Independent Research & Further Reading

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Contents

Education in Female Sexual Health

> *“your gynecologist has never been taught about the clitoris, about the vulva, sexual health, about sexual pain, about libido, arousal, and orgasm.”*

Research suggests that education on female sexual health remains limited and inconsistent across medical school and residency training. While physicians receive some instruction on female sexual anatomy and function, curricula are often focused on reproduction, sexually transmitted infections, and general gynaecological care rather than sexual function and dysfunction. Studies have found that detailed teaching on clitoral and vulvar anatomy is frequently incomplete, with many programmes providing little or no coverage of anatomical variations, clitoral abnormalities, or specialised examinations related to female sexual dysfunction (FSD).

Training on sexual pain conditions, including dyspareunia (pain during intercourse), vaginismus (involuntary tightening of the pelvic floor muscles), and vulvodynia (chronic vulvar pain), is also commonly limited. Similarly, disorders of desire, arousal, and orgasm are often minimally addressed, leaving many trainees feeling underprepared to assess and manage these concerns. Research further indicates that sexual history-taking is frequently restricted to topics such as pregnancy prevention and sexually transmitted infections, with less emphasis on pleasure, satisfaction, libido, arousal, orgasm, or pain. Although targeted educational programmes and workshops have been shown to improve knowledge and confidence in sexual medicine, these approaches are not yet routinely incorporated into medical training.

References 1-5

Anatomy of the External Female Genitalia

> *“This is your labia majora. This is your labia minora. This is your clitoris. This is your urethra, the tube that you pee through," because women can't see it, and they don't know about it (...)*
>
> *women didn't know that their labia shrivel up and disappear (...) The truth is science has never studied it. We don't even know why it changes in shapes and size.”*

The external female genitalia are collectively known as the vulva, which includes several structures that surround and protect the urinary and reproductive openings. The labia majora are the larger, outer folds of skin that extend from the mons pubis towards the perineum and form much of the visible external genitalia. These folds contain fatty tissue and are typically hair-bearing after puberty. Situated inside the labia majora are the labia minora, smaller folds of skin that border the vestibule, the area containing the urethral and vaginal openings. The labia minora help protect these openings and surround the clitoris.

The clitoris is a highly innervated erectile organ located at the front of the vulva and plays a central role in sexual arousal and orgasm. Although only the glans, or external tip, is visible, the clitoris extends internally and includes erectile structures known as the body and crura. The urethra, in this context, refers to the external urethral opening, or meatus, through which urine exits the body. It is located within the vulvar vestibule, between the clitoris and the vaginal opening. Together, the labia majora, labia minora, clitoris, and urethral opening form key components of the vulva and contribute to the protection and function of the urinary and reproductive systems.

Age-Related Changes in the Labia

Research indicates that the labia do not disappear with age, but they can undergo significant changes in appearance and tissue composition, particularly during and after menopause. Declining hormone levels are associated with atrophy (thinning of tissue), hypotrophy (reduction in tissue size), and lipoatrophy (loss of fatty tissue), which can make the labia appear smaller, flatter, or less prominent. The labia majora, which normally contain fatty tissue, may lose volume and elasticity over time, while the labia minora can become thinner and, in some cases, adhere more closely to surrounding tissues.

These changes are commonly described in conditions such as genitourinary syndrome of menopause and vulvovaginal atrophy, where dryness and tissue thinning are frequently observed. However, the medical literature does not describe the labia as disappearing. Rather, they undergo gradual, hormone-related changes in volume and tissue quality that occur within a wide range of normal anatomical variation. Some individuals seek treatments aimed at restoring fullness or improving comfort because of these changes, reflecting tissue loss rather than the absence of the labia themselves.

References 6-14.

The Role of Testosterone in Health and Ageing

"[testosterone] is incredibly important for bone health, potentially heart health when you start it early, for sexual health for sure, and then for UTI prevention, which is killing a lot of the people that we love.

Now, testosterone's really interesting 'cause it actually isn't at menopause that you lose testosterone. It happens in your 30s.

medications for acne, medications for hair loss that people are using can affect your testosterone levels (...)

we know we have global consensus actually that testosterone helps for libido in post-menopausal women. Okay? Now, there is also data in perimenopausal women as well, and that is clear data. It helps with libido, but it also helps with arousal (...)

Actually, there's data to shows that testosterone can help with women's sexual body image, which is like a cool a really cool thing."

Testosterone and Bone Health

Research indicates that testosterone plays an important role in skeletal development and the maintenance of bone mass throughout adulthood. Low testosterone levels in men are associated with reduced bone mineral density and an increased risk of fractures, particularly among those with hypogonadism, a condition characterised by insufficient testosterone production. Testosterone replacement therapy (TRT) has been shown to increase bone mineral density, especially in the spine and cortical bone, although evidence that it reduces fracture risk remains limited.

Testosterone and Heart Health

Low testosterone levels have been associated with poorer cardiometabolic health and a higher risk of cardiovascular events in observational studies, particularly when levels are markedly reduced. However, evidence regarding the cardiovascular effects of TRT remains mixed. A large randomised clinical trial found that TRT was neither more harmful nor more beneficial than placebo for major cardiovascular events over approximately three years. Some research suggests

testosterone may support cardiovascular health through improvements in body composition and blood vessel function, but the long-term clinical significance of these effects remains uncertain.

Testosterone and Sexual Health

Testosterone is closely linked to sexual function. In men, low testosterone is strongly associated with reduced libido and erectile dysfunction. Clinical trials and meta-analyses have found that TRT can improve sexual desire, erectile function, orgasm, and overall sexual satisfaction in hypogonadal men. Research in postmenopausal women has also shown that testosterone therapy can improve several aspects of sexual function and reduce sexual distress, highlighting its role in sexual health across both sexes.

Testosterone and Urinary Tract Infection Prevention

Current evidence does not support testosterone as a proven strategy for preventing urinary tract infections (UTIs). Research involving people assigned female at birth found that testosterone use did not significantly increase or decrease UTI rates compared with cisgender women. Small studies of vaginal testosterone in postmenopausal women have shown improvements in vaginal tissue health, but have not demonstrated a clear reduction in UTI occurrence. Animal research has even suggested that androgens may increase the severity of UTIs under certain conditions. Overall, while testosterone may influence aspects of genitourinary health, its role in UTI prevention remains unproven.

Age-Related Changes in Testosterone Levels in Women

Research indicates that testosterone levels in women begin to decline during the reproductive years, with measurable reductions already apparent by the 30s. Studies consistently show that androgen levels, including testosterone, are highest in early adulthood and decrease progressively with age. Comparisons between women in their late teens and twenties and those aged 35–39 years demonstrate significantly lower testosterone levels in the older group, suggesting that the decline begins well before menopause.

Additional research has found a gradual, stepwise reduction in testosterone and other androgens throughout the 30s and 40s, with some studies describing a particularly marked decline after the twenties. Testosterone concentrations continue to decrease through midlife, with further reductions observed between the ages of 40 and 60. Longitudinal data also suggest that while total

testosterone may stabilise or change slightly after menopause, bioavailable testosterone, the fraction available for use by tissues, continues to decline with age. Importantly, evidence indicates that these hormonal changes are primarily related to ageing rather than menopause itself. Studies comparing women of similar ages before and after menopause have found relatively small differences in testosterone levels, suggesting that the gradual decline begins earlier and progresses independently of the menopausal transition. Overall, testosterone levels are generally lower in women during their 30s than in their 20s and continue to decrease gradually throughout midlife.

