# Independent Research & Further Reading

**Guest**: Rena Malik

**Disclaimer 1:** The sources presented here, directly (or as closely as possible), look at statements made by the guest in this episode. To report on each topic thoroughly, an extensive search and review (beyond the scope of this document) would be required.

**Disclaimer 2:** This podcast and its associated materials do not aim to substitute professional medical advice. For any medical concerns, it is essential to consult a qualified health professional.

## Contents

## Declining Sexual Activity in Contemporary Populations

> *“when you look at some of these stats, and I'll throw this graph that I've, I found here up on screen and it shows that people are (...) having increasingly less sex and becoming more sexless, especially young, amongst young adults, between 18 and 30”*

Research indicates a measurable decline in partnered sexual activity across several high-income countries, particularly among younger adults and those in early to midlife. Large-scale survey data from the United States, including the General Social Survey (GSS) and the National Survey of Sexual Health and Behavior (NSSHB), show that individuals report fewer sexual encounters per year compared to previous decades, alongside a notable rise in sexual inactivity, defined as no sexual activity within the past year. This trend is especially pronounced among younger men and adults aged 25–34, and is observed across both married and unmarried groups.

Comparable patterns have been documented in Britain and other countries, where repeated national surveys reveal declining frequency of partnered sex, fewer individuals reporting high-frequency sexual activity, and a growing proportion reporting no recent sexual activity. These changes appear to reflect both an increase in the number of adults without steady partners and a reduction in sexual frequency within established relationships. Associated factors include socioeconomic constraints, such as lower income and unemployment, as well as poorer health and rising rates of singlehood. Notably, commonly proposed explanations such as increased pornography use or longer working hours do not consistently account for the observed decline.

***References 1-4.***

## Prevalence of Sexual Choking in Young Adults

> *“So young people, if you look at data, like 60% of women, and I think it's 20% of men have been choked during sex of that age group, like college age group. And um, and it's very, very common. Oftentimes they're being choked more than one time during a sexual encounter. And of those people who get choked, 20% have been choked 25 times or more.”*

Current research indicates that relatively high proportions of young adults report having experienced choking during sex, particularly among women. Findings from studies of undergraduate and young adult populations suggest that lifetime prevalence rates for women can approach or exceed 50%, with some samples reporting figures near 60% among college-aged or 18–35-year-old women. In contrast, prevalence among men is generally lower, though still substantial, typically ranging from approximately 25% to 45% depending on the sample and country.

Broader national datasets show lower overall prevalence when wider age ranges are included, with estimates such as 21% of women and 11% of men aged 18–60 in the United States, though rates are higher among those under 40. Evidence also indicates that repeated exposure is not uncommon, with a notable proportion of individuals, particularly women, reporting having experienced choking multiple times. For instance, studies of undergraduate populations show that over one-third of women who are sexually active report being choked more than five times, compared to a much smaller proportion of men. These findings suggest that while figures such as 60% for young women are consistent with certain high-prevalence samples, estimates for men are generally lower and vary more widely across studies..

***References 5-10.***

## High Daily Screen Time Among Young People

> *"People 30 and below, around that age, the extreme outliers are spending eight to 10 hours on their phones, on social media, and on the internet. And roughly about 15 to 20% of young people describe their usage as almost constant,"*

Research suggests that a substantial minority of young people report very high levels of daily screen use, including time spent on smartphones, social media, and the internet. Some studies of university-aged populations indicate that over half of participants report using their smartphones for nine or more hours per day, with many others falling within the 3–8 hour range. Within these same samples, a notable proportion report spending between 7 and 10 hours per day specifically on social media, indicating that extended usage is not uncommon in certain cohorts.

Larger national and cohort studies provide a more moderate overall picture, while still confirming the presence of high-use groups. For example, UK adolescent data show that around

one-fifth of participants report five or more hours of daily social media use, and Korean national survey data identify a distinct subgroup exceeding eight hours of daily smartphone use. Objective tracking studies, which measure actual device usage rather than self-report, tend to find average daily use around five to six hours, though with considerable variation and some individuals reaching or exceeding ten hours per day. Taken together, the evidence indicates that while most young people do not consistently reach 8–10 hours of daily use, a meaningful minority do.

***References 11-17.***

## Autonomic Regulation of Sexual Arousal and Sexual Fow State

> *"Basically, when you get aroused, you need to be in a parasympathetic nervous system state. So in order to get an erection, you need to be in the state, which is like rest and digest (...)*
>
> *"You can also get into a sexual flow state. So when you look at flow state data, and this is by Dr. Emily Jamea, when you look at the flow state data, you need things to be slightly challenging to get into the flow state."*

Sexual arousal is not dependent on a purely parasympathetic state, but rather emerges from a coordinated interaction between parasympathetic and sympathetic nervous system activity. Parasympathetic processes play a central role in genital arousal, particularly through sacral outflow via the pelvic nerves, which promotes vasocongestion (increased blood flow to genital tissues), erection in males, and lubrication in females. These mechanisms involve smooth muscle relaxation and are foundational to the physiological aspects of sexual response.

However, evidence consistently shows that sympathetic activation is also integral to sexual arousal. In women, moderate levels of sympathetic activity appear to facilitate genital arousal, while both very low and very high levels may inhibit it, suggesting an optimal mid-range rather than a purely parasympathetic condition. In men, markers of sympathetic activation, such as increased heart rate, blood pressure, and noradrenaline release, are observed during sexual arousal, and acute stress can enhance certain aspects of sexual stimulus processing. Contemporary models therefore conceptualise sexual arousal as a dual autonomic process, in which parasympathetic activity supports genital response, while sympathetic activation contributes to psychological and

physiological arousal. This integrated perspective indicates that a parasympathetic state alone is neither necessary nor sufficient for sexual arousal.

**Sexual Flow State: Deep Absorption in Sexual Experience**

The notion of a "sexual flow state" is derived from the broader concept of flow, originally articulated by Mihaly Csikszentmihalyi, and refers to a deeply immersive and optimal form of sexual experience. Although not a formal clinical term, it is used in research to describe moments during sex in which attention, arousal, and pleasure become fully aligned, producing a state of intense absorption. This state is characterised by focused concentration, diminished self-consciousness, a distorted sense of time, and a feeling of effortless engagement, often accompanied by heightened enjoyment and intrinsic reward.

When applied to sexual contexts, this form of absorption may involve a heightened awareness of bodily sensations, reduced distraction, and a sense that the experience unfolds naturally without deliberate control. Such experiences are sometimes described as "transcendent," reflecting their overlap with broader flow phenomenology. Importantly, sexual flow is not synonymous with general sexual arousal; rather, it represents a more specific subset of experiences in which conditions such as attentional focus, emotional engagement, and a balance between stimulation and responsiveness are optimally aligned.

Empirical work suggests that individuals who are more dispositionally inclined to experience flow during sex report higher levels of both personal and partner-related sexual satisfaction. This aligns with broader findings in attentional research, which indicate that focusing on embodied, emotionally engaged aspects of erotic experience enhances both subjective arousal and depth of involvement, whereas detached or overly cognitive attention may diminish these effects. In this sense, sexual flow can be understood as a state of unified attention and embodied presence that contributes to particularly meaningful and satisfying sexual encounters.

***References 18-26.***

## Sexual Frequency and All-Cause Mortality

*"Sex (...) helps you live longer. So there's been a few studies looking at sex and longevity. And when you look at people who have sex once a week, compared to people who have sex once a year (...) the difference in all-cause mortality is 49%. They live 49% longer than people who only have sex once a year (...)*

*There was actually an interesting study in 1997, I think it was, where they looked at the number of orgasms men had, and they found that (...) for every a hundred orgasms men had, they lived like 13% longer. They had a 13% increase in life expectancy."*

Observational research suggests a notable association between sexual frequency and all-cause mortality, particularly when comparing very low frequency (approximately once per year) with more regular activity (around once per week). Large cohort studies, including analyses of United States population data, indicate that individuals reporting sexual activity on a weekly basis, typically defined as around 52 or more times per year, show approximately a 40–50% lower relative risk of death over the follow-up period compared to those reporting extremely infrequent activity, such as 0–1 times per year. For example, one analysis found a hazard ratio of 0.51, meaning a roughly 49% lower risk of all-cause mortality after adjusting for multiple confounding variables.

