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Review

Psychiatric disorders, psychopharmacology and sexual dysfunction from the fifth international consultation on sexual medicine (ICSM 2024)

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Abstract

Introduction: The World Health Organization estimates that one in eight people around the world is living with a psychiatric disorder. An under-recognized consequence of psychiatric disorders is the detrimental impact they may have on sexuality. Psychotropic medications to treat psychiatric disorders can also have a significant, negative effect on sexual functioning.

Objective: This review is a consensus International Consultation on Sexual Medicine (ICSM) report developed to outline and describe these impacts.

Methods: The committee members first discussed the structure of the manuscript. Literature searches were conducted for each section, seeking key words related to sexual dysfunction and each specific psychiatric condition and for psychiatric medications and classes of drugs. A modified Delfi approach was used when making decisions on content and recommendations. The content and recommendations were presented at ICSM where experts across the field of sexual medicine provided feedback. These comments were reviewed by the co-chairs of the 24 committees for discussion and feedback.

Results: In relation to psychiatric disorders and sexual dysfunction, there are two primary themes evident in the literature, (1) there are significant methodological weaknesses, and (2) the prevalence of sexual dysfunction across all psychiatric disorders is generally high and elevated compared to controls (when available). Additionally, sexual dysfunction associated with psychotropic medications is a frequent treatment-emergent side effect, for antidepressants ranging from 25% to 80% and for antipsychotics ranging from 16% to 60% depending on the underlying mechanism of action of a particular medication. Sexual dysfunction is also associated with many of the co-morbid conditions related to a psychiatric diagnosis.

Conclusion: The committee outlined three recommendations which highlight the importance of assessing for sexual dysfunction in those who report psychiatric disorders, evaluating sexual dysfunction across treatment while treating these disorders with psychotropic medication, and recognizing that the causes of sexual dysfunction are often multi-factorial.

Keywords: psychiatric disorder; sexual dysfunction; psychotropic medication; anxiety; depression; psychotic disorder.

Recommendations

- **Recommendation 1:** Clinicians should consistently assess for sexual dysfunction in patients who are diagnosed with psychiatric disorders (Strong Recommendation).
- **Recommendation 2:** Clinicians should be aware the etiology of sexual dysfunction in patients with psychiatric disorders is multifactorial. While much of the etiology remains unknown and speculative, sexual dysfunctions in these patients may arise from the psychosocial implications and/or the biological aspects of the psychiatric disorder, as well as the medications used to treat the psychiatric disorder (see Section 2). Sexual dysfunctions may also arise from physical or medical aspects unrelated to the psychiatric disorder (Strong Recommendation).
- **Recommendation 3:** Clinicians should be alert to the risk of sexual dysfunction related to particular psychotropic drugs at the stage of treatment planning and in longitudinal follow-up of changes observed in sexual functioning during the course of the therapy (Strong Recommendation).

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Introduction

The World Health Organization (WHO) characterizes psychiatric disorders as "clinically significant disturbance in an individual's cognition, emotional regulation, or behaviors . . . [which] is usually associated with distress or impairment in important areas of functioning.[1]" WHO estimates that one in every eight people around the world are living with a psychiatric disorder.[1] An under-recognized consequence of psychiatric disorders is the detrimental impact they may have on sexuality. Sexual dysfunction (SD) is common in those with psychiatric disorders.[2] It can encompass a spectrum of specific dysfunctions including hypoactive sexual desire disorder, erectile disorder, female orgasmic disorder, premature ejaculation, delayed ejaculation, dyspareunia, and vaginismus. SD often leads to emotional distress and interpersonal relationship concerns, which only reinforce mental health conditions.[3]

The first section of this article reviews the literature investigating SD in those with psychiatric disorders. There are two primary themes that run through this literature: (1) there are significant methodological weaknesses, and (2) the prevalence of SD across all psychiatric disorders is generally high and elevated compared to controls (when available). In terms of methodological concerns, the studies of SD in psychiatric conditions are few and far between. There are several reasons for the scarcity of these studies, such as lack of interest from mental health researchers, difficulties obtaining a "pure" sample of one psychiatric condition without co-morbid other psychiatric disorders, and some psychiatric disorders (e.g, personality disorders [PDs]) which are frequently studied in terms of traits or dimensions rather than strictly defined diagnoses. Other methodological concerns include the lack of comparison groups in many studies, poor and inconsistent assessment of sexual functioning/limited use of valid instruments, and small sample sizes.

The second section reviews the literature on the impact of psychotropic medications, and co-morbid conditions related to a psychiatric diagnosis, on sexual functioning. SD associated with various psychotropic, and other medications, is a frequent treatment-emergent side effect depending on the underlying mechanism of action of a particular medication.

The third section briefly outlines the issues of management of SD in patients with psychiatric illness.

Despite methodological concerns, the conclusion which does emerge across psychiatric disorders is the detrimental impact psychiatric conditions can have on sexual functioning. While the rates and specific sexual impact vary between psychiatric disorders, the overall prevalence of SD across these psychiatric disorders leads to the recommendations stated above.

Methods

The committee members first discussed the structure of the manuscript. Literature searches were conducted for each section, seeking key words related to SD and each specific psychiatric condition and for psychiatric medications and classes of drugs. A modified Delfi approach was used when making decisions on content of the sections and recommendations. The content and recommendations were presented at the International Consultation on Sexual Medicine where experts across the field of sexual medicine provided feedback and comments. These comments were then reviewed by the co-chairs of the 24 committees for continued discussion and feedback. The final result is a narrative review which outlines the constructs described.

Section 1: Sexual dysfunction with specific psychiatric disorders

In the first section, we briefly review the literature related to SD in common psychiatric disorders and discuss possible etiology. Spontaneous reports of SD in clinical trials and practice grossly underestimate the prevalence of SD related to psychiatric disorders and medications that treat them. To more accurately evaluate SD, we recommend using scales specifically developed and validated in patients with psychiatric disorders. Two suggested scales are: the Arizona Sexual Experiences Scale (ASEX)[4] and the Changes in Sexual Functioning Scale (CSFQ).[5,6] The FDA draft guidance/Consensus Statement[7] on design of clinical trials to demonstrate differences in sexual functioning for psychotropic medications, specifically supports the use of the ASEX or CSFQ for this purpose and that the development of additional scales are not necessary. The ASEX uses a dichotomous analysis (cut-off score for presence or absence of SD), while the CSFQ validation supports both dichotomous and continuous analyses. Some studies of disorder-related SD use these scales, and nearly all studies evaluating sexual side effects of psychotropic medication utilize ASEX or CSFQ data. While these scales are suggested to assess SD, as we review psychiatric disorders, it is acknowledged that there are various patient reported outcomes and clinical assessments to assess these conditions.

Depression

Major Depressive Disorder (MDD) is characterized by both physical and psychological symptoms, such as depressed mood, diminished interest/pleasure, sleep disturbances, low self-esteem, fatigue, and decreased libido.[8,9] MDD affects approximately 12% of the population,[10] and in 2012 WHO declared depression a global crisis.

Sexual dysfunction in depression

Patients with depression exhibit a higher prevelance of SD than the general population.[2] The bidirectional association between depression and SD was confirmed by Atlantis and Sullivan,[11] with a 50% to 70% increased risk of SD in those with depression and a 130% to 210% increased risk of depression in those with SD. In a recent systematic review, Gonçalves et al.[12] reviewed 12 cross-sectional studies of subjects diagnosed with MDD from seven different countries with study sample sizes ranging from 24 to 1637. The authors found a high prevalence of various SDs, including problems with desire/drive (29% to 85%), erections (18% to 46%), lubrication (18% to 79%) and orgasm (81%).