The Effects of Acne and Hair-Loss Medications on Testosterone

Research suggests that some medications used to treat acne and androgen-related hair loss can affect testosterone pathways, although their effects vary depending on the treatment. Many of these therapies are designed not to eliminate testosterone entirely, but to reduce androgen activity or alter the way androgens act in specific tissues such as the skin and hair follicles.

For acne treatment, oral isotretinoin has been shown to reduce total testosterone and dihydrotestosterone (DHT), a potent androgen derived from testosterone, in some women with severe acne. Hormonal treatments used for acne, including certain combined oral contraceptives, can also lower circulating testosterone levels. These effects are generally intentional and contribute to the therapeutic benefits of reducing androgen-driven skin symptoms.

Hair-loss medications such as finasteride and dutasteride primarily target DHT rather than testosterone itself. These drugs inhibit the enzyme 5-alpha-reductase, which converts testosterone into DHT, resulting in substantial reductions in DHT levels while having a more limited effect on testosterone concentrations. Topical antiandrogen treatments are designed to exert their effects locally within the scalp or skin, reducing androgen activity with less impact on hormone levels throughout the body.

Testosterone and Sexual Function in Perimenopausal and Postmenopausal Women

Research from multiple randomised controlled trials and meta-analyses suggests that low-dose testosterone therapy can improve several aspects of sexual function in postmenopausal women, particularly those with hypoactive sexual desire disorder (HSDD), a condition characterised by persistently low sexual desire accompanied by personal distress. Studies have found improvements in libido, arousal, pleasure, orgasm, responsiveness, sexual satisfaction, and the frequency of satisfying sexual experiences, alongside reductions in sexual distress. Most of the

evidence comes from the use of low-dose transdermal testosterone formulations designed to achieve physiological hormone levels.

Evidence for perimenopausal and late reproductive-age women is more limited. While some reviews and clinical guidelines suggest testosterone may benefit women experiencing HSDD before menopause, fewer studies have been conducted in these populations and results are less consistent than those observed in postmenopausal women. As a result, current recommendations are more cautious and generally regard HSDD in postmenopausal women as the primary evidence-based indication for testosterone therapy.

Available research suggests that treatment is generally well tolerated when administered at physiological doses. Reported side effects include acne and increased hair growth, while serious adverse events have not been shown to increase in short-term studies. However, long-term data on outcomes such as breast health and cardiovascular risk remain limited. Consequently, clinical guidelines recommend careful patient selection, monitoring, and the use of doses that maintain testosterone levels within the normal premenopausal range.

Testosterone and Women's Sexual Self-Image

Research suggests that testosterone therapy can improve women's sexual self-image, particularly among women experiencing low sexual desire or sexual dysfunction. Large meta-analyses of randomised controlled trials have found that testosterone treatment improves multiple aspects of sexual wellbeing, including sexual desire, arousal, pleasure, orgasm, responsiveness, and self-image, while also reducing sexual distress and concerns. These benefits have been observed most consistently in peri- and postmenopausal women receiving treatment for hypoactive sexual desire disorder (HSDD).

Evidence from individual clinical trials has also reported improvements in measures of sexual self-perception and overall wellbeing among premenopausal women with low libido treated with transdermal testosterone. Other studies have found that women who received androgen-containing hormone therapy after oophorectomy experienced less deterioration in body image and better sexual functioning over time than women who did not receive androgen therapy.

However, the evidence is stronger for sexual self-image than for body image considered independently of sexual function. Most studies assess self-image as part of broader measures of sexual wellbeing rather than as a separate outcome. While some observational and clinical data

suggest a possible relationship between androgen levels and body-image perception, particularly in specific populations such as women with Turner syndrome or women who have undergone oophorectomy, evidence that testosterone directly improves body image alone remains limited. Overall, current research supports a role for testosterone in improving sexual self-image as part of the treatment of female sexual dysfunction, while evidence for effects on general body image is less established.

References 15-44.

Testosterone Use and Male Fertility

> *“When [men] take high doses of testosterone, you become infertile because your testicles say, "Oh, I don't need to produce, I don't need to produce sperm right now 'cause there's plenty of testosterone around.”*

Research shows that continuous use of high-dose testosterone can significantly impair male fertility by suppressing sperm production. Exogenous testosterone, whether prescribed as testosterone replacement therapy (TRT) or used in the form of anabolic-androgenic steroids, suppresses the hypothalamic–pituitary–gonadal axis, the hormonal system responsible for regulating testosterone production and spermatogenesis, the process by which sperm are produced. This suppression reduces levels of luteinising hormone (LH) and follicle-stimulating hormone (FSH), leading to a marked decrease in intratesticular testosterone and, in many cases, a cessation of sperm production.

Studies have found that azoospermia, the complete absence of sperm in the ejaculate, is common among men using exogenous testosterone. Research involving infertile men taking testosterone reported that the majority became azoospermic while on treatment. In many cases, sperm production recovers after testosterone is discontinued, with hormone levels and sperm counts often improving over several months. However, recovery is variable, and some men continue to experience severe reductions in sperm count or persistent azoospermia despite stopping treatment and receiving supportive therapy. Because of these effects, clinical guidelines generally advise against testosterone therapy in men who wish to preserve fertility. Alternative approaches, including the use

of human chorionic gonadotropin (hCG) or fertility-preserving testosterone regimens, may help maintain or restore sperm production in selected individuals.

References 45-48.

Estrogen Levels After Menopause

> *"every woman over the age of 50, her estrogen goes to essentially zero (...)"*

Research shows that estrogen levels decline substantially after menopause, but they do not fall to zero. As ovarian estrogen production decreases, circulating levels of estradiol and estrone, the two primary forms of estrogen, stabilise at lower but generally measurable concentrations. Studies of healthy postmenopausal women consistently report estradiol levels that are markedly lower than those seen before menopause, typically ranging from approximately 5–25 pg/mL, while estrone remains detectable in most women. Evidence from large population studies indicates that these low levels of estrogen persist well into older age. Although estradiol may fall below laboratory detection limits in some women aged 70 years and older, estrone is usually still present and becomes the predominant circulating estrogen after menopause. Unlike premenopausal women, whose estrogen is primarily produced by the ovaries, postmenopausal women derive most of their estrogen from the conversion of hormones in fat tissue and other peripheral tissues.

References 49-53.

How Birth Control Works

> *“so how do birth control pills work? Um, when you take a combined birth control pill, it has a fake amount of estrogen and a fake amount of progestin in it, so high that it tricks your body into not ovulating. So when you have so much hormone around, your body says, "Oh, I don't need to make my own 'cause there's plenty around," and so the ovaries shut down. So your ovaries are no longer making their own hormones because (...)*
>
> *up to 27% of people on birth control report a decrease in their libido/sex drive.”*

Birth control methods prevent pregnancy by interrupting one or more stages of the reproductive process. Most methods work before fertilisation occurs by preventing the release of an egg, blocking sperm from reaching the egg, or preventing sperm from successfully fertilising the egg. Routine contraceptive methods are generally not considered abortifacient because they act before pregnancy is established.

Hormonal contraceptives, including the pill, patch, vaginal ring, injection, implant, and hormonal intrauterine device (IUD), primarily work by altering hormone levels to prevent ovulation, meaning no egg is released for fertilisation. Many hormonal methods also thicken cervical mucus, making it more difficult for sperm to enter the uterus, and may alter the endometrium, the lining of the uterus, in ways that reduce its suitability for implantation. However, evidence suggests that the primary contraceptive effects of routine hormonal methods occur before fertilisation.

Intrauterine devices work through different mechanisms depending on the type. Copper IUDs release copper ions that impair sperm movement and function, making fertilisation unlikely. Hormonal IUDs release levonorgestrel, a progestin that thickens cervical mucus and produces local changes in the uterus that hinder sperm from reaching and fertilising an egg.

