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Review article

Female sexual dysfunction as an adverse effect of drugs: a narrative review☆

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ABSTRACT

Sexual adverse effects of drugs are common and can compromise adherence to pharmacotherapy. Drugs can disrupt any or all phases of the sexual response cycle, potentially causing significant distress, which can amount to clinically relevant sexual dysfunction. Psychotropic and neurotropic agents are the best-characterised culprits in drug-related sexual dysfunction in females, although sexual dysfunction has been defined in various ways in the relevant literature. Specifically, serotonergic antidepressants, prolactin-increasing antipsychotics, long-term opioid therapy, and enzyme-inducing antiepileptics are associated with decreased desire, arousal dysfunction, orgasmic dysfunction, and more. In addition, progestin-containing contraceptives, antioestrogenic drugs, and beta blockers appear to increase the risk of sexual dysfunction. Possible mechanisms by which drugs interfere with sexual functions include alterations in neurotransmitter systems, increases in prolactin levels, increased sedation, and inhibition of the hypothalamic-pituitary-ovarian axis. Many medical conditions themselves can also cause sexual symptoms, and these are difficult to distinguish from pharmacological sexual adverse effects. However, different drugs for the same diseases can have substantially different sexual safety profiles, which often allows the clinician to choose a less-offending alternative. In some cases, drugs can exert even long-term adverse effects on sexual function. Therefore, sexual adverse effects must be taken into consideration when weighing the benefits and risks of different treatment modalities.

1. Introduction

Sexual dysfunction is an ambiguous term referring to any disruption in adequate and satisfactory psychological, physiological, and interpersonal sexual responses and experiences [1]. In women, different anomalies in sexual function are typically defined by the phase of the sexual cycle that they affect, namely reduced desire for sexual activity; impaired genital and non-genital arousal in response to sexual stimuli; delayed, absent or weak orgasms; and sexual pain [1,2]. For clinical purposes, the definition of sexual dysfunction should involve subjective distress associated with the sexual symptom [1]. However, several different definitions have been used in the literature. For instance, a recent meta-analysis found an overall prevalence of 41 % of sexual dysfunction in the general female population [3], but two large epidemiological studies estimated much lower rates of 12 % [4] and 21 % [5] when sexual distress was considered a criterion of true dysfunction. While the prevalence of all sexual symptoms seems to steadily increase with ageing, the rates of distress and therefore dysfunction decline after middle age [4].

The pathophysiology of sexual dysfunction is often multifactorial. Common risk factors include various diseases and pharmacotherapy. [1] Since sexual activity involves both the central and peripheral nervous systems, hypothalamic-pituitary-gonadal axis, and cardiovascular system [2], a large number of medical conditions and drugs may potentially have a detrimental impact. For example, in addition to depression itself, antidepressants can have adverse effects on sexual function [6]. Usually, it is impossible to reliably distinguish the effect of the drug from that of the disease; nevertheless, different rates of sexual dysfunction between different agents used in the management of the same condition may reveal substantial sexual adverse effect potential. Through many intended and off-target effects, drugs may compromise sexual desire,

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Abbreviations

FSFI	Female Sexual Function Index
PGAD	persistent genital arousal disorder
PSSD	post-SSRI sexual dysfunction
SGLT2	sodium-glucose cotransporter 2
SSRI	selective serotonin reuptake inhibitor

arousal, orgasms, and satisfactory intercourse [7,8]. Since sexual dysfunction has been shown to reduce compliance to pharmacotherapy [9], understanding of the sexual adverse effect profiles of drugs is essential for the clinician (Table 1). This narrative review aims to provide an overview of the most commonly implicated drug classes that contribute to female sexual dysfunction.

