Pituitary Gland
Pituitary gland

Pituitary gland is connected to the hypothalamus by the pituitary (or hypophysial) stalk. Physiologically, the pituitary gland is divisible into two distinct portions:

The anterior pituitary, also known as the adenohypophysis,

The posterior pituitary, also known as the neurohypophysis.

Between these is a small, relatively avascular zone called the pars intermedia, which is much less developed in the human being but is larger and much more functional in some lower animals.
Hypothalamus Controls Pituitary Secretion

Almost all secretion by the pituitary is controlled by either hormonal or nervous signals from the hypothalamus. Indeed, when the pituitary gland is removed (Hypophysectomy) from its normal position beneath the hypothalamus and transplanted to some other part of the body, its rates of secretion of the different hormones (except for prolactin) fall to very low levels.

Secretion from the posterior pituitary is controlled by nerve signals that originate in the hypothalamus and terminate in the posterior pituitary. The bodies of the cells that secrete the posterior pituitary hormones are not located in the pituitary gland itself but are large neurons, called magnocellular neurons, located in the supraoptic and paraventricular nuclei of the hypothalamus. The hormones are then transported in the axoplasm of the neurons’ nerve fibers passing from the hypothalamus to the posterior pituitary gland.
In contrast, secretion by the anterior pituitary is controlled by hormones called hypothalamic releasing and hypothalamic inhibitory hormones (or factors) secreted within the hypothalamus and then conducted, to the anterior pituitary through minute blood vessels called Hypothalamic-hypophysial portal vessels. In the anterior pituitary, these releasing and inhibitory hormones act on the glandular cells to control their secretion. About 30 to 40 percent of the anterior pituitary cells are somatotropes that secrete growth hormone, and about 20 percent are corticotropes that secrete ACTH. Each of the other cell types accounts for only 3 to 5 percent of the total; Somatotropes stain strongly with acid dyes and are therefore called acidophils.
<table>
<thead>
<tr>
<th>Hypothalamic hormone</th>
<th>Pituitary cell</th>
<th>Pituitary hormone</th>
<th>Organ</th>
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<tr>
<td>Growth hormone–releasing hormone (GHRH), which causes release of growth hormone, and</td>
<td>Somatotrope</td>
<td>human growth hormone (hGH)</td>
<td>Total body</td>
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<tr>
<td>growth hormone inhibitory hormone (GHIH), also called somatostatin, which inhibits</td>
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<tr>
<td>release of growth hormone</td>
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<tr>
<td>Corticotropin</td>
<td>Corticotropes</td>
<td>Adreno-corticotropin hormone (ACTH)</td>
<td>Adrenal cortex</td>
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<tr>
<td>Thyrotropin-releasing hormone (TRH)</td>
<td>Thyrrotropes</td>
<td>thyroid-stimulating hormone (TSH)</td>
<td>Thyroid gland</td>
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<td>Gonadotropin-releasing hormone (GnRH)</td>
<td>Gonadotrope</td>
<td>luteinizing hormone (LH) and follicle-stimulating hormone (FSH)</td>
<td>Gonads</td>
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<tr>
<td>Prolactin inhibitory hormone (PIH) found to be dopamine inhibit prolactin production</td>
<td>Lactotropes</td>
<td>prolactin (PRL)</td>
<td>Brest</td>
</tr>
<tr>
<td>thyrotropin-releasing hormone (TRH) that stimulate prolactin production</td>
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</table>
Growth hormone

Growth hormone, also called somatotropic hormone or somatotropin, is a small protein molecule that contains 191 amino acids in a single chain and has a molecular weight of 22,005.