Barrier methods, such as condoms, diaphragms, cervical caps, spermicides, and contraceptive sponges, prevent sperm from reaching the egg through physical or chemical means. Fertility awareness-based methods, sometimes referred to as natural family planning, rely on identifying fertile days within the menstrual cycle and avoiding intercourse during those periods.

Emergency contraception works primarily by delaying or preventing ovulation when used shortly after unprotected intercourse. Emergency contraceptive pills containing levonorgestrel or ulipristal acetate act mainly by ensuring that no egg is available for fertilisation. A copper IUD can also be used as emergency contraception and remains highly effective when inserted within several days of unprotected intercourse.

References 54-58.

Antidepressants and Sexual Side Effects

> *"Antidepressants is a perfect example (...) we know there are sexual side effects."*

Research consistently shows that antidepressant medications can contribute to sexual side effects, although the type and severity of these effects vary considerably between medications. Commonly reported problems include reduced libido, difficulties with sexual arousal, erectile dysfunction, vaginal dryness, delayed orgasm, and delayed or absent ejaculation. These symptoms can arise from both the underlying depressive disorder and the medications used to treat it, making it important to consider both factors when evaluating sexual difficulties.

The risk of sexual side effects is highest with antidepressants that strongly affect serotonin signalling, particularly selective serotonin reuptake inhibitors (SSRIs), serotonin–norepinephrine reuptake inhibitors (SNRIs), and clomipramine. Studies have found that certain medications, including paroxetine, escitalopram, duloxetine, and clomipramine, are associated with especially high rates of sexual dysfunction. By contrast, antidepressants such as bupropion, mirtazapine, agomelatine, vortioxetine, vilazodone, and reboxetine generally demonstrate lower rates of sexual side effects and are sometimes used as alternative treatment options when sexual functioning is a concern.

The underlying mechanisms are thought to involve increased serotonin activity, which can suppress sexual desire, reduce arousal, and interfere with orgasm and ejaculation. Research also suggests that serotonergic antidepressants may indirectly reduce dopamine activity within brain reward pathways and affect neural circuits involved in sexual function.

Sexual side effects are common, with some studies reporting prevalence rates of 40–70% or higher among individuals taking SSRIs or SNRIs. These effects can significantly affect quality of life and are a major reason why some patients discontinue treatment. While symptoms often improve following medication adjustment or discontinuation, a small number of individuals may experience persistent symptoms after stopping treatment, a phenomenon referred to as post-SSRI sexual dysfunction. Clinical guidelines therefore emphasise the importance of discussing sexual function before treatment begins, monitoring for changes during therapy, and considering dose adjustments, medication switches, or other management strategies when sexual side effects occur.

References 59-66.

GLP-1 Medications and Men: Testosterone and Erectile Function

> *"The GLP-1s are the weight loss drugs that everybody's talking about (...) there's some mixed data about it helping with increasing testosterone levels [in men] and sometimes bettering and worsening erectile dysfunction. "*

Research suggests that glucagon-like peptide-1 (GLP-1) receptor agonists, including medications such as semaglutide, liraglutide, dulaglutide, and exenatide, generally do not have harmful effects on male reproductive hormones and may improve testosterone levels in certain populations. The most consistent benefits have been observed in men with obesity, type 2 diabetes, or functional hypogonadism, conditions that are commonly associated with reduced testosterone levels and erectile dysfunction. Multiple systematic reviews and meta-analyses have reported increases in total testosterone and, in some cases, free testosterone among men treated with GLP-1 medications. These hormonal improvements often occur alongside weight loss and improvements in metabolic health, although some evidence suggests that GLP-1 drugs may also exert direct effects on the reproductive hormonal axis.

The relationship between GLP-1 medications and erectile function appears more variable. Several studies have reported modest improvements in erectile function, particularly among men with obesity-related metabolic dysfunction. In some research, liraglutide was associated with greater improvements in erectile function than alternative hormonal treatments. However, not all studies

have demonstrated statistically significant benefits, and the overall effect appears to be moderate rather than dramatic.

Concerns that GLP-1 medications may cause erectile dysfunction have received attention, but current evidence remains limited. Observational studies have identified weak associations between GLP-1 use and erectile dysfunction diagnoses, although these findings do not establish causation and may be influenced by underlying health conditions. Pharmacovigilance data have similarly identified only weak signals for erectile dysfunction, reduced libido, or orgasmic difficulties. Overall, available evidence suggests that GLP-1 medications are generally hormonally neutral or beneficial in men with obesity, diabetes, or low testosterone, while evidence that they directly cause erectile dysfunction remains limited and inconclusive.

References 67-75.

GLP-1 Medications and Women: PCOS, Fertility, and Sexual Health

> *"We are starting to look at these drugs in women, but (...) nobody's looking at it for sexual health. Everyone's looking at it for PCOS (...) for fertility (...)*
>
> *there are [sexual health] side effects."*

GLP-1 Agonists and Polycystic Ovary Syndrome (PCOS)

Research suggests that glucagon-like peptide-1 (GLP-1) receptor agonists, including medications such as semaglutide, liraglutide, dulaglutide, and exenatide, are emerging as a promising treatment option for some of the metabolic and reproductive features of polycystic ovary syndrome (PCOS). Originally developed for the treatment of type 2 diabetes and obesity, these medications have been shown to improve several key aspects of PCOS, particularly in women who experience overweight, obesity, or insulin resistance.

The strongest evidence relates to metabolic health. Multiple systematic reviews and meta-analyses have found that GLP-1 receptor agonists can reduce body weight, body mass index (BMI), waist circumference, and visceral fat, often producing greater improvements than metformin or lifestyle interventions alone. These medications have also been associated with improvements in

insulin resistance and glucose regulation, which are central features of PCOS and contribute to many of its long-term health risks.

Evidence also suggests potential benefits for reproductive and hormonal outcomes. Studies have reported reductions in androgen levels, including free testosterone, alongside improvements in markers of androgen excess. Some research indicates that GLP-1 receptor agonists may help regulate menstrual cycles, increase ovulation frequency, and improve the likelihood of natural conception. These effects are thought to be mediated largely through weight loss and improvements in metabolic function, although direct effects on reproductive hormone regulation have also been proposed.

GLP-1 Agonists and Female Fertility

Research suggests that GLP-1 receptor agonists may improve fertility-related outcomes in women with obesity and polycystic ovary syndrome (PCOS). Studies have reported improvements in menstrual regularity, increased ovulation, and higher rates of natural conception among women with PCOS treated with these medications. A meta-analysis of 11 randomised controlled trials involving 840 women found that GLP-1 receptor agonists increased natural pregnancy rates, although no significant benefit was observed for in vitro fertilisation (IVF) outcomes.

These reproductive improvements have been observed alongside reductions in body weight, improved insulin sensitivity, and lower androgen levels. Experimental research also suggests that GLP-1 receptors are present in ovarian and uterine tissues, where GLP-1 signalling may influence follicle development, steroid hormone production, and endometrial function.

However, important uncertainties remain. Human data on the safety of GLP-1 receptor agonists during pregnancy are limited and inconclusive, while animal studies have reported fetal harm. As a result, these medications are contraindicated during pregnancy and are generally recommended to be discontinued before conception. In addition, there is currently very little human evidence regarding their effects on fertility in women without PCOS, which remains a significant gap in the research.

