**-Pages 52 & 53 “endocrine”: Table 75.1:**

FSH & LH: Remember that these hormones are secreted from the anterior pituitary gland which is stimulated by the hormones.

\*GRH (Gonadotropin Releasing Hormones) stimulate the release of FSH & LH.

Recall that there are some types of cells that secrete LH only, some other types that secrete FSH only, & some other types that secrete both. All 3 cell types have GRH receptors.

**-Page containing Fig. 53.3:**

There are 2 important cells in the primary sex organs of the male:

1) Leydig cells

2) Sertoli cells

There’s an intimate relationship between those two cell types, meaning, neither can function properly without the other. They only work when they are functioning together.

-Leydig cells are affected by LH. They produce the male hormone “testosterone”. Testosterone then passes into sertoli cells.

-Sertoli cells are affected by FSH & they have 4 functions:

1) Produce the androgen binding protein.

2) Produce acromatase enzymes (these enzymes are not present in all cells & their function is to produce estrogen/estradiols from testosterone in the testes).

3) Produce growth factors, & other products. (Those support sperm cells & are involved in spermatogenesis).

4) Produce inhibin.

Note that estrogen produced passes into the leydig cells, where they play a role in spermatogenesis.

-95% of testosterone is made by leydig cells, while the remaining 5% is made by the adrenal glands. Although the major secretion of testes is testosterone; they also secrete progesterone, androgen & dihydrogesterone.

-Androstenedione is a steroid hormone produced in the adrenal glands & the gonads as an intermediate step in the biochemical pathway that produces the androgen “testosterone” and the estrogens “estrone” & “estradiol”. It is an important hormone because it serves as a precursor for the extraglandular formation.

-Leydig cells also contain receptors for prolactin. Hyperprolactinemia in men with pituitary tumors, usually microadenomas, is associated with decreased testosterone levels. This condition is a result of a direct effect of elevated circulating levels of prolactin on leydig cells, reducing the number of LH receptors. In addition, hyperprolactinemia may decrease LH secretion by affecting the nature of its release.

-As mentioned in the paragraph, hyperprolactinemia in men is associated with a decrease in the testosterone level. Why is that?

When prolactin levels increase, LH receptors will become occupied by prolactin (recall that prolactin can bind to LH receptors). Therefore, no more receptors would be available for LH binding. Since LH cannot bind anymore, it will not be able to stimulate the production of testosterone, & thus, decreasing testosterone level.

-Decreasing testosterone levels will cause a decrease in sperm production, & thus decreasing the fertility.

\*Note: Prolactin, as mentioned before, can bind to LH receptors. However, LH can NOT bind to prolactin receptors.

**-Page containing Fig. 37.1:** Regulation of the reproductive system in males:

Notice that the brain centre affects the hypothalamus. The hypothalamus subsequently affects the anterior pituitary gland. The anterior pituitary gland then affects the testes.

Factors that may affect the brain centre are: age, hormonal state, stress levels, environment, various diseases, & drugs.

-Gonadotropin hormones (LH & FS) are affected by: testosterone, inhibin, activin, estradiol & follistatin.

Generally testosterone, estradiol, & inhibin reduce the secretion of LH & FSH in the male. Activin, however, stimulates the secretion of FSH. FSH secretion is inhibited by follistatin.

Note: Inhibin can act DIRECTLY to inhibit FSH but NOT LH. In order for it to inhibit LH secretion it has to be bound to follistatin.

**-Page containing Fig. 36.2:**

GRH (Gonadotropin Releasing Hormone) & GAP (Gonadotropin Associated Protein) work together & can inhibit prolactin secretion.

**-Page containing Fig. 52.14:**

Remember from previous lectures when we have talked about hormones which have alpha & beta subunits. Recall that the alpha subunit is the unspecific subunit (present in many hormones) whereas the beta subunit is specified for a certain hormone.

**-Page containing Fig. 10.1**: Determination of genetic sex:

Whether a zygote is a male or a female only depends upon the sex chromosomes (whether they are XX, or XY) present in the cell. These chromosomes determine whether ovaries or testes develop. There is no other difference between a male & a female.

The differentiation of primitive gonads into testes or ovaries is therefore genetically determined, but the formation of male genatalia depends upon the presence of functional testis. Thus, if, for some reason, the testes were removed surgically during development, the male genital organs would not be able to function properly.

The gonads in both sexes (male & female) have two functions:

\*production of sex hormones.

\*production of germ cells (eggs or sperms).

The proper functioning of testes & ovaries depends upon the normal functioning of the anterior pituitary gland & the hypothalamus.

**-Page titled “Male Reproductive System”:**

The primary sex organs in a male are the two testes. Note that one testis is sufficient. Recall that testes produce androgens & sperms.

**-Page titled “Male Secondary Sex Organs”:**

Androgen (testosterone) is responsible for the development of the secondary sex organs such as: epididymis , vas deferens, seminal vesicle & the prostate gland. It is also responsible for the appearance of secondary sex characteristics such as: axillary & fascial hair, thickening of the voice, & broad shoulders.

Any hypertrophy (enlargement) in the prostate gland would affect the urethra, thus affecting urination either by forming a malignant tumor or a benign tumor.

There are two ways to estimate the degree of hypertrophy in a person:

\*The first way is to measure the concentration of PSA (Prosthetic Specific Antigen) in a sample of serum. If the concentration is high, the person probably suffers from hypertrophy his prostate gland. The higher the concentration, the higher the degree of enlargement, This method is 95% accurate.

\*The second way is to examine the prostatic enlargement physically, & this is a much more accurate way.

**-Page containing Fig. 36.8**: The Process of Spermatogenesis:

Spermatogenesis occurs in three phases:

\*Meiosis (28 days): the production of daughter cells having half the number of chromosomes.

\*Mitosis (25 days): the production of daughter cells having the same number of chromosomes.

\*Spermatogenesis (21 days): Production of mature sperms.

Throughout the three phases, changes in division, metabolism, function, morphology, motility & fertility of the cells occur. These are fine processes that are very important where any error could affect the production of healthy sperms.

Each of these 3 phases can be affected by factors such as; chemical therapy, certain drugs, extreme temperature, exposure to radiation… etc.

The normal duration of the whole process of spermatogenesis can be estimated to be 65 to 75 days.

Hormones can affect the number of sperms produced, but can NOT affect the durations of these phases (cannot prolong or shorten the time of each phase).

Spermatogenesis starts occurring at puberty, & a new cycle begins every 16 days.

Vasectomy (the ligation of vas deferens), an infection, or physical injury in testis may cause the production of antibodies against sperms. If these antibodies’ concentrations were mild or moderate, no problems would be faced. However, if their concentrations were high, they would probably cause infertility.

Sometimes these antibodies are produced, for a reason or another, in both spouses (partners; husband & wife). This would most probably cause infertility unless treated.