However, this relationship must be interpreted with caution. The evidence is correlational rather than causal, and sexual frequency likely functions as a proxy for broader indicators of health and wellbeing, including physical fitness, mental health, relationship status, and social integration. Additional analyses suggest that the association may follow a non-linear, or U-shaped, pattern, where both very low and very high frequencies are linked to less favourable outcomes. Furthermore, the strength of the association can diminish when factors such as depression and comorbid health conditions are taken into account. As such, while the data indicate a significant statistical difference, they do not establish that increasing sexual frequency directly reduces mortality risk.

***References 27-31.***

## Loneliness as a Global Health Priority

> *"The WHO made loneliness like an epidemic. So they've said that loneliness is as bad as having like 15 cigarettes."*

The World Health Organization has not formally classified loneliness as an "epidemic." Instead, it has framed loneliness and social isolation as a significant and pressing global health concern. In 2023, the organisation launched an International Commission on Social Connection, explicitly identifying loneliness as a major public health issue and a social determinant of health, meaning it has measurable effects on physical and mental wellbeing across populations.

The WHO's language is deliberate and technical. Rather than using the term "epidemic," which carries specific epidemiological criteria, it refers to loneliness as a "global health challenge" and a policy priority, particularly in relation to ageing populations and broader societal wellbeing. This positioning places loneliness alongside other major health risk factors, such as physical inactivity and obesity, in terms of its potential impact.

The characterisation of loneliness as an "epidemic" originates primarily from other sources, most notably the United States Surgeon General, who used this term in 2023 to emphasise the scale and urgency of the issue. Similarly, academic and media discourse often adopts this language rhetorically. However, within formal WHO classifications and communications, loneliness is not designated as an epidemic, but rather as a critical and growing public health concern.

***References 32-37.***

## Sexual Activity and Cardiovascular Health

> *"sex is a cardiovascular workout, right? For many people. As a cardiovascular workout, you are getting a physical activity with your partner. you are increasing your heart rate. You are doing these things that are also good for your body."*

Evidence suggests that sexual activity can confer modest cardiovascular benefits, though these effects vary depending on age, sex, baseline health, and relationship context. Physiologically,

partnered sex produces short-term increases in heart rate and blood pressure comparable to mild to moderate physical activity, such as climbing one or two flights of stairs. In healthy individuals, these responses are transient, with rapid return to baseline, and the risk of triggering acute cardiac events is very low, accounting for less than 1% of heart attacks. Clinical guidance generally considers sexual activity safe for individuals with stable cardiovascular conditions following appropriate evaluation.

Over the longer term, observational studies indicate potential associations between more frequent sexual activity and improved cardiovascular outcomes, particularly in men. Higher intercourse frequency has been linked to reduced incidence of cardiovascular disease and major cardiac events, and among patients recovering from myocardial infarction (heart attack), those who resume sexual activity relatively early tend to show lower mortality within the following year, even after accounting for physical health status. Additional findings suggest that sexual activity may be associated with lower levels of inflammatory markers, such as C-reactive protein (CRP, a marker of systemic inflammation), and improved heart rate variability, which reflects healthier autonomic regulation of the heart.

There is also evidence that sexual activity may indirectly support cardiovascular health through related mechanisms, including improved sleep quality, reduced stress, and lower next-day blood pressure. However, these associations are not uniform across all populations. Some studies indicate that very frequent or physically intense sexual activity in older men may be associated with increased cardiovascular risk, while in women, particularly in high-quality relationships, sexual activity may be linked to reduced hypertension risk.

***References 38-45.***

## Mediterranean Diet and Erectile Dysfunction Risk

> *“there's a study called The Health Professionals Follow Up Study. They look at 20,000 men and they saw that men who adhered to a Mediterranean diet had a 22% lower risk of erectile dysfunction.”*

Findings from the Health Professionals Follow-up Study do support an association between adherence to a Mediterranean diet and reduced risk of erectile dysfunction (ED), but the commonly cited figure of a 22% reduction applies specifically to younger men, rather than to all men overall.

In a large analysis published in JAMA Network Open, men under the age of 60 with the highest adherence to a Mediterranean diet had a hazard ratio of 0.78 for incident ED compared to those with the lowest adherence, corresponding to approximately a 22% lower risk. However, this effect diminished with age, with reductions of around 18% in men aged 60 to under 70, and only about 7% in those aged 70 and above. Earlier findings from the same cohort, reported in The Journal of Urology, showed a more modest overall association, with men in the highest Mediterranean diet category having approximately a 14% lower risk of ED without age stratification.

***Reference 46.***

## Pistachio Consumption and Erectile Function

> *"they actually had a study on pistachios. They looked at a hundred grams of pistachios. Guys did a hundred grams of pistachios every day. And they saw a decrease in erectile dysfunction, so better erectile function. And they also looked at it on an ultrasound. So they took, uh, their, the penis and they took a doppler ultrasound to look at blood flow and they saw improvements in blood flow on the penis after adhering to this pistachio diet."*

A small study does report that consuming 100 grams of pistachios daily was associated with improvements in erectile function and penile blood flow, but the strength of this evidence is limited. In a prospective study conducted in Turkey, 17 men with chronic erectile dysfunction followed a pistachio-enriched diet for three weeks. After this period, participants showed significant increases in erectile function scores, measured using the International Index of Erectile Function (IIEF-15), alongside improvements in penile blood flow, assessed via Doppler ultrasound as increased peak systolic velocity, which reflects enhanced arterial inflow to erectile tissue.

The study also reported favourable changes in lipid profiles, including reductions in total cholesterol and low-density lipoprotein (LDL, often termed "bad cholesterol"), and increases in high-density lipoprotein (HDL, "good cholesterol"). These changes may plausibly contribute to vascular improvements relevant to erectile function, given the role of blood flow in erection physiology.

However, the study's design imposes significant limitations. It lacked a control or placebo group, involved a very small sample size, and was conducted over a short duration. As a result, it is

not possible to determine whether the observed improvements were directly caused by pistachio consumption or influenced by placebo effects, behavioural changes, or other confounding factors.

***References 47-49.***

## Fruit Consumption and Erectile Function

> *"colorful fruit, like blueberries, citrus fruits, lycopene, which is red fruits. All of these things improve antioxidants and also have been shown to reduce the incidence of erectile dysfunction (...) specifically blueberries actually came out to have, I think something around 20% also improvement in erectile function when you're eating blueberries regularly."*

Evidence suggests that regular consumption of fruit, particularly flavonoid-rich varieties such as blueberries, citrus fruits, and other red or dark-coloured fruits, is associated with a lower risk of erectile dysfunction (ED). Large prospective cohort studies involving tens of thousands of men have found that higher intake of specific flavonoids, including flavones, flavanones, and anthocyanins (plant compounds with antioxidant properties), is linked to an approximately 11–16% reduction in ED risk. Greater overall fruit intake has also been associated with reduced risk, with some analyses reporting around a 14% lower likelihood of developing ED. When focusing on key fruit sources such as blueberries, strawberries, apples, pears, and citrus, the reduction in risk may approach 19%, with particularly strong associations observed for citrus fruits and blueberries.

Broader dietary patterns reinforce this association. Meta-analyses and observational studies indicate that diets rich in fruits and vegetables are linked to lower rates of ED and, in some cases, improved erectile function scores. In younger men, higher flavonoid intake has been associated with substantially lower odds of ED, while studies in clinical populations, such as men with diabetes, suggest that each additional daily serving of fruits and vegetables may reduce ED risk by around 10–13%. However, not all studies find a significant relationship, particularly in older populations, where factors such as reduced sexual activity and comorbidities may obscure associations.

Fruits rich in flavonoids, antioxidants, and related compounds may support endothelial function, the health of blood vessels, and enhance nitric oxide availability, which is essential for vasodilation and penile erection. While regular fruit consumption is consistently associated with

reduced risk of developing ED, there is less direct evidence that it substantially improves established erectile dysfunction.

***References 50-55.***

## Fibre Intake, Gut Health, and Erectile Function

> *"When you eat fiber in your gut, it converts to short chain fatty acids. These short chain fatty acids then sort of have these endothelial protective mechanisms. They protect the blood vessels, they make them healthier. And so when your blood vessels are healthier, you get better metabolic health. So you get less diabetes, less high blood pressure, less high cholesterol. And these, all of these things together improve erectile function"*

Current evidence suggests that the benefits of dietary fibre may contribute to improved erectile function, but primarily through indirect cardiometabolic and vascular pathways rather than direct gut-specific mechanisms. High-fibre, plant-rich diets are consistently associated with improvements in insulin sensitivity, lipid profiles, body weight, and overall cardiovascular health. These effects are partly mediated by short-chain fatty acids (SCFAs), which are produced by gut microbiota during fibre fermentation and help regulate inflammation, glucose metabolism, and vascular function. Because erectile dysfunction (ED) is closely linked to conditions such as type 2 diabetes, metabolic syndrome, and cardiovascular disease, improvements in these domains are likely to reduce ED risk by enhancing endothelial function, which is critical for penile blood flow.