GLP-1 Agonists and Female Sexual Function

Emerging evidence suggests that GLP-1 receptor agonists may be associated with changes in sexual function in some women, although the available data remain limited and are largely based on surveys, case reports, and observational research. Reported effects include reductions in sexual desire, arousal, orgasm, lubrication, and overall sexual satisfaction. In a survey of 914 cisgender

women using GLP-1 or GLP-1/GIP agonists, approximately one-quarter reported changes in sexual function, with a subset describing decreases that they found distressing. Reduced desire and arousal were among the most commonly reported concerns.

Some evidence suggests that these effects may become more common with longer treatment duration. Women aged 31–50 years have also been reported to experience declines in sexual function more frequently than older age groups. In addition, a published case report described the onset of complete anorgasmia, the inability to achieve orgasm, and reduced arousal shortly after starting GLP-1 therapy, with symptoms resolving after treatment discontinuation.

At the same time, not all women report negative effects. Some studies and reviews note that improvements in body weight, mood, and overall health may contribute to improved sexual function in certain individuals. As a result, the overall impact of GLP-1 receptor agonists on sexual health appears to vary between individuals.

Proposed mechanisms remain theoretical. Researchers have suggested that GLP-1 receptor agonists may influence brain pathways involved in reward, motivation, and sexual desire, although direct evidence in humans is limited. Current reviews conclude that the available evidence is heterogeneous and insufficient to establish a clear causal relationship.

References 76-93.

Ferritin and Iron Levels in Women

> *"women should be over 50 or 75 in their ferritin levels (...)*
>
> *there was a recent JAMA study that found (...) 21% of US women age 25 to 54 are iron deficient. And actually, in Black women, they have the highest prevalence with over 31% of them being iron deficient. Pregnant women on average 36 to 37% are iron deficient and anemic, and non-pregnant women of reproductive age between 15 and 49 globally suffer from anemia at about a 30% range."*

Research suggests that there is no single normal ferritin level for all women, as ferritin concentrations vary substantially with age and menopausal status. Ferritin, a protein that stores iron,

is generally lower in premenopausal women and tends to increase after menopause. As a result, age-specific reference ranges may be more informative than a single adult female reference interval.

In premenopausal women, laboratory reference ranges commonly report lower limits of approximately 10–13 µg/L. However, several studies suggest that ferritin levels below 25–30 µg/L may still be consistent with iron deficiency, even when they fall within some laboratory-defined normal ranges. In postmenopausal women, ferritin levels typically rise, with reported lower reference limits ranging from approximately 20–37 µg/L and upper limits extending considerably higher depending on the population studied and the laboratory assay used.

Interpretation of ferritin levels can be complex because ferritin is also an acute-phase reactant, meaning that levels can increase in response to inflammation, infection, liver disease, kidney disease, obesity, and metabolic syndrome, regardless of iron stores. Reviews have noted that some traditional laboratory reference ranges may underestimate the ferritin level at which iron deficiency begins to affect red blood cell production. Consequently, a ferritin level that appears normal according to a laboratory report may still be associated with iron deficiency, particularly in younger women or those with heavy menstrual bleeding. Clinical interpretation should therefore consider age, menopausal status, symptoms, and other relevant health conditions.

Iron Deficiency and Anaemia in Women

A recent study published in *JAMA Network Open* (Barton et al., 2024) reported that iron deficiency among US women aged 25–54 years ranged from 4.46% to 21.23%, depending on the diagnostic definition used. The highest estimate, 21.23%, was based on a ferritin threshold associated with iron-deficient erythropoiesis (ferritin <25 ng/mL), highlighting how prevalence estimates vary substantially according to the criteria applied. The study also found that iron deficiency was more common among Black and Hispanic women than among White and Asian women, although the data provided do not confirm a prevalence exceeding 31% among Black women.

Among pregnant women aged 25–44 years, the same study reported iron deficiency prevalence ranging from 5.44% to 36.10%, again depending on the definition used. The frequently cited figure of approximately 36% therefore refers to iron deficiency rather than iron-deficiency anaemia. Separate evidence summarised by the US Preventive Services Task Force has estimated iron deficiency during pregnancy at approximately 18% overall and iron-deficiency anaemia at around 5%.

The study further emphasised that estimates of iron deficiency can vary by a factor of two to five depending on the ferritin threshold selected, making it important to interpret prevalence figures in the context of the diagnostic criteria used. While the claim that approximately 30% of non-pregnant women of reproductive age globally experience anaemia is widely cited in international health literature, the sources provided here do not contain data that allow this figure to be independently verified. Overall, the available evidence supports that iron deficiency is common among women of reproductive age in the United States, particularly when more physiologically based ferritin thresholds are applied.

References 94-100.

Endometriosis

> *"There's endometriosis where you have painful periods."*

Endometriosis is a chronic, estrogen-dependent inflammatory disease characterised by the presence of endometrial-like tissue outside the uterus. These lesions most commonly occur within the pelvis, affecting structures such as the ovaries and peritoneum, but they can also develop in other locations including the bladder, bowel, and, more rarely, distant organs. The condition affects an estimated 5–10% of women of reproductive age worldwide and is a leading cause of chronic pelvic pain and infertility.

Symptoms vary considerably between individuals. Common manifestations include painful menstrual periods (dysmenorrhoea), chronic pelvic pain, pain during sexual intercourse (dyspareunia), painful bowel movements (dyschezia), painful urination (dysuria), fatigue, and infertility. Some individuals experience severe symptoms, while others may have minimal or no symptoms despite having extensive disease. Symptom severity does not always correspond to the amount of visible endometriosis.

The causes of endometriosis are not fully understood. Retrograde menstruation, the flow of menstrual tissue through the fallopian tubes into the pelvic cavity, is considered an important contributing mechanism, but it does not explain all cases. Current evidence suggests that genetic, immune, endocrine, and inflammatory factors help ectopic endometrial-like tissue implant, survive, and produce ongoing inflammation and fibrosis.

Diagnosis is often delayed because symptoms can overlap with those of other conditions and there are currently no reliable diagnostic biomarkers. Imaging techniques such as ultrasound and magnetic resonance imaging (MRI) can identify some forms of the disease, particularly ovarian endometriomas and deep infiltrating endometriosis, but definitive diagnosis has traditionally relied on surgical visualisation and histological confirmation. Increasingly, endometriosis is being viewed as a systemic condition associated not only with pelvic symptoms, but also with chronic inflammation, pain sensitisation, and broader effects on physical and psychological wellbeing.

References 101-105.

Endometrial Cancer

> *"Endometrial cancer happens when there's too much estrogen and not enough progesterone."*

Endometrial cancer is the most common gynecologic malignancy in developed countries and is largely driven by hormonal imbalances, especially estrogen unopposed by progesterone. Many lifestyle, reproductive, and medical factors modify this hormonal environment and thus influence risk. Most cases (Type I, ~80%) are estrogen-dependent and linked to a hyperestrogenic state, whereas Type II tumours are less hormone-related and less common.

The core "unopposed estrogen" hypothesis states that sustained estrogen exposure without adequate progesterone increases endometrial cell proliferation, mutation risk, and eventually cancer. Obesity is a major contributor, roughly tripling risk and explaining over 40% of cases in affluent settings, mainly by increasing peripheral estrogen production and lowering sex hormone–binding globulin.

References 106-110.

Progesterone, Estrogen, and The Menstrual Cycle

"I don't know, progesterone, and what does that word mean?

[estrogen] is a molecule (...) It acts on all forms of your body to help it be more healthy (...)

