

Anatomy of the female reproductive system assignment



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Anatomy of the Female Reproductive System The reproductive role of the female is far more complex than that of a male. Not only must she produce gametes, but her body must prepare to nurture a developing embryo for a period of approximately nine months. Ovaries, the female gonads, are the primary reproductive organs of a female, and like the male testes, ovaries serve a dual purpose: They produce the female gametes (ova) and sex hormones, the estrogens * and progesterone (pro-ges? te-ron). The accessory ducts (uterine tubes, uterus, and vagina) transport or otherwise serve the needs of the reproductive cells and a developing fetus.

As illustrated in Figure 27. 11, the ovaries and duct system, collectively known as the internal genitalia, are mostly located in the pelvic cavity. The female's accessory ducts, from the vicinity of the ovary to the body exterior, are the uterine tubes, the uterus, and the vagina. The external sex organs of females are referred to as the external genitalia. **The Ovaries** The paired ovaries flank the uterus on each side. Shaped like an almond and about twice as large, each ovary is held in place in the fork of the iliac blood vessels within the peritoneal cavity by several ligaments.

The ovarian ligament anchors the ovary medially to the uterus; the suspensory ligament anchors it laterally to the pelvic wall; and the mesovarium (mez? o-va? re-um) suspends it in between. The suspensory ligament and the mesovarium are part of the broad ligament, a peritoneal fold that “ tents” over the uterus and supports the uterine tubes, uterus, and vagina. The ovarian ligaments are enclosed by the broad ligament. The ovaries are served by the ovarian arteries, branches of the abdominal aorta, and by the ovarian branch of the uterine arteries.

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The ovarian blood vessels reach the ovaries by traveling through the suspensory ligaments and mesovaria. Like each testis, each ovary is surrounded externally by a fibrous tunica albuginea which is in turn covered externally by a layer of cuboidal epithelial cells called the germinal epithelium, actually a continuation of the peritoneum. The ovary has an outer cortex, which houses the forming gametes, and an inner medullary region, containing the largest blood vessels and nerves, but the relative extent of each region is poorly defined.

Embedded in the highly vascular connective tissue of the ovary cortex are many tiny saclike structures called ovarian follicles. Each follicle consists of an immature egg, called an oocyte (o? o-sit; oo= egg), encased by one or more layers of very different cells. The surrounding cells are called follicle cells if a single layer is present, and granulosa cells when more than one layer is present. Follicles at different stages of maturation are distinguished by their structure. In a primordial follicle, one layer of squamouslike follicle cells surrounds the oocyte.

A primary follicle has two or more layers of cuboidal or columnar-type granulosa cells enclosing the oocyte; it becomes a secondary follicle when fluid-filled spaces appear between the granulosa cells and then coalesce to form a central fluid-filled cavity called an antrum. At the mature vesicular follicle, or Graafian follicle (graf? e-an), stage, the follicle bulges from the surface of the ovary. The oocyte of the vesicular follicle “sits” on a stalk of granulosa cells at one side of the antrum.

Each month in adult women, one of the ripening follicles ejects its oocyte from the ovary, an event called ovulation. After ovulation, the ruptured follicle is transformed into a very different looking glandular structure called the corpus luteum (lu? te-um; plural: corpora lutea), which eventually degenerates. As a rule, most of these structures can be seen within the same ovary. In older women, the surfaces of the ovaries are scarred and pitted, revealing that many oocytes have been released.

The Female Duct System

The Uterine Tubes

The uterine tubes (u? er-in), also called fallopian tubes or oviducts, form the initial part of the female duct system. They receive the ovulated oocyte and are the site where fertilization generally occurs. Each uterine tube is about 10 cm (4 inches) long and extends medially from the region of an ovary to empty into the superolateral region of the uterus via a constricted region called the isthmus (is? mus). The distal end of each uterine tube expands as it curves around the ovary, forming the ampulla; fertilization usually occurs in this region. The ampulla ends in the infundibulum (in? un-dib? u-lum), an open, funnel-shaped structure bearing ciliated, fingerlike projections called fimbriae (fim? bre-e; “ fringe”) that drape over the ovary. Unlike the male duct system, which is continuous with the tubules of the testes, the uterine tubes have little or no actual contact with the ovaries. An ovulated oocyte is cast into the peritoneal cavity, and many oocytes are lost there. However, the uterine tube performs complex movements to capture oocytes??? it bends to drape over the ovary while the fimbriae stiffen and sweep the ovarian surface.

The beating cilia on the fimbriae then create currents in the peritoneal fluid that tend to carry an oocyte into the uterine tube, where it begins its journey

toward the uterus. The uterine tube contains sheets of smooth muscle, and its thick, highly folded mucosa contains both ciliated and nonciliated cells. The oocyte is carried toward the uterus by a combination of muscular peristalsis and the beating of the cilia. Nonciliated cells of the mucosa have dense microvilli and produce a secretion that keeps the oocyte (and sperm, if present) moist and nourished.

Externally, the uterine tubes are covered by visceral peritoneum and supported along their length by a short mesentery (part of the broad ligament) called the mesosalpinx (mez? o-sal? pinks; "mesentery of the trumpet"; salpin = trumpet), a reference to the trumpet-shaped uterine tube it supports. The Uterus The uterus (Latin for "womb") is located in the pelvis, anterior to the rectum and posterosuperior to the bladder. It is a hollow, thick-walled, muscular organ that functions to receive, retain, and nourish a fertilized ovum.

In a premenopausal woman who has never been pregnant, the uterus is about the size and shape of an inverted pear, but it is usually somewhat larger in women who have borne children. Normally, the uterus flexes anteriorly where it joins the vagina, causing the uterus as a whole to be inclined forward, or anteverted. However, the organ is frequently turned backward, or retroverted, in older women. The major portion of the uterus is referred to as the body. The rounded region superior to the entrance of the uterine tubes is the fundus, and the slightly narrowed region between the body and the cervix is the isthmus.

The cervix of the uterus is its narrow neck, or outlet, which projects into the vagina inferiorly. The cavity of the cervix, called the cervical canal, communicates with the vagina via the external os (os = mouth) and with the cavity of the uterine body via the internal os. The mucosa of the cervical canal contains cervical glands that secrete a mucus that fills the cervical canal and covers the external os, presumably to block the spread of bacteria from the vagina into the uterus. Cervical mucus also blocks the entry of sperm, except at midcycle, when it becomes less viscous and allows sperm to pass through.

Supports of the Uterus The uterus is supported laterally by the mesometrium (“mesentery of the uterus”) portion of the broad ligament (Figure 27. 14a). More inferiorly, the lateral cervical (cardinal) ligaments extend from the cervix and superior vagina to the lateral walls of the pelvis, and the paired uterosacral ligaments secure the uterus to the sacrum posteriorly. The uterus is bound to the anterior body wall by the fibrous round ligaments, which run through the inguinal canals to anchor in the subcutaneous tissue of the labia majora.