Large-scale analyses support this indirect relationship. Diets rich in fruits, vegetables, whole grains, legumes, and nuts, all of which are high in fibre, have been associated with reduced risk of ED and modest improvements in erectile function scores. These dietary patterns are understood to support vascular health, reduce systemic inflammation, and improve nitric oxide availability, all of which are central to erectile physiology. In this sense, fibre contributes to a broader physiological environment that is more conducive to healthy sexual function.

Emerging research points to a possible role of the gut microbiome in reproductive health more broadly. SCFAs influence not only metabolic processes but also gut–brain and endocrine signalling pathways, which may affect hormonal balance and inflammatory status. Animal studies suggest that high-fibre diets can improve reproductive parameters such as sperm quality, potentially

via microbiome-mediated mechanisms. However, direct evidence linking gut-specific effects of fibre to erectile function in humans remains limited.

***References 56-61.***

## Effects of Cardiovascular Exercise on Erectile Function

> *"Cardiovascular exercise. When you look at the data, there's multiple different sources of data. The one study that's quoted very often is 150 minutes of exercise, of cardiovascular exercise, moderate intensity. When you look at the improvement in erectile function scores, it is the same amount of improvement as you would see when you take a medication like Viagra (...)*
>
> *So there was a group where they looked at men who had heart disease and they weren't really able to do moderate intensity exercise. So they did like a five minute warmup. They did 20 minutes of walking and five minutes of a cool down. And with this supervised protocol, they still improve erectile function by 70% (...)"*

Moderate-intensity aerobic exercise, such as 150 minutes per week, can meaningfully improve erectile function, though current evidence indicates that its effects are generally smaller than those achieved with phosphodiesterase type 5 (PDE5) inhibitors, such as Sildenafil. Randomised controlled trials show that aerobic exercise programmes, typically involving 120–180 minutes per week, produce moderate to large improvements in erectile function, often reflected as increases of approximately 3–5 points on the International Index of Erectile Function (IIEF). A recent meta-analysis reported a standardised mean difference of 0.81, indicating a clinically meaningful benefit, particularly in men not already using medication. Higher volumes of exercise tend to yield greater improvements, suggesting a dose–response relationship.

However, PDE5 inhibitors demonstrate substantially larger effect sizes. Large meta-analyses of randomised trials show that medications such as sildenafil typically increase IIEF scores by approximately 7–13 points compared to placebo, with markedly higher rates of restored erectile function. These drugs act directly on the nitric oxide–cyclic GMP pathway, producing rapid and reliable improvements in penile blood flow, and are therefore considered first-line treatments for many forms of erectile dysfunction. Evidence indicates that combining structured aerobic exercise

with PDE5 inhibitor therapy produces greater improvements than medication alone, suggesting a synergistic effect. Exercise contributes to underlying vascular and metabolic health, while PDE5 inhibitors provide more immediate functional enhancement.

**Study: Post-Myocardial Infarction Walking and Erectile Function**

A randomised controlled trial by Begot et al. (2015) examined whether a simple, home-based walking programme could improve erectile function in men recovering from acute myocardial infarction (MI, commonly known as a heart attack). Eighty-six men with recent MI and low cardiovascular risk were assigned either to a progressive, unsupervised outdoor walking intervention or to usual post-MI care. At hospital discharge, erectile dysfunction (ED) was highly prevalent, affecting 84% of participants. After 30 days, the walking group showed a marked reduction in ED prevalence, decreasing by 71% from baseline, whereas the control group exhibited a non-significant increase of 9%. Improvements in functional capacity, measured by the 6-minute walk test (a standard assessment of aerobic fitness), were also significantly greater in the walking group. Notably, there was a strong inverse relationship between walking distance and ED severity, indicating that higher aerobic capacity was associated with better erectile function. These findings align with broader evidence from cardiac rehabilitation and exercise-based interventions, suggesting that aerobic activity can produce clinically meaningful improvements in erectile function, likely through enhancements in vascular health and endothelial function (the ability of blood vessels to dilate effectively).

***References 62-67.***

## Resistance Training, Muscle Mass, and Testosterone

> *“we know that resistance training is significantly correlated with testosterone. So when you do heavy resistance training of your large muscle groups (...) you actually see improvements in testosterone (...)*
>
> *So we know that muscle mass decreases about 7% every decade of life after around 40. And so if you maintain your muscle mass, compared to people who lose muscle mass, when you maintain it through resistance exercise, they're three times less likely to have erectile dysfunction (...)*
>
> *like 80% of people over 70 have sarcopenia (...)”*

Resistance training reliably produces short-term increases in testosterone, but its effects on long-term, resting testosterone levels are modest and inconsistent. Acute responses are well established: exercises involving large muscle groups, higher volume, and shorter rest periods typically lead to transient rises in testosterone, peaking shortly after the workout and returning to baseline within approximately one hour. Similar, though generally smaller and more variable, responses are observed in women, particularly following high-intensity protocols.

In contrast, the chronic effects of resistance training on baseline testosterone levels are less clear. Some studies report modest increases or improvements in free testosterone (the biologically active fraction), particularly in younger men, while others find no significant change despite substantial gains in muscle mass and strength. Reviews of the literature suggest that resistance training may help maintain testosterone levels with age rather than meaningfully elevate them over time.

Although testosterone plays a role in muscular adaptation, the magnitude of acute post-exercise testosterone increases does not appear to predict strength or hypertrophy gains. Even when baseline testosterone remains stable, resistance training can still produce significant improvements in muscle and performance. Overall, resistance training induces reliable short-term hormonal responses, but does not consistently lead to sustained increases in resting testosterone levels.

**Age-Related Muscle Loss and Erectile Function**

Research indicates that skeletal muscle mass declines gradually from midlife, with the rate of loss increasing with age. From approximately 40–50 years onwards, men typically lose around 4–5% of muscle mass per decade, while women lose approximately 3–4%. This decline accelerates in later life, with individuals over 60–70 experiencing losses closer to 6–10% per decade, and in some cohorts, even higher. Longitudinal data suggest that by the mid-70s, annual muscle loss may reach approximately 0.8–1.0% in men and 0.6–0.7% in women. However, there is substantial individual variability depending on physical activity, nutrition, and overall health.

There is also consistent evidence linking muscle mass and strength with erectile function. Observational studies show that men with sarcopenia (age-related loss of muscle mass and function) have significantly higher odds of erectile dysfunction (ED), in some cases up to 2.5–2.7 times greater risk of moderate to severe ED compared to those with preserved muscle mass. Conversely, greater muscle strength and participation in muscle-strengthening activities are associated with lower odds of ED. For example, increases in hand-grip strength, a common proxy for overall muscular health, are associated with reduced likelihood of ED, and population-level data suggest that men engaging in regular resistance training have approximately 30% lower odds of ED. Muscle mass and strength are closely tied to metabolic health, insulin sensitivity, vascular function, and hormonal regulation, all of which play key roles in erectile physiology.

**Prevalence of Sarcopenia in Adults Over 70**

Sarcopenia, defined as age-related loss of muscle mass and function, is relatively common in older adults, though estimates vary depending on diagnostic criteria and population. In community-dwelling adults aged 70 and above, a reasonable overall estimate is that approximately 15–25% have sarcopenia. This aligns with findings from multiple cohort studies and meta-analyses, which typically place prevalence within this range for relatively healthy, independent older populations.

However, prevalence increases substantially with advancing age and frailty. Among individuals aged 80 and above, rates often rise to 30–50% or higher, particularly in populations with greater comorbidity or reduced physical function. In more vulnerable settings, such as nursing homes or hospitalised populations, prevalence can reach as high as 30–70%, reflecting the strong association between sarcopenia, illness, and functional decline. Variability in estimates is largely driven by differences in diagnostic frameworks, such as whether definitions prioritise muscle mass alone or include strength and physical performance, as well as regional and demographic differences.

***References 68-89.***

## Erectile Dysfunction Prevalence After 50

> *"above the age of 50, we see 52% of men having erectile dysfunction, which is half 52% men. Yeah. And it goes up 10% every decade. So 60% of 60-year-old. 70% of 70 year olds."*

Erectile dysfunction (ED) is common in men over 50 and increases markedly with age, though prevalence varies across studies and populations. Broadly, evidence indicates that approximately one-third of men in their early 50s experience some degree of ED, with estimates typically ranging from about 25% to 40%. This rises progressively with age, such that by the 60s, roughly 50–75% of men report at least mild ED, and in men aged 70 and above, prevalence often exceeds 50%, with some studies reporting rates approaching 80–90% or higher.