So when women have their period, they bleed for a few days, right? And that's when their hormones, their estrogen and their progesterone is at its lowest. Okay? So hormones are at their lowest, and then the hormones start to increase. Your estrogen starts to go up. You don't make progesterone yet in the beginning. Your estrogen starts to go up (...) There's a follicle in your ovary which has an egg, right? It's gonna pop out an egg. Ovulation is when the egg pops out. So you get this big surge of estrogen, and then when the egg pops out of the ovary, right, that's what's gonna make a baby if it gets fertilized, there is a shell of the egg, right? The egg has a shell which makes progesterone. So the second half of the cycle, there's progesterone around. Okay, first half of the cycle, no progesterone. And so that second half of the cycle, the shell is making progesterone, and then when you don't have fertilization, the shell sort of, um- starts to break down. And that natural breakdown is a drop in progesterone, which causes the lining of the uterus to shed, and you get a period again. And so its estrogen goes high in the beginning and then pops out an egg. Progesterone gets high in the second half, then they both fall and you have a period. And so the hormones being low in the beginning, estrogen is not zero (...) it is when the inner lining of the uterus sheds that that's what a menstrual cycle is."

Progesterone and estrogen are steroid hormones that play central roles in reproduction and the regulation of many physiological processes throughout the body. Estrogen refers to a group of related hormones, including estradiol, estrone, and estriol, with estradiol being the predominant and most biologically active form in non-pregnant women. Progesterone is another steroid hormone that is essential for preparing the uterus for pregnancy and supporting embryo implantation and development.

In women, both hormones are produced primarily by the ovaries, although smaller amounts are also produced by the adrenal glands and adipose (fat) tissue. Progesterone is produced by ovarian granulosa and luteal cells, particularly within the corpus luteum after ovulation, and later by the placenta during pregnancy. Both hormones exert their effects by binding to specific receptors that regulate gene expression and cellular activity throughout the body.

Estrogen is involved in the regulation of the menstrual cycle and reproductive organs, while also influencing bone health, cardiovascular function, brain activity, skin, and the urinary tract. Progesterone plays a key role in the menstrual cycle, implantation, pregnancy maintenance, breast development, and the production of neurosteroids, steroid compounds that affect brain function. Together, estrogen and progesterone coordinate the hormonal processes that regulate ovulation, prepare the uterus for pregnancy, and support reproductive health, while also contributing to the function of multiple organ systems beyond reproduction.

Estrogen and Progesterone in the Menstrual Cycle

Estrogen and progesterone work together to regulate the menstrual cycle by coordinating ovulation and the cyclical changes that occur in the uterine lining. Their levels fluctuate throughout the cycle, with each hormone playing distinct but complementary roles at different stages.

During the early follicular phase, both estrogen and progesterone levels are relatively low. As ovarian follicles develop, estrogen production increases, particularly from the dominant follicle. Rising estrogen levels stimulate the growth and thickening of the endometrium, the lining of the uterus, during the proliferative phase. When estrogen levels become sufficiently elevated, they trigger a surge in luteinising hormone (LH), which induces ovulation, the release of an egg from the ovary.

Following ovulation, the ruptured follicle transforms into the corpus luteum, which produces progesterone along with smaller amounts of estrogen. Progesterone converts the thickened endometrium into a secretory lining that is receptive to embryo implantation. It also limits further estrogen-driven proliferation of the uterine lining, helping to prepare the uterus for a potential pregnancy.

If pregnancy does not occur, the corpus luteum regresses and progesterone levels decline. This loss of hormonal support causes the endometrium to break down and shed, resulting in menstruation. The coordinated rise and fall of estrogen and progesterone therefore regulate endometrial growth, ovulation, implantation readiness, and menstrual bleeding. Disruptions in this hormonal balance have been associated with conditions such as heavy menstrual bleeding, endometriosis, and other gynaecological disorders.

References 111-121.

Hormone Replacement Therapy for Women

“But hormone therapy in general is this idea of giving back hormones when you have hot flashes, night sweats, osteoporosis, you know, sort of this over 50 crowd that has these declining estrogen and progesterone levels. And so typically classic hormone therapy is estrogen and progesterone (...)

vaginal hormones are microdoses of estrogen or what we call DHEA vaginally that supports the bladder and the vagina, so it helps with pain with sex, dryness, urinary frequency, urinary urgency, leakage, and it prevents urinary tract infections massively (...)

a billion dollars went into the NIH to study this in women, and they, it was called the Women's Health Initiative, and the study was shut down early. It was thousands and thousands of people age 50 to 79. They gave a hormone pill, like a birth control pill almost, to all of these women, and they followed them, and they stopped the study early in the early 2000s, and they did a press conference (...) And at this press conference, they said, "We're shutting down this, this study early. Hormone therapy causes cardiovascular disease and breast cancer." (...) After the press conference, they actually published the study, so it hadn't even been published yet, and when people actually looked at the study, it didn't say any of those things. It... in fact, women [who] didn't have a uterus and just took the estrogen form, had a less chance of getting and dying from breast cancer. There was a decreased risk of fractures, of diabetes (...) In fact, the same authors of this study back in the early 2000s, published this year, in 2025 actually, that below age 70, that type of hormone therapy, which we don't really use anymore, has no increased risk of cardiovascular disease or stroke (...)

doctors (...) in their mind they're told hormones (...) cause breast cancer.

Only 1.7% of women... are getting prescriptions for hormone therapy who should be offered prescriptions.”

Hormone replacement therapy (HRT), also known as menopausal hormone therapy, is a treatment used to replace hormones that decline during the menopausal transition and after menopause. It primarily involves the administration of estrogen, often together with progesterone or another progestogen, to reduce symptoms associated with estrogen deficiency and to help maintain certain aspects of long-term health.

HRT is most commonly used to treat vasomotor symptoms such as hot flashes and night sweats, as well as genitourinary symptoms including vaginal dryness, pain during sexual intercourse, and urinary complaints. It may also help improve sleep disturbances, mood-related symptoms, and quality of life in women experiencing significant menopausal symptoms. In selected women, HRT is also used to help prevent bone loss and reduce fracture risk.

The specific form of HRT depends in part on whether a woman still has a uterus. Women who have undergone hysterectomy generally receive estrogen alone, whereas women with an intact uterus typically require both estrogen and a progestogen to protect the endometrium, the lining of the uterus. Hormones can be administered in several forms, including tablets, skin patches, gels, sprays, and vaginal preparations. Treatment regimens are usually tailored to the individual and adjusted to the lowest effective dose.

Research consistently identifies HRT as the most effective treatment for menopausal vasomotor and genitourinary symptoms. However, treatment decisions must also consider potential risks, including venous thromboembolism, stroke, and breast cancer, which vary according to a woman's age, health status, hormone regimen, route of administration, and duration of use. Current clinical guidance emphasises an individualised approach that balances potential benefits and risks according to each woman's symptoms, medical history, and preferences.

Vaginal Hormones and Genitourinary Symptoms of Menopause

Research supports the use of low-dose vaginal hormones for the treatment of genitourinary syndrome of menopause (GSM), a condition characterised by symptoms such as vaginal dryness, pain during sexual intercourse, urinary urgency and frequency, urinary leakage, and recurrent urinary tract infections (UTIs). Among the available treatments, low-dose vaginal estrogen has the strongest evidence base.

Systematic reviews and clinical guidelines consistently report that vaginal estrogen improves vaginal dryness and dyspareunia, the medical term for pain during sexual intercourse. Evidence also indicates benefits for urinary symptoms, including urgency, frequency, nocturia (waking at night to urinate), and some forms of urinary incontinence. In addition, multiple reviews have found that vaginal estrogen reduces the frequency of recurrent UTIs in postmenopausal women.

Vaginal dehydroepiandrosterone (DHEA), also known as prasterone, has also been studied as a local hormonal treatment. Randomised controlled trials and meta-analyses have shown that

vaginal DHEA improves symptoms of vaginal dryness and dyspareunia compared with placebo, although the magnitude of benefit is generally modest. Some studies have also reported improvements in sexual function. However, evidence regarding its effects on urinary symptoms and recurrent UTIs is more limited, and direct evidence supporting UTI prevention has not been established.