Men with MDD commonly experience reduced sexual desire, with 25% also reporting erectile dysfunction (ED) and association with premature ejaculation.[13] Among 40 women of reproductive age with MDD who had not started antidepressant medication, a study by Sreelakshmy et al.[14] found a 90% prevalence of SD. While Thakurta et al.[15] found in a sample of 36 women and 24 men diagnosed with MDD a prevalence of SD of 75% in women and 67% in men, the most common was decreased interest in sexual relations.

The complex and unclear etiological mechanisms linking depression and SD stem from the heterogeneous nature and overlapping symptomatology of both conditions.[11] Although treating depression may improve SD, it is not always guaranteed, and antidepressant therapies, notably selective serotonin reuptake inhibitors (SSRIs), can cause or exacerbate SD (see Section 2). Studies indicate the incidence between 30% and 50% of patients treated with an antidepressant medication experience some form of sexual impairment,[16,17] affecting all phases of the sexual response cycle; men usually report adverse effects with desire, orgasm, and arousal, and women more frequently report decreased arousal.[2,13]

Bipolar disorder

Bipolar Disorder (BD) is characterized by mood swings alternating between periods of mania or hypomania and episodes of depression. BD occurs in approximately 2.5% of the population.[8]

Sexual dysfunction in bipolar disorder

While some bipolar patients report increased sexual interest during mania or hypomania, the limited research in this area suggests BD can have significant negative implications on sexual functioning.[8] Dell'Osso et al.[18] studied 82 men and women with BD compared to 101 controls, and reported the lifetime impairment in sexual function was significantly higher in the BD patients. In this study, over 40% of BD patients reported SDs, specifically in problems with sexual interest/arousal and achieving an orgasm. Namli et al.[19] compared 50 patients, both men and women, with BD to 50 healthy controls, and estimated over half of the patients with BD experience SD. Sorensen et al.[20] who studied 61 women with BD, found 25% reported at least one SD, 54% of the group was sexually distressed, and 39% were unsatisfied with their sexual life.

The severity of the BD also seems to be related to an increased chance of SDs. In a recent study, Dargel et al.[21] investigated sexual functioning in a sample of 80 BD patients, both men and women, who were in remission on stable pharmacological treatment for at least three months compared to 70 healthy controls. The BD patients reported significantly lower sexual functioning scores compared to the healthy controls. Additionally, the chance of SD doubled for each increase in the number of suicide attempts, and the chance of SD increased by 60% for each additional hospitalization. Depressive symptoms were related to SD as the odds of orgasmic disturbances increased by 60% while the odds of sexual interest/frequency dysfunctions increased by 20% with higher levels of subthreshold depressive symptoms.

Aspects of BD such as affective symptoms, age at onset, BD type, and predominant polarity may be related to sexuality.[21] Dargel et al.[21] highlight systems related to BD such as the hypothalamic–pituitary–adrenal (HPA) axis, the amygdala, and the serotonergic, noradrenergic, dopaminergic neurotransmitters all of which are implicated in the biological systems of desire, arousal, and erection/ejaculation.

Anxiety

Anxiety is a complex response to stress or perceived threats, characterized by fear, uneasiness, and worry. Anxiety disorders involve persistent and excessive worry, significantly disrupting daily functioning. WHO estimates that 4% of the world population currently experiences an anxiety disorder.[22]

Sexual dysfunction in anxiety

Anxiety profoundly affects sexual functioning, presenting challenges related to sexual desire, arousal, and satisfaction in both men and women. For instance, manifestations include decreased libido, ED in men, and issues like vaginal dryness and pain in women.[23] Anxiety's effects on sexual arousal are significant. Through increasing activity in the sympathetic system, anxiety can distract individuals from erotic sensations, inhibiting their arousal.[24,25] This can reflect on men by having erection problems and on women through decrease in lubrication and blood flow to the clitoris. The cycle of anxiety-driven SD leading to performance anxiety leading to anticipatory dysfunction is a contributing factor in arousal disorders.[26] This co-presence is frequently seen in clinical practice, with patients often presenting with both SDs and anxiety disorders, making it challenging to identify the primary disorder.[23]

Anxiety triggers the release of stress hormones, such as cortisol and adrenaline, influencing blood flow, muscle tension, and overall physiological arousal.[27] The fight or flight state caused by anxiety negatively involves the arousal and orgasm phases, potentially interfering with sexual desire.[28,29] Psychologically, negative thought patterns, self-doubt, and fear around performance associated with anxiety create mental barriers to sexual enjoyment. Additionally, the strain that anxiety places on relationships contributes to a cycle of increased stress and further exacerbation of sexual difficulties. This necessitates a holistic approach considering biological, psychological, and social factors to enhance overall well-being and foster healthier relationships among professionals.

Obsessive compulsive disorder (OCD) and body dysmorphic disorder (BDD)

In the Diagnostic and Statistical Manual of Mental Disorders (DSM-5-TR), OCD and BDD are recognized as within the diagnostic category of obsessive-compulsive and related disorders. OCD is characterized by the presence of obsessive (intrusive, repetitive, or unwanted) thoughts and compulsive behaviors or mental acts.[30] A meta-analysis of 34 studies showed a prevalence of 1.1% in the general population, with women 1.6 times more likely to experience OCD compared to men.[31] BDD involves preoccupation with one or more perceived defects or flaws in physical appearance that are not observable or appear slight to others.[30] BDD has a lifetime prevalence of approximately 2% with an almost equal distribution among genders.[30]

Sexual dysfunction in OCD and BDD

In a recent metanalysis of 24 studies, the prevalence of SD was between 25% and 81% in OCD patients.[3] Forty to 73% reported low sexual satisfaction. Although desire and arousal difficulties have been reported in most studies, the most common complaint was orgasm difficulties by 24% to 44% of patients. Limitations include the small number of studies, small sample sizes, and the use of multiple questionnaires (some not validated), which may contribute to bias. One study showed the frequency of SD in women with OCD, assessed by the Female Sexual Function Index (FSFI), to be 81%, and 25%, assessed with the International Index of Erectile Function (IIEF) in men with OCD.[3,32] BDD may involve any part of the physical appearance such as the muscles, the nose, breast size, etc., but of specific interest is genital dysmorphic

disorder, which involves preoccupation and an altered perception of the size and shape of the genitals, which causes shame and distress.[33] Although the relationship of BDD with SD is poorly studied, this population is likely to experience low sexual satisfaction and arousal difficulties.[34,35]

The association between OCD and SD is not well understood but has been suggested to be bidirectional. Vulnerability to anxiety may play a role in the pathogenesis and maintenance of both SD and OCD. Specific OCD symptoms, i.e, obsessions related to contamination, religion, or sexuality, along with emotions like disgust, and doubting and checking behaviors, can interfere with sexual function.[3,32] Additionally, co-morbid depression and medication side effects may further link SDs with OCD.[3,32] SD in BDD is mainly due to feelings of shame and distress with body appearance during sexual activities.[3,32,34,35]

Post-traumatic stress disorder

Post-traumatic stress disorder (PTSD) can occur following a traumatic event which threatens one's life or that of another, or seeing someone die/dead. PTSD is characterized by symptoms of intrusive thoughts, flashbacks, and avoidance of places or activities related to the traumatic event.[8] It is estimated that 3.9% of the world's population will experience PTSD during their lifetime.[36]