These ligaments allow the uterus a good deal of mobility, and its position changes as the rectum and bladder fill and empty. **The Uterine Wall** The wall of the uterus is composed of three layers. The perimetrium, the incomplete outermost serous layer, is the peritoneum. The myometrium (mi? o-me? tre-um; “muscle of the uterus”) is the bulky middle layer, composed of interlacing bundles of smooth muscle, that contracts rhythmically during childbirth to expel the baby from the mother’s body. The endometrium is the

mucosal lining of the uterine cavity; it is a simple columnar epithelium underlain by a thick lamina propria.

If fertilization occurs, the young embryo burrows into the endometrium (implants) and resides there for the rest of its development. The endometrium has two chief strata (layers). The stratum functionalis (funk-shun-a? lis), or functional layer, undergoes cyclic changes in response to blood levels of ovarian hormones and is shed during menstruation (approximately every 28 days). The thinner, deeper stratum basalis (ba-sa? lis), or basal layer, forms a new functionalis after menstruation ends. It is unresponsive to ovarian hormones. The endometrium has numerous uterine glands that change in length as endometrial thickness changes.

To understand the cyclic changes of the uterine endometrium (discussed later in the chapter), it is essential to understand the vascular supply of the uterus. The uterine arteries arise from the internal iliacs in the pelvis, ascend along the sides of the uterus, and send branches into the uterine wall. These branches break up into several arcuate arteries (ar? ku-at) within the myometrium. The arcuate arteries send radial branches into the endometrium, where they in turn give off straight arteries to the stratum basalis and spiral (coiled) arteries to the stratum functionalis.

The spiral arteries repeatedly degenerate and regenerate, and it is their spasms that actually cause the functionalis layer to be shed during menstruation. Veins in the endometrium are thin-walled and form an extensive network with occasional sinusoidal enlargements. The Vagina The vagina (“sheath”) is a thin-walled tube, 8??? 10 cm (3??? 4 inches) long. It

lies between the bladder and the rectum and extends from the cervix to the body exterior. The urethra is embedded in its anterior wall. Often called the birth canal, the vagina provides a passageway for delivery of an infant and for menstrual flow.

Since it receives the penis (and semen) during sexual intercourse, it is the female organ of copulation. The distensible wall of the vagina consists of three coats: an outer fibroelastic adventitia, a smooth muscle muscularis, and a mucosa marked by transverse ridges or rugae, which stimulate the penis during intercourse. The mucosa is a stratified squamous epithelium adapted to stand up to friction. Certain mucosal cells (dendritic cells) act as antigen-presenting cells and are thought to provide the route of HIV transmission from an infected male to the female during sexual intercourse.

The vaginal mucosa has no glands; it is lubricated by the cervical mucous glands. Its epithelial cells release large amounts of glycogen, which is anaerobically metabolized to lactic acid by resident bacteria. Consequently, the pH of a woman's vagina is normally quite acidic. This acidity helps keep the vagina healthy and free of infection, but it is also hostile to sperm.

Although vaginal fluid of adult females is acidic, it tends to be alkaline in adolescents, predisposing sexually active teenagers to sexually transmitted diseases.

In virgins (those who have never participated in sexual intercourse), the mucosa near the distal vaginal orifice forms an incomplete partition called the hymen (hi? men). The hymen is very vascular and tends to bleed when it is ruptured during the first coitus (sexual intercourse). However, its durability

varies. In some females, it is ruptured during a sports activity, tampon insertion, or pelvic examination. Occasionally, it is so tough that it must be breached surgically if intercourse is to occur. The upper end of the vaginal canal loosely surrounds the cervix of the uterus, producing a vaginal recess called the vaginal fornix.

The posterior part of this recess, the posterior fornix, is much deeper than the lateral and anterior fornices. Generally, the lumen of the vagina is quite small and, except where it is held open by the cervix, its posterior and anterior walls are in contact with one another. The vagina stretches considerably during copulation and childbirth, but its lateral distension is limited by the ischial spines and the sacrospinous ligaments. The External Genitalia As mentioned earlier, the female reproductive structures that lie external to the vagina are called the external genitalia (see Figure 27. 16).

The external genitalia, also called the vulva (vul? vah; “ covering”) or pudendum (“ shameful”), include the mons pubis, labia, clitoris, and structures associated with the vestibule. The mons pubis (mons pu? bis; “ mountain on the pubis”) is a fatty, rounded area overlying the pubic symphysis. After puberty, this area is covered with pubic hair. Running posteriorly from the mons pubis are two elongated, hair-covered fatty skin folds, the labia majora (la? be-ah mah-jor? ah; “ larger lips”). These are the female counterpart, or homologue, of the male scrotum (that is, they derive from the same embryonic tissue).

The labia majora enclose the labia minora (mi-nor? ah; “ smaller”), two thin, hair-free skin folds, homologous to the ventral penis. The labia minora

enclose a recess called the vestibule (“entrance hall”), which contains the external openings of the urethra and the vagina. Flanking the vaginal opening are the pea-size greater vestibular glands, homologous to the bulbourethral glands of males. These glands release mucus into the vestibule and help to keep it moist and lubricated, facilitating intercourse. At the extreme posterior end of the vestibule the labia minora come together to form a ridge called the fourchette.

Just anterior to the vestibule is the clitoris (klit? o-ris; “hill”), a small, protruding structure composed largely of erectile tissue, which is homologous to the penis of the male. Its exposed portion is called the glans of the clitoris. It is hooded by a skin fold called the prepuce of the clitoris, formed by the junction of the labia minora folds. The clitoris is richly innervated with sensory nerve endings sensitive to touch, and it becomes swollen with blood and erect during tactile stimulation, contributing to a female’s sexual arousal.

Like the penis, the body of the clitoris has dorsal erectile columns (corpora cavernosa) attached proximally by crura, but it lacks a corpus spongiosum that conveys a urethra. In males the urethra carries both urine and semen and runs through the penis, but the female urinary and reproductive tracts are completely separate. Instead, the bulbs of the vestibule, which lie along each side of the vaginal orifice and deep to the bulbospongiosus muscle, are the homologues of the single penile bulb and corpus spongiosum of the male, and during sexual stimulation the bulbs of the vestibule engorge with blood.

This may help grip the penis within the vagina and also squeezes the urethral orifice shut, which prevents semen (and bacteria) from traveling superiorly into the bladder during intercourse. The female perineum is a diamond-shaped region located between the pubic arch anteriorly, the coccyx posteriorly, and the ischial tuberosities laterally. The soft tissues of the perineum overlie the muscles of the pelvic outlet, and the posterior ends of the labia majora overlie the central tendon, into which most muscles supporting the pelvic floor insert

The Mammary Glands The mammary glands are present in both sexes, but they normally function only in females. The biological role of the mammary glands is to produce milk to nourish a newborn baby, so they are important only when reproduction has already been accomplished. Developmentally, mammary glands are modified sweat glands that are really part of the skin, or integumentary system. Each mammary gland is contained within a rounded skin-covered breast within the hypodermis (superficial fascia), anterior to the pectoral muscles of the thorax.