2. Methods

The format, focus, and scope of the review were agreed on by the authors in a planning meeting on 17 October 2024. To achieve the aims of the review, a narrative approach was chosen to overview the effects of numerous drug classes on female sexual function, and to equip clinicians with means to manage drug-induced sexual dysfunction. Based on the consensus of the authors, drug classes specifically chosen for review were antidepressants, antipsychotics, opioids, cardiovascular drugs, hormonal contraceptives, anticancer drugs, antiparkinson drugs, other neurotropic agents, and sodium-glucose cotransporter 2 (SGLT2) inhibitors. All phases of the female sexual cycle, including desire, arousal, orgasm, as well as intercourse itself, were considered potential victims of drug adverse effects. Thereafter, between October 2024 and January 2025, the PubMed database was searched for publications on female sexual dysfunction associated with the use of various drugs, using search strings consisting of different combinations of full and truncated terms pertaining to the aforementioned drug classes and female sexual dysfunction. Reference lists of reviewed articles were also searched for additional relevant publications. No publication time restrictions were applied, although more recent papers were preferred. The titles and abstracts of the search results were screened to identify relevant publications. Primarily, recent meta-analyses, systematic and comprehensive reviews, and clinical and epidemiological studies were included. Secondarily, narrative reviews, smaller clinical studies, and older publications were included when stronger evidence was lacking. Papers not written in English or reporting outcomes on males only were excluded. Due to the narrative nature of this review, documenting the numbers of publications retrieved, excluded, reviewed, and included was not considered feasible. All authors participated in the screening, reviewing, and interpreting of the literature.

Table 1
Common drug classes associated with risk of female sexual dysfunction.

Drug class	Frequency of sexual dysfunction	Comment
Antidepressants	4–80 % [12]	Non-serotonergic agents, e.g. bupropion and agomelatine, associated with smaller risk
Antipsychotics	16–60 % [18]	Agents associated with smaller increases in prolactin, e.g. aripiprazole and quetiapine, associated with smaller risk
Opioids	60–70 % [22]	In long-term opioid therapy
Progestin-containing hormonal contraceptives	Unclear	Hormonal intrauterine devices might be associated with smaller risk
Beta blockers	Unclear	
Antioestrogens	30–50 % [33]	Tamoxifen associated with slightly smaller risk than e.g. letrozole
SGLT2 inhibitors	Unknown	Genital infections might contribute to sexual pain
Benzodiazepines	Unknown	
Antiepileptics	Unknown	Enzyme-inducing agents, e.g. carbamazepine and phenytoin, associated with greater risk than non-enzyme-inducing agents, e.g. oxcarbazepine, lamotrigine, gabapentin and pregabalin
Antiparkinson agents	Unknown	Dopaminergic agents, i.e. levodopa and dopamine agonists, associated with compulsive sexual behaviour

SGLT2: sodium-glucose cotransporter 2.

3. Results

3.1. Antidepressants

Antidepressants are prominent culprits in drug-induced sexual dysfunction, although the reported incidence rates vary greatly [10]. In a Spanish cross-sectional study, an overall frequency as high as 80 % was reported in women taking various antidepressants [11]. However, there are substantial differences between different antidepressants: a meta-analysis involving both male and female patients and assessing 16 different agents found total sexual dysfunction rates varying from 4 % to 80 %, translating to odds ratios ranging from 0.2 to 27 compared with placebo. The most serotonergic drugs, such as sertraline, venlafaxine, citalopram, paroxetine, and fluoxetine, had the highest rates of sexual dysfunction overall, as well as in the specific domains of desire, arousal, and orgasm (Table 2). [12] Indeed, serotonin is thought to mediate a negative effect across all phases of the sexual cycle [10]. In women specifically, these drugs were associated with rates of 71–74 % for desire dysfunction, 78–84 % for arousal dysfunction, and 40–45 % for orgasmic dysfunction. Furthermore, tricyclic antidepressants are also serotonergic, and in males and females, imipramine was associated with a total sexual dysfunction rate of about 45 %, while clomipramine had an orgasm dysfunction rate up to 90 %. On the contrary, in males and females, non-serotonergic antidepressants such as agomelatine and bupropion were associated with the lowest rates of sexual dysfunction across all domains. [12] Vortioxetine, an antidepressant with complex effects on the serotonin system, is also associated with a low risk of female sexual dysfunction [13]. More recent real-world studies have confirmed this pattern in women: the rate of female sexual dysfunction across all domains is much higher with selective serotonin reuptake inhibitors (SSRI) and serotonin-norepinephrine reuptake inhibitors compared with non-serotonergic antidepressants [8,11].