Sexual dysfunction in PTSD

SDs are frequently reported by those with PTSD.[37] For example, Bentsen et al. (2015) conducted a systematic review of publications including male veterans with PTSD and multiple measures of SD. The authors reviewed 11 studies, 5 case–controls, and 6 cross-sectional studies. While there were various methods used, the authors were able to estimate SD was observed in 60% to 80% of these men.[38] Other studies that examined war veterans showed a link between PTSD and ED or sexual pain.[38–40] PTSD has been linked to SDs regardless of the trauma. PTSD is often associated with physical and sexual abuse, and not surprisingly, studies have found that women who had been sexually abused suffer from SDs and dissatisfactions.[41,42] In a study of 372 female sexual assault survivors compared to 99 women with no sexual assault history, where all women completed the Sexual Arousability Inventory, 59% of women with a sexual assault history reported SD compared to 17% of women without a sexual assault history. Of the 59% with a sexual assault history who reported SDs, 71% reported that experiencing sexual assault was related to the development of their sexual problems.[41] Additionally, sexually abused men have demonstrated increases in ED, premature ejaculation, and low sexual desire.[43]

Each of the symptoms of PTSD can be linked to its effect on sexual function. Intrusive thoughts such as dreams, flashbacks, or memories can resurface when a traumatized individual is having a sexual encounter, reminding them of the experience and the abuser.[44,45] As a result of this intense emotional arousal, SDs such as avoidance or fear of sex, low libido, and low arousal may become ways of coping.[44,45] The physical changes in arousal and reactivity can also impact sexual function.[37] A hyperarousal state can make it difficult for individuals to be mindful of pleasure during sexual activity. It can also heighten sensations of pain or discomfort. In time, sex may start to be associated with tension and/or anxiety.[46,47]

Compulsive sexual behavior disorder (CSBD)

The WHO recently introduced Compulsive Sexual Behavior Disorder as the new clinical entity in the ICD-11. CSBD includes features such as a persistent pattern of failure to control intense, repetitive sexual impulses or urges resulting in repetitive sexual behavior.[48] These individuals may engage in sexual behaviors despite knowing they cause physical, emotional, or interpersonal problems.[49–51] Research on the epidemiology of CSBD is still evolving as the diagnostic guidelines were recently released, however, it is estimated that 4.9% of men and 3% of women report experiences fulfilling the ICD-11 requirements for lifetime diagnosis. ICD-11 criteria-based questionnaires such as CSBD-19 or CSBD-DI may be used for screening purposes.[52,53] As some individuals may present clinically with guilt and shame over masturbation, pornography use, or other kinds of sexual expression that is not accepted due to restrictive moral attitudes, it is important to acknowledge that distress that is entirely related to moral judgments and disapproval about sexual impulses, urges, or behaviors is not sufficient to meet diagnostic requirements.[48] The frequency of sexual behavior or quantity of pornography use alone are also not sufficient indicators of CSBD and should not be overused in making clinical diagnoses.

Sexual dysfunction in compulsive sexual behavior disorder

While the inability to control sexual behaviors can lead to negative feelings of guilt and shame, preventing a healthy sexual self-image,[54] the impact of CSBD on sexual functioning is controversial. While some authors proport CSBD is associated with ED when accompanied by excessive pornography use,[55] there is no specific evidence to support this claim. Although patients who compulsively masturbate may report difficulties in attaining genital arousal during solitary or partnered sexual encounters, these are likely related to a physiological state of refractoriness and should not be misinterpreted as "pornography-induced sexual dysfunction" that is commonly mentioned in popular media, but not appropriately evidence-based.[56] Based on cross-sectional studies, ED was linked to self-reported problematic pornography use defined as impaired control over the behavior despite negative consequences in important areas of life, including work and relationships,[57,58] rather than to the frequency of pornography use or its variability.[59–61] Delayed ejaculation was reported in 30% of a small sample of hypersexual men who masturbated more than 7 hours a week.[62]

Studies have also shown co-morbidity between CSBD and paraphilic disorders in up to 30% of the studied samples.[63–65] In these cases, both can increase the clinical severity the patient is experiencing, causing further distress and increasing forensic risk,[66,67] particularly in paraphilic disorders such as sexual sadism, pedophilic, and exhibitionistic disorders.[66] It is essential to note that while the two disorders, CSBD and paraphilic disorders involve intense sexual urges, CSBD is characterized by conventional sexual preferences or inclinations.[50,63,68] For risk assessment and prevention purposes, it is relevant that CSBD was found predictive for both sexual violence towards one's sexual partner and sexual re-offending in male criminal samples.[69–71]

Compulsive Sexual Behavior has been linked as a coping strategy for negative emotional states such as depressive mood, anxiety, or boredom.[56,63,72,73] Moreover, many sexually compulsive individuals are also suffering from

co-morbid anxiety disorder, depression, Attention-Deficit/Hyperactivity Disorder (ADHD), or substance use disorder (SUD).[74] The proposed mechanisms for CSBD suggest there is an imbalance between sexually excitatory (i.e, dopaminergic) and inhibitory (i.e, serotonergic) factors.[75–79] Dysregulation of emotion may also contribute to reduced inhibition and difficulties in sexual self-control.[73]

Personality disorders

A PD "is an enduring pattern of thinking, feeling, and behavior" manifested in disturbances of cognition, affective reactivity, interpersonal functioning, or impulse control. The overall median prevalence of PDs is reportedly 2.5% to 10.5% depending on the PD.[8]

Sexual dysfunction in personality disorders

SD in PDs has not been well studied, which limits the ability to provide prevalence estimates for PDs in general and also limits the conclusions that can be drawn from the literature. Grauvogl et al.[80] investigated whether various PDs are associated with SD in a sample of 188 women aged 18-25 years. Women with sexual problems reported significantly more cluster A (specifically schizoid) and cluster C (specifically avoidant and obsessive-compulsive) PDs than women without sexual problems. In addition, a higher level of cluster A (namely schizoid) and a lower level of cluster B (namely antisocial PD and borderline personality disorder [BPD]) PDs were associated with lower levels of sexual functioning. The systematic review of 17 studies by Ciocca et al.[81] suggested that either the presence of a personality trait or a PD increases the likelihood of various sexual symptoms.

Three studies focused on sexual functioning in individuals with BPD. Zanarini et al.[82] compared 290 subjects with BPD (mostly white women) to 72 subjects meeting the criteria for at least one non-borderline PD. Of these, 61.0% of those with BPD and 19.4% of control subjects reported difficulties in sexually intimate relationships. Female BPD subjects were significantly more likely to report sexual relationship difficulties than male BPD subjects (though the number of male subjects was small). In a study of 45 women with BPD and 30 healthy women by Schulte-Herbruggen et al.[83] the BPD group showed a significantly higher prevalence of SD. Importantly, the impairment of sexual functioning in BPD subjects was best explained by a history of sexual abuse, as SD occurred in sexually traumatized BPD subjects and not in those BPD subjects without sexual trauma. The authors suggested that SD is not specific to BPD, but rather to a history of sexual trauma. Finally, Frias et al.[84] concluded in their comprehensive review that individuals with BPD display higher sexual identity disturbance, higher sexual impulsivity, and increased risky sexual behaviors.

Ciocca et al.[81] propose that maladaptive personality functioning, regardless of the type of PD, may manifest, among others, through problematic sexuality and sexual impairment. They noted that sexual compulsivity and sexually risky behaviors are typical, especially for antisocial and borderline PDs. In BPD, Zanarini et al.[82] and Schulte-Herbruggen et al.[83] both found that a pattern of sexual relationship difficulties in adults was significantly associated with a reported childhood sexual abuse history and an adult history of rape.