Slightly below the center of each breast is a ring of pigmented skin, the areola (ah-re? o-lah), which surrounds a central protruding nipple. Large sebaceous glands in the areola make it slightly bumpy and produce sebum that reduces chapping and cracking of the skin of the nipple. Autonomic nervous system controls of smooth muscle fibers in the areola and nipple cause the nipple to become erect when stimulated by tactile or sexual stimuli and when exposed to cold. Internally, each mammary gland consists of 15 to 25 lobes that radiate around and open at the nipple.

The lobes are padded and separated from each other by fibrous connective tissue and fat. The interlobar connective tissue forms suspensory ligaments that attach the breast to the underlying muscle fascia and to the overlying dermis. As suggested by their name, the suspensory ligaments provide natural support for the breasts, like a built-in brassiere. Within the lobes are smaller units called lobules, which contain glandular alveoli that produce milk when a woman is lactating. These compound alveolar glands pass the milk into the lactiferous ducts (lak-tif? er-us), which open to the outside at the nipple.

Just deep to the areola, each duct has a dilated region called a lactiferous sinus where milk accumulates during nursing. The description of mammary glands given here applies only to nursing women or women in the last trimester of pregnancy. In nonpregnant women, the glandular structure of the breast is largely undeveloped and the duct system is rudimentary; hence breast size is largely due to the amount of fat deposits. Physiology of the Female Reproductive System Oogenesis Gamete production in males begins at puberty and continues throughout life, but the situation is quite different in females.

It has been assumed that a female's total supply of eggs is already determined by the time she is born, and the time span during which she releases them extends only from puberty to menopause (about the age of 50). However, a recent study in adult mice has indicated that germ stem cells are alive and generating little "egglets" throughout life and there are hints that egg stem cells also exist in adult women. Although these findings seem to overturn the assumption that the number of oocytes (thus, potential <https://assignbuster.com/anatomy-of-the-female-reproductive-system-assignment/>

eggs) is limited??? an idea that has been part of the bedrock of biology??? it is still too early to retire the “ no new eggs” doctrine.

Meiosis, the specialized nuclear division that occurs in the testes to produce sperm, also occurs in the ovaries. In this case, female sex cells are produced, and the process is called oogenesis (o? o-gen? e-sis; “ the beginning of an egg”). The process of oogenesis takes years to complete. First, in the fetal period the oogonia, the diploid stem cells of the ovaries, multiply rapidly by mitosis and then enter a growth phase and lay in nutrient reserves.

Gradually, primordial follicles begin to appear as the oogonia are transformed into primary oocytes and become surrounded by a single layer of flattened follicle cells.

The primary oocytes begin the first meiotic division, but become “ stalled” late in prophase I and do not complete it. By birth, a female has been presumed to have her lifetime supply of primary oocytes; of the original 7 million oocytes approximately 2 million of them escape programmed death and are already in place in the cortical region of the immature ovary. Since they remain in their state of suspended animation all through childhood, the wait is a long one??? 10 to 14 years at the very least!

At puberty, perhaps 250, 000 oocytes remain and beginning at this time a small number of primary oocytes are recruited (activated) each month in response to the LH surge midcycle . However, only one is “ selected” each time to continue meiosis I, ultimately producing two haploid cells (each with 23 replicated chromosomes) that are quite dissimilar in size. The smaller cell is called the first polar body. The larger cell, which contains nearly all the

cytoplasm of the primary oocyte, is the secondary oocyte. The events of this first maturation division are interesting.

A spindle forms at the very edge of the oocyte and a little “ nipple,” into which the polar body chromosomes will be cast, appears at that edge. This ensures that the polar body receives almost no cytoplasm or organelles. The first polar body may continue its development and undergo meiosis II, producing two even smaller polar bodies. However, in humans, the secondary oocyte arrests in metaphase II and it is this cell (not a functional ovum) that is ovulated. If an ovulated secondary oocyte is not penetrated by a sperm, it simply deteriorates.

But, if sperm penetration does occur, it quickly completes meiosis II, yielding one large ovum and a tiny second polar body. The union of the egg and sperm nuclei, described in Chapter 28, constitutes fertilization. What you should realize now is that the potential end products of oogenesis are three tiny polar bodies, nearly devoid of cytoplasm, and one large ovum. All of these cells are haploid, but only the ovum is a functional gamete. This is quite different from spermatogenesis, where the product is four viable gametes??? spermatozoa.

The unequal cytoplasmic divisions that occur during oogenesis ensure that a fertilized egg has ample nutrients for its six- to seven-day journey to the uterus. Without nutrient-containing cytoplasm the polar bodies degenerate and die. Since the reproductive life of a female is at most about 40 years (from the age of 11 to approximately 51) and typically only one ovulation occurs each month, fewer than 500 oocytes out of her estimated pubertal

potential of 250, 000 are released during a woman's lifetime. Again, nature has provided us with a generous oversupply of sex cells.

Perhaps the most striking difference between male and female meiosis is the error rate. As many as 20% of oocytes but only 3??? 4% of sperm have the wrong number of chromosomes, a situation that often results from failure of the homologues to separate during meiosis I. Thus, it appears that faced with meiotic disruption, meiosis in males grinds to a halt but in females it marches on. The Ovarian Cycle The monthly series of events associated with the maturation of an egg is called the ovarian cycle. The ovarian cycle is best described in terms of two consecutive phases.

The follicular phase is the period of follicle growth, typically indicated as lasting from the first to the fourteenth day of the cycle. The luteal phase is the period of corpus luteum activity, days 14??? 28. The so-called typical ovarian cycle repeats at intervals of 28 days, with ovulation occurring midcycle. However, only 10??? 15% of women naturally have 28-day cycles; cycles as long as 40 days or as short as 21 days are fairly common. In such cases, the length of the follicular phase and timing of ovulation vary, but the luteal phase remains constant: It is 14 days between the time of ovulation and the end of the cycle.

The Follicular Phase A Primordial Follicle Becomes a Primary Follicle - When a primordial follicle is activated (a process directed by the oocyte), the squamouslike cells surrounding the primary oocyte grow, becoming cuboidal cells, and the oocyte enlarges. The follicle is now called a primary follicle A Primary Follicle Becomes a Secondary Follicle - Next, follicular cells

proliferate, forming a stratified epithelium around the oocyte. As soon as more than one cell layer is present, the follicle cells take on the name granulosa cells.

The granulosa cells are connected to the developing oocyte by gap junctions, through which ions, metabolites, and signaling molecules can pass, and from this point on, bidirectional “ conversations” occur between the oocyte and granulosa cells, so they guide one another’s development. One of the signals passing from the granulosa cells to the oocyte “ tells” the oocyte to grow. Others dictate asymmetry (polarity) in the future egg. In the next stage, a layer of connective tissue condenses around the follicle, forming the theca folliculi (the? kah fah-lik? u-li; “ box around the follicle”).