Growth hormone and prolactin show about 25% sequences identity

Growth Hormone Effects:

Growth Hormone metabolic effects

Aside from its general effect in causing growth, growth hormone has multiple specific metabolic effects, including

1. Growth hormone increase rate of protein synthesis in most cells of the body
2. Growth hormone enhances fat utilization for energy

Excessive Growth Hormone causes excessive mobilization of fat from the adipose tissue

“Ketogenic” Effect this lead to:

1. Large quantities of aceto-acetic acid are formed by the liver and released into the body fluids, thus causing ketosis
2. Frequently causes a fatty liver.

Therefore,

1. Under the influence of growth hormone, fat is used for energy in preference to the use of carbohydrates and proteins.
2. Growth hormone’s ability to promote fat utilization, together with its protein anabolic effect, causes an increase in lean body mass (mean free fat body mass).
Growth hormone decreased rate of glucose utilization throughout the body.

Growth hormone causes multiple effects that influence carbohydrate metabolism, including:

1. decreased glucose uptake in tissues such as skeletal muscle and fat,
2. increased glucose production by the liver,
3. increased insulin secretion.

For these reasons, growth hormone’s effects are called diabetogenic, and excess secretion of growth hormone can produce metabolic disturbances similar to those found in patients with type II (non-insulin-dependent) diabetes, who are also resistant to the metabolic effects of insulin.

Growth hormone fails to cause growth:

a. in animals that lack a pancreas
b. if carbohydrates are excluded from the diet.

This shows that adequate insulin activity and adequate availability of carbohydrates are necessary for growth hormone to be effective.
Growth Hormone effect on Cartilage and Bone Growth

Growth Hormone causes growth of almost all tissues of the body that are capable of growing. Growth Hormone most obvious effect is to increase growth of the skeletal frame.

This results from multiple effects of growth hormone on bone, including:

1. Increase deposition of protein by the chondrocytic and osteogenic cells that cause bone growth,
2. Increase rate of reproduction of chondrocytic and osteogenic cells,
3. A specific effect of converting chondrocytes into osteogenic cells, thus causing deposition of new bone.

There are two principal mechanisms of bone growth.

**First,** in response to growth hormone stimulation, the long bones grow in length at the epiphyseal cartilages, where the epiphyses at the ends of the bone are separated from the shaft by epiphyseal plate. This growth first causes deposition of new cartilage, followed by its conversion into new bone, thus elongating the shaft and pushing the epiphyses farther and farther apart. At the same time, the epiphyseal cartilage itself is progressively used up, so by late adolescence, no additional epiphyseal cartilage remains to provide for further long bone growth.
Epiphyseal plate is found in children and adolescents; in adults, who have stopped growing, epiphyseal plate is replaced by an epiphyseal line. This replacement is known as epiphyseal closure.

As soon as bony fusion occurs between the shaft and the epiphysis at each end, so no further lengthening of the long bone can occur.

Flat bone for instance, the jaw bones can be stimulated to grow even after adolescence, causing forward protrusion of the chin and lower teeth. Likewise, the bones of the skull can grow in thickness and give rise to bony protrusions over the eyes.

Epiphyseal plate sometime called growth plate or epiphyseal growth plate

Second, osteoblasts in the bone periosteum and in some bone cavities deposit new bone on the surfaces of older bone. Simultaneously, osteoclasts in the bone remove old bone. When the rate of deposition is greater than that of resorption, the thickness of the bone increases.

Growth hormone strongly stimulates osteoblasts. Therefore, the bones can continue to become thicker throughout life under the influence of growth hormone; this is especially true for the membranous bones.
Growth Hormone exerts much of its effect through intermediate substances called “Somatomedins” (Also Called “Insulin-Like Growth Factors”)

Growth hormone causes the liver (and, to a much less extent, other tissues) to form several small proteins called somatomedins that have the potent effect of increasing all aspects of bone growth. Many of the somatomedin effects on growth are similar to the effects of insulin on growth. Therefore, the somatomedins are also called insulin-like growth factors (IGFs: somatomedin A, which is another name for insulin-like growth factor 2 (IGF-2) somatomedin C another name is insulin-like growth factor 1 (IGF-1)

somatomedin C (IGF-1) molecular weight is about 7500

somatomedin C (IGF-1) concentration in the plasma closely follows the rate of growth hormone secretion.