The notion that ED increases by "10% per decade" is a simplification and not strictly accurate. This figure originates from analyses showing that the *odds* of ED may increase by approximately 10% per year of age, rather than prevalence rising by a fixed 10 percentage points every decade. In reality, the increase in ED prevalence is non-linear. For example, some datasets show prevalence rising from around 20% at age 50 to 35% by the late 50s, and then accelerating more sharply in later decades, reaching substantially higher levels by age 70 and beyond. This age-related pattern reflects cumulative changes in vascular health, hormonal regulation, and comorbid conditions such as diabetes, hypertension, obesity, and cardiovascular disease. Thus, while ED clearly becomes more common with age, it does not follow a simple linear increase of 10% per decade, but rather a progressively accelerating trajectory influenced by overall health status.

***References 90-95.***

## Erectile Dysfunction and Future Heart Attack Risk

> *"within three to five years [of developing erectile dysunction], you will start developing issues with your heart. And so it precedes those issues. And if seven years later, 14% of those guys will have a heart attack."*

Evidence does not support the specific claim that approximately 14% of men will have a heart attack within seven years of developing erectile dysfunction (ED). Instead, research consistently shows that ED is associated with an increased *relative* risk of cardiovascular events, including myocardial infarction (heart attack), rather than a fixed absolute risk applicable to all individuals. Large cohort studies and meta-analyses indicate that men with ED have approximately a 1.4 to 1.6 times higher risk of cardiovascular events compared to men without ED, even after adjusting for other risk factors. This reflects a meaningful elevation in risk, but it does not translate into a universal percentage such as "14% over seven years." Absolute risk varies considerably depending on factors such as age, baseline cardiovascular health, and the presence of comorbidities like diabetes, hypertension, and obesity.

Some studies reporting figures in the range of 10–15% refer to longer follow-up periods, such as 10–12 years, and often include broader cardiovascular outcomes rather than heart attacks specifically. In contrast, shorter-term data, including seven-year follow-up estimates, typically show lower absolute event rates, often in the range of a few percent annually when accumulated over time. What is well established is that ED can function as an early marker of underlying vascular disease, often preceding cardiovascular events by several years.

***References 96-104.***

## Nocturnal Erections and Clitoral Tumescence

> *"when you're a young boy, you get erections three to five times a night, and they can last up to 40 minutes long (...) When you get older, like in your forties, that drops to about half the time. (...)*
>
> *women also have nocturnal clitoral tumescence."*

Night-time erections, medically termed nocturnal penile tumescence (NPT), show a clear and progressive decline with age. In younger men, particularly those under 50, objective sleep-laboratory studies indicate an average of approximately 2 to 3 effective erections per night. In contrast, men aged 50 and above typically experience fewer episodes, averaging closer to 1 to 2 per night, with statistically significant reductions observed across studies.

Beyond frequency, the quality of nocturnal erections also changes with age. Younger men tend to exhibit longer durations of tumescence (periods of erection during sleep), greater rigidity, and more rapid onset. In older men, these erections are generally shorter in duration, with reduced maximal circumference changes and slower development. While the basic capacity for tumescence may be relatively preserved into midlife, more fully rigid erections during sleep become less common in later decades, particularly after the age of 60–65.

These changes are thought to reflect a combination of physiological factors, including age-related declines in androgen levels, alterations in vascular function, and structural changes in penile tissue. Additionally, the coordination between REM sleep (the stage of sleep most associated with dreaming), nocturnal erections, and hormonal rhythms, such as testosterone secretion, becomes less synchronised with age.

**Nocturnal Genital Arousal in Women**

Women do experience nocturnal genital arousal, including clitoral tumescence, analogous in principle to nocturnal penile tumescence in men. Sleep laboratory studies using physiological measures, such as vaginal photoplethysmography (a method for assessing blood flow to genital tissues), show that women exhibit cyclic increases in genital blood flow during sleep, particularly during rapid eye movement (REM) sleep. These episodes occur in the vast majority of REM periods and reflect genuine vasocongestion, the same underlying mechanism responsible for sexual arousal during wakefulness.

Although most studies measure vaginal rather than clitoral blood flow directly, these responses are part of a unified genital arousal system. The clitoris contains erectile tissue that responds to increased blood flow in a manner analogous to the penis, and clitoral and vaginal engorgement typically occur together as part of the same physiological process. Notably, the magnitude of these nocturnal responses can be comparable to levels observed during conscious sexual stimulation, indicating that they represent substantial physiological arousal.

These patterns are considered normal and distinct from pathological conditions such as persistent genital arousal disorder or clitoral priapism, which involve prolonged or distressing arousal outside typical regulatory mechanisms. While there is no standardised clinical test equivalent to male nocturnal penile tumescence, the evidence clearly supports the presence of regular, sleep-related genital engorgement in women, including clitoral involvement, particularly during REM sleep.

***References 105-112-.***

## Testosterone, Circadian Rhythm, and Morning Erections

> *"And so [testosterone] is highest in the morning, which is why very often you get a morning erection (...)"*

Testosterone levels follow a clear circadian rhythm, peaking in the early morning and declining throughout the day, with morning values typically 20–35% higher than later measurements in younger men. This diurnal variation diminishes with age and is often blunted in men with testosterone deficiency. Adequate testosterone is important for normal nocturnal and morning erections, as hypogonadal men show reduced nocturnal penile tumescence that can improve with testosterone therapy. Mechanistically, androgens support erectile function through effects on nitric oxide signalling and central nervous system regulation. However, testosterone is not the sole determinant of morning erections. Normal erectile function can occur across a range of testosterone levels, and once a sufficient threshold is reached, higher levels confer little additional benefit. Testosterone therefore appears to play a regulatory role, aligning sexual drive and physiological readiness, rather than acting as a direct trigger of individual erections.

***References 113-122.***

## Embryological Homology of External Genital Structures

> *"So the penis and the clitoris are homologs. The scrotum and the labia majora are homologs."*

The penis and clitoris, as well as the scrotum and labia majora, are homologous structures in typical human development. During early fetal life, the external genitalia exist in an undifferentiated or “indifferent” state, meaning they share a common anatomical foundation. The penis and clitoris both arise from the genital tubercle, with differentiation occurring under the influence of androgens around the ninth week of gestation; in their presence, the structure develops into a penis, and in their relative absence, into a clitoris. Both organs retain comparable structural features, including erectile corpora cavernosa, a distal glans, and similar neurovascular organisation, alongside broadly comparable histological composition. Likewise, the scrotum and labia majora originate from the labioscrotal swellings; these fuse in males to form the scrotum and remain separate in females as the labia majora. This shared developmental origin accounts for their anatomical and tissue similarities, and is reflected in clinical practice, where labia majora tissue can be used in scrotal reconstruction.

***References 123-128.***

## Neuroendocrine Changes During Orgasm and Post-Coital Dysphoria

> *“during an orgasm is you have this release of dopamine and then your prolactin goes up (...)*
>
> *some people who feel post-coital dysphoria, they actually feel sad or they feel depressed after the orgasm.”*

Orgasm is reliably associated with a marked increase in prolactin (a hormone involved in sexual satiety and refractory processes), alongside a transient inhibition of dopaminergic activity (dopamine, a neurotransmitter central to motivation and reward). Laboratory studies consistently show that plasma prolactin rises substantially following orgasm in both men and women, whether induced through masturbation or intercourse, with elevations persisting for at least an hour and in some cases showing a secondary increase the following day. This effect appears specific to orgasm itself, as sexual arousal without orgasm does not produce the same prolactin response, and greater increases are typically observed following intercourse, consistent with enhanced subjective sexual satisfaction.

With respect to dopamine, direct human measurements during orgasm are limited, but available evidence does not support a simple increase at the moment of climax. Instead, dopamine activity is understood to rise during the anticipatory and arousal phases, facilitating sexual

motivation and physiological readiness. At orgasm, however, the pronounced prolactin surge suggests a temporary suppression of hypothalamic dopaminergic signalling, as dopamine normally exerts an inhibitory effect on prolactin release. This shift is further supported by theoretical and animal research indicating that orgasm involves opioid-mediated processes that dampen dopamine activity, contributing to the transition from heightened arousal to post-orgasmic satiety and refractoriness.