The high burden of sexual adverse effects of antidepressants highlights the priority of non-pharmacological treatment modalities. When,

Table 2
Antidepressant-specific risk of total sexual dysfunction and binding to serotonin reuptake transporter.

Antidepressant	Risk of total sexual dysfunction compared to placebo (odds ratio) [12]	Binding potency to serotonin reuptake transporter (K_D, nmol/l) [14][a]
Sertraline	27	0.29
Venlafaxine	25	8.9
Citalopram	20	1.16
Paroxetine	17	0.13
Fluoxetine	16	0.81
Imipramine	7	1.40
Fluvoxamine	3	2.2
Mirtazapine	2	>100,000
Bupropion	0.8	9,100
Nefazodone	0.5	200
Agomelatine	0.3	N/A

K_D: equilibrium dissociation constant, N/A: not applicable.
[a] Smaller K_D values indicate greater binding potency.

however, pharmacotherapy is warranted, the favourable sexual safety profiles of bupropion, agomelatine, mirtazapine, and vortioxetine should be considered as one factor when selecting the most appropriate antidepressant. These less-offending drugs can also provide alternatives in treatment-emergent sexual dysfunction. Sometimes sexual adverse effects also resolve spontaneously over time. [15] Interestingly, treatment-emergent sexual dysfunction has also been reported to persist in some patients after discontinuation of serotonergic antidepressants. These conditions, known as "post-SSRI sexual dysfunction" (PSSD) and "persistent genital arousal disorder" (PGAD), are receiving increasing attention. In women, PSSD manifests as decreased desire, orgasmic dysfunction, and arousal dysfunction such as vaginal dryness and reduced genital sensation. PGAD, on the other hand, typically presents with unwanted and uncomfortable symptoms of sexual arousal, including but not limited to the genitals, that may continue for up to several days, even following orgasm and in the absence of sexual desire. The prevalence and incidence, as well as the exact pathophysiology, of both PSSD and PGAD are unknown. [16]

3.2. Antipsychotics

Antipsychotics are another drug class with a well-characterised sexual adverse effect profile. Antipsychotics can modulate several neurotransmitter pathways in the central and peripheral nervous systems, including inhibition of dopaminergic, histaminergic, cholinergic, and α-adrenergic signalling. Importantly, antipsychotics antagonise dopamine D2 receptors in the hypothalamic tuberoinfundibular system, which may lead to increased prolactin secretion. Hyperprolactinaemia interferes with the hypothalamic-pituitary-gonadal axis, which reduces sex hormone synthesis. [17] A meta-analysis involving both male and female patients found frequencies of total sexual dysfunction varying from 16 % to 60 %, depending on the antipsychotic. Quetiapine, ziprasidone, and aripiprazole appeared to be less-offending than risperidone, olanzapine, and haloperidol. An essentially similar pattern was observed for the specific domains of desire, arousal, and orgasmic dysfunction. Regarding females specifically, limited data also points to greater frequency of sexual dysfunction associated with risperidone than quetiapine. [18] Interestingly, this is consistent with the magnitudes of prolactin elevations associated with antipsychotics (quetiapine and aripiprazole << haloperidol and risperidone) [19].

3.3. Opioids

Opioids are well-known for suppressing the hypothalamic-pituitary-gonadal axis, also in women, and for increasing prolactin secretion [20]. However, studies focusing on the effect of opioids on female sexual function are scarce. In a cross-sectional setting, 25 % of female non-cancer chronic pain patients receiving long-term opioid therapy reported sexual dysfunction spontaneously. However, sexual impairment was as prevalent as 64 % among sexually active patients. In these patients, all domains of the Female Sexual Function Index (FSFI) were reduced, especially those of pain and orgasm. These results should be interpreted cautiously, as the authors did not specify the indications for opioid therapy, one of which could have been sexual pain itself. [21] In another cross-sectional study among patients suffering from chronic non-cancer pain and receiving long-term opioid treatment, 71 % of women reported decreased sexual desire, and of those sexually active, 59 % suffered from decreased capability, 64 % from decreased sexual frequency, and 59 % from decreased satisfaction [22]. Although pain itself also impairs sexual function, a population-based survey involving both women and men found an increased risk of decreased sexual desire in those non-cancer chronic pain patients receiving long-term opioid therapy compared with those not receiving opioids [23]. Sexual dysfunction is also prevalent in patients abusing opioids [24], as well as in those receiving methadone maintenance therapy for opioid dependence [25]. Although it is unknown whether there are within-class differences in the risk of sexual dysfunction, other pharmacodynamic properties, such as the serotonergic effects of tramadol [26], might also contribute to the risk. In any case, the deleterious sexual adverse effects of opioids should prompt clinicians and patients to adhere primarily to other treatment modalities for chronic pain.

