best conceptualize these patterns of co-occurrence among mental disorders, we need to understand how and why disorders covary. One way to understand comorbidity among a group of disorders is to determine which statistical models offer the strongest explanation of empirical data that document the comorbidity (Krueger & Markon, 2006).

[2]In this review, mental disorders (including sexual dysfunction) are conceptualized as syndromes indicated by groups of observable clinical symptoms and signs that commonly occur together. In this paradigm, the disorder itself is not observable; it is only measureable via its manifestations, which indicate the severity of the disorder on a continuous spectrum from 'normal' to 'disordered'. The studies we review based on diagnoses (coded as present or absent) therefore have better criterion validity to the *DSM* disorder constructs, but lose valuable information about symptom variation above and below the diagnostic threshold (Krueger, Caspi, Moffit, & Silva, 1998). As such, we synthesize studies that focused on diagnosed disorders alongside studies that focused on continuous symptom measures, interpreting them together (i.e., with diagnoses interpreted as dichotomous indicators of disorder severity).

Studies that have statistically analyzed the patterns of co-occurrence among symptoms of mental disorders have consistently found that the best explanation for these patterns is the presence of shared underlying factors (dimensions, or "spectra") that are transdiagnostic — that is, they cut across traditional diagnostic boundaries and represent the stable core psychological processes shared by groups of disorders (Achenbach & Edelbrock, 1978, 1984; Markon, 2010; Wright & Simms, 2015). For example, in early work on adult psychopathology, Clark and Watson (1991) proposed a tripartite model to account for the comorbidity between anxiety and depression: The overlap between these disorder constructs is accounted for by a general heritable factor they term neuroticism —with a defining characteristic of negative affect (see Andrews, Morris-Yates, Holt & Henderson, 1990)— and they are distinguished by unique factors of anxious arousal (specific to anxiety) and anhedonia or low positive affect (specific to depression).

With a broader focus on the comorbidity among a variety of mental disorders, Krueger and colleagues (1998) found evidence of two correlated spectra of psychopathology that describe the structure of common mental disorders (see Figure 1): The externalizing spectrum accounts for the comorbidity among substance use disorders and antisocial behavior, where disinhibition is prominent; and the internalizing spectrum accounts for the comorbidity among depression, anxiety, and other disorders characterized by prominent negative affect and distress. The internalizing spectrum splits into fear and distress sub-factors in many structural studies, which primarily differentiate between disorders with fear-based characteristics (e.g., phobias, social anxiety) and with sadness/worry-based characteristics (e.g., major depression, generalized anxiety disorder [GAD]) respectively (Eaton, Rodriguez-Seijas, Carragher & Krueger, 2015a; Eaton, South & Krueger, 2010; Krueger & Markon, 2006). This descriptive structure groups disorders according to actual empirical relationships, rather than through the focus on shared phenomenological features in the DSM (Krueger, Markon, Patrick & Iacono, 2005). Through use of factor analytic techniques, researchers can separate the correlations among disorders that indicate the latent comorbidity spectra (e.g., what major depression *has in common* with GAD) from the disorder-specific variance that indicates the uniqueness of each disorder (e.g., what makes major depression *different* from GAD and all other internalizing disorders) (Krueger & Piasecki, 2002). The resulting internalizing-externalizing model of common mental disorder comorbidity is consistent with current genetic and personality research and has been replicated across sociodemographic groups, cultures, diagnostic time frames, at various measurement levels, using a variety of analytic approaches, and for clinical and community samples (see Carragher, Krueger, Eaton & Slade, 2015; Eaton, 2014; Eaton et al., 2015a).

The discovery of internalizing and externalizing spectra in adult psychopathology has fostered a rapidly growing literature on the underlying hierarchical and dimensional structure of psychopathology. A Hierarchical Taxonomy of Psychopathology (HiTOP) has subsequently been established to integrate the vast body of research that has tested factor analytic models of dimensional spectra into a single descriptive model of the meta-structure that includes 11 of the 19 DSM chapters, and six spectra, including internalizing and externalizing (Kotov et al., under review).

Utility of the spectra

While the label “internalizing disorders” can be used simply to refer to the disorders that indicate the latent internalizing spectrum —for example, as a collective label for depressive and anxiety disorders[3]— the spectra of psychopathology also convey a wealth of information beyond descriptive labels for groups of symptoms and disorders. The internalizing spectrum, for example, can be thought of as a continuous underlying dimension of risk where someone with a high level of internalizing is likely to experience multiple chronic and severe internalizing symptoms (e.g., symptoms of depressive and anxiety disorders) over their lifetime, and someone with a low level of internalizing may not experience any symptoms (e.g., Krueger & Finger, 2001; Krueger, Markon, Patrick, Benning & Kramer, 2007). The spectra also represent the conceptual overlap between the disorders, capturing their shared processes and mechanisms, and acting as channels for core psychopathological processes (Forbes, Tackett, Markon & Krueger, 2016). For example, spectra can account for the roles of genetic and environmental risk and protective factors, predict disorder onset, course and treatment response, and predict adaptive functioning (Eaton et al., 2015a; Kessler et al., 2011; Kim & Eaton, in press; Krueger & Eaton, 2015; Lahey, Zald, Hakes, Krueger & Rathouz, 2014; Rodriguez-Seijas, Stohl, Hasin & Eaton, 2015). These spectra also frame the development of psychopathology across the lifespan, and can act as a bridge between multiple streams of research (e.g., nosological research, developmental psychopathology research, neuropsychology research, and clinical practice; Forbes et al., 2016; Kotov et al., under review). As such, the empirically derived spectra of psychopathology offer ideal phenotypes to frame future research. As a result of the findings that dimensional spectrum models offer the best explanation of the broad structure of psychopathology, spectra of psychopathology have been incorporated into the DSM-5 to reflect the empirical relationships and similarities between larger groups of disorders (e.g., the Depressive Disorders and Anxiety Disorders chapters are adjacent; APA, 2013).

Many analyses of the structure of psychopathology have relied upon datasets from large epidemiological surveys. These data sets are strong in their sheer size and in the quality and hence representativeness of their sampling. Unfortunately, sexual dysfunctions are almost invariably excluded from epidemiological research on psychopathology, and as a result sexual dysfunctions have not been included in any of the large-scale analyses of the structure of mental disorders (Kendler, 2009; Krueger, Watson & Barlow, 2005; Markon, 2010). Sexual dysfunctions have been tentatively included in HiTOP as part of the internalizing spectrum based on work by Forbes and colleagues on self-report data from convenience samples (Forbes, Baillie & Schniering, 2015a, 2015b, 2016; Forbes & Schniering, 2013), which are reviewed in detail in Part 3. However, empirical evidence for the inclusion of sexual dysfunctions in HiTOP is at the lowest standard (i.e., emerging evidence with scant validation; Kotov et al., under review), and they are at risk of being left behind.

While the HiTOP model is based on the results of studies that have empirically analyzed the factor structure of the descriptive features of psychopathology (Kotov et al., under review), an earlier framework outlined a substantive approach with which to build hypotheses about

[3]This is how we use the term internalizing disorders in subsequent sections.

the classification of mental disorders into spectra in the lead-up to the DSM-5 (Andrews et al., 2009). The APA Diagnostic Spectra Study Group of the DSM-5 suggested eleven criteria to support the validity of a spectrum: (1) comorbidity, (2) abnormalities of cognitive/emotional processing, (3) temperamental antecedents, (4) treatment response, (5) symptoms, (6) neural substrates, (7) biomarkers, (8) illness course, (9) genetic risk factors, (10) familial risk factors, and (11) environmental risk factors. Part 2 will review evidence in this framework that supports the formation of the hypothesis that sexual dysfunctions are part of the internalizing spectrum. We will focus primarily on the phenotypic features of low sexual function beyond shared symptoms (i.e., comorbid disorder presentation; and cognitive, emotional, temperamental, and affective features), and touch briefly on neurobiological and psychophysiological factors, but we will not discuss genetic, familial and environmental risk factors because there is scant direct evidence in this area. Part 3 will then review the studies that have tested the hypothesis that sexual dysfunctions are part of the internalizing spectrum.