**Post-Coital Dysphoria: Definition and Psychological Features**

Research describes post-coital dysphoria (PCD) as the experience of unexpected negative emotions following consensual and typically satisfactory sexual activity, often referred to as “post-sex blues.” Despite the sexual encounter being desired and pleasurable, individuals may experience tearfulness, sadness, or depression-like affect, alongside anxiety (a state of heightened nervousness or unease), irritability, agitation, or even feelings such as guilt, emptiness, disgust, or a desire to withdraw from a partner. These responses are often perceived as paradoxical, given the preceding positive experience, and may last from a few minutes to over an hour.

Evidence suggests that PCD is relatively common as an occasional experience, with a substantial proportion of both men and women reporting at least one episode across their lifetime, although recurrent or persistent cases are less frequent. It has been observed across different contexts, including within relationships, casual encounters, and solitary sexual activity. While the underlying causes remain unclear, PCD is understood as multifactorial, with statistical associations observed between its occurrence and broader psychological factors such as distress, anxiety, prior sexual trauma, and other sexual difficulties.

***References 129-145.***

## Affectionate Touch, Stress Regulation, and Relationship Quality

> *“there's some evidence, you know, the Gottman's have been on your podcast, doing a 20 second hug (...) helps alleviate stress (...) doing a six second kiss with your partner (...) these sort of small things can help alleviate stress.”*

Research suggests that brief forms of affectionate touch, such as hugging and kissing, can contribute to stress reduction and improvements in relationship quality. Experimental studies indicate that even short physical contact can modulate physiological stress responses. For instance, couples who engaged in a brief period of touch, including a 20-second hug, prior to a stress-inducing task exhibited lower heart rate and blood pressure responses compared to those without physical contact. Similarly, short embraces before stress exposure have been associated with reductions in cortisol (a hormone released in response to stress), particularly in women. Controlled trials further show that a single 20-second hug, or even self-directed soothing touch, can attenuate cortisol reactivity, while observational studies suggest that more frequent daily hugging predicts lower next-day cortisol patterns and improved emotional resilience following interpersonal conflict. Evidence also indicates that being hugged may buffer the relationship between conflict and susceptibility to illness.

In relational terms, affectionate behaviours such as kissing and cuddling appear to support bonding and perceived relationship quality. Interventions that increased the frequency of romantic kissing over several weeks have been linked to reductions in perceived stress and improvements in relationship satisfaction, alongside physiological markers such as cholesterol. Similarly, structured increases in physical closeness, such as cuddling, have been shown to enhance relationship satisfaction beyond simply spending more time together. More broadly, affectionate touch is associated with improved individual well-being, greater perceived partner support, and enhanced relational security.

***References 146-155.***

# Sleep Restriction, Sleep Apnea, and Testosterone Levels

> *"Just so much abundance of data on how sleep affects hormonal health. So when you sleep, they looked at data on men sleeping five hours a night versus eight hours a night. Guys who sleep five hours a night (...) testosterone drops by 15% (...)*
>
> *If you have one really easy way to check is take a measuring tape and measure your neck circumference. If it's more than 17 inches for a guy or 16 inches for a female, it means it's very likely that you may have sleep apnea (...)*
>
> *when you improve sleep apnea, we've seen improvements in testosterone as high as 200 nanograms per deciliter.*

Research suggests that short-term sleep restriction can reduce testosterone levels, although the often-cited figure of a 15% decrease is context-specific rather than universal. A small controlled trial involving healthy young men found that restricting sleep to five hours per night for one week led to a reduction in daytime testosterone of approximately 10–15% compared to a well-rested condition. This study, conducted under tightly controlled laboratory conditions, provides evidence that acute sleep loss can meaningfully affect hormonal regulation.

However, broader evidence presents a more mixed picture. Other experimental studies using different sleep restriction protocols, such as more severe but shorter durations or more moderate chronic restriction, do not consistently replicate this degree of testosterone decline. Larger population-based and genetic studies likewise do not support a simple or uniform causal relationship between sleep duration alone and testosterone levels. Instead, the evidence suggests that factors such as overall sleep quality, insomnia (difficulty falling or staying asleep), and total sleep deprivation may play a more significant role than sleep duration.

**Neck Circumference and Obstructive Sleep Apnoea**

Larger neck size is strongly associated with obstructive sleep apnoea (OSA), a condition characterised by repeated interruptions in breathing during sleep. Neck circumference (NC) has been shown in multiple studies to correlate with the apnoea–hypopnoea index (AHI), which measures the frequency and severity of breathing disturbances. Individuals with more severe OSA tend to have larger neck circumferences, and in some clinical cohorts, NC has been found to be a stronger predictor of OSA presence and severity than body mass index (BMI). Threshold values, often around

40–41 cm in men and approximately 36–40 cm in women, have demonstrated moderate accuracy in identifying elevated risk.

In paediatric populations, similar associations are observed. Among children and adolescents, particularly those with obesity, measures such as neck circumference and neck-to-height ratio independently predict OSA severity even when accounting for developmental factors such as age and puberty. Meta-analytic evidence further supports that individuals with OSA tend to have, on average, slightly larger neck measurements than those without the condition.

However, neck size should be understood as a risk indicator rather than a diagnostic tool. It partly reflects patterns of fat distribution, especially around the upper airway, which can contribute to airway obstruction during sleep. Its predictive value may vary across populations and is sometimes reduced when other factors, such as BMI, are taken into account, particularly in women. As such, while a larger neck circumference is a useful clinical marker of increased risk, formal assessment of OSA requires validated screening tools and, ultimately, objective sleep studies.

**Sleep Apnoea Treatment and Testosterone Levels**

While obstructive sleep apnoea (OSA) is associated with lower testosterone levels, particularly in more severe cases, treating the condition does not reliably lead to increases in testosterone. Meta-analyses consistently show that men with OSA tend to have reduced total testosterone compared to controls, although this relationship is partly influenced by confounding factors such as obesity (excess body fat), which itself is strongly linked to lower testosterone.

With respect to treatment, continuous positive airway pressure (CPAP), the standard therapy for OSA, has not been shown to produce consistent or significant increases in testosterone levels, even when used for several months. Large reviews and meta-analyses report no meaningful changes in total testosterone, free testosterone, or sex hormone-binding globulin following CPAP therapy. While some smaller or less controlled studies suggest possible improvements, these findings are not robust or consistently replicated. Evidence more strongly supports weight loss as a key factor in improving testosterone levels in men with OSA. Reductions in body weight are associated with clear, often proportional increases in testosterone, suggesting that metabolic factors play a central role.

***References 156-173.***

# Plastics (BPA, Phthalates, PFAS) and Endocrine Disruption

> *"plastic water bottles have things like phthalates and BPAs, which can affect hormonal health (...) Also, things like plastics in the environment, PFAS (...) can affect hormone health (...)*
>
> *if you look at the data, like people are (...) consuming quite a bit of microplastics and we're seeing them actually even in testicles and penis tissue samples."*

BPA (bisphenol A), phthalates, and PFAS (per- and polyfluoroalkyl substances) are widely recognised as endocrine-disrupting chemicals (EDCs), meaning they can interfere with the body's hormonal systems.

BPA exhibits oestrogen-like and anti-androgen effects, interacting with multiple hormone receptors, including oestrogen, androgen, thyroid, and glucocorticoid receptors. It can alter hormone synthesis, signalling, and regulation, with effects observed across reproductive, metabolic, neuroendocrine, and immune systems. Related compounds (such as BPS and BPF) show similar or sometimes stronger endocrine-disrupting activity.

Phthalates are also well-established endocrine disruptors. They can act as xenoestrogens (synthetic compounds that mimic oestrogen), anti-androgens, and anti-thyroid agents, interfering with hormone receptor binding and steroid hormone production. Evidence from human and animal studies links phthalate exposure to altered reproductive development, changes in puberty timing, fertility issues, and broader endocrine and developmental effects.

PFAS are similarly classified as endocrine-disrupting pollutants. They affect multiple hormonal systems, including thyroid hormones (T3, T4, TSH) and sex hormones, and are associated with metabolic, reproductive, and developmental changes. Mechanistically, PFAS can bind to hormone transport proteins, activate nuclear receptors such as PPARs (involved in metabolism), and disrupt key regulatory axes such as the hypothalamic–pituitary–gonadal and thyroid systems.