As the follicle grows, the thecal and granulosa cells cooperate to produce estrogens (the inner thecal cells produce androgens, which the granulosa cells convert to estrogens). At the same time, the oocyte secretes a glycoprotein-rich substance that forms a thick transparent membrane, called the zona pellucida (pe-lu? sid-ah), that encapsulates it (see Figure 27. 12). In phase, clear liquid accumulates between the granulosa cells and eventually coalesces to form a fluid-filled cavity called the antrum (“ cave”). The presence of an antrum distinguishes the new secondary follicle from the primary follicle.

A Secondary Follicle Becomes a Vesicular Follicle - The antrum continues to expand with fluid until it isolates the oocyte, along with its surrounding capsule of granulosa cells called a corona radiata (“ radiating crown”), on a stalk on one side of the follicle. When a follicle is full size (about 2. 5 cm, or 1

inch, in diameter), it becomes a vesicular follicle and bulges from the external ovarian surface like an “ angry boil. ” This usually occurs by day 14. As one of the final events of follicle maturation, the primary oocyte completes meiosis I to form the secondary oocyte and first polar body.

Once this has occurred , the stage is set for ovulation. At this point, the granulosa cells send another important signal to the oocyte that says, in effect, “ Wait, do not complete meiosis yet! ” Ovulation occurs when the ballooning ovary wall ruptures and expels the secondary oocyte (still surrounded by its corona radiata) into the peritoneal cavity . Some women experience a twinge of pain in the lower abdomen when ovulation occurs. This episode, called mittelschmerz (mit? el-shmarts; German for “ middle pain”), is caused by the intense stretching of the ovarian wall during ovulation.

In the ovaries of adult females, there are always several follicles at different stages of maturation. As a rule, one follicle outstrips the others to become the dominant follicle and is at the peak stage of maturation when the hormonal (LH) stimulus is given for ovulation. How this follicle is selected, or selects itself, is still uncertain, but it is probably the one that attains the greatest FSH sensitivity the quickest. The others degenerate (undergo programmed cell death, or apoptosis) and are reabsorbed. In 1??? 2% of all ovulations, more than one oocyte is ovulated.

This phenomenon, which increases with age, can result in multiple births. Since, in such cases, different oocytes are fertilized by different sperm, the siblings are fraternal, or nonidentical, twins. Identical twins result from the

fertilization of a single oocyte by a single sperm, followed by separation of the fertilized egg's daughter cells in early development. Additionally, it now appears that in some women, oocytes may be released at times unrelated to the woman's hormone levels, which may help to explain why a rhythm method of contraception sometimes fails and why some fraternal twins have different conception dates.

The Luteal Phase After ovulation, the ruptured follicle collapses, and the antrum fills with clotted blood. This corpus hemorrhagicum is eventually absorbed. The remaining granulosa cells increase in size and along with the internal thecal cells they form a new, quite different endocrine gland, the corpus luteum ("yellow body"), that begins to secrete progesterone and some estrogen. If pregnancy does not occur, the corpus luteum starts degenerating in about ten days and its hormonal output ends. In this case, all that ultimately remains is a scar called the corpus albicans (al? i-kans; "white body"). On the other hand, if the oocyte is fertilized and pregnancy ensues, the corpus luteum persists until the placenta is ready to take over its hormone-producing duties in about three months. The last two or three days of the luteal phase, when the endometrium is just beginning to erode, is sometimes called the luteolytic or ischemic phase.

Hormonal Regulation of the Ovarian Cycle Ovarian events are much more complicated than those occurring in the testes, but the hormonal controls set into motion at puberty are similar in the two sexes.

Gonadotropin-releasing hormone (GnRH), the pituitary gonadotropins, and, in this case, ovarian estrogen and progesterone interact to produce the cyclic events occurring in the ovaries. However, in females another hormone

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plays an important role in stimulating the hypothalamus to release GnRH. The onset of puberty in females is linked to adiposity, and the messenger from fatty tissue to the hypothalamus is leptin. If blood levels of lipids and leptin (better known for its role in energy production and appetite) are low, puberty is delayed. Establishing the Ovarian Cycle

During childhood, the ovaries grow and continuously secrete small amounts of estrogens, which inhibit hypothalamic release of GnRH. As puberty nears, the hypothalamus becomes less sensitive to estrogen and begins to release GnRH in a rhythmic pulsatile manner. GnRH, in turn, stimulates the anterior pituitary to release FSH and LH, which prompt the ovaries to secrete hormones (primarily estrogens). Gonadotropin levels continue to increase for about four years and, during this time, pubertal girls are still not ovulating and thus are incapable of getting pregnant.

Eventually, the adult cyclic pattern is achieved, and hormonal interactions stabilize. These events are heralded by the young woman's first menstrual period, referred to as menarche (me-nar-ke; men = month, arche = first). Usually, it is not until the third year postmenarche that the cycles become regular and all are ovulatory. Hormonal Interactions During the Ovarian Cycle The waxing and waning of anterior pituitary gonadotropins (FSH and LH) and ovarian hormones and the positive and negative feedback interactions that regulate ovarian function are as described next. A 28-day cycle is assumed. On day 1 of the cycle, rising levels of GnRH from the hypothalamus stimulate increased production and release of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) by the anterior pituitary. ??? FSH and LH stimulate follicle growth and maturation and <https://assignbuster.com/anatomy-of-the-female-reproductive-system-assignment/>

estrogen secretion. FSH exerts its main effects on the follicle cells, whereas LH (at least initially) targets the thecal cells. (Why only some follicles respond to these hormonal stimuli is still a mystery. However, there is little doubt that enhanced responsiveness is due to formation of more gonadotropin receptors. As the follicles enlarge, LH prods the thecal cells to produce androgens. These diffuse through the basement membrane, where they are converted to estrogens by the granulosa cells. Only tiny amounts of ovarian androgens enter the blood, because they are almost completely converted to estrogens within the ovaries. ??? The rising estrogen levels in the plasma exert negative feedback on the anterior pituitary, inhibiting its release of FSH and LH, while simultaneously prodding it to synthesize and accumulate these gonadotropins.

Within the ovary, estrogen increases estrogen output by intensifying the effect of FSH on follicle maturation. Inhibin, released by the granulosa cells, also exerts negative feedback controls on FSH release during this period. ??? Although the initial small rise in estrogen blood levels inhibits the hypothalamic-pituitary axis, high estrogen levels have the opposite effect. Once estrogen reaches a critical blood concentration, it exerts positive feedback on the brain and anterior pituitary. ??? High estrogen levels set a cascade of events into motion.