Part 2: Evidence that supports the formation of the hypothesis that sexual dysfunctions are part of the internalizing spectrum

While specific manifestations of internalizing are phenotypically distinguishable, they are proposed to be maintained by similar processes, such as a prominence of negative affect, and aversive reactions to negative affect that often include situational, cognitive, and behavioral avoidance (Allen, McHugh & Barlow, 2008; Barlow et al., 2010). There is currently evidence that a variety of DSM-5 disorders may be manifestations of the internalizing spectrum, including: depressive and anxiety disorders, obsessive-compulsive and related disorders, trauma and stressor-related disorders, bipolar and related disorders, somatic symptom disorders, and feeding and eating disorders (Kotov et al., under review). Many of these disorders share traits like repetitive negative thinking, perfectionism, and emotion avoidance (Barlow et al., 2010; Ehring & Watkins, 2008; Mills et al., 2016). Notably, symptoms of these disorders have multifaceted relationships with low sexual function, which are explored below. For the sake of brevity, Part 2 will focus on the relationships between diagnoses and symptoms of sexual dysfunctions and the core internalizing disorders (i.e., depressive and anxiety disorders), starting with the common patterns of comorbidity.

Comorbidity

Internalizing disorders have long been known to have high rates of co-occurrence with sexual dysfunctions (Derogatis, Meyer & King, 1981; Kaplan, 1979; Wolpe, 1958). Over the last thirty-five years (i.e., since the release of DSM-III), a vast body of research has consistently found systematic patterns of comorbidity between internalizing disorders and sexual dysfunctions, and these findings have been documented in detail elsewhere (e.g., Atlantis & Sullivan, 2012; Laurent & Simons, 2009). To briefly offer some specific examples, diagnoses and symptoms of major depressive disorder, GAD, panic disorder, obsessive-compulsive disorder (OCD), and posttraumatic stress disorder have been found to have multifaceted relationships with lower sexual desire, arousal, and orgasmic function for men and women, as well as with sexual pain for women (e.g., Aksaray, Yelken, Kaptanoglu, Oflu & Ozaltin, 2001; Althof et al., 2005; Balon, 2006a; Breyer et al., 2014; Cheng, Ng &

Ko; Figueira, Possidente, Marques & Hayes, 2001; Hirsch, 2009; Kaya et al., 2006; Kendurkar & Kaur, 2008; Rajkumar & Kumaran, 2015; van Lankveld & Grotjohann, 2000; van Minnen & Kampman, 2000; Vulink, Denys, Bus & Westenberg, 2006). Social anxiety has been found to have more specific relationships with orgasmic disorders and erectile function in men (Bodinger et al., 2002; Corretti, Pierucci, De Scisciolo & Nisita, 2006; Figueira et al., 2001; Tignol, Martin-Guehl, Aouizerate, Grabot & Auriacombe, 2006), but also with desire, arousal, and pain disorders in women (Bodinger et al., 2002; Corretti & Baldi, 2007; Figueira et al., 2001). A small subset of studies has found mixed results (e.g., Dèttore, Pucciarelli & Santarnecchi, 2013; McCabe & Connaughton, 2014) or results contrary to the patterns described above (e.g., Munoz & Stravynski, 2010). However, these findings represent a small minority of the body of research, and are outweighed by the consistent and frequently replicated findings of comorbidity in the extant literature.

The high rates of comorbidity between sexual dysfunctions and internalizing disorders are particularly striking in the absence of overlapping symptoms (vs. between depressive and anxiety disorders, for instance, which have some overlapping diagnostic criteria; APA, 2013), and have been replicated across demographic groups, and in populations from most cultures around the world (e.g., Laurent & Simons, 2009). Further, these relationships hold independent of the side effects of medication[4] (Aksoy, Aksoy, Maner, Gokalp & Yanik, 2012; Araujo, Durante, Feldman, Goldstein & McKinlay, 1998; Baldwin, 2001; Bodinger et al., 2002; Breyer et al., 2014; Kennedy, Dickens, Eisfeld & Bagby, 1999; Nicolosi, Moreira, Villa & Glasser, 2004; Vulink et al., 2006; Williams & Reynolds, 2006).

Shared cognitive and emotional processes

Looking beyond the high comorbidity rates among sexual dysfunctions and internalizing disorders, there is a large body of literature highlighting parallels in their cognitive and affective processes and characteristics. For example, people with hypoactive sexual desire disorder, and men with erectile dysfunction or premature ejaculation share the same difficulty in identifying and communicating emotions (i.e., alexithymia; Madioni & Mammana, 2001; Michetti et al., 2007) that is common in emotional disorders like depression and anxiety (Marchesi, Brusamonti & Maggini, 2000).

Nobre and colleagues built on Barlow's (1986) model of sexual response, and their work highlights detailed evidence for consistency between the cognitive and emotional processes in low sexual function that mirror cognitive models of depression and anxiety (cf. Beck, 1974, 1979). For example, the three key processes highlighted in SexLab's work on the cognitive and affective features of low sexual function are: (1) people with sexual dysfunctions have unrealistic and inflexible beliefs about sex (e.g., a real man should be able to get an erection whenever he wants to); (2) when negative sexual events occur that are inconsistent with these beliefs, negative sexual self-schemas develop (e.g., I'm not a real man), which (3) generate negative automatic emotions and thoughts (e.g., I'll never be able to get an erection) that interfere with sexual response as part of an ongoing cycle that

[4]In contrast, the co-occurrence of sexual dysfunctions with other domains of psychopathology has been attributed to the medications that are commonly used to treat schizophrenia and mania, for example (e.g., lithium and antipsychotics; Baggaley, 2008; de Boer, Castelein, Wiersma, Schoevers & Knegtering, 2015; Elnazer, Sampson & Baldwin, 2015).

maintains low sexual function (Nobre & Pinto-Gouveia, 2009; Oliveira et al., 2014; Oliveira & Nobre, 2013b; Peixoto & Nobre, 2014, 2015; Quinta Gomes & Nobre, 2012).

There are striking similarities between these three processes in sexual dysfunctions and internalizing disorders. Sexual dysfunctions are characterized by the same sorts of unconditional beliefs (e.g., conservative, unrealistic, rigid, and/or negative ideas about sex; Bach, Wincze & Barlow, 2007; Dosch, Belayachi & Van der Linden, 2016; Nobre & Pinto-Gouveia, 2006a, 2008a, 2009; Peixoto & Nobre, 2014; Sbrocco & Barlow, 1996; Strub, 2013) that act as vulnerability factors for the development of internalizing disorders (e.g., Beck, 1979; Hollon, Stewart & Strunk, 2006). Men and women with sexual dysfunction also have been found to have more negative sexual schemas (i.e., deep, persistent beliefs about sex) than people without sexual dysfunction (Nobre & Pinto-Gouveia, 2009; Peixoto & Nobre, 2015). These maladaptive schemas, such as failure expectancies, incompetence self-schemas, perceived lack of control, and hyper-vigilance (Nobre, 2009; Nobre & Pinto-Gouveia, 2008a, 2009; Oliveira & Nobre, 2013a; Peixoto & Nobre, 2014; Quinta Gomes & Nobre, 2012; Wiegel, Scepkowski & Barlow, 2007) are also characteristic of internalizing disorders (Pinto-Gouveia, Castilho, Galhardo & Cunha, 2006; Schmidt, Joiner, Young & Telch, 1995; Young, Klosko & Weishaar, 2003). Perhaps as a consequence of these negative schemas, people with a sexual dysfunction have been found to endorse cognitive distortions (Rosen, Leiblum & Spector, 1994), and report negative automatic thoughts (Nobre & Pinto-Gouveia, 2008c) that are associated with impaired arousal and orgasmic function in particular (Nobre & Pinto-Gouveia, 2008a, 2008b).

Additional parallels between cognitive and emotional processing in low sexual function and internalizing disorders are evident in the context of Barlow et al.'s (2010) recent cognitive model of internalizing psychopathology where aversive reactions to negative emotions lead to attempts to control, avoid, or suppress these emotions (e.g., situational, cognitive, and behavioral avoidance; Brown & Barlow, 2009), which ultimately fail and maintain the cycle of distress (Barlow et al., 2010). Behavioral and cognitive avoidance are also evident in sexual dysfunctions; for example, behavioral intimacy avoidance is often characteristic of chronic low sexual function (Lewis et al., 2010). Cognitive avoidance is also central to the disruption of sexual response, where cognitive biases orient towards the threat in erotic cues, triggering negative automatic thoughts and cognitive distraction and avoidance (Carvalho & Nobre, 2011a; Cuntim & Nobre, 2011; Peixoto & Nobre, 2012; Strub, 2013). This effect is particularly potent for impaired male sexual desire (Carvalho & Nobre, 2011b), male and female subjective arousal (Nobre & Pinto-Gouveia, 2008a; Oliveira et al., 2014), and female orgasm (Cuntim & Nobre, 2011). These maladaptive cognitive processes have been found to act for men and women of different sexual orientations (e.g., Peixoto & Nobre, 2014, 2015), in a variety of cultures (Abdolmanafi et al., 2015; Beard & Amir, 2010; Dosch et al., 2016; Sbrocco & Barlow, 1996; van den Hout & Barlow, 2000; Weisberg, Brown, Wincze & Barlow, 2001), and operate independently from the effect of co-occurring symptoms of depression and anxiety (Carvalho & Nobre, 2011a; Oliveira & Nobre, 2013a; Quinta Gomes & Nobre, 2012).