**Microplastic Exposure and Detection in Male Reproductive Tissues**

Research suggests that human exposure to microplastics (tiny plastic particles typically smaller than 5 mm) has increased over recent decades, alongside rising environmental plastic accumulation. Modelling studies across multiple countries indicate that combined dietary and

inhalational exposure has risen substantially since the 1990s, in some regions by several-fold. Estimates of annual intake vary, but suggest tens of thousands of particles consumed through food and beverages, with substantially higher totals when inhalation is included. Environmental records, including long-term studies in wildlife, generally support the view that microplastic presence has increased since the mid-20th century, although trends may vary by region and ecosystem.

In parallel, emerging evidence shows that microplastics have been detected in human male reproductive tissues. Studies have identified microplastics in testicular tissue, semen, and penile tissue samples. In testicular analyses, microplastics have been found in a high proportion of samples, with various polymer types present, and some studies reporting associations with reduced testicular weight, lower sperm count, and markers of inflammation (the body's immune response to injury or foreign material). Microplastics have also been detected in semen across both general and high-exposure populations. More recently, microplastics have been directly identified in penile tissue, including in samples from men undergoing surgical procedures and in cases of penile cancer, where higher concentrations and diversity of particles have been observed in tumour tissue compared to adjacent non-cancerous tissue.

***References 194-174.***

## Pornography Use Patterns and Relationship Satisfaction

> *"We see that when couples watch porn together, they actually have higher satisfaction (...) better sex lives (...) When they watch it together, they are more likely to be more satisfied in their relationship. And when there's a discord, like one person really doesn't like it or doesn't use it and the other one uses it a lot, that's where we see the problem come up."*

Studies using dyadic (partner-paired) data and large samples consistently indicate that shared pornography use, where both partners engage together, is linked to higher levels of relationship satisfaction, sexual satisfaction, and perceived closeness. This pattern is also associated with more open sexual communication, suggesting that mutual engagement may function as a form of shared experience that supports intimacy. In some large population samples, couples who either both use pornography together or both abstain tend to report the highest levels of satisfaction, sexual desire, and frequency of sexual activity.

In contrast, discordant or discrepant pornography use, where one partner engages frequently and the other does not, is associated with lower relationship satisfaction and greater relational strain. Studies report that such mismatches are linked to poorer communication, reduced emotional closeness, and, in some cases, increased relational conflict or aggression. Sexual satisfaction appears lowest in these discordant pairings, whereas couples with aligned patterns of use, whether high or low, tend to report more favourable outcomes.

***References 195-198.***

## Prevalence and Composition of Squirting

> *"about 40% of women squirt. Squirting is the emission of fluid at the time of orgasm (...)*
>
> *there's been a lot of research on this. So there's people who say it's pee there's people who say it's not, and there's a couple studies. So one is they put dye into the bladder and they took women who said they were squirters and they had them orgasm and they saw is there dye in the fluid. And yeah, there was dye in fluid."*

Research suggests that a substantial proportion of women report having experienced squirting (the expulsion of fluid during sexual arousal or orgasm), with estimates commonly falling around 40% in large survey-based studies. Some population studies report even higher lifetime prevalence, although regular or frequent squirting appears to be relatively uncommon. Variability in estimates partly reflects differences in how squirting is defined and whether it is distinguished from related phenomena such as female ejaculation.

With respect to composition, the evidence distinguishes between "squirting" and "female ejaculation" as physiologically distinct processes. Squirting refers to the expulsion of a larger volume of clear fluid via the urethra, and studies using imaging and biochemical analysis indicate that this fluid is primarily diluted urine (urine that is less concentrated due to fluid accumulation during arousal). Measurements of substances such as urea, creatinine, and uric acid closely resemble those found in urine. However, many samples also contain small amounts of prostate-specific antigen (PSA), a marker of secretions from the Skene's glands (sometimes referred to as the female prostate), suggesting a minor contribution from these glands. In contrast, female ejaculation in a stricter sense involves the release of a much smaller volume of thicker, whitish fluid originating primarily from the

Skene's glands, with a distinct biochemical profile that includes higher concentrations of PSA and differs from urine.

***References 199-203.***

## Post-Coital Urinary Tract Infections: Prevalence and Mechanisms

> *"a subset of women who get UTIs after sex. Not everybody, but some do. And it's, it's not because of the ejaculate or because of the male harboring some bacteria. It's because of the actual thrusting of the penis. It's taking bacteria from the outside and making it more easy for it to go through the urethra into the bladder."*

A substantial subset of women experience urinary tract infections (UTIs) following sexual activity, commonly referred to as post-coital UTIs. UTIs (infections typically involving the bladder, most often caused by bacteria such as *Escherichia coli*) are highly prevalent, with over half of women experiencing at least one in their lifetime. Among sexually active women, a significant proportion of UTIs occur shortly after intercourse, with early studies suggesting that many cases arise within 24 hours. Prospective research further demonstrates a dose–response relationship, whereby increased frequency of sexual activity is associated with a markedly higher risk of infection. This association is observed across age groups, including postmenopausal women.

The primary mechanism involves the mechanical transfer of bacteria. Due to the relatively short length of the female urethra (the tube connecting the bladder to the outside of the body) and its proximity to the anus, bacteria from the gastrointestinal tract, particularly *E. coli*, can be more easily introduced into the urinary tract. Sexual intercourse can facilitate this process by physically moving bacteria from the perineal region toward and into the urethra, allowing ascent into the bladder. Additional contributing factors include the presence of bacteria around the vaginal and urethral opening prior to intercourse, as well as the use of spermicides or diaphragms, which can disrupt protective vaginal microbiota (beneficial bacteria, such as lactobacilli, that help prevent infection).

However, not all women experience post-coital UTIs, indicating variability in susceptibility. Differences in the vaginal and periurethral microbiome, individual immune and genetic factors, prior history of UTIs, and behavioural factors (such as hygiene practices or delayed urination after sex) all

appear to influence risk. While post-coital urination may offer modest protective benefit, the condition is best understood as the result of an interaction between anatomical, microbial, and behavioural factors rather than a single cause.

***References 204-209.***

## Orgasm Without Genital Stimulation: Evidence and Key Studies

> *"there's actually a famous paper about non genital orgasms and like how people orgasm without any genital stimulation (...)*
>
> *There was a study where they took 19 couples, a small study (...) and they basically told them to stimulate an erogenous zone and a non erogenous zone. So it was, um, the forehead for the non erogenous zon and the erogenous zone [was the neck] And so they had the couple stimulate and they told them, stimulated at levels of 18 centimeters per second in terms of how fast you're caressing the arm or body part, and at three centimeters per second. And what they found was those who stimulate at three centimeters per second had more sexual arousal, had more pleasant stimulation compared to those who were stimulating it at 18 centimeters per second."*

Research has documented the phenomenon of orgasm occurring without direct genital stimulation, and several well-known studies provide empirical support for this. One of the earliest laboratory investigations, conducted by Whipple et al. (1992), examined women who reported the ability to reach orgasm through sexual imagery alone. The study found that these imagery-induced orgasms produced physiological responses, including increases in heart rate, blood pressure, pupil dilation, and pain thresholds (reduced sensitivity to pain), comparable to those observed during orgasm induced by genital stimulation. This provided early evidence that orgasm can be generated centrally, through cognitive and neural processes, rather than requiring direct physical stimulation.

More recent work has extended these findings using hormonal markers. Case studies by Pfaus and colleagues have shown that individuals can induce orgasms without genital contact through practices such as yoga, pelvic floor control, or hypnosis. These studies measured prolactin (a hormone that reliably increases following orgasm) and found substantial post-orgasmic elevations, in some cases exceeding 100% of baseline levels. These hormonal changes, alongside subjective reports

and observed muscle contraction patterns, closely matched those seen in conventionally stimulated orgasms, while control conditions (such as reading or non-sexual exercise) did not produce similar effects.

Further experimental work has demonstrated that hypnotically induced orgasms, in fully clothed participants without genital touch, can elicit both the physiological and subjective features of orgasm, including significant prolactin increases and characteristic pelvic floor activity. Complementing this, broader reviews, such as those by Komisaruk and Whipple, have documented a range of non-genital orgasmic experiences, including those arising from imagery, nipple stimulation, exercise, sleep, neurological conditions, and direct brain or spinal stimulation. Taken together, this body of research supports the view that orgasm is a centrally mediated neurophysiological process that can, under certain conditions, be initiated without direct genital stimulation.

