Hormones and male fertility: testosterone and hormonal imbalance

The hormonal axis behind sperm production

Spermatogenesis is controlled by the hypothalamic-pituitary-gonadal axis. The hypothalamus releases gonadotropin-releasing hormone, or GnRH, in pulses. GnRH signals the pituitary gland to release luteinizing hormone, or LH, and follicle-stimulating hormone, or FSH. LH stimulates Leydig cells in the testes to produce testosterone. FSH acts mainly on Sertoli cells, which support developing sperm cells and help coordinate sperm maturation.

This pathway is regulated by feedback loops. When circulating testosterone and estradiol rise, the brain reduces GnRH, LH, and FSH output. When testosterone is low because the testes are underactive, the pituitary may increase LH and FSH in an attempt to stimulate the testes. These patterns help clinicians distinguish between primary testicular problems and central, or hypothalamic-pituitary, causes.

Importantly, the testes require a very high local concentration of testosterone within the seminiferous tubules for normal sperm production. This intratesticular testosterone level is much higher than the testosterone measured in routine blood testing. A man can have a normal or high serum testosterone level while sperm production is suppressed if LH and FSH signaling

to the testes has been turned down.

Testosterone is essential, but more is not always better

Testosterone contributes to male reproductive health in several ways. It supports sexual desire, erectile physiology, ejaculation, maintenance of secondary sexual characteristics, muscle and bone health, and the testicular environment needed for spermatogenesis. However, fertility is not improved simply by raising testosterone in the bloodstream.

In men producing testosterone naturally, LH-driven testicular testosterone production helps maintain intratesticular testosterone. FSH and Sertoli cell function provide additional signals needed for sperm development. When testosterone is supplied from outside the body, the brain often interprets this as sufficient androgen exposure and reduces GnRH, LH, and FSH secretion. That reduction can lower intratesticular testosterone dramatically and impair sperm output.

This is why testosterone can function as a male contraceptive. Exogenous testosterone may lead to oligospermia, meaning low sperm concentration, or azoospermia, meaning no sperm seen in the ejaculate. Recovery after stopping testosterone can occur, but it may take months and is not always predictable, especially after prolonged use or combined anabolic steroid exposure.

Patterns of hormonal imbalance that can affect fertility

Low testosterone with high LH and FSH: This pattern may suggest primary testicular dysfunction, where the pituitary is trying to stimulate the testes but the testes respond inadequately.
Low testosterone with low or inappropriately normal LH and FSH: This may point toward secondary hypogonadism, involving the hypothalamus or pituitary gland, or functional suppression from obesity, illness, medications, sleep disorders, or stress physiology.
High testosterone with low LH and FSH: This pattern is concerning for exogenous androgen exposure, including prescribed testosterone, anabolic steroids, or some supplements contaminated with androgenic compounds.
Abnormal prolactin: Elevated prolactin can suppress GnRH and reduce LH and FSH signaling, potentially affecting testosterone and sexual function.

Thyroid or metabolic disorders: Thyroid dysfunction, poorly controlled diabetes, severe obesity, and systemic illness can influence sex hormone-binding globulin, testosterone availability, libido, erectile function, and semen quality.

External testosterone and anabolic steroids: a major fertility caution

One of the most important fertility messages is simple: men who desire pregnancy should not start testosterone therapy without discussing fertility goals with a qualified clinician. This includes testosterone injections, gels, patches, pellets, oral androgens, and non-prescribed anabolic-androgenic steroids. It also includes "performance," "muscle-building," or "testosterone boosting" products that may contain undisclosed androgenic substances.

The mechanism is well established. Exogenous testosterone suppresses pituitary LH and FSH. With less LH, Leydig cells reduce local testosterone production inside the testes. With less FSH, Sertoli cell support for spermatogenesis declines. The result can be a substantial reduction in sperm concentration and sometimes azoospermia.

This can be emotionally difficult because testosterone therapy may improve some symptoms such as low libido, fatigue, or mood in appropriately selected men, yet simultaneously undermine sperm production. Fertility-preserving alternatives may exist for certain patients, such as medications that stimulate the body’s own gonadotropin production or support testicular function, but these require individualized medical supervision. The right approach depends on the cause of low testosterone, semen findings, timing of pregnancy goals, and medical risks.