Growth hormone attaches only weakly to the plasma proteins in the blood. Therefore, it is released from the blood into the tissues rapidly, having a half-time in the blood of less than 20 minutes. By contrast, somatomedin C (IGF-1) attaches strongly to a carrier protein in the blood that, like somatomedin C (IGF-1), is produced in response to growth hormone. As a result, somatomedin C (IGF-1) is released only slowly from the blood to the tissues, with a half-time of about 20 hours. This greatly prolongs the growth-promoting effects of the bursts of growth hormone secretion. It has been postulate that most, if not nearly all of the growth effect of GH results from somatomedin C (IGF-1) and other somatomedins, rather than from direct effects of growth hormone on the bone and other tissues.
- somatomedin C (IGF-1) has insulin-like effect

<table>
<thead>
<tr>
<th>Treatment</th>
<th>GH</th>
<th>IGF-I</th>
<th>GH+IGF-I</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>↑</td>
<td>↓ or No effect</td>
<td>No effect</td>
</tr>
<tr>
<td>Insulin secretion</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Insulin sensitivity</td>
<td>↓</td>
<td>↑ or ↑↑</td>
<td>↑</td>
</tr>
<tr>
<td>Lipolysis</td>
<td>↑</td>
<td>No effect</td>
<td>↑</td>
</tr>
<tr>
<td>Protein synthesis</td>
<td>↑</td>
<td>No effect or ↑</td>
<td>↑</td>
</tr>
<tr>
<td>Protein breakdown</td>
<td>↑</td>
<td>No effect or ↑</td>
<td>Not known</td>
</tr>
<tr>
<td>Net protein balance</td>
<td>↑</td>
<td>No effect or ↑</td>
<td>↑↑</td>
</tr>
</tbody>
</table>

↑ Stimulating; ↓ inhibiting
Growth hormone release is episodic and pulsatile (regular release). Plasma Growth hormone levels may change as much as 10-fold within minutes. Diurnal variations in growth hormone secretion superimposed on these developmental stages. Growth hormone is found at relatively low levels during the day, unless specific triggers for its release are present.

During sleep, on the other hand, large pulsatile bursts of growth hormone secretion occur. One of the largest increases occur shortly after the onset of sleep.

The stimulator of Growth hormone secretion is Growth Hormone Releasing Hormone (GHRH). Growth Hormone Releasing Hormone (GHRH) increases slow wave sleep and feeding behavior.
Controlling Growth Hormone Secretion

It is known that growth hormone secretion is controlled by two factors secreted in the hypothalamus and then transported to the anterior pituitary gland through the hypothalamic-hypophysial portal vessels. They are growth hormone–releasing hormone (GHRH) and growth hormone inhibitory hormone (also called somatostatin). Both of these are polypeptides; GHRH is composed of 44 amino acids, and somatostatin is composed of 14 amino acids.

Regulation of growth hormone secretion

(1) Hypothalamic control:

- Growth Hormone Releasing Hormone (GHRH) stimulates the synthesis and secretion of growth hormone.
- Ghrelin. The main site of ghrelin synthesis and secretion is the stomach, but it is also produced in the hypothalamus and has marked growth hormone-stimulating activity. Ghrelin appears to be involved in the regulation of food intake.
- Somatostatin inhibits secretion of growth hormone by blocking the response of the anterior pituitary to GHRH.
(2) Negative feedback control by somatomedin C (or Insulin like growth Factor-I: IGF-I)
- Somatomedins inhibit the secretion of growth hormone
  ① By acting directly on the anterior pituitary
  ② By stimulating the secretion of somatostatin from the hypothalamus.