3.4. Hormonal contraceptives

The effect of hormonal contraceptives, i.e. various progestins alone or in combination with ethinyl oestradiol, on female sexual function has been a matter of considerable debate. Studies investigating the effect of oral contraceptives on sexual desire have obtained mixed results, and while combination oral contraceptives have quite consistently been shown to substantially reduce the total and bioavailable concentrations of androgens, the relationship between androgens and female sexual desire appears less consistent and might depend on individual susceptibility to androgenic effects. [27] According to a recent meta-analysis, however, hormonal intrauterine devices might have a superior sexual safety profile compared with oral contraceptives. Overall, decreased sexual desire was associated with the use of oral and intrauterine hormonal contraceptives, and tendencies towards orgasmic dysfunction and worse total FSFI score were found. Interestingly, when the only study with patients using exclusively intrauterine devices was removed in a sensitivity analysis, the total FSFI score became statistically significantly worse in users of contraception. In this study, the use of intrauterine device was associated with superior FSFI scores of desire, arousal, lubrication, orgasm, satisfaction, and pain compared with non-use. [28] Finally, some studies have linked combination oral contraceptives, especially those containing antiandrogenic progestins, with arousal dysfunction such as vaginal dryness [27], but this was not supported by the meta-analysis [28].

3.5. Cardiovascular drugs

In men, certain antihypertensive drugs have been shown to be associated with an increased risk of sexual dysfunction; however, this connection remains somewhat unclear in women. Available evidence was systematically reviewed by Choy et al. [29]. Conflicting evidence suggests that beta blockers, specifically atenolol and metoprolol, might be associated with deterioration of some domains, namely desire, sexual fantasy, and lubrication, of sexual function in hypertensive females. Some studies have also reported worse total FSFI scores in users of non-specified beta blockers. On the other hand, angiotensin converting enzyme inhibitors and angiotensin receptor blockers appear to have no adverse effects on female sexual function. In fact, some studies suggest beneficial effects on sexual desire, fantasy, arousal, and satisfaction. Calcium channel blockers and diuretics seem to have essentially neutral effect. [29]

3.6. Anticancer drugs

The treatment of cancer, as well as cancer itself, may result in neural, vascular, hormonal, and psychological complications following surgery, radiotherapy, and chemotherapy. Treatment-emergent peripheral neuropathy, genital vascular injuries, and hypogonadism may put cancer patients at a great risk of any or all kinds of sexual dysfunction. [30] Breast cancer, for example, is the most common malignancy among women [31], and it is associated with sexual dysfunction prevalence rates of >80 % according to a review in Asian patient populations [32]. Among other insults, chemotherapy can result in premature ovarian failure and early menopause in younger breast cancer patients. Moreover, approximately 75 % of breast cancer cases are oestrogen receptor positive and often require 5–10 years of adjuvant antioestrogen therapy to prevent recurrence. Endocrine therapies, namely selective oestrogen receptor modulators (e.g. tamoxifen) and aromatase inhibitors (e.g. letrozole), significantly reduce oestrogenic effects and are associated

with substantial sexual adverse effects, including sexual pain, decreased desire, orgasmic dysfunction, and overall poor sexual satisfaction. These adverse effects generally appear to be somewhat more pronounced in women treated with aromatase inhibitors compared with selective oestrogen receptor modulators. [31,33]

Since cancer patients are typically treated with a multimodal approach, it is difficult to delineate the exact contribution of anticancer drugs to sexual dysfunction. Besides, cancer itself is a devastating, existential crisis with potential to impair all aspects of sexuality. The patient and the clinician must select between effective but often debilitating treatment, and disease progression. At any rate, it is important to always acknowledge the detrimental sexual adverse effects that the patients might be suffering from. While these adverse effects might not be avoidable, they can be manageable with non-pharmacological modalities such as psychoeducation, lifestyle improvements, vaginal moisturisers, pelvic floor muscle training, and counselling [34].