There is a sudden burstlike release of accumulated LH (and, to a lesser extent, FSH) by the anterior pituitary about midcycle ??? The LH surge stimulates the primary oocyte of the dominant follicle to complete the first meiotic division, forming a secondary oocyte that continues on to metaphase

II. LH also triggers ovulation at or around day 14. Whatever the mechanism, <https://assignbuster.com/anatomy-of-the-female-reproductive-system-assignment/>

blood stops flowing through the protruding part of the follicle wall and within 5 minutes, that region of the follicle wall bulges out, thins, and then ruptures.

The role (if any) of FSH in this process is unknown. Shortly after ovulation, estrogen levels decline. This probably reflects the damage to the dominant estrogen-secreting follicle during ovulation. ??? The LH surge also transforms the ruptured follicle into a corpus luteum (hence the name “luteinizing” hormone), and stimulates the newly formed endocrine gland to produce progesterone and estrogen almost immediately after it is formed. ??? Rising progesterone and estrogen blood levels exert a powerful negative feedback effect on anterior pituitary release of LH and FSH.

Corpus luteum release of inhibin enhances this inhibitory effect. Declining gonadotropin levels inhibit the development of new follicles and prevent additional LH surges that might cause additional oocytes to be ovulated. As LH blood levels fall, the stimulus for luteal activity ends, and the corpus luteum degenerates. As goes the corpus luteum, so go the levels of ovarian hormones, and blood estrogen and progesterone levels drop sharply. The marked decline in ovarian hormones at the end of the cycle (days 26??? 28) ends their blockade of FSH and LH secretion, and the cycle starts anew.

Although the ovarian events are described as if we are following one follicle through the 28-day cycle, this is not really the case. What is happening is that the increase of FSH at the beginning of each cycle activates several follicles to mature. Then, with the midcycle LH surge, one (or more) Graafian follicles undergo ovulation. However, the ovulated oocyte would actually have been activated about 110 days (some three months) before, not 14

days before. The Uterine (Menstrual) Cycle Although the uterus is where the young embryo implants and develops, it is receptive to implantation for only a short period each month. Not surprisingly, this brief interval is exactly the time when a developing embryo would normally begin implanting, six to seven days after ovulation. The uterine, or menstrual (men? stroo-al), cycle is a series of cyclic changes that the uterine endometrium goes through each month as it responds to the waxing and waning of ovarian hormones in the blood. These endometrial changes are coordinated with the phases of the ovarian cycle, which are dictated by gonadotropins released by the anterior pituitary.

The events of the uterine cycle, are as follows: 1. Days 1??? 5: Menstrual phase. In this phase, menstruation (men? stroo-a? shun), the uterus sheds all but the deepest part of its endometrium. (At the beginning of this stage, ovarian hormones are at their lowest normal levels and gonadotropins are beginning to rise. Then FSH levels begin to rise.) The thick, hormone-dependent functional layer of the endometrium detaches from the uterine wall, a process that is accompanied by bleeding for 3??? 5 days.

The detached tissue and blood pass out through the vagina as the menstrual flow. By day 5, the growing ovarian follicles are starting to produce more estrogen. 2. Days 6??? 14: Proliferative (preovulatory) phase. In this phase, the endometrium rebuilds itself: Under the influence of rising blood levels of estrogens, the basal layer of the endometrium generates a new functional layer. As this new layer thickens, its glands enlarge and its spiral arteries increase in number. Consequently, the endometrium once again becomes velvety, thick, and well vascularized.

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During this phase, estrogens also induce synthesis of progesterone receptors in the endometrial cells, readying them for interaction with progesterone.

Normally, cervical mucus is thick and sticky, but rising estrogen levels cause it to thin and become crystalline, forming channels that facilitate the passage of sperm into the uterus. Ovulation, which takes less than five minutes, occurs in the ovary at the end of the proliferative stage (day 14) in response to the sudden release of LH from the anterior pituitary. LH also converts the ruptured follicle to a corpus luteum. . Days 15??? 28: Secretory (postovulatory) phase. This 14-day phase is the most constant timewise.

During the secretory phase the endometrium prepares for implantation of an embryo. Rising levels of progesterone from the corpus luteum act on the estrogen-primed endometrium, causing the spiral arteries to elaborate and converting the functional layer to a secretory mucosa. The uterine glands enlarge, coil, and begin secreting nutritious glycogen into the uterine cavity. These nutrients sustain the embryo until it has implanted in the blood-rich endometrial lining.

Increasing progesterone levels also cause the cervical mucus to become viscous again, forming the cervical plug, which blocks sperm entry and plays an important role in keeping the uterus “ private” in the event an embryo has begun to implant. Rising progesterone (and estrogen) levels inhibit LH release by the anterior pituitary. If fertilization has not occurred, the corpus luteum begins to degenerate toward the end of the secretory phase as LH blood levels decline. Progesterone levels fall, depriving the endometrium of hormonal support, and the spiral arteries kink and go into spasms.

Denied oxygen and nutrients, the ischemic endometrial cells die, setting the stage for menstruation to begin on day 28. The spiral arteries constrict one final time and then suddenly relax and open wide. As blood gushes into the weakened capillary beds, they fragment, causing the functional layer to slough off. The menstrual cycle starts over again on this first day of menstrual flow. Notice that the menstrual and proliferative phases overlap the follicular stage and ovulation in the ovarian cycle, and that the uterine secretory phase corresponds to the ovarian luteal phase.

Extremely strenuous activity can delay menarche in girls and can disrupt the normal menstrual cycle in adult women, even causing amenorrhea (a-men? o-re? ah), cessation of menses. Female athletes have little body fat, and fat deposits help convert adrenal androgens to estrogens and are the source of leptin which, as noted above, plays a critical permissive role in the onset of puberty in females. In mature females, leptin keeps the hypothalamus informed about whether energy stores are sufficient to support the high energy demands of reproduction. If not, the reproductive cycles are shut down.

These effects are usually totally reversible when the athletic training is discontinued, but a worrisome consequence of amenorrhea in young, healthy adult women is that they suffer dramatic losses in bone mass normally seen only in old age. Once estrogen levels drop and the menstrual cycle stops (regardless of cause), bone loss begins. Although menses has traditionally been viewed as a somewhat messy way of discarding a uterine lining “fattened” in anticipation of a baby that was never conceived, its adaptive

value and expense to the female body in terms of tissue, blood, and nutrient (particularly iron) loss have been questioned.

Why not just keep the prepared endometrium for the next cycle? A controversial view of menses is that the uterus is a hospitable receptacle for bacteria and viruses delivered via the male penis and piggy-backing in semen. This being so, menses is an aggressive way of cleaning house. Bleeding rids the body of the uterine lining where pathogens are likely to linger, and menstrual blood is loaded with macrophages which provide active protection. Extruterine Effects of Estrogens and Progesterone With a name meaning “ generators of sexual activity,” estrogens are analogous to testosterone, the male steroid.