When to consider hormone testing in a fertility evaluation

A semen analysis is usually the first-line test for male fertility because it directly evaluates sperm concentration, motility, morphology, volume, and other parameters. Hormone testing becomes especially relevant when semen analysis is abnormal, when sperm concentration is very low, when there is sexual dysfunction, or when clinical signs suggest endocrine disruption.

Clinicians may consider hormonal evaluation when there is low libido, erectile

or ejaculatory difficulty, reduced shaving frequency, loss of muscle mass, gynecomastia, infertility with small or soft testes, delayed puberty history, prior testicular injury, chemotherapy or radiation exposure, pituitary symptoms, headaches or visual field problems, obesity with hypogonadal symptoms, or known use of testosterone or anabolic steroids.

Common laboratory tests may include total testosterone, free testosterone or calculated free testosterone, LH, FSH, prolactin, estradiol, thyroid-stimulating hormone, and sometimes inhibin B or other tests depending on the case. Genetic testing, scrotal ultrasound, pituitary imaging, or referral to reproductive urology may be appropriate in selected situations, particularly with azoospermia or severe oligospermia.

Low testosterone symptoms and the fertility trade-off

Low testosterone symptoms can overlap with many common health conditions. Fatigue, low mood, poor sleep, reduced libido, erectile changes, reduced exercise tolerance, and weight gain are real concerns, but they are not specific to testosterone deficiency. Sleep apnea, depression, thyroid disease, diabetes, medication effects, overtraining, undernutrition, chronic illness, alcohol use, and relationship stress may all produce similar symptoms.

For a man actively trying to conceive, the evaluation should explicitly include fertility goals. A clinician may ask: Are you trying now, in the next year, or in the future? Have you had a semen analysis? Have you previously used testosterone or anabolic steroids? Have you ever fathered a pregnancy? These questions change the risk-benefit discussion.

The key distinction is between replacing testosterone from outside the body and stimulating endogenous production. In some men with secondary hypogonadism, specialist-directed treatments may help increase internal LH and FSH activity or mimic gonadotropin signaling, potentially preserving or restoring sperm production. These decisions should be managed by clinicians experienced in male infertility because hormone manipulation can have side effects and requires monitoring.

Lifestyle and medical factors that interact with hormones

Hormones do not operate in isolation. General health can influence reproductive signaling and semen quality. Severe obesity is associated with lower testosterone, altered estradiol balance, inflammation, insulin resistance, and sleep apnea. Excess alcohol, some opioids, glucocorticoids, certain psychiatric medications, and chronic systemic disease can affect the hypothalamic-pituitary-gonadal axis. Heat exposure to the testes, varicocele, infections, and environmental toxins may also contribute to impaired spermatogenesis through hormonal and non-hormonal pathways.

Supportive steps often include addressing sleep quality, metabolic health, smoking cessation, avoidance of anabolic steroids, moderation of alcohol, review of medications with a clinician, and management of chronic diseases. These measures are not a substitute for evaluation, especially if semen parameters are severely abnormal, but they can support the broader reproductive environment.

Couples often feel pressure to "optimize" everything at once. A more sustainable approach is to identify modifiable factors, obtain objective testing, and work with professionals who can prioritize what is most likely to matter. Because sperm development takes roughly three months, improvements in semen parameters, when they occur, may lag behind changes in hormones or lifestyle.

Working with a fertility-focused clinician

If pregnancy has not occurred after 12 months of regular unprotected intercourse, or after 6 months when the female partner is 35 or older, both partners generally deserve evaluation. Earlier assessment is reasonable if there is a known male factor risk, prior abnormal semen analysis, irregular cycles in the female partner, prior pelvic or testicular surgery, chemotherapy history, or suspected endocrine disorder.

A reproductive urologist, and sometimes an endocrinologist, can interpret hormone patterns alongside semen results. This is particularly valuable when testosterone is low, LH or FSH are abnormal, or there is a history of testosterone use. The goal is not to assign blame; male factor infertility is common, medically meaningful, and often treatable or manageable. A compassionate evaluation can reduce uncertainty and help couples choose next

steps with clearer information.

For some men, management may involve stopping fertility-suppressing substances under supervision, treating reversible endocrine disorders, addressing varicocele or obstruction, using fertility-preserving hormonal strategies, sperm retrieval procedures, or assisted reproductive technologies. The appropriate path is highly individualized and should be guided by qualified healthcare professionals.