Temperamental antecedents

Internalizing disorders and sexual dysfunctions also have overlap in their associated personality and emotional traits. For example, high levels of neuroticism appear to confer a risk for internalizing psychopathology, in line with their conceptual overlap (Krueger & Markon, 2006); and people presenting with sexual dysfunctions have been found to have high levels of neuroticism, even after controlling for other psychopathology (Harris, Cherkas, Kato, Heiman & Spector, 2008; Quinta Gomes & Nobre, 2011; Watts & Nettle, 2010). Neuroticism scores on the NEO-Five Factor Inventory have also been found to predict the overall frequency of negative sexual cognitions (Moyano & Sierra, 2013). More broadly, sexual dysfunctions share the high level of negative affect that characterizes internalizing disorders, which is a general dimension of fear, anger, sadness, guilt, worry, and disgust (Clark & Watson, 1991); high levels of these emotions are related broadly to low sexual function (Oliveira & Nobre, 2013b; Peixoto & Nobre, 2012), and specifically to lower sexual arousal (Nobre & Pinto-Gouveia, 2008a; Oliveira et al., 2014; Peixoto & Nobre, 2012; Vilarinho et al., 2014) and sexual desire (Carvalho & Nobre, 2011b; Nobre & Pinto-Gouveia, 2008b) in men and women. Consistent with this, men and women with sexual dysfunctions report higher levels of negative affect —particularly during sexual activity— compared to people without sexual dysfunctions (Nobre & Pinto-Gouveia, 2006b; Peixoto & Nobre, 2016), and sexual dysfunctions are also generally related to both temporary and recurrent negative affect (Wiegel et al., 2007).

Treatment response

These shared cognitive and affective processes are possible reasons for the response of sexual dysfunctions to treatments shared with internalizing disorders (Strub, 2013). For example, female sexual desire, arousal, orgasm, and sexual pain disorders; as well as male premature ejaculation, erectile dysfunction, and delayed ejaculation have been found to respond to cognitive-behavioral and mindfulness therapies (Andersson et al., 2011; Brotto, Basson & Luria, 2008; Brotto, Basson, Smith, Driscoll & Sadownik, 2015; Brotto & Smith, 2014; Brotto, Woo & Leiblum, 2010; Frühauf, Gerger, Schmidt, Munder & Barth, 2013; Jones & McCabe, 2011; Mohammadi, Mohammadkhani, Dolatshahi & Dadkhah, 2013). This is not conclusive evidence, as cognitive-behavioral therapy (CBT) covers a broad variety of therapeutic techniques that can be effective for most mental disorders. However, the fact that these same techniques have been applied to treat sexual dysfunctions, with similar effects (e.g., McCabe, 2001; Trudel et al., 2001), is consistent with shared treatment response between the disorders.

Stronger evidence for shared treatment response between the disorders is found in experimental studies that have shown that sexual response can be manipulated by altering schemas and moods (Kuffel & Heiman, 2006; ter Kuile, Both & Van Uden, 2010), which are specifically targeted in CBT for internalizing disorders (e.g., Allen et al., 2008; Barlow et al., 2010; Boisseau, Farchione, Fairholme, Ellard & Barlow, 2010). These studies offer preliminary evidence for specific shared mechanisms and processes that may foster comorbidity between the disorders, which is a necessary step before it is possible to develop and test interventions to prevent their comorbidity (Kessler & Price, 1993).

Finally, there is additional evidence for shared treatment response in studies that have found sexual function to improve with treatment for internalizing disorders (e.g., Hoyer, Uhmann, Rambow & Jacobi, 2009; Schnurr et al., 2009; ter Kuile, Both & van Lankveld, 2010), and in studies that have found internalizing disorders to improve with treatment for sexual dysfunctions (e.g., Bocchio et al., 2009; Chudakov, Matar & Kaplan, 2008).

The effect of selective serotonin reuptake inhibitors (SSRIs) is a notable exception to this pattern of shared treatment response: SSRIs are the most widely prescribed class of antidepressant medication used to treat depressive and anxiety disorders in many countries, such as the United States (e.g., Preskorn, Ross, & Stanga, 2004), but are associated with a high risk for antidepressant-induced sexual dysfunction (Keks, Hope & Culhane, 2014; Balon, 2006b). The precise mechanisms underlying this relationship are not known. One specific mechanism that may account for antidepressant-induced sexual dysfunction is the SSRI activation of the 5-HT_2 receptor, which impairs all stages of sexual response for men and women (Zemishlany & Weizman, 2008). More broadly, the sexual dysfunction side effects of SSRIs are likely due to a combination of their effects on serotonergic tone, reduction in dopaminergic activity and reuptake, reduction in noradrenergic transmission, and reduction in nitric oxide levels (e.g., Bredt et al., 1991; Schatzberg, Cole & DeBattista, 2007; Schatzberg & Nemeroff, 2009); these neurotransmitter systems are involved in both sexual dysfunctions and internalizing disorders, as discussed below. Interestingly, given serotonergic tone predicts ejaculatory latency (in animal models; Waldinger, 2002), SSRIs have been found to be an effective treatment for premature ejaculation (e.g., Giuliano & Hellstrom, 2008).

Neural substrates

Given the complexity of the relationships between neurobiological mechanisms that interact to allow for positive sexual function (e.g., Clayton, 2007; Yehuda, Lehrner & Rosenbaum, 2015), this area of the literature is largely beyond the scope of our review. Briefly, many of the brain systems that are involved in internalizing disorders have also been found to have a role in sexual response. For example, activation of the hypothalamic-pituitary-adrenal axis and amygdala may interfere with sexual function (Baird, Wilson, Bladin, Saling & Reutens, 2007; Hamilton & Julian, 2014). Further, high levels of the hormone cortisol are associated with lower subjective desire and arousal in women, where high cortisol may predispose women to have a negative assessment of sexual stimuli (Dundon, 2014).

In particular, there is substantial overlap in the neurotransmitter systems that have a role in internalizing disorders and sexual response. As mentioned above, dopaminergic, noradrenergic, and serotonergic neurotransmitter systems are involved in sexual dysfunction: Desire and arousal function are increased by disinhibition of dopaminergic systems (Meston & Frohlich, 2000; Bancroft & Janssen, 2000). Noradrenergic neurophysiology is also related to male erection and ejaculations, and may explain the link between anxiety and spontaneous ejaculation (Redmond, Kosten & Reiser, 1983; Segraves, 1989). The role of serotonergic neurotransmission is more complex, with serotonin likely modulating both excitatory and inhibitory pathways for sexual behavior (e.g., Bancroft & Janssen, 2000), and generally thought to impede sexual function (Frohlich & Meston, 2000; Miner & Seftel,

2007; Segraves, 1989). An early formulation of the monoamine theory of depression postulated that the effectiveness of antidepressant agents owed to a fundamental deficit of neurotransmitters (primarily serotonin and noradrenaline) in individuals with depression (see Hirschfield, 2000). This deficit was thought to be corrected by blocking transporters that would normally remove serotonin from the synaptic cleft. Recent research, however, now attributes the effectiveness of serotonergic antidepressants to downstream effects that increase synaptogenesis and neurogenesis by modulating neuronal growth factors (e.g. brain-derived neurotrophic factor; Lee & Kim, 2010). Thus, the monoamine hypothesis of depression is likely misguided, and the apparent contradiction in increased serotonin impeding sexual function but alleviating depression is unlikely to hold.

In short, all of the neural substrates mentioned here have a prominent role in internalizing disorders as well as sexual function (Goldberg, Krueger, Andrews & Hobbs, 2009; Hirschfeld, 2000; Levine, 2000; Pariante & Lightman; Stein, Westenberg & Liebowitz, 2002; Vaidyanathan, Patrick & Cuthbert, 2009; Vreeburg et al., 2010). As such, it seems likely that there are shared substrates between internalizing disorders and low sexual function.