As estrogen levels rise during puberty, they (1) promote oogenesis and follicle growth in the ovary and (2) exert anabolic effects on the female reproductive tract. Consequently, the uterine tubes, uterus, and vagina grow larger and become functional??? ready to support a pregnancy. The uterine tubes and uterus exhibit enhanced motility; the vaginal mucosa thickens; and the external genitalia mature. Estrogens also support the growth spurt at puberty that makes girls grow much more quickly than boys during the ages of 12 and 13.

But this growth is short lived because rising estrogen levels also cause the epiphyses of long bones to close sooner, and females reach their full height between the ages of 15 and 17 years. In contrast, the aggressive growth of males continues until the age of 19 to 21 years, at which point rising estrogen levels cause epiphyseal closure. The estrogen-induced secondary

sex characteristics of females include (1) growth of the breasts; (2) increased deposit of subcutaneous fat, especially in the hips and breasts; and (3) widening and lightening of the pelvis (adaptations for childbirth).

Estrogens have several metabolic effects, including maintaining low total blood cholesterol levels (and high HDL levels) and facilitating calcium uptake, which helps sustain the density of the skeleton. (These metabolic effects, though initiated under estrogen's influence during puberty, are not true secondary sex characteristics.) Progesterone works with estrogen to establish and then help regulate the uterine cycle and promotes changes in cervical mucus. Its other effects are exhibited largely during pregnancy, when it inhibits uterine motility and takes up where estrogen leaves off in preparing the breasts for lactation.

Indeed, progesterone is named for these important roles (pro = for; gestation = pregnancy). However, the source of progesterone and estrogen during most of pregnancy is the placenta, not the ovaries. Female Sexual Response The female sexual response is similar to that of males in most respects. During sexual excitement, the clitoris, vaginal mucosa, and breasts engorge with blood; the nipples erect; and increased activity of the vestibular glands lubricates the vestibule and facilitates entry of the penis. These events, though more widespread, are analogous to the erection phase in men.

Sexual excitement is promoted by touch and psychological stimuli and is mediated along the same autonomic nerve pathways as in males. The final phase of the female sexual response, orgasm, is not accompanied by

ejaculation, but muscle tension increases throughout the body, pulse rate and blood pressure rise, and the uterus begins to contract rhythmically. As in males, orgasm is accompanied by a sensation of intense pleasure and followed by relaxation. Orgasm in females is not followed by a refractory period, so females may experience multiple orgasms during a single sexual experience.

A man must achieve orgasm and ejaculate if fertilization is to occur, but female orgasm is not required for conception. Indeed, some women never experience orgasm, yet are perfectly able to conceive. Although the female libido was formerly believed to be prompted by testosterone, new studies indicate that dehydroepiandrosterone (DHEA), an androgen produced by the adrenal cortex, is in fact the male sex hormone associated with desire or lack of it in females. Pregnancy and Human Development From Egg to Zygote Accomplishing Fertilization Before fertilization can occur, sperm must reach the ovulated secondary oocyte.

The oocyte is viable for 12 to 24 hours after it is cast out of the ovary; the chance of pregnancy drops to almost zero the next day. Most sperm retain their fertilizing power for 24 to 48 hours after ejaculation. Consequently, for successful fertilization to occur, coitus must occur no more than two days before ovulation and no later than 24 hours after, at which point the oocyte is approximately one-third of the way down the length of the uterine tube. Fertilization occurs when a sperm fuses with an egg (actually a secondary oocyte) to form a fertilized egg, or zygote (zi? got; "yoked together"), the first cell of the new individual.

Let's look at the events leading to fertilization. Sperm Transport and Capacitation During copulation, a man expels millions of sperm with considerable force into his partner's vaginal canal. Despite this "head start," most sperm don't reach the oocyte, even though it is only about 5 inches away. Millions of sperm leak from the vagina almost immediately after being deposited there. Of those remaining, millions more are destroyed by the vagina's acidic environment and, unless the thick "curtain" of cervical mucus has been made fluid by estrogens, millions more fail to make it through the cervix.

Those that do reach the uterus by their whiplike tail movements are then subjected to forceful uterine contractions that act in a washing machine??? like manner to disperse them throughout the uterine cavity, where thousands more are destroyed by resident phagocytes. Only a few thousand (and sometimes fewer than 200) sperm, out of the millions in the male ejaculate, are conducted by reverse peristalsis into and up the uterine tubes, where the oocyte may be moving leisurely toward the uterus. These difficulties aside, there is still another hurdle to overcome. Sperm freshly deposited in the vagina are incapable of penetrating an oocyte.

They must first be capacitated over the next 6 to 8 hours; that is, their mobility is enhanced and their membranes must become fragile so that the hydrolytic enzymes in their acrosomes can be released. As sperm swim through the cervical mucus, uterus, and uterine tubes, secretions of the female tract cause some of their membrane proteins to be removed, and the cholesterol that keeps their acrosomal membranes "tough" and stable is

depleted. Thus, even though the sperm may reach the oocyte within a few minutes, they must “ wait around” (so to speak) for capacitation to occur.

This is an elaborate mechanism for preventing the spilling of acrosomal enzymes. But consider the alternative??? fragile acrosomal membranes in the male reproductive tract could rupture prematurely, causing some degree of autolysis (self-digestion) of the male reproductive system. How sperm navigate to find a released oocyte in the uterine tube is an area of active research. It now appears that they “ sniff” their way to the oocyte. Sperm are known to bear proteins called olfactory receptors that respond to chemical stimuli and it is presumed that the oocyte or its surrounding cells release signaling molecules that direct the sperm.

Acrosomal Reaction and Sperm Penetration The ovulated oocyte is encapsulated by the corona radiata and the deeper zona pellucida, a transparent layer of glycoprotein-rich extracellular matrix secreted by the oocyte, and both must be breached before the oocyte can be penetrated. Once a sperm gets to the immediate vicinity of the oocyte, it penetrates the corona radiata, assisted by a cell-surface hyaluronidase that digests the intercellular cement between the granulosa cells in the immediate area, causing them to fall away from the oocyte.

After breaching the corona, the sperm head binds to the ZP3 glycoprotein of the zona pellucida, which functions as a sperm receptor and helps trigger the acrosomal (ak? ro-som-al) reaction. The acrosomal reaction involves the breakdown of the plasma membrane and the acrosomal membrane, and release of acrosomal enzymes (acrosin, proteases, and others) which digest

holes through the zona pellucida. Hundreds of acrosomes must undergo exocytosis to digest holes in the zona pellucida. This is one case that does not bear out the adage, “ The early bird catches the worm. A sperm that comes along later, after hundreds of sperm have undergone acrosomal reactions to expose the oocyte membrane, is in the best position to be the fertilizing sperm. Once a path has been cleared and a single sperm fuses with the oocyte’s membrane receptors, its nucleus is pulled into the oocyte cytoplasm. Each sperm carries a special two-part binding apparatus on its surface. The beta protein part acts first as it binds to a receptor on the oocyte membrane. This event engages the alpha protein part, causing it to insert into the membrane.