Schizophrenia

Schizophrenia is characterized as a mental illness with positive symptoms (delusions, hallucinations, and disorganized thoughts/speech), negative symptoms (restricted emotional expression and avolition), and cognitive impairment. The lifetime prevalence of schizophrenia is approximately 0.3% to 0.7%.[8]

Sexual dysfunction in schizophrenia

Two recent systematic reviews have pooled data to determine estimates of SD in patients with schizophrenia. Zhao et al.[85] conducted a meta-analysis of 10 observational studies including 1161 patients with schizophrenia compared to 2409 healthy controls, while Korchia and colleagues[86] reviewed 72 observational studies which included 21 076 patients with schizophrenia. The rates for all SDs in patients with schizophrenia for both genders ranged from 50% to 56%. For men, the rates ranged from 57% to 66%, and for women, from 60% to 61%. Zhao and colleagues[85,87] reported that both men and women with schizophrenia had a 2-fold greater risk of SD as compared with the healthy controls. Korchia et al.[86] estimated the prevalence of specific SDs in patients with schizophrenia as ED (44%), loss of libido in men (41%), ejaculation dysfunction in men (39%), orgasm dysfunction in women (28%), and amenorrhea in women (25%).

The etiology of SDs in schizophrenia is not well understood. In general, the cause is considered multifactorial potentially due to three primary reasons. First, the disorder itself may lead to SD. The disorder can include neurocognitive symptoms such as negative symptoms, decreased initiative, lack of motivation, social withdrawal, and the presence of co-morbid depression.[30,88] The second primary etiology may be the use of antipsychotic medications, which can have a significant impact on sexual functioning (see Section 2 for specific discussion). Third, SDs may be related to psychosocial factors or medical aspects that may arise from the condition. These may include addictions, decreased physical activity, sleep disorders, and unhealthy diets as well as chronic metabolic disorders (diabetes, hypertension, overweight)[89] and peripheral inflammation.[90]

Substance use disorder

SUD involves symptoms such as impaired control, physical dependence/withdrawal, social problems, and risky behaviors associated with using a substance despite its negative effects.[8] In the United States, approximately 16% of the population meets the criteria for having a SUD in a year.[91]

Sexual dysfunction in SUD

There may be a general misconception that alcohol, marijuana, opioids and stimulants improve sexual functioning because the antianxiety, and disinhibitory effects of these substances may increase risky sexual behavior, especially during early, intermittent use and in lower quantities.[92] However, long-term SUD/chronic daily use has consistently been associated with SD. In a recent scoping review (overview of existing research and any gaps), Ghosh et al.[93] identified 65 relevant articles, five prospective studies, and 60 cross-sectional studies. The majority of these studies were conducted in men and in alcohol and opioid use disorders. The five prospective studies, all in men, revealed a prevalence of SD of 75% in alcohol use disorder and 61% in opioid use disorders.

In women, Salari et al.[94] published a recent meta-analysis of studies focusing on alcohol use disorder and SD. This analysis included seven studies involving a total of 50 225 women and found a significant association between alcohol use disorder

and SD (odds ratio of 1.74). Kumar et al.[95] In a study of 40 women with alcohol dependence syndrome compared to 40 matched healthy controls, found that women with alcohol dependence syndrome reported lower scores in all sexual domains. Specifically, on the Sexual Dysfunction Checklist, these women reported low libido (55%), difficulty reaching orgasm (52.5%), and, if they did reach orgasm, assessment with the ASEX indicated it was often unsatisfactory (50%).

In cannabis and opioid use, Pizzol et al.[96] conducted a systematic review of cannabis use and the presence of ED assessed with IIEF-5 in 2019. These authors included five case–control studies, ranging in publication date from 2008 to 2017, with data from 3395 subjects (all men), 1035 using cannabis (smoking), and 2360 nonusers, and found a significantly higher prevalence of ED in cannabis users of 69% compared to 35% in controls (odds ratio = 3.83). However, the relationship between cannabis use and SD ultimately is unclear as the concentration of the active ingredient in cannabis (THC) is much higher today (2017) than during the early studies.[97] In terms of opioids, Zhao and colleagues[87] conducted a meta-analysis of 10 studies including 8829 men. In this analysis, 2456 men were using opioids and the remainder were non-opioid user controls. The use of opioids was significantly associated with an increased relative risk of ED of 1.96.

Substances of abuse can have significant impact on brain function and brain chemistry. These substances can impact the HPA axis; receptors in the paraventricular nucleus of the hypothalamus or receptors located in the corpus cavernosum; production of luteinizing hormone; and reduced androgen production by the adrenal glands. These effects can interrupt the hormonal milieu, with specific implications for the suppression of testosterone and estradiol.[92]

Neurodevelopmental disorders

Neurodevelopmental disorders (NDDs) usually manifest in early ages of childhood and are characterized by deficits in personal, social, academic, or occupational functioning. Two important NDDs are Autism Spectrum Disorder (ASD), characterized by impairments in social interactions and communication skills, as well as repetitive and stereotyped behaviors; and ADHD manifested by deficits in attention, cognition, mood, movement, and psychomotor activity.[8]

The prevalence of ASD is estimated at 1.7% of the general population, and like in other neurodevelopmental disorders, it affects mostly men,[98] while the prevalence of ADHD is estimated at 2.5% to 6.7% of adults.[99]

Sexual dysfunction in neurodevelopmental disorders

The literature in ASD, although scarce, reveals a significant proportion of individuals with ASD experience dissatisfaction in their relationships and sexual lives, as well as SDs.[100] In a systematic review that included 17 studies with 1939 individuals with ASD and 13 193 neurotypical peers assessed with various sexual functioning scales, Young & Cocalllis[100] reported significantly lower scores in sexual excitation and problems regarding desire, arousal, orgasm, satisfaction, pain, and lubrication in women with ASD compared to neurotypical peers. ASD men also reported more issues with erectile function and sexual intercourse satisfaction than their neurotypical counterparts, but significantly higher scores for sexual excitation.

The available literature on the relationship investigating ADHD and SDs presents mixed results. While Anderson-Schmidt et al.[101] who studied 32 ADHD subjects and 293 peers in the neurotypical control group and Hertz et al.[102] whose sample included 139 ADHD subjects and 67 subjects in the control group, did not find any significant difference in sexual function between ADHD subjects and controls, Bijlenga et al.[103] reported a significantly higher prevalence of SDs among 136 participants with ADHD compared to the 4147 subjects in the control group. The later study found that 39% of men and 43% of women with ADHD had SDs, compared to 17% of men and 20% of women in the control group. The most common SDs were orgasmic problems, premature ejaculation, sexual aversion, and negative emotions during or after sexual activity. In these three studies, the percentages of patients receiving treatment (psychotherapy or psychopharmacology) varied, with some patients not receiving any treatment.

Findings suggest that individuals with ASD and/or ADHD often see themselves as "different" in their sexual orientation, behavior, and experiences compared to neurotypical peers.[98,100] In ASD, the hypo- and hyper-sensitivities present may lead to the need for more intense sexual stimulation to become and stay sexually aroused and to reach orgasm.[100,104] The higher prevalence of some SDs in ADHD may be related to symptoms like distractibility, inattention, impulsivity, sensation seeking, and hyperactivity as well as characteristics such as low self-esteem, fear of intimacy, and poor relational skills. Additionally, the medicines prescribed for treating ADHD have been found to impair sexual functioning.[100,103]

Eating disorders

Eating disorders encompass anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder and other specified food intake disorders.[30] The lifetime prevalence is 1.69%, and they are much more common in women compared to men.[105] They are also more frequent in sexual and gender minority adults compared to cis gendered heterosexual adults.[106]

Sexual dysfunction in eating disorders

Studies on women with eating disorders indicate higher rates of SD than controls. In a study comparing 111 women with anorexia nervosa to 120 controls, the AN group scored lower in all FSFI subscales.[107] Another study, including 44 women with anorexia nervosa, 44 with bulimia nervosa, and 72 controls, found both AN and BN groups to score significantly lower in all FSFI subscales than controls.[108] A study comparing 242 women with eating disorders to normative data found that more women with eating disorders had a loss of libido, higher sexual anxiety, and detached relationships compared to controls. In the same study, loss of libido was almost 5 times greater in participants with restrictive anorexia nervosa and purging anorexia nervosa compared with the BN group.[109] Unfortunately, data on SDs in men with eating disorders is lacking.