(3) Negative feedback control by GHRH and growth hormone
- GHRH inhibits its own secretion from the hypothalamus.
- Growth hormone also inhibits its own secretion by stimulating the secretion of somatostatin from the hypothalamus.

<table>
<thead>
<tr>
<th>Several factors related to a person’s state of nutrition or stress are known to stimulate secretion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stimulate Growth Hormone Secretion</strong></td>
</tr>
<tr>
<td>1. Decreased blood glucose</td>
</tr>
<tr>
<td>2. Decreased blood free fatty acids</td>
</tr>
<tr>
<td>3. Increased blood amino acids (arginine)</td>
</tr>
<tr>
<td>5. Starvation or fasting, protein deficiency</td>
</tr>
<tr>
<td>7. Trauma, stress, excitement</td>
</tr>
<tr>
<td>8. Exercise</td>
</tr>
<tr>
<td>9. Testosterone, estrogen</td>
</tr>
<tr>
<td>10. Deep sleep (stages II and IV)</td>
</tr>
</tbody>
</table>
Abnormalities of Growth Hormone Secretion
Pan-hypo-pituitarism.
This term means decreased secretion of all the anterior pituitary hormones.
Dwarfism.
Most instances of dwarfism result from generalized deficiency of anterior pituitary secretion (pan-hypo-pituitarism) during childhood. In general, all the physical parts of the body develop in appropriate proportion to one another, but the rate of development is greatly decreased.
Gigantism.
Occasionally, the acidophilic become excessively active. As a result, large quantities of growth hormone are produced.
① All body tissues grow rapidly, including the bones
② The giant ordinarily has hyperglycemia
Acromegaly.

If an acidophilic tumor occurs after adolescence the person cannot grow taller, but the bones can become thicker and the soft tissues can continue to grow. This condition is known as acromegaly.

1. Enlargement is especially marked in the Bones of the ① Hands ② Feet
2. Thicken and increase size in the membranous bones, including the ① cranium, ② nose ③ bosses on the forehead, ④ supraorbital ridges ⑤ lower jawbone
3. Changes in many soft tissue organs, such as the tongue, the liver, and especially the kidneys, become greatly enlarged.
Physiology of growth

Growth period:
In human, there are two periods of rapid growth:
The first in infancy is partly a continuation of the fetal growth period.
The second in late puberty just before growth stops: is due to hormonal cause and the subsequent cessation is due in part to closer of the epiphyses by (estrogen, testosterone).
Since girls mature earlier than boys, this growth spurt appears earlier in girls.
Of course, in both sexes the rate of growth of individual tissue varies.
The factors affecting growth including:

1. Genetic effects:
2. Nutrition effects: The food supply is the most important extrinsic factor affecting growth.
3. Hormonal effects:

The hormones affecting growth are:

1. Growth hormone:
2. Sex hormones:
   Although androgen and estrogen initially stimulate growth, estrogen ultimately terminate growth by causing the epiphyses to fuse to the long bones (*epiphysial colures*).
3. Thyroid hormone:
   Thyroid hormone effect is permissive to that of growth hormone
4. Insulin: affect growth through the effects of insulin on *protein metabolism*.
5. Adreno- cortical hormones:
Prolactin Hormone

Although estrogen and progesterone are essential for the physical development of the breasts during pregnancy, a specific effect of both these hormones is to inhibit the actual secretion of milk. Conversely, the hormone prolactin has exactly the opposite effect on milk secretion—promoting it.