This somehow causes the egg and sperm membranes to open and fuse together with such perfect contact that the contents of both cells are combined within a single membrane??? all without spilling a drop.

Interestingly, the region of the oocyte where the sperm enters determines the future right and left axes of the embryo’s body. Blocks to Polyspermy
Polyspermy (entry of several sperm into an egg) occurs in some animals, but in humans only one sperm is allowed to penetrate the oocyte, ensuring monospermy, the one-sperm-per-oocyte condition.

Once the sperm has entered the oocyte, waves of Ca^{2+} are released by the oocyte’s endoplasmic reticulum into its cytoplasm, which activates the oocyte to prepare for cell division. These calcium surges also cause the cortical reaction, in which granules located just inside the plasma membrane spill their enzymes into the extracellular space beneath the zona pellucida.

These enzymes, called zonal inhibiting proteins (ZIPs), destroy the sperm receptors, preventing further sperm entry.

Additionally, the spilled material binds water, and as it swells it detaches all sperm still bound to receptors on the oocyte membrane, accomplishing the so-called slow block to polyspermy. In rare cases of polyspermy that do occur, the embryos contain too much genetic material and are nonviable (die). Completion of Meiosis II and Fertilization After a sperm enters the oocyte it loses its tail and midpiece, and the centrosome in its midpiece elaborates microtubules which the sperm uses to find the oocyte nucleus.

The sperm then locomotes its DNA-rich nucleus toward the oocyte, its nucleus swelling to about five times its normal size to form the male pronucleus (pro-nu? kle-us; pro = before) on the way. Meanwhile the secondary oocyte, stimulated into activity by the calcium surges, completes meiosis II, forming the ovum nucleus and the second polar body (Figure 28. 3a and b). This accomplished, the ovum nucleus swells, becoming the female pronucleus, and the two pronuclei approach each other. A mitotic spindle develops between them (Figure 28. 3c), and the pronuclei membranes rupture, releasing their chromosomes into the immediate vicinity of the newly formed spindle. The true moment of fertilization occurs as the maternal and paternal chromosomes combine and produce the diploid zygote, or fertilized egg. Almost as soon as the male and female pronuclei come together, their chromosomes replicate. Then, the first mitotic division of the conceptus begins Cleavage and Blastocyst Formation Cleavage is a period of fairly rapid mitotic divisions of the zygote following fertilization. Cleavage produces

small cells with a high surface-to-volume ratio, which enhances their uptake of nutrients and oxygen and the disposal of wastes.

It also provides a large number of cells to serve as building blocks for constructing the embryo. Consider, for a moment, the difficulty of trying to construct a building from one huge block of granite. If you now consider how much easier it would be if instead you could use hundreds of bricks, you will quickly grasp the importance of cleavage. Some 36 hours after fertilization, the first cleavage division has produced two identical cells called blastomeres. These divide to produce four cells, then eight, and so on.

By 72 hours after fertilization, a loose collection of cells that form a berry-shaped cluster of 16 or more cells called the morula (mor? u-lah; "little mulberry") has been formed. All the while, transport of the embryo toward the uterus continues. By day 3 or 4 after fertilization, the embryo consists of about 100 cells and floats free in the uterus. By this time, it has tightened its connections between neighboring cells (a process called compaction) and begins accumulating fluid within an internal cavity. The zona pellucida now starts to break down and the inner structure, now called a blastocyst, "hatches" from it. The blastocyst (blas? o-sist) is a fluid-filled hollow sphere composed of a single layer of large, flattened cells called trophoblast cells (trof? o-blast) and a small cluster of 20 to 30 rounded cells, called the inner cell mass, located at one side. Trophoblast cells begin to display L-selectin (adhesion) molecules on their surface soon after the blastocyst hatching.

They also take part in placenta formation, a fact hinted at by the literal translation of "trophoblast" (nourishment generator), and secrete and display several factors with immunosuppressive effects that protect the

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trophoblast (hence the developing embryo) from attack by the mother's cells.

The inner cell mass becomes the embryonic disc, which forms the embryo proper (and the extraembryonic membranes except the chorion, a trophoblast derivative). **Implantation** While the blastocyst floats in the uterine cavity for two to three days, it receives nourishment from glycogen-rich uterine secretions. Then, some six to seven days after ovulation, given a properly prepared endometrium, implantation begins. The receptivity of the endometrium to implantation??? the so-called window of implantation??? is opened by the surging levels of ovarian hormones (estrogens and progesterone) in the blood.

If the mucosa is properly prepared, integrin and selectin proteins on the trophoblast cells bind respectively to the extracellular matrix components (collagen, fibronectin, laminin, and others) of the endometrial cells and to selectin-binding carbohydrates on the inner uterine wall, and the blastocyst implants high in the uterus. If the endometrium is not yet optimally mature, the blastocyst detaches and floats to a lower level, implanting when it finds a site with the proper receptors and chemical signals.

The trophoblast cells overlying the inner cell mass adhere to the endometrium and secrete digestive enzymes and growth factors against the endometrial surface. The endometrium quickly thickens at the point of contact and takes on characteristics of an acute inflammatory response??? the uterine blood vessels become more permeable and leaky, and inflammatory cells including lymphocytes, natural killer cells, and

macrophages invade the area. The trophoblast then proliferates and forms two distinct layers. The cells in the inner layer, collectively called the cytotrophoblast (si? o-trof? o-blast) or cellular trophoblast, retain their cell boundaries. The cells in the outer layer lose their plasma membranes and form a multinuclear cytoplasmic mass called the syncytiotrophoblast (sin-sit? e-o-trof? o-blast; syn = together, cyt = cell) or syncytial trophoblast, which invades the endometrium and rapidly digests the uterine cells it contacts. As the endometrium is eroded, the blastocyst burrows into this thick, velvety lining and is surrounded by a pool of blood leaked from degraded endometrial blood vessels.

Shortly, the implanted blastocyst is covered over and sealed off from the uterine cavity by proliferation of the endometrial cells. Placentation Placentation (plas? en-ta? shun) refers to the formation of a placenta (" flat cake"), a temporary organ that originates from both embryonic and maternal (endometrial) tissues. Cells from the original inner cell mass give rise to a layer of extraembryonic mesoderm that lines the inner surface of the trophoblast ; together these become the chorion. The chorion develops fingerlike chorionic villi, which are especially elaborate where they are in contact with maternal blood.