Biomarkers

Studies that have examined biomarkers like cardiovascular reactivity, galvanic skin response, and vaginal pulse amplitude have found commonalities among the physiological responses that characterize anxiety and low sexual function. In particular, the sympathetic nervous system (SNS) has a prominent role in sexual function that is similar to its role in internalizing disorders (Hoehn-Saric & McLeod, 1988; Veith et al., 1994; Villacres, Hollifield, Katon, Wilkinson & Veith, 1987). For example, high and low levels of SNS activation are associated with impaired desire, arousal, and orgasm (Corretti & Baldi, 2007; Lorenz, Harte & Meston, 2015), but moderate levels of SNS activation may facilitate physiological sexual arousal in particular (Lorenz et al., 2015; Meston, 2000; Meston & Gorzalka, 1996). This inverted U-shaped relationship appears to be limited to healthy and sexually functional people who mislabel their physiological arousal as sexual arousal (i.e., excitation transfer; Elliot & O'Donohue, 1997; Palace & Gorzalka, 1990; van den Hout & Barlow, 2000).

In contrast, SNS activation appears to have a positive linear relationship with disrupted sexual response in people with existing sexual difficulties, and people with higher levels of anxiety, who are more likely to focus attention selectively on non-erotic and/or threatening cues, which increases anxiety and impairs subjective and physiological arousal (van den Hout & Barlow, 2000; van Minnen & Kampman, 2000). Correspondingly, many of the same experimental studies that have examined the impact of SNS activation and state anxiety have found distraction (i.e., a shared cognitive component of state and trait anxiety) to have a negative impact on sexual arousal for sexually functional *and* dysfunctional participants, as it increases the salience of non-erotic cues, preventing positive cognitive and emotional feedback and subsequent subjective arousal (Basson, 2002; Corretti & Baldi, 2007; Corretti et al., 2006; Elliot & O'Donohue, 1997).

Another shared biomarker is low resting heart rate variability —a measure of autonomic nervous system (ANS) activity— which is a marker of depression and anxiety (Kemp, Quintana, Felmingham, Matthews, & Jelink, 2012; Kemp, Quintana, Gray, Felmingham, Brown, & Gatt, 2010), and has recently been found to predict female sexual arousal and orgasmic function (Costa & Brody, 2012; Stanton, Lorenz, Pulverman & Meston, 2015) and erectile dysfunction (Lee, Joo, Kim, Cho, Cho, Won & Choi, 2011). Serum markers of inflammation that are associated with depression (e.g., Ramsey et al., 2016) have also been found to predict erectile dysfunction (e.g., Vlachopoulos et al., 2006). Taken together, these findings suggest there are shared biomarkers for internalizing psychopathology and low sexual function.

Disorder course

Lastly, the course of internalizing disorders and sexual dysfunctions is similar. For example, the latent internalizing spectrum —as a statistical representation of the level of risk for developing an internalizing disorder— is stable over time (Eaton, Krueger & Oltmanns, 2011; Krueger, 1999; Sunderland, Slade, Carragher, Batterham & Buchan, 2013; Vollebergh et al., 2001). In other words, although individual disorders may remit and recur over time, the underlying liability for disorders to develop as manifestations of the internalizing spectrum remains relatively stable (Eaton et al., 2011). This course of internalizing psychopathology as a whole is consistent with the course of individual depressive and anxiety disorders, as well as sexual dysfunctions, which tend to be persistent with high rates of recurrence and are often chronic across the lifespan (APA, 2013).

Conflicting evidence

There is one consistent finding that stands in contrast to the studies reviewed above: Three studies have found that some men and women experiencing depressive symptoms report elevated desire (Bancroft et al., 2003a; Frohlich & Meston, 2002; Nofzinger et al., 1993). Nofzinger et al. (1993) found that this relationship was specific to episodes of atypical depression, and was not evident in anhedonic, endogenous, or melancholic depression. It seems likely that these studies may be describing the same phenomenon, as Frohlich and Meston (2002) and Bancroft et al. (2003a) did not test for features of atypical depression, which can manifest as mild symptoms of major depression on self-report inventories (e.g., Fabre & Smith, 2012). A specific relationship between atypical depression and elevated desire would also be consistent with the finding that atypical depression is associated with lower rates of sexual dysfunction than major depression in men and women (Fabre, Clayton, Smith, Goldstein & Derogatis, 2013; Fabre & Smith, 2012), and could be explained by the mood reactivity to potential positive events in atypical depression, which may reinforce sexual approach behaviors. In contrast, in other types of depression, the ongoing dysphoria and anhedonia may disrupt the process by which pleasurable behaviors are reinforced (Frohlich & Meston, 2002). Either way, these findings are important to consider, as they highlight the importance of nuanced analyses that do not presume the same interrelationships in all groups (e.g., factor mixture analyses, which are explored in more detail in Part 3).

Integrating the substantive evidence

There are gaps in the research, particularly regarding the validators related to shared risk factors (i.e., genetic, familial, and environmental risk factors), which require epidemiological twin studies that assesses common mental disorders and sexual function at the same time. However, there is evidence from multiple streams of research that highlight robust multifaceted relationships between the established core internalizing disorders and low sexual function. If we discount the possibility that these relationships occur by chance, there are three types of relationships that could account for these findings: 1) internalizing disorders cause sexual dysfunctions, 2) sexual dysfunctions cause internalizing disorders, and/or 3) internalizing disorders and sexual dysfunctions share a set of common causes, which can be summarized by the internalizing spectrum. In fact, all three of these statements can hold simultaneously. The only way to adjudicate which hypothesis best accounts for the robust multifaceted relationships is to analyse longitudinal data: Prominent unidirectional relationships between internalizing psychopathology and sexual function—in either direction—would support the former two hypotheses; systematic bidirectional relationships would be consistent with either a shared set of common causes or reciprocal causality accounting for their overlap; and an absence of directional relationships would suggest that a shared set of common causes accounts for their overlap. Longitudinal research to date has consistently found small bidirectional relationships or no directional relationships between these constructs (Atlantis & Sullivan, 2012; Forbes et al., 2015a; Kalmbach, Pillai, Kingsberg & Ciesla, 2015; Peleg-Sagy & Shahar, 2013), in line with the internalizing hypothesis, but no longitudinal study to date has found evidence for substantial unidirectional or bidirectional relationships to the best of our knowledge. Many researchers have correspondingly proposed that internalizing symptoms and low sexual function may be manifestations of the same underlying processes (Atlantis & Sullivan, 2012; Corretti et al., 2006; Kalmbach et al., 2015; Oliveira & Nobre, 2013b; Peixoto & Nobre, 2012; van Lankveld & Grotjohann, 2000; Watts & Nettle, 2010). In short, the multifaceted relationships between the disorders combined with the lack of any evident unidirectional causal relationships suggest that conceptualizing sexual dysfunctions as manifestations of the internalizing spectrum is both a plausible and parsimonious explanation for these patterns of associations.

We note again that we are not proposing the internalizing spectrum is the only —or even the most influential— determinant of low sexual function. Krueger (1999) found that the internalizing spectrum accounted for 32–58% of the variance in the internalizing disorders, and Eaton et al. (2013) found that the lower-order fear and distress spectra accounted for 48–80% of the variance in the internalizing disorders. There is evidence for a similar relationship between internalizing and low sexual function: Forbes et al. (2015a) found that 28–80% of the variance in most domains of male and female sexual function was accounted for by a longitudinal internalizing spectrum model[5]. This means that in our proposed model,

[5]These estimates included the domains of desire, arousal sensation, lubrication, orgasm, and sexually-related distress for women; and the domains of erectile function, ejaculatory control and associated distress, and sexual satisfaction (operationalising low sexually-related distress) for men. The cognitive component of female sexual arousal had stronger relationships with the model (75–84% of its variance was accounted for by a longitudinal internalizing spectrum model), as expected. In contrast, female sexual pain and male sexual desire had weaker relationships (8–11% and 10–16% of their variance was accounted for, respectively), which is discussed in detail in Part 3.

an average of a fifth to two-thirds of the variance in sexual function would still be attributable to other factors, such as biological and medical vulnerabilities (e.g., hormone levels, diabetes), social context (e.g., poor communication, relationship problems, sex-roles, religious ideas) and environmental factors (e.g., lack of privacy, pets, work schedules; Wincze & Weisberg, 2015). Further, the amount of variance accounted for by the internalizing spectrum —and other determinants of low sexual function— would vary from person to person such that for some individuals low sexual response might only be an adaptive reaction to their social context, or due to a medical condition, for example.

In summary, the extant literature offers robust evidence consistent with the hypothesis that the internalizing spectrum is a core underlying vulnerability for sexual dysfunctions. There are eight studies that have been published in the last five years that provide direct support for this hypothesis. We will now turn to review these studies.