3.7. Other drugs

Although not always well-documented, it is conceivable that several other drug classes could also cause sexual adverse effects. A group of new antidiabetics, SGLT2 inhibitors, function by increasing urinary excretion of glucose. Consequently, SGLT2 inhibitors such as empagliflozin and dapagliflozin have been associated with a markedly increased risk—up to 6–20-fold according to a recent network meta-analysis [35]—of female genital infections, particularly vulvovaginal candidiasis [36]. This may add to the burden of sexual pain in SGLT2 inhibitor users.

Moreover, sedation through various neuropharmacological mechanisms is a wanted or unwanted effect of numerous drugs, with possible negative sequelae for sexual function. For instance, there is some evidence that benzodiazepine use is associated with an increased risk of female sexual dysfunction, specifically decreased desire and orgasmic dysfunction [37]. Lithium, a mood stabiliser, has also been associated with sexual dysfunction in males and females according to some studies [38,39]. Enzyme-inducing antiepileptic drugs, particularly carbamazepine and phenytoin, can accelerate the metabolism of sex hormones and increase the levels of sex hormone-binding globulin, potentially reducing bioavailable sex hormones. Indeed, carbamazepine has been associated with sexual dysfunction in both women and men. On the other hand, antiepileptics not inducing enzymes, such as oxcarbazepine, lamotrigine, pregabalin, and gabapentin, do not seem to be associated with increased rates of sexual dysfunction. Valproate, on the other hand, has been associated with sexual dysfunction. [40] Finally, dopaminergic antiparkinson drugs can increase sexual desire to the point of compulsive sexual behaviour, although this is much less common in women than in men [41].

4. Conclusions

Drug-related female sexual dysfunction is very common and can occur within many different drug classes, particularly antidepressants, antipsychotics, opioids, progestin-containing oral hormonal contraceptives, beta blockers, anticancer agents, antioestrogenic agents, and antiepileptics. It should be noted, however, that the medical conditions themselves that are managed with these drugs can have substantial effects on sexual function, so distinguishing between drug adverse effect and disease symptom is difficult. Sexual adverse effects can be severe and even have long-term impacts. Fortunately, in many cases, pharmacological and non-pharmacological treatment options with smaller risk of sexual dysfunction are available. It is also worthwhile to remember that even unavoidable sexual adverse effects can be alleviated with various methods such as lifestyle changes, vaginal moisturisers, and counselling. Drug-related sexual dysfunction in females can cause considerable distress and compromise compliance to treatment and should therefore be recognised and taken into account when prescribing and reviewing medication.

Contributors

Mikael O.W. Piha contributed to conceptualisation, data acquisition, analysis and interpretation, and writing of the manuscript.

Katja Kero contributed to conceptualisation, data acquisition, analysis and interpretation, and writing of the manuscript.

Aleksi Tornio contributed to conceptualisation, data analysis and interpretation, and writing of the manuscript.

All authors saw and approved the final version and no other person made a substantial contribution to the paper.

Ethical statement

The present work is a review of literature and therefore exempt from ethical review.

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Declaration of competing interest

The authors declare that they have no competing interest.