Soon the mesodermal cores of the chorionic villi become richly vascularized by newly forming blood vessels, which extend to the embryo via the allantois as the umbilical arteries and vein. The continuing erosion produces large, blood-filled lacunae, or intervillus spaces, in the stratum functionalis of the endometrium, and the villi come to lie in these spaces totally immersed in maternal blood. The part of the endometrium that lies between the chorionic <https://assignbuster.com/anatomy-of-the-female-reproductive-system-assignment/>

villi and the stratum basalis becomes the decidua basalis (de-sid? -ah), whereas that surrounding the uterine cavity face of the implanted embryo forms the decidua capsularis. Together, the chorionic villi and the decidua basalis form the disc-shaped placenta. The fetal side of the placenta is easily recognized because it is slick and smooth, and the umbilical cord projects from its surface. In contrast, the maternal side is “ bumpy,” revealing the shape of the chorionic villus masses. The placenta detaches and sloughs off after the infant is born, so the name of the maternal portion??? decidua (“ that which falls off”)??? is appropriate.

The decidua capsularis expands to accommodate the fetus, which eventually fills and stretches the uterine cavity. As the developing fetus grows, the villi in the decidua capsularis are compressed and degenerate, and the villi in the decidua basalis increase in number and branch even more profusely. The placenta is usually fully functional as a nutritive, respiratory, excretory, and endocrine organ by the end of the third month of pregnancy. However, well before this time, oxygen and nutrients are diffusing from maternal to embryonic blood, and embryonic metabolic wastes are passing in the opposite direction.

The barriers to free passage of substances between the two blood supplies are embryonic barriers??? the membranes of the chorionic villi and the endothelium of embryonic capillaries. Although the maternal and embryonic blood supplies are very close, they normally do not intermix. While the placenta secretes hCG from the beginning, the ability of its syncytiotrophoblast cells (the “ hormone manufacturers”) to produce the estrogens and progesterone of pregnancy matures much more slowly.

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If, for some reason, placental hormones are inadequate when hCG levels wane, the endometrium degenerates and the pregnancy is aborted.

Throughout pregnancy, blood levels of estrogens and progesterone continue to increase, encouraging growth and further differentiation of the mammary glands and readying them for lactation. The placenta also produces other hormones, such as human placental lactogen, human chorionic thyrotropin, and relaxin. The effects of these hormones on the mother are described shortly. Events of Fetal Development

The main events of the fetal period??? weeks 9 through 38??? are listed chronologically in Table 28. 2. The fetal period is a time of rapid growth of the body structures that were established in the embryo. During the first half of this period, cells are still differentiating into specific cell types to form the body's distinctive tissues and are completing the fine details of body structure. During the fetal period, the developing fetus grows from a crown-to-rump length of about 22 mm (slightly less than 1 inch) and a weight of approximately 2 g (0. 6 ounce) to about 360 mm (14 inches) and 3. 2 kg (7 lb) or more. (Total body length at birth is about 550 mm, or 22 inches.) As you might expect with such tremendous growth, the changes in fetal appearance are quite dramatic (Figure 28. 14). Nevertheless, the greatest amount of growth occurs in the first 8 weeks of life, when the embryo grows from one cell to a fetus of 1 inch. Anatomical Changes to the Mother As pregnancy progresses, the female reproductive organs become increasingly vascular and engorged with blood, and the vagina develops a purplish hue (Chadwick's sign).

The enhanced vascularity increases vaginal sensitivity and sexual intensity, and some women achieve orgasm for the first time when they are pregnant. The breasts, too, engorge with blood and, prodded by rising levels of estrogen and progesterone, they enlarge and their areolae darken. Some women develop increased pigmentation of facial skin of the nose and cheeks, a condition called chloasma (klo-az? mah; “to be green”) or the “mask of pregnancy.” The degree of uterine enlargement during pregnancy is remarkable. Starting as a fist-sized organ, the uterus fills most of the pelvic cavity by 16 weeks.

Though the fetus is only about 140 mm long (crown-to-rump) at this time, the placenta is fully formed, uterine muscle is hypertrophied, and amniotic fluid volume is increasing. As pregnancy continues, the uterus pushes higher into the abdominal cavity, exerting pressure on both abdominal and pelvic organs. As birth nears, the uterus reaches the level of the xiphoid process and occupies most of the abdominal cavity. The crowded abdominal organs press superiorly against the diaphragm, which intrudes on the thoracic cavity. As a result, the ribs flare, causing the thorax to widen.

The increasing bulkiness of the anterior abdomen changes the woman’s center of gravity, and many women develop lordosis (accentuated lumbar curvature) and backaches during the last few months of pregnancy.

Placental production of the hormone relaxin causes pelvic ligaments and the pubic symphysis to relax, widen, and become more flexible. This increased motility eases birth passage, but it may result in a waddling gait in the meantime. Additionally, relaxin relaxes the springy ligaments supporting the

bones of the foot and many women need to buy larger shoes during pregnancy.

Considerable weight gain occurs during a normal pregnancy. Because some women are over- or underweight before pregnancy begins, it is almost impossible to state the ideal or desirable weight gain. However, summing up the weight increases resulting from fetal and placental growth, growth of the maternal reproductive organs and breasts, and increased blood volume during pregnancy, a weight gain of approximately 13 kg (about 28 lb) usually occurs. Obviously, good nutrition is necessary all through pregnancy if the developing fetus is to have all the building materials (especially proteins, calcium, and iron) needed to form its tissues.

Additionally, multivitamins containing folic acid reduce the risk of having a baby with neurological problems, including such birth defects as spina bifida and anencephaly. However, a pregnant woman needs only 300 additional calories daily to sustain proper fetal growth. The emphasis should be on eating high-quality food, not just more food. Not surprisingly, effects of the fetal environment may not show up until decades later. Below-normal birth weight, for instance, places females at risk for gestational diabetes and increases the general risk of cardiovascular disease later in life. Metabolic Changes

As the placenta enlarges, it secretes increasing amounts of human placental lactogen (hPL), also called human chorionic somatomammotropin (hCS), which works cooperatively with estrogens and progesterone to stimulate maturation of the breasts for lactation. hPL also promotes growth of the fetus

and exerts a glucose-sparing effect in the mother. Consequently, maternal cells metabolize more fatty acids and less glucose than usual, sparing glucose for use by the fetus. Gestational diabetes mellitus occurs in about 10% of pregnancies, but over half of those women go on to develop type II diabetes later in life.

The placenta also releases human chorionic thyrotropin (hCT), a glycoprotein hormone similar to thyroid-stimulating hormone of the anterior pituitary. hCT activity increases the rate of maternal metabolism throughout the pregnancy, causing hypermetabolism. Because plasma levels of parathyroid hormone and activated vitamin D rise, pregnant women tend to be in positive calcium balance throughout pregnancy. This ensures that the developing fetus will have adequate calcium to mineralize its bones.

Physiological Changes Gastrointestinal System

Many women suffer nausea, commonly called morning sickness, during the first few months of pregnancy, until their system adjusts to the elevated levels of progesterone and estrogens. (Nausea is also a side effect of many birth control pills.) Heartburn, due to reflux of stomach acid into the esophagus, is common because the esophagus is displaced and the stomach is crowded by the growing uterus. Constipation occurs because motility of the digestive tract declines during pregnancy. Urinary System The kidneys produce more urine during pregnancy because they