Current clinical guidelines recognise low-dose vaginal estrogen as a first-line treatment for GSM and related urinary symptoms. Vaginal DHEA is also considered an effective option, particularly for moderate to severe vaginal symptoms and pain during sexual intercourse. Overall, the evidence suggests that both treatments can improve symptoms of GSM, although vaginal estrogen remains the more extensively studied option for urinary symptoms and recurrent UTI prevention.

Women's Health Initiative Hormone Therapy Studies and Subsequent Re-analyses

The Women's Health Initiative (WHI) hormone therapy trials examined the effects of estrogen plus progestin and estrogen alone in postmenopausal women. The original studies found that combined estrogen–progestin therapy was associated with increased risks of coronary heart disease, stroke, pulmonary embolism, and invasive breast cancer, although it also reduced hip fracture and colorectal cancer risk and improved menopausal symptoms. These findings led to the early termination of the combined-therapy trial, and subsequent follow-up showed a persistent increase in breast cancer incidence.

In contrast, the estrogen-only trial, conducted in women who had undergone hysterectomy, found no reduction in coronary heart disease risk and an increased risk of stroke, resulting in early discontinuation. However, long-term follow-up demonstrated lower breast cancer incidence and mortality among women receiving estrogen alone compared with placebo.

Subsequent analyses emphasised that the effects of hormone therapy vary according to the type of hormone regimen, age at treatment initiation, and time since menopause. Overall, the evidence did not support the use of hormone therapy for the prevention of cardiovascular disease or other chronic conditions. However, the balance of benefits and risks appeared more favourable among women who initiated treatment before age 60 or within 10 years of menopause. In some younger subgroups, particularly women who underwent oophorectomy and received estrogen alone, hormone therapy was associated with lower all-cause mortality.

Long-term follow-up and re-analyses have consistently reported divergent breast cancer outcomes between the two regimens, with estrogen-only therapy associated with reduced breast cancer incidence and mortality and combined estrogen–progestin therapy associated with increased breast cancer risk.

2025 Women's Health Initiative Re-analysis of Hormone Therapy and Cardiovascular Risk

A 2025 secondary analysis of the Women's Health Initiative (WHI) hormone therapy trials re-examined cardiovascular outcomes among women with moderate to severe vasomotor symptoms who received conjugated equine estrogen (CEE) alone or combined with medroxyprogesterone acetate (MPA). The analysis found no statistically significant increase in a composite measure of atherosclerotic cardiovascular disease (ASCVD) among women aged 50–59 years receiving either hormone therapy regimen. In this age group, both CEE alone and CEE plus MPA were associated with neutral ASCVD risk estimates compared with placebo.

However, the study did not conclude that hormone therapy carries no cardiovascular or stroke risk in all women under 70 years of age. Among women aged 60–69 years, risk estimates were higher for some outcomes, although confidence intervals included the possibility of no effect. In women aged 70 years and older, the analysis found a clear increase in ASCVD risk with both hormone therapy regimens.

These findings refine, rather than overturn, earlier WHI results. Previous WHI reports and subsequent reviews found that standard-dose oral hormone therapy was associated with an increased risk of stroke, and some analyses concluded that this risk was not substantially modified by age or time since menopause. The 2025 study focused specifically on women with vasomotor symptoms and assessed a composite cardiovascular outcome rather than stroke alone.

Hormone Replacement Therapy and Breast Cancer Risk

Research indicates that hormone replacement therapy (HRT) is associated with a modest increase in breast cancer risk, although the magnitude of this risk varies according to the type of therapy used, duration of treatment, and individual patient characteristics. Large cohort studies and meta-analyses have consistently found that current HRT use is associated with a higher risk of breast cancer compared with non-use, with absolute increases typically amounting to a small number of additional cases per 10,000 women each year.

The risk appears to differ by treatment regimen. Combined estrogen–progestogen therapy is consistently associated with a greater increase in breast cancer risk than estrogen-only therapy. Evidence also suggests that different progestogens may be associated with different levels of risk, with some formulations showing lower relative risks than others.

Duration of use is an important factor. Breast cancer risk increases with longer periods of treatment and is most clearly observed among women who use HRT for five years or more. The risk generally decreases after treatment is discontinued and may approach that of never-users over time, although some residual increase may persist following prolonged use of combined hormone therapy.

Certain factors appear to modify risk. Studies have reported higher relative risks among lean women and women with dense breast tissue. In women with a previous history of breast cancer, systemic HRT has been associated with an increased risk of cancer recurrence, particularly in hormone receptor-positive disease.

Hormone Therapy Use Among Midlife and Postmenopausal Women

Studies from several countries indicate that menopausal hormone therapy (MHT) use among midlife and postmenopausal women is relatively low and has declined substantially over recent decades. In the United States, national survey data found that current MHT use decreased from 26.9% in 1999 to 4.7% between 2017 and early 2020. A large UK primary-care study reported annual prescribing prevalence rates of 7.9% in 2010 and 6.9% in 2020, while Korean claims data found MHT use ranging from 6.3% to 7.8% among women aged 40 years and older.

Reported rates vary according to the population studied and the methods used to measure treatment use. For example, analyses based on healthcare visits have reported hormone therapy prescribing in 3.8% of ambulatory visits among midlife and older women, while studies of selected populations have found higher rates. Some investigations restricted to new prescriptions, specific healthcare settings, or narrow medication categories have reported substantially lower prescribing frequencies.

References 122-145.

Urinary Tract Infections: Risks, Treatment Gaps, and Prevention

“urinary tract infections (...) can also go into your bloodstream and cause fevers and chills and cause kidney infections. It can cause something called urosepsis, where you have to go into the intensive care unit and need antibiotics through an IV, and it can kill you if you have an infection go through your whole body (...)

There is, and hor- there's, there's no probiotic on earth that is proven to [01:02:00] do what the vagina needs quite like hormones. Hormones make the tissue go from not acidic to quite acidic, and it is that acidic environment that protects it from infection (...)

women are dying of UTIs and they have an option, but no one is writing the prescriptions. there's published data that less than nine percent of Medicare patients are getting prescriptions for [DHEA]. More than 75% of people in large database collections are not getting prescriptions for this.

with vaginal hormones (...) there's no increased risk of any problems (...)

there is some data that cranberry pills can help, uh, with preventing UTIs (...) but the, uh, amount that you'd have to drink is, uh, very sugary and diabetes inducing, and it wouldn't taste that good. And so they, they do make pills, but it's a small, these things are small things that help”

Urinary Tract Infections, Kidney Infections, and Urosepsis

Urinary tract infections (UTIs) can progress beyond the bladder and, in some cases, lead to more serious complications. When bacteria ascend from the bladder into the kidneys, the resulting infection is known as pyelonephritis, a severe form of UTI that can cause significant illness and may become life-threatening if left untreated. Pyelonephritis can lead to kidney injury and, in some cases, allow bacteria to enter the bloodstream.

Urosepsis is a form of sepsis that originates from a urinary tract infection and is considered the most severe complication of UTI. It typically develops when an ascending urinary infection reaches the kidneys and is accompanied by bacteraemia, the presence of bacteria in the bloodstream. UTIs are recognised as a common source of sepsis, accounting for a substantial proportion of sepsis cases across clinical studies.