References

[1] S.R. Davis, Sexual dysfunction in women, N. Engl. J. Med. 391 (2024) 736–745, https://doi.org/10.1056/NEJMcp2313307.
[2] R.J. Levin, The human sexual response cycle, in: W.W. IsHak (Ed.), The textbook of clinical sexual medicine, Springer Nature, Cham, 2017, pp. 39–51, https://doi.org/10.1007/978-3-319-52539-6.
[3] J. Ramírez-Santos, D. Cristóbal-Cañadas, T. Parron-Carreño, D. Lozano-Paniagua, B.J. Nievas-Soriano, The problem of calculating the prevalence of sexual dysfunction: a meta-analysis attending gender, Sex. Med. Rev. 12 (2024) 116–126, https://doi.org/10.1093/sxmrev/qead058.
[4] J.L. Shifren, B.U. Monz, P.A. Russo, A. Segreti, C.B. Johannes, Sexual problems and distress in United States women: prevalence and correlates, Obstet. Gynecol. 112 (2008) 970–978, https://doi.org/10.1097/AOG.0b013e3181898cdb.
[5] J. Zheng, M.A. Skiba, R.J. Bell, R.M. Islam, S.R. Davis, The prevalence of sexual dysfunctions and sexually related distress in young women: a cross-sectional survey, Fertil. Steril. 113 (2020) 426–434, https://doi.org/10.1016/j.fertnstert.2019.09.027.
[6] R. Basson, T. Gilks, Women's sexual dysfunction associated with psychiatric disorders and their treatment, Womens Health (Lond.) 14 (2018), https://doi.org/10.1177/1745506518762664, 1745506518762664.
[7] A.V. Gueldini de Moraes, A.L. Ribeiro Valadares, J.F. Lui Filho, L. Costa-Paiva, A. O. Pedro, Medication use and sexual function: a population-based study in middle aged women, J. Sex. Med. 16 (2019) 1371–1380, https://doi.org/10.1016/j.jsxm.2019.06.004.
[8] C. Valeiro, C. Matos, J. Scholl, F. van Hunsel, Drug-induced sexual dysfunction: an analysis of reports to a National Pharmacovigilance Database, Drug Saf. 45 (2022) 639–650, https://doi.org/10.1007/s40264-022-01174-3.
[9] L. Souaiby, F. Kazour, M. Zoghbi, R. Bou Khalil, S. Richa, Sexual dysfunction in patients with schizophrenia and schizoaffective disorder and its association with adherence to antipsychotic medication, J. Ment. Health 29 (2020) 623–630, https://doi.org/10.1080/09638237.2019.1581333.
[10] A. La Torre, G. Giupponi, D. Duffy, A. Conca, Sexual dysfunction related to psychotropic drugs: a critical review–part I: antidepressants, Pharmacopsychiatry 46 (2013) 191–199, https://doi.org/10.1055/s-0033-1345205.
[11] A.L. Montejo, J. Calama, F. Rico-Villademoros, L. Montejo, N. González-García, J. Pérez, SALSEX Working Study Group, A real-world study on antidepressant-associated sexual dysfunction in 2144 outpatients: the SALSEX I study, Arch. Sex. Behav. 48 (2019) 923–933, https://doi.org/10.1007/s10508-018-1365-6.
[12] A. Serretti, A. Chiesa, Treatment-emergent sexual dysfunction related to antidepressants: a meta-analysis, J. Clin. Psychopharmacol. 29 (2009) 259–266, https://doi.org/10.1097/JCP.0b013e3181a5233f.
[13] P.L. Jacobsen, A.R. Mahableshwarkar, W.A. Palo, Y. Chen, M. Dragheim, A. H. Clayton, Treatment-emergent sexual dysfunction in randomized trials of vortioxetine for major depressive disorder or generalized anxiety disorder: a

