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**DEPARTMENT: ANATOMY**

**COURSE CODE: PHS 204**

**Discuss lactation and gestation period in a normal female**

**I expect more on the physiology of lactation and details on the physiology of pregnancy in a normal woman**

* **Alveoli** secrete milk and contract when stimulated
* **Oxytocin** stimulates milk secretion and is released during the ‘let down’ or milk ejection reflex
* After let down, milk travels into the **ductules**, then to the larger – **lactiferous or mammary** ducts
* **Prolactin** levels rise with nipple stimulation
* Alveolar cells make milk in response to prolactin when the baby sucks
* **Oxytocin** causes the alveoli to squeeze the newly produced milk into the duct system

Latch On and sucking



Oxytocin Release



Releases Milk



Infant Empties Breast



Production Increases



Milk Production Occurs

The mechanism by which this inhibitory effect is brought about, or by which lactation is initiated at delivery, has long been the subject of an argument that revolves around the opposing actions of estrogen, progesterone, and prolactin, as studied in laboratory animals, goats, and cattle. During pregnancy the combination of estrogen and progesterone circulating in the blood appears to inhibit milk secretion by blocking the release of prolactin from the pituitary gland and by making the mammary gland cells unresponsive to this pituitary hormone.

Prolactin stimulation is promoted by suckling and is a neuro-endocrine reflex. Suckling mechanically stimulates receptors in the nipple and impulses pass up to the brain stem and to the hypothalamus to reduce the secretion of dopamine and increase vasoactive intestinal protein (promotes prolactin secretion). Suckling at one feed promotes prolactin release which causes production for the next feed which accumulates in alveoli and ducts (turgor).

**Physiology of pregnancy in a normal woman**

**Fetal-placental unit**

Levels of progesterone and estrogen rise continually throughout pregnancy, suppressing the hypothalamic axis and subsequently the menstrual cycle. The progesterone is first produced by the corpus luteum and then by the placenta in the second trimester. Women also experience increased human chorionic gonadotropin (β-hCG), which is produced by the placenta.

**Pancreatic Insulin**

The placenta also produces human placental lactogen (hPL), which stimulates maternal lipolysis and fatty acid metabolism. As a result, this conserves blood glucose for use by the fetus. It can also decrease maternal tissue sensitivity to insulin, resulting in gestational diabetes.

**Pituitary gland**

The pituitary gland grows by about one-third as a result of hyperplasia of the lactrotrophs in response to the high plasma estrogen. Prolactin, which is produced by the lactrotrophs increases progressively throughout pregnancy. Prolactin mediates a change in the structure of the breast mammary glands from ductal to lobular-alveolar and stimulates milk production.

**Parathyroid**

Fetal skeletal formation and then later lactation challenges the maternal body to maintain their calcium levels. The fetal skeleton requires approximately 30 grams of calcium by the end of pregnancy. The mother's body adapts by increasing parathyroid hormone, leading to an increase in calcium uptake within the gut as well as increased calcium reabsorption by the kidneys. Maternal total serum calcium decreases due to maternal hypoalbuminemia, but the ionized calcium levels are maintained.

**Adrenal glands**

Total cortisol increases to three times of non-pregnant levels by the third trimester.[2] The increased estrogen in pregnancy leads to increase corticosteroid-binding globulin production and in response the adrenal gland produces more cortisol. The net effect is an increase of free cortisol. This contributes to insulin resistance of pregnancy and possibly striae.Despite the increase in cortisol, the pregnant mom does not exhibit Cushing syndrome or symptoms of high cortisol. One theory is that high progesterone levels act as an antagonist to the cortisol.

The adrenal gland also produces more aldosterone, leading to an eight-fold increase in aldosterone.Women do not show signs of hyperaldosterone, such as hypokalemia, hypernatremia, or high blood pressure.

The adrenal gland also produces more androgens, such as testosterone, but this is buffered by estrogen's increase in sex-hormone binding globulin (SHBG). SHBG binds avidly to testosterone and to a lesser degree DHEA.

**Thyroid**

The thyroid enlarges and may be more easily felt during the first trimester. The increase in kidney clearance during pregnancy causes more iodide to be excreted and causes relative iodine deficiency and as a result an increase in thyroid size. Estrogen-stimulated increase in thyroid-binding globulin (TBG) leads to an increase in total thyroxine (T4), but free thyroxine (T4) and triiodothyronine (T3) remain normal.