Although most UTIs do not progress to urosepsis, the risk is higher in individuals with factors such as urinary obstruction, kidney stones, urinary catheters, structural abnormalities of the urinary tract, chronic kidney disease, diabetes, or other complicated urinary infections. Research has shown that urosepsis can progress to septic shock and multiple organ dysfunction, both of which are associated with a high risk of death. Mortality rates reported for urosepsis vary considerably depending on the population studied and the severity of illness, ranging from a few percent in some cohorts to 30–40% or higher in severe cases.

Vaginal Estrogen Use in Medicare Beneficiaries With Genitourinary Syndrome of Menopause (GSM)

The available evidence does not provide an estimate of the proportion of Medicare patients with urinary tract infection (UTI) diagnoses who are prescribed vaginal DHEA. The cited study examined vaginal estrogen use among 1,838,732 Medicare women aged 66 years and older with a diagnosis related to genitourinary syndrome of menopause (GSM). Overall, 9.0% filled at least one vaginal estrogen prescription during a median follow-up of eight years. Women presenting with recurrent UTIs were among the least likely to receive vaginal estrogen treatment compared with those presenting with other GSM-related symptoms.

Vaginal DHEA (Prasterone) Prescribing and Treatment Uptake

The available evidence does not provide a direct estimate of the proportion of women who are prescribed vaginal DHEA (prasterone). Studies of prasterone have primarily focused on efficacy and safety rather than population-level prescribing patterns.

However, several sources indicate that genitourinary syndrome of menopause (GSM), a common indication for vaginal DHEA, is substantially undertreated. One review reported that fewer than 10% of women with menopausal genitourinary symptoms receive treatment despite the availability of multiple therapeutic options. Position statements and consensus reviews similarly describe GSM as underdiagnosed and undertreated.

These findings suggest that many women who could potentially benefit from treatments such as vaginal DHEA are not receiving therapy. However, the available studies do not quantify prasterone prescribing rates and do not directly demonstrate that more than 75% of women are not prescribed vaginal DHEA. Overall, the evidence supports substantial under-treatment of GSM but does not provide a precise estimate of vaginal DHEA utilisation.

Cranberry Pills and Urinary Tract Infection Prevention

Research suggests that cranberry products can provide a modest reduction in the risk of recurrent urinary tract infections (UTIs), particularly among women with a history of recurrent infections. A 2023 Cochrane review of 50 trials involving 8,857 participants found that cranberry products reduced the risk of symptomatic, culture-confirmed UTIs by approximately 30% overall. Benefits were most evident in women with recurrent UTIs, children, and some individuals at increased risk of procedure-related infections.

Evidence from clinical trials indicates that cranberry capsules and extracts can reduce UTI recurrence, delay the time to first recurrence, and decrease antibiotic use. Several studies have reported lower rates of culture-confirmed UTIs among women taking cranberry capsules compared with placebo. Meta-analyses focused on women have similarly found a reduction in recurrent UTIs of approximately 20–30%, with capsules and tablets appearing at least as effective as cranberry juice.

The effectiveness of cranberry supplements appears to depend on product formulation and dose. Reviews have highlighted the importance of standardized levels of proanthocyanidins (PACs), particularly A-type PACs, with doses of approximately 36 mg per day or higher most commonly associated with benefit. Earlier studies using low-dose or poorly standardised products often reported little or no effect.

Cranberry capsules are generally well tolerated and are associated with few side effects. However, current evidence does not support a clear benefit in pregnant women or frail older adults living in institutional settings. Overall, the available evidence suggests that cranberry pills containing adequate amounts of standardized PACs can modestly reduce the risk of recurrent UTIs and may offer a useful non-antibiotic preventive option for some individuals.

Cranberry Capsules, Cranberry Juice, and Blood Glucose

Research suggests that cranberry capsules generally contain less sugar and have more favourable effects on glucose metabolism than standard cranberry juices, particularly those that are sweetened. While most studies compare the metabolic effects of different cranberry preparations rather than directly measuring glucose content, the overall evidence indicates important differences between juice and capsule formulations.

Cranberry juice often contains naturally occurring sugars and, in some products, added sugars that increase caloric and carbohydrate intake. By contrast, cranberry capsules, powders, and

tablets typically provide cranberry-derived compounds with minimal sugar and energy content. Reviews have noted that some cranberry juice interventions delivered substantial amounts of fructose and glucose, which may reduce potential metabolic benefits.

Studies examining glycaemic outcomes have found that dried cranberry preparations, including capsules, are associated with improvements in markers of insulin resistance, such as fasting insulin levels and the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), a measure of how effectively the body responds to insulin. These effects appear more consistent than those observed with cranberry juice. Low-calorie or unsweetened cranberry juices generally have little effect on blood glucose and behave similarly to water or other low-calorie beverages, whereas standard-calorie juices are more likely to produce short-term increases in glucose and insulin levels.

Overall, the available evidence suggests that cranberry capsules provide the potential benefits of cranberry supplementation with substantially lower sugar exposure than most cranberry juices. Dried cranberry preparations also appear to have more favourable effects on insulin resistance, while the metabolic impact of cranberry juice depends largely on its sugar and calorie content.

References 146-158.

Painful Sex (Dyspareunia) in Women

> *"There are some published reports that up to 75% of women will say at some point in their life sex is painful (...) during menopause it climbs drastically, with estimates ranging to 20 to almost half of women having pain during sex."*

Prevalence estimates for painful sex, or dyspareunia, vary according to the population studied and the definition used. A narrative review reported a worldwide prevalence of approximately 15%, while another review estimated that 10–28% of women experience dyspareunia during their lifetime.

Large population-based studies have reported similar findings. A British probability survey found that 7.5% of sexually active women reported painful sex lasting at least three months during the previous year, while a nationwide French study found that 7.9% of reproductive-age women

experienced painful sex often or always. Among premenopausal women, prevalence estimates have been reported in the range of 12–21%.

Systematic reviews and meta-analyses have reported substantially wider prevalence ranges because of differences in study methods, definitions, and assessment tools. In representative community samples, reported rates generally range from approximately 6% to 32%. Much higher rates have been observed in specific clinical populations. For example, one study found that 75.7% of women with endometriosis reported dyspareunia.

Dyspareunia Prevalence in Menopausal Women

Painful intercourse (dyspareunia) is a common symptom during the peri- and postmenopausal years, particularly in the context of genitourinary syndrome of menopause (GSM), a condition characterised by vulvovaginal and urinary symptoms related to hormonal changes. Prevalence estimates vary considerably across studies due to differences in populations, definitions, and whether analyses are limited to sexually active women.

However, research consistently indicates that dyspareunia is a frequent and clinically significant concern during menopause. Reported prevalence rates generally range from approximately 15–45% in menopausal populations, with some studies finding that around 20–40% of sexually active postmenopausal women experience painful intercourse. Reviews have also reported prevalence estimates as high as 45% among postmenopausal women, compared with approximately 10% in women of childbearing age. Although menopause-related hormonal changes contribute to symptoms, dyspareunia is recognised as a multifactorial condition and may also be influenced by other physical, psychological, and pain-related factors. Within GSM, painful intercourse is consistently identified as one of the most common symptoms alongside vaginal dryness and urinary complaints.

References 159-164.

Prevalence of Anorgasmia in Women

> *“about 20% of women will say that they can't have an orgasm (...)”*

Anorgasmia, the persistent difficulty or inability to achieve orgasm, is a relatively common sexual concern among women. Research suggests that prevalence is typically reported in the range of 20–35%, although estimates vary substantially depending on how anorgasmia is defined and the population being studied. Reviews of female sexual dysfunction have reported prevalence estimates for orgasmic disorders ranging from approximately 11–41%, reflecting differences in measurement methods, cultural factors, and study design. Population-based studies have found rates around 24–26% in some community samples, while higher estimates are often reported in clinical settings and among women with specific medical conditions. Researchers also note that studies do not always distinguish between complete inability to achieve orgasm and broader orgasm-related difficulties, which contributes to variability in reported rates. Overall, the available evidence indicates that orgasmic difficulties are common, affecting roughly one in four to one in three women, although prevalence estimates can vary considerably across populations and definitions.