**Study: Erogenous Touch, Expectation, and the Social Brain**

Panagiotopoulou et al. (2018) demonstrated that stimulation of erogenous zones (areas of the body associated with heightened sexual sensitivity, such as the neck) was rated as more pleasant and sexually arousing than touch to non-erogenous regions, while slow, C-tactile (CT) optimal stroking (gentle touch at approximately 3 cm/s, associated with specialised nerve fibres linked to affective touch) independently enhanced both pleasantness and arousal compared to faster, CT-suboptimal contact. Notably, these factors did not interact statistically, suggesting that anatomical sensitivity and tactile dynamics contributed distinct pathways to erotic experience.

The findings further show that imagined touch alone can increase perceived pleasantness, indicating a significant role for top-down processes (cognitive influences such as expectation, memory, and imagination) in shaping sensory experience. Additionally, touch perceived as arousing by the receiver was also judged as more arousing for the giver, pointing to a shared, intersubjective mapping of erogeneity within social contexts.

***References 210-214.***

## Contributing Factors to Declining Testosterone Levels

*"So testosterone is declining (...)*

*when you have more fat mass (...) more testosterone is being converted to estrogen (...) a rise in diabetes and insulin resistance, which also causes a decrease in testosterone. We're seeing a rise in ultra processed food intake and that doesn't have the optimal nutrition that you need to optimize testosterone (...)*

*when your testosterone is low, but below two 14 nanograms per deciliter, that your risk of mortality goes up by two (...) But when you go super high, super physiologic, meaning like 1800 or higher (...) Now you're putting yourself at risk for other things (...) you can have blood thickening, which is a known side effect of testosterone replacement (...)*

*the data would suggest, you know, depending on the data, you look at 20 to 40% of guys have low testosterone. And when you look at the number that get treated, it's like 2%"*

Testosterone levels have declined at a population level over recent decades, with multiple large-scale studies demonstrating a consistent downward trend that cannot be fully explained by ageing or obesity. A major meta-analysis encompassing over one million men reports a gradual decline of approximately 0.6 nmol/L per decade after adjusting for key factors such as age, body mass index (BMI, a measure of body fat based on height and weight), and assay differences. Longitudinal cohort studies, including well-known ageing studies and large national health datasets, similarly show that more recent generations of men tend to have lower testosterone levels than earlier cohorts at the same age, suggesting a secular (time-related) decline.

The magnitude of this reduction varies across studies and populations. For example, large healthcare data from Israel indicate roughly a 10% decrease in testosterone among young men over a little more than a decade, while other cohorts report steady annual declines in younger adult populations. These changes appear to occur independently of traditional explanatory factors such as obesity or smoking in some analyses, although metabolic health still plays a significant role at the individual level. While earlier research suggested a steady age-related decline in testosterone within individuals, more recent evidence indicates that much of this decline may instead be attributable to comorbid conditions (such as diabetes or metabolic syndrome) rather than ageing alone.

**Adipose Tissue, Aromatase Activity, and Hormonal Conversion**

Excess fat mass promotes the conversion of testosterone to oestrogen through increased activity of aromatase (an enzyme, CYP19A1, that converts androgens such as testosterone into oestrogens like estradiol). Adipose tissue is a major site of this process outside the gonads, and its contribution becomes more significant as fat mass increases. Studies show that aromatase expression and activity rise with greater adiposity, leading to enhanced conversion of testosterone into oestrogen, particularly in men. This process is further amplified by obesity-related inflammation, which upregulates aromatase at the genetic level, forming what has been described as an "obesity–inflammation–aromatase axis."

This mechanism contributes to a shift in hormonal balance, with reduced circulating testosterone and relatively increased oestrogenic activity. Some evidence suggests that these effects may be especially pronounced at the tissue level, meaning that local oestrogen concentrations within fat may rise even when changes in blood levels appear modest. In men with obesity, this increased conversion often occurs alongside reduced testosterone production by the testes, indicating that both impaired synthesis and enhanced aromatisation contribute to lower testosterone levels.

**Insulin Resistance, Ultra-Processed Diets, and Testosterone**

Insulin resistance (a condition in which cells respond poorly to insulin, leading to impaired glucose regulation) is consistently associated with lower testosterone levels, with evidence suggesting a bidirectional relationship. Clinical and epidemiological studies show that men with insulin-resistant states, such as type 2 diabetes and metabolic syndrome, frequently exhibit reduced total and free testosterone, with some estimates indicating that up to half of men with type 2 diabetes meet criteria for hypogonadism (clinically low testosterone). These associations often persist even after adjusting for body mass index and waist circumference, indicating an independent link. Prospective and interventional data further suggest a feedback loop, whereby low testosterone contributes to increased fat accumulation and worsening insulin sensitivity, while insulin resistance, particularly in the context of visceral adiposity (fat stored around internal organs), accelerates testosterone decline.

Tthe relationship between ultra-processed foods (UPFs, industrially formulated foods high in additives, refined carbohydrates, and unhealthy fats) and testosterone is less direct and more heterogeneous. Observational studies have linked dietary patterns rich in processed and fried foods to lower testosterone levels and poorer reproductive parameters, although causality is not firmly established. Some large-scale analyses suggest that UPF consumption is associated with metabolic

changes, including increased inflammation and reduced sex hormone-binding globulin (SHBG, a protein that regulates testosterone availability), but findings on total testosterone are inconsistent.

**Testosterone Levels, Mortality Risk, and Cardiovascular Effects**

Low testosterone has been consistently linked to increased all-cause and cardiovascular mortality. Large cohort studies and meta-analyses show that men with markedly low testosterone, particularly below approximately 250–350 ng/dL, have a higher risk of death from multiple causes, including cardiovascular disease. More recent analyses indicate that this association is strongest at very low levels, suggesting that low testosterone may function both as a marker of underlying ill health and as a contributing factor to adverse outcomes.

Very high testosterone levels, particularly those achieved through supraphysiological dosing (levels well above the normal physiological range, such as ~1500–1800 ng/dL), are associated with an increased risk of polycythaemia (an abnormal increase in red blood cell concentration). This condition leads to elevated haematocrit (the proportion of blood made up of red cells), resulting in thicker, more viscous blood. Increased blood viscosity is biologically linked to a higher risk of thrombosis (blood clots), which can contribute to major adverse cardiovascular events (such as heart attack or stroke). Clinical data indicate that individuals who develop polycythaemia during testosterone therapy have a significantly elevated risk of such events.

**Prevalence of Low Testosterone and Treatment Rates**

Research suggests that low testosterone (typically defined as total testosterone below ~300 ng/dL) is relatively common, although prevalence varies depending on age, health status, and diagnostic criteria. Large population-based studies, including analyses of U.S. national health data, estimate that approximately 20–30% of adult men meet biochemical criteria for low testosterone, with higher figures, approaching 30–40%, observed in older or clinical populations, particularly those over 45 or with comorbid conditions such as obesity. In contrast, prevalence is substantially lower in younger, otherwise healthy men, especially those in their 40s. When stricter criteria are applied, requiring both low testosterone and clinical symptoms (such as reduced libido, fatigue, or low mood), the proportion of affected men is considerably smaller. Despite this relatively high prevalence, only a minority of men with low testosterone receive treatment. Estimates suggest that approximately 10–30% of men meeting biochemical criteria are treated with testosterone therapy in general population.

***References 215-244.***

## Estrogen Deficiency and Osteoporosis Risk

> *"when your estrogen gets very low, you get a higher risk for fractures, higher risk for osteoporosis."*

Low oestrogen (a key sex hormone involved in bone maintenance) is strongly associated with an increased risk of osteoporosis (a condition characterised by reduced bone mineral density, or BMD, and increased fracture risk) in both women and men. In postmenopausal women, oestrogen deficiency is recognised as the primary driver of osteoporosis, with the decline in hormone levels leading to accelerated bone loss and higher fracture incidence. Time since menopause, which reflects the duration of low oestrogen exposure, is independently associated with increased osteoporosis risk. Similar patterns are observed in younger women with conditions such as primary ovarian insufficiency (POI), where prolonged oestrogen deficiency is linked to significantly reduced BMD.

Evidence also shows that premenopausal women with hypoestrogenic states (low oestrogen levels), such as those with amenorrhoea (absence of menstrual cycles), exhibit lower bone density and increased bone resorption (the breakdown of bone tissue) compared to oestrogen-replete peers. In men, circulating estradiol (a form of oestrogen) appears to be a stronger predictor of bone health than testosterone, with low estradiol levels associated with increased fracture risk and bone turnover. Clinically, conditions involving early or prolonged oestrogen deficiency, such as early menopause or untreated POI, significantly elevate osteoporosis risk. Maintaining oestrogen above very low thresholds is associated with protective effects on bone, and hormone therapy has been shown to improve BMD and reduce fracture risk in appropriately selected individuals.