pooled analysis, CNS Spectr. 21 (2016) 367–378, https://doi.org/10.1017/S1092852915000553.
[14] M. Tatsumi, K. Groshan, R.D. Blakely, E. Richelson, Pharmacological profile of antidepressants and related compounds at human monoamine transporters, Eur. J. Pharmacol. 340 (1997) 249–258, https://doi.org/10.1016/s0014-2999(97)01393-9.
[15] T. Lorenz, J. Rullo, S. Faubion, Antidepressant-induced female sexual dysfunction, Mayo Clin. Proc. 91 (2016) 1280–1286, https://doi.org/10.1016/j.mayocp.2016.04.033.
[16] L. Tarchi, G.P. Merola, O. Baccaredda-Boy, F. Arganini, E. Cassioli, E. Rossi, M. Maggi, D.S. Baldwin, V. Ricca, G. Castellini, Selective serotonin reuptake inhibitors, post-treatment sexual dysfunction and persistent genital arousal disorder: a systematic review, Pharmacoepidemiol. Drug Saf. 32 (2023) 1053–1067, https://doi.org/10.1002/pds.5653.
[17] A. La Torre, A. Conca, D. Duffy, G. Giupponi, M. Pompili, M. Grözinger, Sexual dysfunction related to psychotropic drugs: a critical review part II: antipsychotics, Pharmacopsychiatry 46 (2013) 201–208, https://doi.org/10.1055/s-0033-1347177.
[18] A. Serretti, A. Chiesa, A meta-analysis of sexual dysfunction in psychiatric patients taking antipsychotics, Int. Clin. Psychopharmacol. 26 (2011) 130–140, https://doi.org/10.1097/YIC.0b013e328341e434.
[19] Y. Zhu, C. Zhang, S. Siafis, K. Zhuo, D. Zhu, H. Wu, D. Liu, K. Jiang, J. Wang, S. Leucht, C. Li, Prolactin levels influenced by antipsychotic drugs in schizophrenia: a systematic review and network meta-analysis, Schizophr. Res. 237 (2021) 20–25, https://doi.org/10.1016/j.schres.2021.08.013.
[20] F. de Vries, M. Bruin, D.J. Lobatto, O.M. Dekkers, J.W. Schoones, W.R. van Furth, A.M. Pereira, N. Karavitaki, N.R. Biermasz, A.H. Zamanipoor Najafabadi, Opioids and their endocrine effects: a systematic review and meta-analysis, J. Clin. Endocrinol. Metab. 105 (2020) 1020–1029, https://doi.org/10.1210/clinem/dgz022.
[21] R. Ajo, A. Segura, M.M. Inda, B. Planelles, L. Martínez, G. Ferrández, A. Sánchez, C. Margarit, A.M. Peiró, Opioids increase sexual dysfunction in patients with non-cancer pain, J. Sex. Med. 13 (2016) 1377–1386, https://doi.org/10.1016/j.jsxm.2016.07.003.
[22] T.-C. Lin, S.-T. Ho, L.-P. Ger, H.-H. Liou, S.-L. Hwang, Gender difference in long-term use of opioids among Taiwan officially registered patients with chronic noncancer pain, Medicine (Baltimore) 97 (2018) e10805, https://doi.org/10.1097/MD.0000000000010805.
[23] H. Birke, O. Ekholm, J. Højsted, P. Sjøgren, G.P. Kurita, Chronic pain, opioid therapy, sexual desire, and satisfaction in sexual life: a population-based survey, Pain Med. 20 (2019) 1132–1140, https://doi.org/10.1093/pm/pny122.
[24] A. Ghosh, S. Kathiravan, K. Sharma, S.K. Mattoo, A scoping review of the prevalence and correlates of sexual dysfunction in adults with substance use disorders, J. Sex. Med. 19 (2022) 216–233, https://doi.org/10.1016/j.jsxm.2021.11.018.
[25] H.A. Ortman, J.A. Siegel, The effect of methadone on the hypothalamic pituitary gonadal axis and sexual function: a systematic review, Drug Alcohol Depend. 207 (2020) 107823, https://doi.org/10.1016/j.drugalcdep.2019.107823.
[26] Orion Corporation, Tramal summary of product characteristics. https://fimea.fi/laakehaut_ja_luettelot/laakehaku, 2024. (Accessed 14 February 2025).
[27] J.M. Krapf, A.T. Goldstein, Combined estrogen-progestin oral contraceptives and female sexuality: an updated review, Sex. Med. Rev. 12 (2024) 307–320, https://doi.org/10.1093/sxmrev/qeae011.