Both eating disorders and SDs can stem from a sense of alienation and an inability to accurately perceive one's body.[107] In this respect, studies support that body image disturbance is the shared factor that explains both eating disorders and SDs. The cognitive distraction caused by the discomfort of showing one's body during sexual activity may be a factor behind the increased prevalence of SDs in

Table 1 Examples of factors involved in regulation of sexual functioning.

Biological: central nervous system, sympathetic and parasympathetic innervation, blood flow regulation, neuroendocrine system (sexual hormones, thyroid hormones), genetics (related to metabolism)
Psychological: general mood, the level of relaxation or anxiety (general and performance-related), self-image, positive and negative sexual experiences in the past, history of sexual trauma, stress
Interpersonal: quality of current and past relationships, differences in approach to sex, expectations in the dyadic relationship, perception of partner's attractiveness
Sociocultural: cultural, familial, and religious norms and views of sexuality

this group. Psychological factors that seem to mediate the presence of SDs in patients with eating disorders are interpersonal difficulties, disturbed intimacy, and insecure attachment style.[107] Eating disorders may be associated with amenorrhea, menstrual irregularities, and low estrogen levels. In addition, hormonal changes that occur because of weight changes may induce low sexual desire. SDs are interconnected with the psychopathology of eating disorders and therefore should be considered as a core aspect in the clinical management of persons with eating disorders.

Section 2: Psychopharmocology and sexual dysfunction

The second section reviews the literature on the impact of psychotropic medications on sexual functioning. It is important to obtain a baseline assessment of sexual function, preferably using a validated sexual function questionnaire such as the ASEX or CSFQ, to determine the impact of the psychiatric disorder on sexual functioning and to monitor longitudinal outcomes with the addition of psychopharmacologic treatment—both for efficacy and adverse effects such as SD. Determining patient preferences about what side effects to avoid (e.g, SD, weight gain, sleep disturbance, cognitive difficulties) is important for adherence and/or quality of life.

SD associated with various psychotropic medications[110–112] is a frequent treatment-emergent side effect, for antidepressants ranging from approximately 25% to 80%[111] and for antipsychotics ranging from 16% to 60% depending on the underlying mechanism of action of a particular medication.[110,112]

Regulation of sexual functioning, including impairment, is complex and derives from biological, psychological, interpersonal, and sociocultural factors.[113–117] Examples are presented in Table 1. SD is frequently the result of impairment of more than one of these factors.

Sexual functioning may be impacted at various levels and systems—central and peripheral nervous systems, neuroendocrine system (central and peripheral influence), or peripheral blood flow. All these factors can change over the lifespan, and some can also change across the menstrual cycle and reproductive status in women.[116]

Various neurotransmitter and hormonal systems exist in a dynamic balance between excitatory and inhibitory influences.[116,118] The excitatory factors include, among others, physiological and organic factors such as neurotransmitters (dopamine, norepinephrine, nitrous oxide), hormonal neuromodulators (oxytocin, vasopressin, melanocortins) and sex steroids (testosterone, estrogen) in combination with psychological and cultural factors. Sexual excitation involves dopamine and melanocortins which stimulate attention and desire, and noradrenaline and oxytocin which promote sexual arousal, all activated in the hypothalamus and limbic system in response to sexual cues and stimulation.[116] Sexual stimulation can be primed internally by steroids and or externally by drugs stimulating sexual function or sexual incentives.[116]

The inhibitory factors include neurotransmitters (serotonin), hormones (prolactin), hormone modulators (opioids), neuromodulators (endocannabinoids), again in combination with some psychological and cultural factors. These neurochemical inhibitory systems usually blunt the excitatory systems at the end of the sexual response cycle. However, these inhibitory systems could be also tonically activated by variables such as stress or drugs that augment their actions (e.g, SSRIs), or when excitatory systems are endogenously blunted, e.g, in hypogonadal individuals or in those taking drugs that inhibit the excitatory systems.[116]

It is important to note that this view of sexual excitation and inhibition on neurotransmitter and neuromodulator levels is fairly simplified and that there are many other neurotransmitters and neuromodulators that may affect sexual functioning, such as gamma-aminobutyric acid (GABA), prostaglandins, and adrenocorticotropic hormone (ACTH).

Peripheral regulation of sexual function is similarly complex. The clitoral and penile tissues are impacted by gonadal steroids, such as estrogen, progestin, and testosterone. These hormones influence bioavailability and function of each other.[114] Their secretion from peripheral gonadal tissue is under central control from the hypothalamus and pituitary gland. Genital sensation is influenced by serotonin. The vasocongestion is modulated by a number of substances such as nitric oxide, norepinephrine, prostaglandin E, vasoactive intestinal peptide (VIP), substance P, and cholinergic fibers. Orgasm can be inhibited through stimulation of 5-HT2 receptors by various substances. For some of these substances, bioavailability and adequate levels of hormones are required for their proper regulatory function. For instance, nitric oxide is known to be released from endothelium by stimulation and then activates production of cyclic guanosine monophosphate that relaxes penile smooth muscles. However, for nitric oxide to be fully effective it requires adequate levels of estrogen and bioavailable testosterone. Cholinergic nerves seem to be involved in the erectile process via several mechanisms.[110] For instance, they modulate adrenergic nerves that tend to decrease tumescence. They also facilitate the vasodilation response to nitric oxide.

The impact of hormones on sexual functioning is well-known, though not completely understood. For instance, the role of testosterone in sexual desire is significant. Interestingly, supraphysiological levels of testosterone in eugonadal men have a very little effect on their desire, while supraphysiological levels of testosterone increase sexual

desire in women.[114,116] However, it is unclear whether and what threshold level of testosterone is necessary for sexual desire. Prolactin's effect on central and peripheral regulation of sexual function is not fully understood and it is thought to involve dopamine via negative feedback. Hyperprolactinemia in men is associated with decreased desire and ED, and in women impacts the reproductive cycle and induces galactorrhea. Changes in thyroid hormones can also impact sexual function, as well as levels of circulating sex hormones.[114,116]

Regulation of sexual function can be negatively impacted at various levels: central and peripheral loci, hormones and neurotransmitters, and in different phases of the sexual response cycle. Medications and substances of abuse bind to various receptors or compete with various neurotransmitters involved in regulation of sexual function, for example, decrease in sexual desire via binding to subtypes of serotonin receptors, or by blocking dopamine receptors and thus increasing prolactin.[116] Alpha-adrenergic medications may negatively impact arousal. Serotonergic substances delay orgasm or may cause anorgasmia. Some mental disorders may be associated with impairment of sexual regulation via involvement of various neurotransmitters such as of serotonin in pathophysiology of depression and SD, or by the negative impact of the HPA axis hyperactivity in mood disorders and on sexual functioning. Understanding of both the underlying pathophysiology of sexual function regulation and mechanism of action of various medications and drugs of abuse is important for recognizing SD associated with medications and could serve as a guiding principle in management of medications and substances of abuse associated with SD.[116]

Explanation of mechanisms of drug-induced sexual dysfunction

To understand why psychotropic medication is so commonly associated with sexual side effects, it is necessary to understand the role of neural pathways and neurotransmitters in the regulation of human sexual response. A common theoretical explanation of how sexual responsivity is modulated between excitation and inhibition was described by Bancroft and Janssen (2000) and Perelman (2009).[119,120] Dopamine and serotonin are the two monoamines that play the most prominent role in neuronal activation and inhibition of sexual response, respectively. Dopamine release in the mesolimbic area mediates sexual desire, motivation, and attention towards cues that predict sexual rewards, while, in the hypothalamus, it activates the parasympathetic pathway that evokes and sustains genital arousal. Serotonin centrally mediates the physiological state of satiety and relaxation after orgasm and inhibits further dopamine release, while in the periphery, it inhibits spinal reflexes leading to prolongation of ejaculatory and orgasmic latency time.[116,121–123]

Mechanisms responsible for psychotropic medication-related problems in sexual functioning can be explained using the 3 Levels of Interaction Model illustrated in Table 1.