Part 3: Studies that have tested the hypothesis that sexual problems are part of the internalizing spectrum

In their review, Laurent and Simons (2009) proposed that sexual dysfunctions may form part of the internalizing spectrum, and that specific types of low sexual function might have differential relationships with narrower facets of internalizing. Specifically, they suggested that low sexual function related to physiological arousal (e.g., erectile function, lubrication, and sexual pain) could be differentially related to anxiety disorders —that is, with the fear sub-factor of internalizing in Krueger's (1999) model or the anxious arousal factor in Clark and Watson's (1991) tripartite model. Similarly, they suggested that low sexual function with more prominent cognitive, motivational, and pleasure-seeking components (e.g., low desire and orgasmic function) might be related to the distress sub-factor of internalizing in Krueger's model or the anhedonia factor in Clark and Watson's model.

This set of hypotheses has largely been tested in the context of Clark and Watson's (1991) tripartite model. For example, Kalmbach and colleagues have examined these hypotheses for self-reported female sexual function —mostly in young women— in daily diary studies (Kalmbach, Kingsberg & Ciesla, 2014; Kalmbach & Pillai, 2014), a large cross-sectional study (Kalmbach, Ciesla, Janata & Kingsberg, 2012), and a prospective longitudinal study over five weekly assessments (Kalmbach et al., 2015). Their regression analyses across the four studies converged on largely consistent patterns of findings. Kalmbach et al. focused on the unique relationships of the three domains in the tripartite model with different domains of sexual function, and found that anhedonia was consistently uniquely related to low levels of desire. Anxious arousal also had consistent relationships with difficulties with lubrication and pain (Kalmbach et al., 2012; Kalmbach et al., 2014; Kalmbach & Pillai, 2014); and negative affect was uniquely related to more difficulties with orgasm in the daily diary studies (Kalmbach et al., 2014; Kalmbach & Pillai, 2014). These findings are broadly consistent with Laurent and Simons' (2009) hypotheses, but it is important to note that they relate only to female sexual function over brief time periods, and are limited by the predominance of women under 45 in the samples. Forbes and Schniering (2013) also tested these hypotheses—using a more diverse sample of men and women—in the context of

Krueger's (1999) internalizing spectrum model. Forbes and Schiering found that the specific relationships hypothesized by Laurent and Simons (2009) were not needed to explain the data; instead, a higher-order internalizing dimension accounted for the shared variance between the disorders to the extent that the specific cross-loadings to fear and distress did not account for any additional variance. It would be interesting for future research to test the fit of a model that does not include a low sexual function latent variable, but treats sexual function domains as direct indicators for the fear and distress spectra.

While Kalmbach and colleagues' studies focused on the unique relationships between anhedonia, anxious arousal, and negative affect with specific domains of sexual response, the internalizing spectrum lies in the shared variance between these factors (i.e., the overlap between them), as well as in their unique components. As such, in these studies it is the predictive validity of the model as a whole that tests whether sexual function is related to internalizing. Although it is not highlighted in Kalmbach and colleagues' papers, it is noteworthy then that they found the combination of the three factors together were consistently associated with lower levels of female sexual desire, subjective and physiological arousal, and orgasm function, and higher levels of pain (Kalmbach et al., 2012; Kalmbach et al., 2014; Kalmbach & Pillai, 2014; Kalmbach et al., 2015). This was the case when examining state (Kalmbach et al., 2015) and trait (Kalmbach et al., 2012) affect, as well as symptoms of anxiety and depression specifically (Kalmbach et al., 2014). In short, these four studies all converge on evidence for multivariate relationships among these constructs for women. In additional support for the sexual problems forming part of the internalizing spectrum, Kalmbach et al. found that these multivariate relationships were almost exclusively concurrent, and did not find any consistent time-lagged relationships between studies, which suggests that there were no evident causal relationships on a daily (Kalmbach et al., 2014; Kalmbach & Pillai, 2014) or weekly (Kalmbach et al., 2015) time scale. While Kalmbach and colleagues' work was largely focused on female sexual function, Kalmbach et al. (2012) also included young men, and found that subjective arousal, erectile function, and orgasmic function were all predicted by the internalizing domains, but desire was not. This aberration is noteworthy in the context of the studies reviewed below, and will be revisited later.

The strongest evidence supporting the hypothesis that sexual problems are part of the internalizing spectrum comes from the work by Forbes and colleagues (Forbes et al., 2015a, 2015b, 2016; Forbes & Schniering, 2013), which specifically tested this hypothesis in the context of Krueger's (1999) internalizing spectrum model for men and women across four studies. These studies included cross-sectional (Forbes et al., 2015b, 2016; Forbes & Schniering, 2013) and longitudinal (Forbes et al., 2015a) data and utilized a variety of self-report measures (Forbes et al., 2015b; Forbes & Schniering, 2013), different inclusion criteria (i.e., penetrative sexual intercourse in the past four weeks in Forbes et al., 2016; sexual activity including masturbation in the past four weeks Forbes & Schniering, 2013; and no specific requirements regarding sexual activity in Forbes et al., 2015a, 2015b), and statistical modeling methods with divergent assumptions and limitations across all four studies. This heterogeneity in the methods between the studies is important because it strengthens our confidence in the findings of the body of research as a whole. Forbes and colleagues measured self-reported symptoms of common internalizing disorders (e.g.,

depression, GAD, social anxiety, panic and agoraphobia, OCD) as well as domains of sexual function (e.g., desire, arousal sensation, cognitive arousal, lubrication, orgasm function, sexual pain, and sexually related distress for women; desire, erectile function, ejaculatory control and related distress, and sexual satisfaction for men; e.g., Forbes et al., 2015b; Forbes & Schniering, 2013) and focused on finding the best statistical model to describe the relationships between these domains. These studies relied on convenience samples from the community (i.e., tending to be young and well-educated), which limits their generalisability (Laumann, Paik, & Rosen, 1999; Lutfey et al., 2009), but they do represent diverse symptom levels similar to or higher than population norms for most symptom measures, including 20–40% of the primary samples reaching clinical cut-offs for moderate or severe impairment for most disorders in Forbes et al. (2015a, 2015b). We introduce these studies individually below, before integrating their findings for men and women in the context of the literature.

Forbes and Schniering (2013) used structural equation modeling (SEM) as the first test of an expanded dimensional model of the internalizing spectrum that included sexual problems alongside symptoms of depression, GAD, social anxiety, panic disorder, and agoraphobia (cf. Figure 2). This model was tested separately for men and women; it provided comparatively better fit than models based on Laurent and Simons' (2009) specific hypotheses described above, as well as models based on the DSM-IV that conceptualized sexual problems as unrelated to depressive and anxiety disorders. However, while the internalizing model provided excellent absolute fit to the data for women, it did not have strong model fit for men. Forbes and Schniering (2013) attributed this to the narrow range of sexual problems in the male sample.

Forbes et al. (2016) subsequently compared latent profile analyses (LPA) of the data with SEM in the same sample. The aim of this study was to explicitly compare the dimensional internalizing model in Forbes and Schniering (2013) to a categorical model of the relationships. In LPA, individuals are grouped by shared symptom profiles. If the profiles are qualitatively distinct (e.g., characterized by specific groups of symptoms or disorders), this suggests that the disorders belong to different diagnostic categories. Alternatively, if the profiles are parallel, this suggests the groups only differ on symptom severity, which is consistent with an underlying dimensional spectrum between the disorders. Forbes at al. (2016) found that the dimensional internalizing (SEM) model fit better —for men and for women— than the categorical LPA models, and the strongest LPA models had largely parallel profiles. Interestingly, while the overwhelming majority of men and women in the sample (96% and 100%, respectively) had strong dimensional relationships consistent with the internalizing spectrum (i.e., depressive and anxiety disorders systematically covaried with low sexual function), symptoms of depressive and anxiety disorders were unrelated to low sexual function for 4% of men. Given these men reported particularly low erectile function, one explanation for this finding may be that they were experiencing primarily organic —rather than psychogenic— erectile dysfunction (i.e., due to substance or medication use, or due to a medical condition; cf. APA, 2013).