***References 245-250.***

## Anabolic Steroid Use and Cardiovascular Risk

> *“There's a 15 times higher risk of having (...) premature heart failure and 122 times more risk of cardiac death when you're taking anabolic steroids.”*

Anabolic–androgenic steroid (AAS) use is associated with substantially increased cardiovascular risk. Large cohort studies provide more moderate, though still clinically significant, estimates. For example, AAS users have been shown to have an approximately 3.6-fold higher risk of heart failure and nearly 9-fold higher risk of cardiomyopathy (disease of the heart muscle) compared to non-users. Other studies report around a twofold increase in combined cardiovascular morbidity and mortality, alongside markedly elevated all-cause mortality, with one study estimating nearly a 19-fold increase, although this includes all causes of death rather than cardiac-specific outcomes.

With respect to sudden cardiac death (SCD), evidence from reviews suggests that AAS use may increase risk by approximately 6–20 times compared to non-using athletes, although these estimates are derived from observational and narrative data rather than precise population-level incidence rates. Case reports and autopsy studies consistently document serious cardiac pathology in AAS users, including myocardial hypertrophy (thickening of the heart muscle), fibrosis (scar tissue formation), atherosclerosis (arterial plaque buildup), and arrhythmias (abnormal heart rhythms), all of which can contribute to fatal outcomes.

***References 251-257.***

## Oral Contraceptives and Sex Hormone-Binding Globulin (SHBG) Elevation

> *“women, SHBG goes up when you take oral contraceptives and it stays up for life if you take oral contraceptives. So it can affect their free testosterone.”*

Combined oral contraceptives (COCs) increase levels of SHBG (a protein that binds sex hormones such as testosterone and regulates their availability), often substantially. The magnitude of this increase varies depending on the formulation, particularly the dose of ethinylestradiol (a

synthetic oestrogen) and the type of progestin used. Studies show that SHBG can rise by approximately 50–80% with second-generation formulations (e.g., levonorgestrel-based pills) and by as much as 200–320% with more oestrogenic or anti-androgenic progestins. This elevation appears to remain stable during continued use, with sustained increases documented over several months of treatment. In contrast, progestin-only contraceptives tend not to increase SHBG and may even reduce it.

Following discontinuation, the trajectory of SHBG levels is less clear. Some prospective evidence suggests that SHBG returns to baseline levels after stopping oral contraceptives. However, other studies, particularly in clinical populations, report that SHBG may remain elevated for extended periods, even several months after cessation, and in some cases may not fully normalise. Reviews of the literature indicate that while SHBG generally declines after discontinuation, complete reversal to pre-treatment levels is not consistently observed, and the long-term persistence of elevated SHBG may vary between individuals.

***References 258-264.***

## Testosterone Replacement Therapy: Benefits and Limitations

> *"[testosterone replacement therapy] a great drug. It's a great option to improve your quality of life. It's obviously preventing bone loss. It's improving your longevity (...) having some benefit in terms of cardiometabolic health (...)*
>
> *causes infertility, right? When you're on testosterone replacement, after about 18 months, 70% of people on testosterone replacement will be infertile."*

Testosterone replacement therapy (TRT) has been shown to produce modest improvements in health-related quality of life, including psychological well-being, libido, mood, and erectile function, with effects generally more pronounced in men with clear biochemical deficiency. In contrast, men with only mild or borderline testosterone reductions may derive little to no benefit, and in some cases, lifestyle interventions such as weight loss may produce comparable or greater improvements.

Regarding longevity and cardiovascular outcomes, observational studies suggest that long-term TRT in hypogonadal men, particularly those with type 2 diabetes or elevated cardiometabolic risk, is associated with reduced all-cause mortality. However, randomised controlled trials (RCTs) have not demonstrated a definitive survival benefit, though they consistently show no increase in cardiovascular events or mortality in the short to medium term.

TRT also appears to improve aspects of the cardiometabolic profile, including reductions in fat mass, blood pressure, lipid levels, glucose, and HbA1c (a marker of long-term blood sugar control), particularly with long-term therapy in hypogonadal men.

In terms of bone health, TRT increases bone mineral density (BMD, a measure of bone strength), particularly in the lumbar spine and cortical bone, and reduces markers of bone resorption (the breakdown of bone tissue). Despite these improvements, current evidence does not show a reduction in fracture risk, and clinical guidelines do not recommend TRT as a standalone treatment for osteoporosis in high-risk individuals.

**Testosterone Therapy and Male Fertility**

Exogenous testosterone (testosterone administered from outside the body, as in testosterone replacement therapy, TRT) commonly suppresses spermatogenesis (the production of sperm), but the claim that “70% of men are infertile after 18 months” is not supported as a general rule. Testosterone therapy suppresses the hypothalamic–pituitary–gonadal axis (the hormonal system regulating reproduction), leading to reduced levels of luteinising hormone (LH) and follicle-stimulating hormone (FSH), both of which are essential for sperm production. As a result, many men on TRT, particularly with long-acting formulations, develop severe oligospermia (very low sperm count) or azoospermia (absence of sperm), often within a few months of treatment.

However, this effect is typically reversible. Data from male contraceptive studies show that the majority of men recover normal or near-normal sperm counts after discontinuing testosterone. Recovery rates increase over time, with approximately two-thirds recovering within six months, around 90% by one year, and nearly all by two years. Clinical studies of men using therapeutic testosterone similarly report substantial recovery of sperm production within several months after cessation, although factors such as age and duration of use can influence recovery time.

***References 265-272.***

# Penile Traction Devices and Length Outcomes

> *They've actually done some research on [traction devise] one 30 minutes twice a day (...) it does show improvements in length about two centimeters.*
>
> *There's one study in Japan where they looked at nose length (...) correlated with the length of the penis."*

Penile traction devices can produce modest increases in penile length, although the magnitude of change is generally small and requires prolonged, consistent use. Clinical studies in men using traction for cosmetic concerns report average gains in the range of approximately 1–2 cm in flaccid or stretched length after several months of daily use, often involving multiple hours per day. Similar findings are observed in medical contexts, such as in men with Peyronie's disease (a condition involving penile curvature due to fibrous scar tissue) or following prostate surgery, where randomised trials show small but measurable improvements in length compared to controls.

These effects appear limited to length, with no consistent evidence of increased penile girth (circumference). The underlying mechanism is thought to involve gradual tissue remodelling in response to sustained mechanical stretching. However, the evidence base is moderate in quality, with many studies being small or non-randomised, and outcomes varying depending on adherence and duration of use. Side effects are typically mild, including discomfort or skin irritation, when devices are used as directed.

**Nose Size and Penile Length: Correlation and Limitations**

Research suggests that there is a positive association between nose size and penile length, although this relationship is correlational and based on limited study populations. Recent studies in Asian cohorts have reported moderate correlations between nose dimensions and both flaccid and erect penile length. For example, one study found that nose size was significantly associated with penile length even after adjusting for factors such as body mass index, while another reported similar findings using stretched penile measurements. These results indicate that individuals with larger noses tended, on average, to have longer penile measurements, although the strength of this relationship is moderate rather than strong.

Some researchers have proposed that this association may reflect shared developmental influences, particularly prenatal androgen exposure (hormones involved in sexual development

before birth), which could affect both facial and genital growth. However, this mechanism remains speculative and has not been conclusively demonstrated. More broadly, studies examining body proportions and penile size consistently show that most anthropometric measures, such as height, weight, or other physical features, have only weak or modest correlations with penile length. level.

***References 273-282.***

## Orgasm from Penetration Alone: Prevalence and Variation

> *"only about 85% of women orgasm through penetration alone, they need clitoral stimulation to achieve climax."*

Research indicates that only a minority of women report being able to orgasm through vaginal penetration alone, with most studies placing this proportion between approximately 15% and 35%. Large and nationally representative surveys commonly report figures at the lower end of this range, around 18–20%, while some studies using different methodologies or samples report higher estimates, up to around one-third of women. Across these datasets, the majority of women indicate that additional clitoral stimulation (stimulation of the clitoris, a highly sensitive organ central to female sexual pleasure) is typically required to achieve orgasm.

These variations in reported prevalence are influenced by methodological differences. Studies that distinguish clearly between orgasm from penetration alone and orgasm with combined stimulation tend to report lower rates for penetration-only orgasm. Additionally, differences in question wording, sample populations, and cultural factors contribute to variability in findings. Many women also report using specific techniques, such as adjusting angle or incorporating clitoral stimulation during intercourse, to enhance pleasure and likelihood of orgasm.

***References 283-287.***

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