The first level represents a direct impact on SD via modification of the neurophysiological response to sexual cues/stimuli. Drugs that inhibit serotonin reuptake and increase serotonergic tone via 5-HT2 and 5-HT3 receptor agonism are likely to decrease sexual desire and delay or inhibit ejaculation and/or orgasm. Dopamine receptor antagonists are related to inhibition of desire and arousal/genital response. Some serotonergic and antidopaminergic medications characterized by additional 5-HT1a receptor agonism, 5-HT2a antagonism or partial dopamine agonism are related to a significantly lower risk of SD. Anticholinergic mechanisms are related to ED through reduction of peripheral vasodilatation while increased noradrenergic activity may adversely affect desire, arousal, and orgasm due to decreased neural activation of the caudate nucleus.[121,124–126]

The second level represents problems in sexual functioning as a consequence of medication adverse effects that are not directly related to physiological sexual response but rather to other symptoms that produce a sort of physical or mental discomfort influencing desire and/or ability to engage in sexual activity. Antidopaminergic drugs (1st generation and some of the 2nd generation antipsychotics) may cause life-disturbing extrapyramidal symptoms that are related to D2 dopamine receptor blockade in the nigrostriatal system. These side effects most commonly include akathisia (psychomotor restlessness), tremor and muscular stiffness (parkinsonism) or dystonia (involuntary muscle contractions). Tardive dyskinesia usually presents as involuntary facial movements (chewing-like, grimacing, buccolingual movements, tongue protrusion, or lip smacking) and may last years after drug discontinuation.[127,128] Apart from physical discomfort, anxiety, or pain related to these symptoms, they may also affect physicial appearance, self-esteem, and social interactions, including sexual and non-sexual partnered activities.

Drugs with anthistaminic properties (including 1st and 2nd generation antipsychotics and some antidepressants) are likely to cause sedation that often limits patients in undertaking a variety of activities.[129] One of many anticholinergic side effects (common for 1st generation antipsychotics and tricyclic antidepressants) is a decrease in saliva production (dry mouth) and vaginal dryness[130] that is likely to make kissing and penetrative sex less pleasurable. Serotonergic medications often cause feelings of emotional blunting or numbness where patients feel unable to engage emotionally or experience intense feelings and related/persistant anhedonia.[131] These experiences apply also to contacts with loved ones and diminished pleasure from sexual activity.

The third level represents other systemic consequences and long-term side effects of psychotropic medications. These especially include weight gain, metabolic syndrome/diabetes mellitus, and related cardiovascular risk factors that have been clearly linked with the pathophysiology of SD.[132–136] Particularly, antimuscarinic/anticholinergic antipsychotics and antidepressants such as tricyclics are linked with weight gain and related cardiometabolic complications. Dopaminergic blockade within the tubero-infundibular pathway is related to treatment-emergent hyperprolactinemia which is also identified as risk factor for SD.[137–139]

The stratification of risk and severity of treatment-emergent SD associated with particular psychotropic medications based on aggregated information from multiple studies and meta-analyses are presented in Table 2.

Antidepressant therapy

According to international, multi-center studies, it has been estimated that 27% to 65% of women and 26% to 57% of men treated for depression with SSRIs or SNRIs reported worsening of pre-existing difficulties or the emergence of new sexual difficulties in the early weeks of treatment.[140,141] Post-marketing studies have reported

Table 2 Mechanisms of psychotropic medication–related problems in sexual functioning—the 3 Levels of Interaction Model.

Level 1 Direct impact on sexual function	•Serotonergic agonists (5-HT2 and 5-HT3 receptors): inhibition of spinal reflexes and ↑orgasmic/ejaculatory latency time •Dopaminergic antagonists: ↓mesolimbic activation of sexual desire and attention toward sexual cues, central inhibition of genital response (MPOA, PVN) •Anticholinergic effects: ↓peripheral vasodilatation
Level 2 Problems in sexual functioning secondary to other adverse symptoms	•Extrapyramidal symptoms include akathisia, tremor, muscle stiffness, dystonia, tardive dyskinesia (dopaminergic antagonism in the nigrostriatal system) •Increased sedation (antihistaminic activity) •Emotional blunting (↑serotonergic activity) •Dry mouth (anticholinergic mechanism)
Level 3 Impact on medical risk factors of sexual dysfunction	•Weight gain and cardiometabolic risk factors (antihistaminic/anticholinergic activity) •Hyperprolactinemia (dopaminergic antagonism in tuberoinfundibular system)

sexual dysfunction associated with antidepressants ranging from approximately 25% to 80%.[111] Sexual side effects are more commonly caused by serotonergic and cholinergic effects and less likely by drugs enhancing dopaminergic and melanotonergic function. Antidepressants that are related to the lowest risk of SD are agomelatine (*melatonin-agonist and 5HT$_{2c}$-antagonist*), bupropion (norepinephrine and dopamine reuptake inhibitor), mirtazapine (*adrenergic alpha2 and 5-HT2 and 5-HT3 receptor antagonist)*, moclobemide (*monoamine oxidase A inhibitor)*, nefazadone (*postsynaptic serotonin (5-HT2) receptor(s) antagonist and moderate presynaptic serotonin and norepinephrine reuptake inhibitor*), selegiline (*selective monoamine oxidase B inhibitor with primarily dopaminergic mechanism)*, trazodone *(partial 5-HT$_{1A}$ agonist and 5-HT$_2$ and alpha-1 adrenergic receptor antagonist)*, vilazodone and vortioxetine *(multimodal serotonergic drugs blocking serotonin transporter but also 5-HT1A agonism among others*), and gepirone-ER (*5-HT$_{1A}$ partial agonist*).[111,142–147]

Antipsychotics

SDs, commonly erectile and orgasmic dysfunction in the short term and decreased sexual desire in the longer term, affect from 38% to 86% of patients treated with antipsychotics (APs).[148] The higher risk is related to use of antidopaminergic and hyperprolactinemia-inducing drugs including first-generation and some of the second generation APs (especially risperidone, paliperidone, and amisulpride). The lowest risk of treatment-emergent SD was related to the use of aripiprazole (*partial agonist of D$_2$ and 5-HT$_{1A}$ receptors*), lurasidone (*partial agonist of 5-HT1A and antagonist of D2, 5-HT2A and 5-HT7 receptors*), quetiapine (*5-HT2A and H1 receptor antagonism and D2 receptor blockade followed by rapid dissociation from the receptor*) and ziprasidone (*antagonist at D2, 5HT1D and 5HT2A receptors*).[112,148–150] However, it should be acknowledged that treatment with drugs like quetiapine or olanzapine often results in significant weight gain and adds to the cardiometabolic risk factors of SD in the long term. Moreover, due to antagonism at the H1 receptor, these drugs have strong sedative effects that may adversely affect sexual motivation.[132,151,152]

Mood stabalizers

Treatment with classical mood stabilizers (other than APs) is generally associated with lower rates of treatment-emergent SD as compared to the former groups of drugs. Antiepileptics such as carbamazepine, valproate, or lamotrigine show mild or no changes in sexual response where lamotrigine is considered to have the lowest impact on sexual functioning. There are some concerns with lithium as about 1/3 of patients reported decreased sexual desire and satisfaction, and ED.[148,153] These studies were small and of limited methodology.