In a larger convenience sample, Forbes et al. (2015b) examined factor mixture analyses of the relationships between depressive and anxiety symptoms and low sexual function. This type of analysis allows for a model to have categorical and dimensional components

simultaneously. For example, within a single factor mixture analysis participants' responses can be split into groups with different structural relationships so that one group can have strong dimensional relationships between internalizing symptoms and low sexual function, while for another group these domains are unrelated. Factor mixture analyses can thus detect common processes for large groups of people, but also allow for different structural relationships within groups that do not share these common processes. This feature is excellent for modeling psychological processes involved in sexual function, as we know that not all experiences of low sexual function will be related to internalizing psychopathology. The findings of Forbes et al. (2015b) reaffirmed the dimensional internalizing structure for all of the women, and found even stronger dimensional relationships for 87% of the men. Interestingly, 13% of men had categorical distinctions between sexual function and symptoms of depressive and anxiety disorders, mirroring the smaller group of men with categorical distinctions in Forbes et al. (2016). We will revisit these findings later.

Finally, Forbes et al. (2015a) used SEM to examine the longitudinal relationships between the disorder constructs at a latent level. An internalizing structure fit best, and was invariant over the course of four weeks for men[6], and over the course of one week, four weeks, and six months for women. There were no evident causal relationships (i.e., cross-lags) in any of these models, which supports the hypothesis of a shared underlying factor accounting for change in the disorders over time, as discussed in Part 2. Taken together with Kalmbach et al.'s (2015) longitudinal study, there is evidence for concurrent change in depression, anxiety, and women's self-reported low sexual function in a variety of time scales, ranging from one day to six months.

Results for women

Overall, the results for women were consistent within and between the eight studies, which all showed dimensional relationships between low sexual function and internalizing psychopathology in community samples. These relationships were evident in women who were sexually active with a partner (Forbes et al., 2016; Forbes & Schniering, 2013) as well as those who were not (Forbes et al., 2015a, 2015b). While Kalmbach et al. (2014a, 2014b, 2012) found desire, arousal, orgasm, and pain to be consistently related to symptoms of depression and anxiety, Forbes and colleagues had some variability in the findings for specific disorders across the studies. For example, initial findings suggested that female sexual desire was weakly associated with the internalizing spectrum (Forbes & Schniering, 2013), and it was subsequently excluded in the next analysis of that sample (Forbes et al., 2016). Forbes and Schniering (2013) hypothesized that this finding was due to the poor measurement of desire in the Female Sexual Function Index (cf. Forbes, Baillie & Schniering, 2014; Rosen et al., 2000). Consistent with this, when the Abbreviated Sexual Function Questionnaire (Williams, Abraham & Symonds, 2010) was used, desire was a strong indicator for the models (Forbes et al., 2015a, 2015b). On balance, these later results —in combination with Kalmbach et al.'s four studies and with the larger literature that has found depression and anxiety to be related to low levels of desire (e.g., see Laurent &

[6]The models were not tested using a one-week or six-month gap for men because the subsample sizes were not large enough (Forbes et al., 2015a).

Simons, 2009 for a review)— suggest that desire is a good indicator of the internalizing spectrum.

Forbes et al. (2015a, 2015b) also found that sexual pain and sexually-related distress were more closely related to depressive and anxiety disorders than to other domains of female sexual function, which means that a model based on Figure 2 may not be an ideal representation of these specific relationships (i.e., where pain and distress are only related to depressive and anxiety disorders via their relationship to the latent sexual function variable). This finding for pain is consistent with research that has found pain to operate independently of other domains of sexual function (e.g., Binik et al., 2002) but to be closely related to depression and anxiety (e.g., Kaya et al., 2006). Similarly, the result for distress is consistent with Bancroft et al.'s (2003b) finding that emotional wellbeing —rather than sexual function specifically— is the best predictor of distress. Future studies could examine these relationships in an exploratory SEM framework to allow for more flexibility in the relationships among the disorders. Overall, the studies based on female samples depict a complex set of interrelationships that are consistent with the body of research reviewed in Part 2, and uniformly show clear dimensional relationships between internalizing disorders and low sexual function.

Results for men

After accounting for the small groups of men that apparently had non-psychogenic low sexual function, 87–96% of the men in Forbes et al. (2015b, 2016) had strong dimensional relationships between depressive and anxiety disorders and low sexual function, consistent with a shared internalizing spectrum. In fact, these dimensional relationships were even stronger than those found for women. Overall, self-reported erectile function, premature ejaculation, and sexual satisfaction (included to operationalize low sexually-related distress) were consistently related to symptoms of depression and anxiety (Forbes et al., 2015a, 2015b, 2016; Forbes & Schniering, 2013; Kalmbach et al., 2012). While the generalisability of these studies is limited by the non-representative samples —particularly by the young male samples in Forbes and Schniering (2013), Forbes et al. (2016), and Kalmbach et al. (2012)— Forbes et al. (2015a, 2015b) had better measurement and more diversity in symptom levels, and these latter studies found the strongest evidence for dimensional relationships for men.

One consistent and puzzling finding was that male sexual desire was a poor indicator of the internalizing spectrum in every study (Forbes et al., 2015a, 2015b, 2016; Forbes & Schniering, 2013; Kalmbach et al., 2012) despite their divergent statistical approaches, and the use of three different self-report measures. As such, it is unlikely that this finding was due solely to poor measurement (cf. Forbes, 2014; Forbes et al., 2014; Forbes et al., 2016). An alternative explanation could be that none of the studies differentiated between dyadic and solitary desire. Spector, Carey, and Steinberg (1996) suggested that solitary desire might serve a different purpose (e.g., tension release) than the emotional and physical intimacy associated with dyadic desire. Thus, we may expect dyadic desire to covary with internalizing symptoms, but solitary desire may play more of a self-soothing role, and be heightened during negative mood states (Frohlich & Meston, 2002). Alternatively, there may

have been insufficient variability in sexual desire (i.e., restriction of range) in the samples, given all of the studies relied predominantly on young, healthy, and educated men from the community. The relationship between low sexual desire and depression in particular has been documented to the extent that a decrease in libido "is an indicator of depression in all patient cohorts except in women older than 70 years" (Baldwin, 1996, p. 31), so it is likely that clinical samples and/or higher symptom levels may be required to identify this relationship.

Integrating the findings of the eight studies

Taken together, the eight studies that have analyzed the overarching relationships between the disorders are consistent with the hypothesis that low sexual function shares a common underlying factor of internalizing psychopathology with depressive and anxiety disorders for men and women. The relationships in Forbes et al.'s (2013, 2015a, 2015b, 2016) models showed that anxiety and depressive symptoms were more strongly related to each other than they were to low sexual function. However, this was expected, and is likely due to the variety of etiologic factors that influence sexual function —as discussed earlier— as well as the shared symptoms between depressive and anxiety disorders, the overlap in the domains that are measured (i.e., cognitive and emotional versus sexual), and the similar stem-and-response measures for depressive and anxiety symptoms (i.e., measurement overlap). The strongest case for interpreting these studies' findings as an indication of a shared liability between the disorders is in their high rates of comorbidity; shared cognitive, affective, and personality characteristics; common neural substrates and biomarkers; shared course and treatment response; and the lack of unidirectional causal relationships between them.

On the whole, there are fewer gender differences in the studies than we might have expected based on a review of the body of research on male and female sexual response cycles. For example, a simple linear sexual response cycle is generally accepted for men (Masters & Johnson, 1970). However, this model has been challenged for women, where motivation to engage in sexual activity and other contextual factors are given a prominent role, and desire and arousal are particularly intertwined with one another (Basson, 2000, 2001, 2005; Brotto, Graham, Binik, Segraves & Zucker, 2010). In contrast, within the literature more broadly, the results of some factor analytic studies suggest that there may in fact be similarities between men and women who experience low sexual function, and between men and women who do not. The sexual response cycle has been found to have distinct desire, arousal, and orgasm phases for men and women without low sexual function (Carvalho, Vieria & Nobre, 2011; Giles & McCabe, 2009), whereas sexual desire, arousal, and orgasm tend to overlap for men and women experiencing low sexual function (Carvalho, Vieira & Nobre, 2012; Carvalho et al., 2011; Corona et al., 2015; Giles & McCabe, 2009). These findings are consistent with the notion of higher symptom severity being associated with greater comorbidity (Kessler et al., 2005; Krueger, 1999), and with the similar dimensional findings for men and women across Forbes et al.'s (2015a, 2015b) later studies in particular.

Overall, these eight studies examined the relationships among depressive and anxiety symptoms and low sexual function in five different statistical frameworks. However, it is important to note that while the studies' findings are all *consistent* with the internalizing

hypothesis, they are by no means conclusive. For example, as we discussed in Part 2, the robust multifaceted relationships among the constructs could be accounted for by a variety of theoretical models, including causal relationships between internalizing disorders and sexual dysfunctions. Forbes et al. (2015a) is the only study to have tested the extended internalizing model using longitudinal data, and found no evidence for unidirectional causal relationships, in line with existing longitudinal studies of the relationships among internalizing symptoms and sexual function (e.g., Atlantis & Sullivan, 2012; Peleg-Sagy & Shahar, 2013). As such, the extant literature is more closely aligned with a shared common causes model (i.e., including sexual dysfunctions in the internalizing spectrum), rather than causal relationships accounting for their interrelationships.