[28] M. Huang, G. Li, J. Liu, Y. Li, P. Du, Is there an association between contraception and sexual dysfunction in women? A systematic review and meta-analysis based on female sexual function index, J. Sex. Med. 17 (2020) 1942–1955, https://doi.org/10.1016/j.jsxm.2020.06.008.
[29] C.L. Choy, H. Sidi, C.S. Koon, O.S. Ming, I.N. Mohamed, N.C. Guan, C.A. Alfonso, Systematic review and meta-analysis for sexual dysfunction in women with hypertension, J. Sex. Med. 16 (2019) 1029–1048, https://doi.org/10.1016/j.jsxm.2019.04.007.
[30] F. Priviero, C. Webb, Biology of iatrogenic sexual dysfunction in men and women survivors of cancer, Urol. Oncol. 40 (2022) 366–371, https://doi.org/10.1016/j.urolonc.2021.01.017.
[31] S. Vegunta, C.L. Kuhle, J.A. Vencill, P.H. Lucas, D.M. Mussallem, Sexual health after a breast cancer diagnosis: addressing a forgotten aspect of survivorship, J. Clin. Med. 11 (2022) 6723, https://doi.org/10.3390/jcm11226723.
[32] M. Jehan, S. Azam, M.A. Taimuri, A. Sumbal, A. Azhar, A. Amir, M.O. Oduoye, A. Zainab, A. Ikram, T. Ali, Care for breast cancer survivors in Asian countries: a review of sexual dysfunction, Womens Health (Lond.) 20 (2024), https://doi.org/10.1177/17455057241237687, 17455057241237687.
[33] E.N. Boswell, D.S. Dizon, Breast cancer and sexual function, Transl. Androl. Urol. 4 (2015) 160–168, https://doi.org/10.3978/j.issn.2223-4683.2014.12.04.
[34] L. Cucciniello, F. Miglietta, V. Guarneri, F. Puglisi, Managing sexual health challenges in breast cancer survivors: a comprehensive review, Breast 76 (2024) 103754, https://doi.org/10.1016/j.breast.2024.103754.
[35] L. Chen, Q. Xue, C. Yan, B. Tang, L. Wang, B. Zhang, Q. Zhao, Comparative safety of different recommended doses of sodium-glucose cotransporter 2 inhibitors in patients with type 2 diabetes mellitus: a systematic review and network meta-analysis of randomized clinical trials [supplementary material], Front. Endocrinol. (Lausanne) 14 (2023) 1256548, https://doi.org/10.3389/fendo.2023.1256548.
[36] K. Engelhardt, M. Ferguson, J.L. Rosselli, Prevention and management of genital mycotic infections in the setting of sodium-glucose cotransporter 2 inhibitors, Ann. Pharmacother. 55 (2021) 543–548, https://doi.org/10.1177/1060028020951928.
[37] B. Hosseinzadeh Zoroufchi, H. Doustmohammadi, T. Mokhtari, A. Abdollahpour, Benzodiazepines related sexual dysfunctions: a critical review on pharmacology and mechanism of action, Rev. Int. Androl. 19 (2021) 62–68, https://doi.org/10.1016/j.androl.2019.08.003.
[38] H.Y. Elnazer, A. Sampson, D. Baldwin, Lithium and sexual dysfunction: an under-researched area, Hum. Psychopharmacol. 30 (2015) 66–69, https://doi.org/10.1002/hup.2457.
[39] A. García-Blanco, M.P. García-Portilla, L. Fuente-Tomás, M. Batalla, M. Sánchez-Autet, B. Arranz, G. Safont, S. Arqués, L. Livianos, P. Sierra, Sexual dysfunction and mood stabilizers in long-term stable patients with bipolar disorder, J. Sex. Med. 17 (2020) 930–940, https://doi.org/10.1016/j.jsxm.2020.01.032.
[40] M. Yogarajah, M. Mula, Sexual dysfunction in epilepsy and the role of anti-epileptic drugs, Curr. Pharm. Des. 23 (2017) 5649–5661, https://doi.org/10.2174/1381612823666171004115230.
[41] C.M. Santa Rosa Malcher, K. Roberto da Silva Gonçalves Oliveira, M.C. Fernandes Caldato, B. Lopes Dos Santos Lobato, J. da Silva Pedroso, M. de Tubino Scanavino, Sexual disorders and quality of life in Parkinson's disease, Sex. Med. 9 (2021) 100280, https://doi.org/10.1016/j.esxm.2020.10.008.