Co-morbid medical conditions impacting sexual function

Many other factors contribute to onset, severity, or persistence of SD in patients with a psychiatric diagnosis and/or receiving medications as a part of their treatment. These factors are derived from the Biology contribution of the Biopsychosocial Model of Sexual Response and include normal changes in pattern and level of sex hormones over the course of a lifetime (biologic sex, puberty, reproductive years, and elders), hormonal/endocrine dysfunctions, and medical conditions and use of substances (prescribed and over-the-counter medications, drugs of abuse). Significant differences in patterns of SD exist between the sexes. Sexual function worsens in men with age (>0.50 OR increase, i.e, greater than 50%, with each decade after 20–29 years), while rates of SD in women remain fairly stable as distress decreases even as sexual complaints increase.[43] Men in poor health have increased risk of SD in all areas whereas women in poor health only have increased risk for sexual pain complaints, likely due to genitourinary syndrome of menopause (GSM).[43] The presence of urinary symptoms increases the risk of arousal dysfunction—specifically ED in men and sexual arousal and pain disorders in women.[43] Also, women are 2.5x more likely to find sexual intercourse not pleasurable than men.[43]

Multiple medical illnesses affect SD, many of which occur with higher frequency in patients with specific psychiatric conditions e.g, depression is bidirectional in co-morbidity in patients with diabetes, migraine, obstructive sleep apnea (OSA), and inflammatory joint disease.[154] In addition, the medications used to treat the medical illnesses may also contribute to SD. Co-morbid medical conditions appear to be the primary driver of the shorter lifespan in people with severe mental illness (SMI). These include cardiovascular diseases, SD, nutritional and metabolic diseases, viral and respiratory tract conditions, pregnancy complications, obesity-related disorders, and cancer, among others. Some of the excess morbidity and mortality is attributable to lack of adequate health care evaluation, medical interventions, and attention to modifiable lifestyle factors.[155] Thus, the presence of SD should prompt us to evaluate for co-morbid medical conditions and make appropriate referrals, and monitor and provide support in medication adherence, lifestyle changes, etc.

The highest rates are seen in patients with cardiovascular disease (CVD), especially in men with the occurrence of SD related to the severity of CVD.[156] Patients with CVD are 3x more likely to complain of SD than those without CVD with a prevalence estimated at nearly 90%.[157] Between 60% and 90% of patients with chronic heart failure report SD.[158] Men most frequently report reduced sexual desire and ED, often with resulting orgasmic difficulties. Women describe impact on all phases of the sexual response cycle including decreased sexual desire, genito-urinary syndrome of menopause (arousal difficulties), resulting pain with penetration, and orgasmic dysfunction.[156,158] As such, when men initially complain of ED, a cardiovascular work-up and evaluation for diabetes mellitus should be initiated. Treatment with sexual rehabilitation, e.g, pelvic floor muscle therapy, appropriate counseling such as addressing fears of resuming sexual activity after myocardial infarction with partner involvement around intimacy, maximizing treatment of the CVD while avoiding medications that specifically contribute to SD, and modifying health behaviors such as smoking, sleep disturbances (e.g, heart failure, obesity/obstructive sleep apnea), alcohol abuse, and physical inactivity may all benefit patients with SD associated with CVD.[157,158]

Neurological disorders are frequently associated with SD[127,159] and include multiple sclerosis (MS) where the prevalence of SD in women is >60% with an OR of 3.05 vs. controls[160] and 77% in men[161] with significantly higher rates of diminished desire in patients with epilepsy than patients with MS.[161] Other neurological conditions that impact sexual functioning/arousal response include traumatic brain injury, stroke, Parkinson's disease, spinal cord lesions, and damage to autonomic nerves with pelvic surgery.[159,162]

The impact on sexual functioning of conditions of the thyroid, the HPA axis, gonads, and other endocrine disorders such as diabetes mellitus/metabolic syndrome, are well-documented. Diabetes mellitus (DM) is strongly associated with the frequency and severity of male SD. Even prediabetes (preDM) has been shown in a meta-analysis of 12 studies to increase the prevalence of ED in men with preDM vs. controls. The severity of ED increases as impaired glucose metabolism progresses and with decreasing testosterone levels, and men with preDM are at increased risk of testosterone deficiency and hypogonadism. The reverse is also true: men with hypogonadism have a higher prevalence of preDM.[163] Diabetic neuropathy can also negatively impact sexual functioning. In premenopausal women with type 2 diabetes, menstrual disturbance, smoking, and duration of diabetes >10 years negatively affected sexual function as measured by the FSFI.[164] Thyroid disease including hypothyroidism and hyperthyroidism is associated with SD. An analysis of 4 studies of 25 519 men, 6429 with hyperthyroidism, demonstrated a prevalence of ED = 31.1% and an OR 1.73, $P < .00001$ vs. controls.[165] The impact of autoimmune thyroid disease (AITD) on sexual function was assessed in 250 women with AITD and 70 healthy young women using the FSFI and the Beck Depression Inventory. The risk of SD increased with the severity of thyroid disease with statistically significant impact on sexual desire, lubrication, and orgasm. Depression co-morbidity and higher thyroid stimulating hormone (TSH) values were identified as risk factors for SD.[166] In a meta-analysis of 68 studies in women with systemic autoimmune rheumatic disorders assessing sexual function with the FSFI, the overall prevalence of SD was >60% in women with Sjogren's syndrome with systemic sclerosis found to have the highest levels of SD.[167] Of 329 patients with rheumatoid arthritis (RA), 250 women and 79 men, 58.1% of women and 33.8% of men indicated overall SD using the Changes in Sexual Functioning Questionnaire (CSFQ). Risk factors for SD in this population were correlated with female gender, older age, moderate–severe depression, loneliness, more than 2 medical co-morbidities, and significant fatigue.[168] Other inflammatory joint diseases such as psoriatic arthritis, ankylosing spondylitis, and juvenile idiopathic arthritis have a bidirectional relationship with depression, so sexual functioning should also be assessed in these populations.[154]

Patients with cancer are at risk for SD from both the disease, the psychological impact of the diagnosis, and the treatments. Among 126 female gynecologic cancer survivors, 43.7% were diagnosed as having female SD with distress based on DSM-5 criteria.[169] Up to 75% of female breast cancer survivors report SD primarily related to chemotherapy, surgery, and radiation therapy where disrupted body image, abrupt onset of genitourinary syndrome of menopause, and diminished quality of life also play a role.[170,171] Consideration of patient preferences in breast cancer treatment does not need to reduce survival, but providers should discuss the potential for SD prior to initiating treatment.[172] Consideration of impact on sexual function, options and quality of life has long been important for planning treatment for men with prostate cancer, especially given earlier diagnosis and improved survival rates.[173] In 2 reviews/meta-analyses of the prevalence of SD in men with cancer, high rates of ED (>40%) were seen and were impacted by cancer treatment, cancer site, and age.[174,175] Use of angiogenesis inhibitors in advanced renal cancer impacted sexual functioning per the IIEF with reduction in scores of 30% to 60% in all phases of the sexual response cycle and satisfaction.[176]

Additional concerns: Post-SSRI sexual dysfunction (PSSD)