To solidify the grounding of this hypothesis, the potential for unidirectional causal relationships should also be examined on shorter and longer time scales. For example, causal relationships could exist on a momentary time scale where emotional stress causes difficulties in becoming aroused, or on a larger time scale where years of chronic sexual dysfunction eventually foster depression or anxiety. The net effects of these time scales are included in extant research, but in order to disentangle the relationships, these models should be examined using data from ecological momentary assessment and longitudinal epidemiological research, respectively. Future research on these questions need not use a SEM framework, but could also use mixed models, longitudinal panel modeling, or factor mixture models (i.e., to allow for groups with different structural relationships; cf. Forbes et al., 2015b), and should focus on elucidating these relationships for men in particular.

As we have alluded to throughout Part 3, these eight studies are also limited by their reliance on convenience samples of predominantly young, educated, and healthy participants, as well as their reliance on self-reported measurement of the constructs of interest. The use of non-clinical samples means that low sexual function was not necessarily accompanied by the clinically significant distress that is needed to make a diagnosis of a sexual dysfunction, and may have been an adaptive response to other factors (e.g., social or environmental context) for some individuals. However, the use of non-clinical samples is also a strength: To date, there has been very little research on these disorders in non-clinical samples, as most studies begin with a base population of participants with diagnosed sexual dysfunctions or depressive or anxiety disorders (Laurent & Simons, 2009). Research that relies on clinical samples is vulnerable to Berkson’s bias (Berkson, 1946), which states that comorbid cases are more common in clinical samples than the population at large, even if the probability of seeking help is independent of disorder severity. These high comorbidity rates are associated with elevated distress, and, importantly, with restricted range in symptom variation due to the nature of the exclusively high-symptom sample (see Edwards, 1976). The inclusion of a broad range of symptom levels (e.g., Forbes et al., 2015a, 2015b) is vital to understand the nature of the relationships that describe the covariation among disorders, so an investigation of these relationships in diverse non-clinical samples provides particularly useful information. The dimensional structure of psychopathology has been found to have high levels of similarity between clinical and non-clinical samples (O’Connor, 2002). As such, the extension of the internalizing spectrum in a sample of participants across a variety of symptom levels offers important insights into the relationships between the disorders, and is relevant to both epidemiological and clinical research.

Another limitation of these eight studies was that while they examined the most common experiences of low sexual function (McCabe et al., 2016), and the five of seven core common internalizing disorders (e.g., Krueger, 1999; Krueger et al., 1998), they did not measure delayed ejaculation, and could not provide divergent evidence that sexual problems are part of the internalizing spectrum specifically, as opposed to, say, the externalizing or thought disorders spectra. As such, future research should include clinical samples and epidemiological samples, as well as broader measurement of psychopathology and sexual function.

In short, the eight studies that have tested whether low sexual function might be an indicator of the internalizing spectrum have found empirical support for this model. These studies, together with the literature reviewed in Part 2, thus provide robust evidence that sexual dysfunctions have a clear place in the meta-structure of psychopathology as part of the internalizing spectrum, and this has important implications for research and practice, as discussed below. The findings of these studies also highlight that the *DSM*—and the *International Classification of Diseases* (World Health Organization, 2008)—do not accurately represent the interrelationships between the disorders. Accurate models of these relationships are important precursors for the development of an empirically based nosology, sensitive diagnostic protocol, and efficacious treatment programs. As such, the internalizing spectrum —as part of the HiTOP framework (Kotov et al., under review)— offers an alternative empirically supported model that provides a springboard to bring sexual dysfunctions into the broad psychopathology research framework.

Part 4: Implications and Future research

Reconceptualizing sexual dysfunctions as part of the internalizing spectrum and including them in HiTOP research could have exciting implications for sex research. For example, if low sexual function were recognized as an indicator of internalizing psychopathology, it might hopefully follow that population studies of mental health would be revised to include measures of sexual function. The lack of epidemiological data on sexual dysfunctions together with common mental disorders has been a big factor perpetuating the absence of sexual problems in psychopathology research, and good quality data on these relationships would be invaluable for sex researchers. This move could also help integrate sex research into mainstream psychology research, where sexual experiences are often only considered as of peripheral importance, as side effects or symptoms of ‘primary disorders’ (e.g., APA, 2013).

An additional benefit of including sexual problems in the new hierarchical and dimensional taxonomy of psychopathology would be to offer sex researchers a dimensional alternative to the categorical *DSM* model. This is important because sexual function is continuous (i.e., it does not naturally separate into discrete categories of ‘function’ and ‘dysfunction’), and phases of sexual response often overlap (Carvalho et al., 2012; Carvalho et al., 2011; Corona et al., 2015; Giles & McCabe, 2009). In contrast, the *DSM* deems sexual dysfunctions as either present or absent, and implies that low levels of arousal (e.g., Female Sexual Interest/Arousal Disorder, or Erectile Disorder) are categorically distinct from orgasmic function (e.g., Female Orgasmic Disorder, Premature [Early] Ejaculation, or Delayed Ejaculation). A

move toward empirically validated models of characterizing low sexual function is also consistent with the *National Institute of Mental Health*'s move away from funding research based on *DSM* categories (Insel & Lieberman, 2013), and allows for the integration of sex research in the corresponding movement toward transdiagnostic treatment protocols, as well as proposed directions for neuroscience (i.e., focusing on the RDoC; Insel et al., 2010) and developmental psychology research (Forbes et al., 2016).

The finding that sexual problems likely share the underlying internalizing spectrum with depressive and anxiety disorders also has important implications for the diagnosis of sexual dysfunctions. Screening for depression and anxiety in presenting sexual dysfunction is already emphasized as important practice (Althof et al., 2005; APA, 2013). However, the reverse is not true, and sexual problems are underrecognized and undertreated (Bossini et al., 2014), despite their prevalence and impact. The presence of a shared transdiagnostic spectrum means that a history or current presentation of depressive or anxiety symptoms acts as an indicator to screen for distressing low sexual function. As mentioned earlier, this is a particularly important pattern of comorbidity to screen for, as it is associated with more chronic and severe symptoms across the board, more distress, resistance to treatment, poor long-term outcomes, and higher rates of suicidality (Bossini et al., 2014; Dell'Osso et al., 2009; Dobkin et al., 2006; Laurent & Simons, 2009; Michael & O'Keane, 2000). If our nosologies explicitly recognized the shared liability between low sexual function and other indicators of the internalizing spectrum, this could formalize the screening process and overcome some of the discomfort associated with initiating a conversation about sexual difficulties in primary care (Bossini et al., 2014).

These relationships also have implications for treatment. The high rates of comorbidity and shared treatment response between sexual dysfunctions and depressive and anxiety disorders undermine the efficacy and logic of treating them separately (cf. Nolen-Hoeksema & Watkins, 2011). The shared cognitive and emotional processing abnormalities noted earlier also highlight the exciting potential for efficacious transdiagnostic treatment programs that target the disorders concurrently. For example, transdiagnostic CBT programs have been developed that reduce the internalizing liability by targeting the shared mechanisms between depression and anxiety (e.g., Ellard, Fairholme, Boisseau, Farchione & Barlow, 2010; Kushner et al., 2013). The Unified Protocol is a transdiagnostic CBT program that distills the common principles of CBT treatment protocols for individual depressive and anxiety disorders, and includes many of the same techniques that are used to treat sexual dysfunctions, such as psychoeducation, mindfulness training, challenging maladaptive cognitions, and identifying and preventing avoidance (Barlow et al., 2010). To our knowledge, there are currently no transdiagnostic CBT programs to treat the comorbid presentation of depressive and anxiety disorders with sexual dysfunctions, so the extension of existing transdiagnostic CBT protocols could be a productive area for future research. It is likely that pharmacologic treatment will be an effective addition to these programs, and this should be investigated, as studies have suggested that neither medication nor psychotherapy alone is maximally effective for treating low sexual function (Althof et al., 2005; Heiman, 2002).