References 165-169.

Non-Surgical Lysis of Clitoral Adhesions and Sexual Function

> *"the clitoris has this hood to it, okay? And about 25, 23% of the time, the hood can get stuck to the head (...) It's called a clitoral adhesion (...) And we published data that if you remove these adhesions in an office-based, very simple procedure, we saw improvements in orgasm, arousal, and satisfaction up to 60 to 70% (...)"*

A 2022 retrospective case series found that non-surgical lysis of clitoral adhesions was associated with improvements in several aspects of sexual function and high levels of patient satisfaction. Among survey respondents, 76% reported reduced pain, 63% reported improved sexual arousal, 64% reported improved ability to achieve orgasm, and 71% reported greater satisfaction with sex following the procedure. Additionally, 83% were satisfied with their decision to undergo treatment, and 93% said they would recommend the procedure to others with clitoral adhesions. The authors concluded that non-surgical lysis may be a promising treatment option for symptomatic clitoral adhesions, while noting that the findings were based on a small retrospective case series from a single clinic and should therefore be interpreted as preliminary evidence.

Reference 170.

Pornography Viewing Among Young Adults

"I've got some data here on a website like Pornhub, which is one of the leading porn websites. It says that roughly 65% of their traffic is men. independent surveys tracking any regular pornography use find a similar gap, but note that women's consumption is highly age-dependent. Men between the age of 18 and 35, 75 to 95% of them report viewing porn regularly, whereas women age 18 to 35, only 34%, roughly, report viewing porn regularly (...)

A couple dozen studies, including a major meta-analysis, show a consistent link between solo porn consumption and lower relationship and sexual satisfaction (...) heavy solo use can desensitize the brain's reward system, leading to performance anxiety and erectile issues during real-life partner sex."

Pornography use is common among young adults, although there is a substantial gender difference in the frequency of viewing. Large population surveys and reviews suggest that approximately half of young adult men engage with pornography on a regular basis, typically defined as weekly use. For example, national survey data indicate that around 46% of men aged 18–39 reported intentionally viewing pornography within a given week, while broader reviews have similarly concluded that roughly half of younger men are regular pornography consumers. In contrast, regular use among women is considerably less common. Survey data suggest that approximately 15–20% of women in the same age range report weekly pornography viewing. Although lifetime and past-year exposure to pornography is relatively common among women, frequent consumption tends to be concentrated within a smaller minority. While prevalence estimates vary across countries and according to how "regular use" is defined, the available evidence consistently indicates that weekly pornography viewing is reported by around half of young adult men and approximately one in six young adult women.

Pornography Use and Relationship Satisfaction

Research suggests that solo pornography use is associated with lower sexual satisfaction and, in some cases, lower relationship satisfaction, although these associations vary according to context, frequency of use, gender, and partner dynamics. Large reviews and meta-analyses have found that pornography consumption is modestly linked to lower sexual and relationship satisfaction, particularly among men. However, the evidence indicates that solitary use, especially when frequent, secretive, or substantially different from a partner's usage patterns, is more consistently associated

with negative outcomes than pornography use itself. In contrast, pornography viewed together by partners is often associated with greater sexual communication, closeness, and satisfaction. Several studies suggest that factors such as masturbation frequency, greater arousal to pornography than to a partner, comparisons between real-life and pornographic sexual experiences, and reduced sexual communication may help explain these associations. Researchers also note that the effects are generally small and complex, with some studies finding no direct relationship between solitary pornography use and satisfaction measures.

Pornography Use, Reward Processing, and Sexual Function

Research suggests that problematic pornography use (PPU), rather than pornography use frequency alone, is associated with alterations in reward processing and a higher likelihood of sexual difficulties. Neuroimaging studies indicate that individuals with PPU show heightened responses to pornography-related cues and may develop a stronger motivational focus on sexual stimuli, alongside reduced responsiveness to other rewards. Some research also suggests that repeated exposure can be associated with desensitization to partnered sexual experiences and a preference for pornography-related stimulation.

However, large population studies generally find that the amount of pornography consumed is not independently associated with erectile dysfunction once factors such as age, mental health, relationship satisfaction, and sexual desire are taken into account. In contrast, problematic or compulsive patterns of use, particularly when accompanied by anxiety, guilt, sexual dissatisfaction, or self-perceived addiction, are more consistently linked to erectile difficulties and sexual performance concerns. Researchers have proposed that reward-system adaptations, combined with psychological factors such as performance anxiety and emotional distress, may contribute to these outcomes in some individuals.

References 171-184.

Spontaneous and Responsive Sexual Desire

> *“men and women have different types of arousal, spontaneous and responsive. And I was looking at some of the data on the variance, which I think is very important for people to know, and it says that men are highly spontaneous in their arousal. Which kind of means from my interpretation, please correct me if I'm wrong, that as a man I can literally be, I can literally think about something and, um, get aroused. And it's not to say that women can't, but it- the data suggests that men are more that way inclined. The data here says that spo- the spontaneous rate in a man is about 70%, whereas in women it's about 10 to 15%. It says the responsive rate (...) is 10 to 15% in men and in women in this particular report is 40 to 50% (...)”*

Research does not provide clear, well-established population estimates for the proportion of men and women who predominantly experience spontaneous versus responsive sexual desire. While influential theories have proposed that women are more likely to experience responsive desire, arising in response to intimacy or sexual stimulation, and men are more likely to experience spontaneous desire, empirical evidence supporting specific prevalence figures remains limited. One large study of men identified a predominantly spontaneous desire pattern in approximately 74% of participants, while only 2.5% were classified as primarily responsive.

However, comparable population-level estimates for women are lacking. Existing studies suggest that responsive desire is common among women, but many women experience both spontaneous and responsive desire rather than fitting into mutually exclusive categories. Reviews of the literature also highlight conceptual and measurement challenges in distinguishing between these forms of desire, and newer assessment tools are still undergoing validation. Overall, current evidence does not clearly support commonly cited figures suggesting that 70% of men experience spontaneous desire, while 40–50% of women experience responsive desire. Instead, the research indicates that sexual desire is more complex and variable than a simple sex-based division between spontaneous and responsive patterns.

References 185-189.

Prevalence of Premature Ejaculation in Men

> *“you know how there's men who premature ejaculate (...) That's probably like 8% of men”*

Premature ejaculation (PE) is one of the most common male sexual concerns, although prevalence estimates vary considerably depending on how it is defined and measured. A large systematic review reported a mean prevalence of 14.2% across 79 studies, while research generally suggests that approximately 5–15% of men meet standardized diagnostic criteria for PE and 20–30% report ejaculating sooner than they would like in broader self-report surveys. Overall, clinically defined PE appears to affect a minority of men, whereas concerns about ejaculating too quickly are reported by roughly one in five to one in four men.

References 190-195.

Declining Sexual Frequency

> *“People are having less sex than ever.”*

Research suggests that sexual frequency has declined in some populations over recent decades, although the trend is not universal. Studies from the United States, Britain, Germany, Japan, Australia, and China have reported increases in sexual inactivity or reductions in partnered sexual activity, particularly among young adults, married or cohabiting couples, and some middle-aged groups. However, several large surveys have found relatively stable rates of sexual activity over time, suggesting that changes in relationship patterns, socioeconomic factors, living arrangements, and partnership status may play an important role in these trends.

References 196-203.

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