PSSD is not formally recognized as a distinct diagnostic entity in any version of the Diagnostic & Statistical Manual (newest is DSM-5-TR) or the International Classification of Diseases (newest is ICD-11), but was recognized by the European Medical Agency in 2019 based on 82 named reports, 32 with supporting documentation and other available sources. Based on reports submitted individually by sufferers, PSSD

Table 3 Stratification of risk/severity of treatment-emergent SD related to psychotropic medications.[111,112,124–126,144–146,148,185]

Antidepressants	
Low risk	Agomelatine, Bupropion, Gepirone-ER,[147] Nefazodone, Selegiline, Trazodone, Vilazodone, Vortioxetine
Intermediate risk	Desvenlafaxine, Duloxetine, Escitalopram, Fluvoxamine, Mirtazapine, Moclobemide, Reboxetine
High risk	Amitriptyline, Citalopram, Clomipramine, Fluoxetine, Imipramine, Isocarboxaside, Nortriptyline, Paroxetine, Sertraline, Venlafaxine
Antipsychotics	
Low risk	*
Intermediate risk	Aripiprazole, Lurasidone, Quetiapine, Ziprasidone
High risk	Amisulpride, Clozapine, Haloperidol, Olanzapine, Paliperidone, Perphenazine, Risperidone, Thioridazine
Non-antipsychotic mood stabilizers	
Low risk	Lamotrigine, lithium
Intermediate risk	Carbamazepine, Valproate
High risk	-

Low risk—influence on SD comparable to placebo, low probability of SD Intermediate risk—may cause less severe SD, usually seen with higher doses High risk—high probability of SD even at lower dose, the risk/severity of SD is usually dose-dependent *Newer antipsychotic medications such as brexpiprazole, cariprazine, lumateperone clinically seem to have low rates of SD, but no controlled studies using validated sexual functioning questionnaires have been published.

has been described as treatment-emergent SD persisting after SSRI discontinuation.[177] Growing numbers of reports on PSSD were subsequently collected in databases such as RxISK.org or Lareb (the Netherlands Pharmacovigilance Centre).[178] Reported duration of antidepressant treatment ranges from 1 day to many years. More recently, diagnostic criteria of PSSD were proposed as "enduring change in somatic (tactile) or erogenous (sexual) genital sensation after treatment stops."[179] Common clinical presentations include genital anesthesia or "genital numbness," pleasureless or weak orgasmic sensations, decreased sexual drive, ED and premature ejaculation in men, and decreased vaginal lubrication and nipple sensitivity in women. Emotional and cognitive problems and poor quality of life are also reported.[180] These symptoms are reported as persisting months or even years after SSRI, SNRI, or tricyclic antidepressant (TCA) discontinuation and should be differentiated from SD associated with recurrence of symptoms of the underlying psychiatric condition such as anxiety- or depression-related SD for which the serotonergic antidepressant was prescribed. The rate of PSSD among antidepressant users is unknown, but the vast majority of patients reporting these symptoms are men. Hypotheses regarding possible pathogenesis include persisting 5-HT1A receptor downregulation, epigenetic or neurohormonal changes, dopamine/serotonin interactions, serotonin neurotoxicity, and alteration of receptor ion channel transduction, but none of these has been confirmed and there are no specific biomarkers identified for PSSD.[178,181,182]

Only recently, a 19-year retrospective cohort analysis of 12 302 men who were 21-49 years old when they presented with ED attempted to estimate the risk of irreversible PSSD (ED only) compared the 7% with a reported history of serotonergic antidepressant use to the 93% without. Unfortunately, this sample of men with ED was enriched—all had ED, so the calculated occurrence rate of 0.46% cannot be used to determine prevalence of PSSD in the general population.[183] Another retrospective study of 13 Caucasian men (mean age 29.53 + 4.57 years) referred for SD with diagnosis of anxiety disorders and/or an adjustment disorder (although no patient-reported outcome measure of anxiety, e.g., GAD-7 was used) with onset of PSSD symptoms within 2 weeks of starting an SSRI treatment and up to 4 weeks after medication discontinuation, were treated empirically with a variety of interventions. The finding that bupropion, a known antidote for TESD with SSRIs was ineffective[180] may suggest an alternative etiology should be considered.

The incidence of PSSD is rare given the number of individuals worldwide taking serotonergic antidepressant medications, so prospective studies would require an unachievable number of participants entering prior to initiation of the SSRI antidepressant.

Assessment should include evaluation for potential underlying modifiable causes, identification of recurrence of the underlying psychiatric disorder (measured by patient-reported outcome measures), as well as taking a careful and detailed sexual history to exclude other etiologies of SD.[178] Interventions may include elimination of identified medical and psychiatric causes of SD (e.g., smoking, SUD, recurrence of anxiety disorder, etc.) and potential treatments, such as treating the recurrent anxiety or other psychiatric disorder with a non-SSRI medication. Several "antidotes" have been proposed to counteract symptoms of PSSD;[177,180,184] however, due to the lack of empirical prospective evidence and the rarity of this complaint, there are no established treatment recommendations. Further studies are needed to provide evidence on specific neurobiological mechanisms of PSSD and possible interventions.

Section 3: Management of Sexual Dysfunction in patients with psychiatric illness

Issues to address in management of SD in patients with psychiatric disorders and/or psychopharmacological treatments include overcoming barriers to discussion or interventions; individualizing the treatment plan; identifying co-morbid medical/psychiatric conditions and the bidirectional nature of the relationship between psychiatric conditions and SD[11]; use of validated measurements in psychiatric and medical conditions (ASEX, CSFQ, FSFI, IIEF); application of diagnostic criteria in ICD-11 Chapter on Conditions Related to Sexual Health that integrates Mental Health & Behavioral and Genitourinary Disorders; addressing impact of co-morbid medical conditions and concomitant medications; management of side effects associated with SD (e.g., weight gain, sleep disturbance, emotional blunting, cognitive dysfunction); eliciting patient preferences; and

monitoring outcomes such as response/nonresponse of the underlying condition, nonadherence or reduced quality of life.

Strategies for management of SD should begin with the provider initiating the conversation around sexual functioning, administration of a validated questionnaire or taking a phase-directed sexual history, and assessment of distress associated with SD. Determination if preexisting SD (primary sexual disorder or related to psychiatric illness) vs. relationship to psychotropic medications or co-morbid medical conditions or treatments vs. psychosocial/relationship problems drives any intervention. If related to the psychiatric illness, initial choice of treatment should consider patient preferences for side effects to avoid. Consider selecting medications less likely to cause SD (see Table 3) in sexually active patients who are starting psychotropic medication(s) for the first time.[16,110,186]

If the patient is already taking a psychotropic medication, assess for efficacy of treatment for the psychiatric condition. If treatment response is inadequate, focus on enhancing treatment to reduce the burden of the psychiatric illness on sexual functioning. Concomitant evaluation and management of modifiable factors such co-morbid medical and psychiatric conditions, concomitant medications and other substance use (alcohol, tobacco, illicit substances), and psychosocial/relationship factors should be addressed prior to changing efficacious psychotropic therapy. Consider evaluating sex steroids and other labs such as TSH, HgbA1c, etc. as indicated.

If the treatment is efficacious, but associated with SD, discuss patient preferences about switching to a medication with fewer sexual side effects, adding an adjunctive medication to reverse the sexual side effects of the initial psychotropic medication,[16,110,186] or utilizing non-pharmacologic interventions such as lifestyle modifications and therapy (cognitive-behavioral therapy, mindfulness-based therapy, trauma-based therapy, other sexual therapy). Longitudinally assess for remission of the psychiatric symptoms and monitor for medication side effects that the patient wants to avoid (SD, weight gain/metabolic syndrome, sleep disturbance, cognitive impairment, emotional blunting) which can further contribute to SD, reduced quality of life or non-adherence to treatment.

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