Of course, before transdiagnostic programs can be applied to sexual dysfunctions, the precise nature of the underlying mechanisms between the disorders will need to be investigated so that targeted treatment programs can be developed. As such, perhaps the most important area for future research is to define the transdiagnostic mechanisms between the disorders, and subsequently to determine whether they can be targeted effectively using transdiagnostic treatment (cf. Kessler & Price, 1993). To date, research on the overarching comorbidity between internalizing disorders and low sexual function has been based on a psychiatric epidemiology approach that focuses on the latent structure of the relationships and on the spectra of psychopathology (cf. Krueger & Markon, 2006). In order to extend this research to efficacious interventions, it will be essential to combine this approach with a focus on how the shared spectra manifest in specific cognitive, affective, and behavioral mechanisms (Harvey, Watkins, Mansell & Shafran, 2004). The shared characteristics found across internalizing disorders and low sexual function (e.g., distraction, worry, guilt, negative automatic thoughts, emotion dysregulation, rigid assumptions, avoidance; Carvalho et al., 2012; Nobre & Pinto-Gouveia, 2009) have been researched extensively in depression and anxiety, and Nobre and colleagues have begun research into these characteristics in sexual dysfunctions. However, the role of these characteristics has not been examined in comorbid disorder presentation. If they prove to be responsive to experimental interventions, as preliminary experimental results suggest may be the case (e.g., Kuffel & Heiman, 2006; ter Kuile et al., 2010), this could potentially lead to the development of transdiagnostic treatment programs (cf. Kessler & Price, 1993).

Lastly, the relationship between low sexual function and the internalizing spectrum highlights the possibility that other types of sexual behavior and experiences could be integrated into a hierarchical and dimensional model of psychopathology. For example, Eaton et al. (2015b) and Rodriguez-Seijas, Arfer, Thompson, Hasin, and Eaton (under review) have highlighted that sex-related drug and alcohol use may form part of the externalizing spectrum. The externalizing spectrum could also be related to other risky sexual behaviors, compulsive sexual behaviors, atypical sexual interests and behavior (e.g., attraction to sexual violence), or paraphilic disorders, as many share the characteristics of disinhibition and behavioral dyscontrol that characterize the externalizing spectrum (Justus, Finn & Steinmetz, 2000; Kafka, 2001; Testa & Collins, 1997; Wilson, 2010). Future research that integrates the range of sexual behavior and experience within the broader transdiagnostic framework would be a valuable avenue for future research.

Conclusion

Depression, anxiety, and sexual problems are highly prevalent. When combined, these phenomena have a profound negative impact, are resistant to treatment, and are associated with poor long-term outcomes for patients. A shared latent liability between the disorders evidently accounts for their comorbidity and interrelationships over time, and this finding offers the valuable opportunity to integrate sexual dysfunctions into an empirically derived taxonomy of psychopathology through the internalizing spectrum. Ultimately, these relationships can form the basis of new classification systems —evidence based nosologies — upon which empirically supported assessments and intervention can be built.

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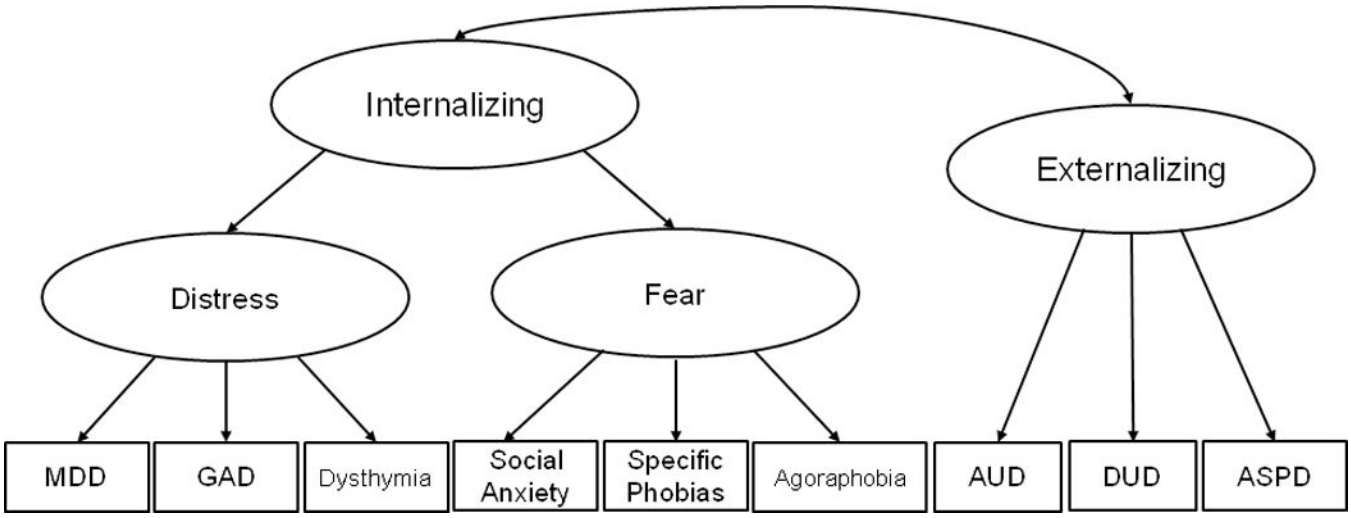

Figure 1.

An example of the internalizing-externalizing model of common mental disorders based on Krueger (1999). MDD = major depressive disorder; GAD = generalized anxiety disorder; AUD = alcohol use disorder; DUD = drug use disorder; ASPD = antisocial personality disorder. In this model the rectangles represent observed variables (e.g., DSM diagnoses rated as present or absent), which act as indicators for the latent variables (i.e., the ellipses). Each latent variable represents the shared variance among its indicator variables, and these relationships are represented by the single-headed arrows. Double-headed arrows represent correlations between variables.

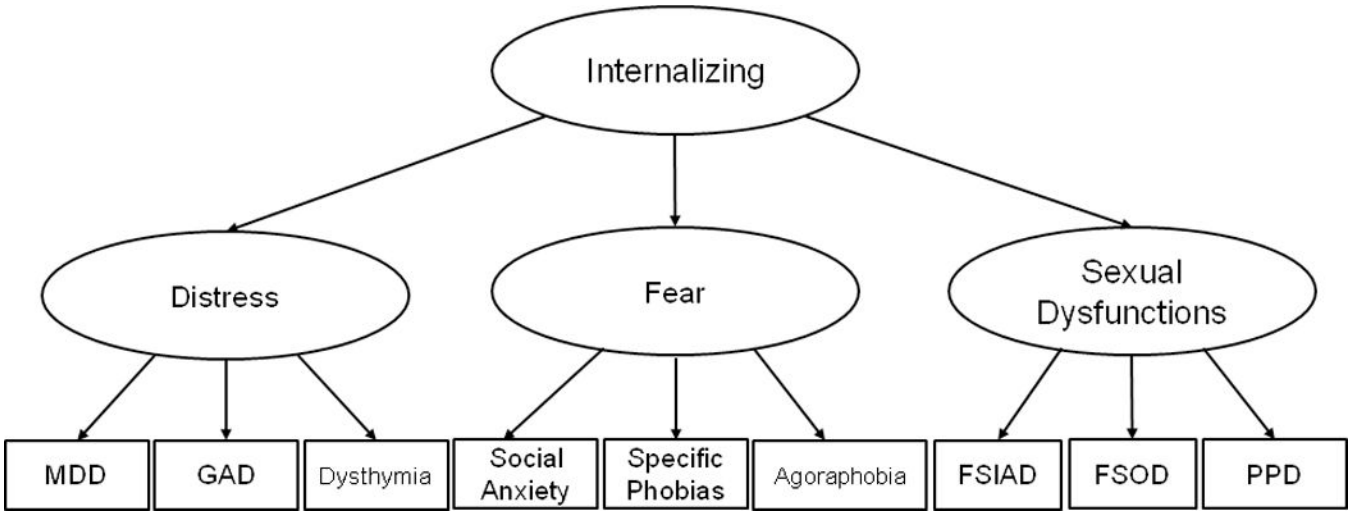

Figure 2.

An example of a model of the Internalizing spectrum that includes female sexual problems, reflecting the findings of Forbes and Schniering (2013) and Forbes et al. (2015a, 2015b, 2016). MDD = major depressive disorder; GAD = generalized anxiety disorder; FSIAD = female sexual interest/arousal disorder; FSOD = female sexual orgasm disorder; PPD = genito-pelvic pain/penetration disorder. As for Figure 1, the rectangles represent observed variables (e.g., DSM diagnoses rated as present or absent), which act as indicators for the latent variables (i.e., the ellipses). Each latent variable represents the shared variance among its indicator variables, and these relationships are represented by the single-headed arrows. Double-headed arrows represent correlations between variables.