This hormone is secreted by the mother’s anterior pituitary gland, and its concentration in her blood rises steadily from the fifth week of pregnancy until birth of the baby, at which time it has risen to 10 to 20 times the normal non-pregnant level. After birth of the baby, the basal level of prolactin secretion returns to the non-pregnant level over the next few weeks. However, each time the mother nurses her baby, nervous signals from the nipples to the hypothalamus (during child milk suckling) causes a 10- to 20-fold surge in prolactin secretion that lasts for about 1 hour. This prolactin acts on the mother’s breasts to keep the mammary glands secreting milk into the alveoli for the subsequent nursing periods. If this prolactin surge is absent or blocked as a result of hypothalamic or pituitary damage or if nursing does not continue, the breasts lose their ability to produce milk within 1 week or so. However, milk production can continue for several years if the child continues to suckle, although the rate of milk formation normally decreases considerably after 7 to 9 months.
a. Regulation of prolactin secretion (Figure 7-7 and Table 7-3)

(1) Hypothalamic control by dopamine and thyrotropin-releasing hormone (TRH)
- Prolactin secretion is tonically inhibited by dopamine [previously called prolactin inhibiting factor (PIF)] secreted by the hypothalamus. Thus, interruption of the hypothalamic-pituitary tract causes increased secretion of prolactin and sustained lactation. TRH increases prolactin secretion.

(2) Negative feedback control
- Prolactin inhibits its own secretion by stimulating the hypothalamic release of dopamine.

<table>
<thead>
<tr>
<th>Factors that Increase Prolactin Secretion</th>
<th>Factors that Decrease Prolactin Secretion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estrogen (pregnancy)</td>
<td>Dopamine</td>
</tr>
<tr>
<td>Breast-feeding</td>
<td>Bromocriptine (dopamine agonist)</td>
</tr>
<tr>
<td>Sleep</td>
<td>Somatostatin</td>
</tr>
<tr>
<td>Stress</td>
<td>Prolactin (by negative feedback)</td>
</tr>
<tr>
<td>TRH</td>
<td>TRH</td>
</tr>
<tr>
<td>Dopamine antagonists</td>
<td>TRH</td>
</tr>
</tbody>
</table>

TRH = thyrotropin releasing hormone
c. Patho-physiology of prolactin

(1) Prolactin deficiency *(destruction of the anterior pituitary)*
- results in the failure to lactate.

(2) Prolactin excess
- results from hypothalamic destruction *(due to loss of the tonic "inhibitory" control by dopamine)*, or from prolactin-secreting tumors *(prolactinomas)*.
- causes
  1. galactorrhea *(Galactorrhea is the spontaneous flow of milk from the breast, unassociated with childbirth or nursing)*
  2. decreased libido.
  3. failure to ovulate and amenorrhea because it inhibits GnRH secretion
- can be treated with bromocriptine, which reduces prolactin secretion by acting as a dopamine agonist
Pituitary Intermediate-lobe

Intermediate-lobe cells and corticotropes of the anterior lobe both synthesize a large precursor protein that is cleaved to form a family of hormones. After removal of the signal peptide, this prohormone is known as pro-opio-melano-cortin (POMC). This molecule is also synthesized in the hypothalamus, the lungs, the gastrointestinal tract, and the placenta.

In corticotropes, it is hydrolyzed to ACTH and a polypeptide of unknown function called β-lipotropin (LPH), plus a small amount of β-endorphin, and these substances are secreted. In the intermediate lobe cells, POMC is hydrolyzed to corticotropin-like intermediate-lobe peptide (CLIP), γ-LPH, and appreciable quantities of β-endorphin. ACTH will hydrolyzes to CLIP and α-MSH indicated similarity in structure between ACTH and α-MSH.

The functions, if any, of CLIP and γ-LPH are unknown, whereas β-endorphin is an opioid that has the five amino acid residues of met-enkephalin at its amino terminal end. The melanotropins α- and β-MSH are also formed. However, the intermediate lobe in humans is rudimentary, and it appears that neither α-MSH nor β-MSH is secreted in adults.

In some animals, an intermediate “lobe” of the pituitary gland, called the pars intermedia, is highly developed, lying between the anterior and posterior pituitary lobes. This lobe secretes an especially large amount of MSH. Furthermore, this secretion is independently controlled by the hypothalamus in response to the amount of light to which the animal is exposed or in response to other environmental factors. For instance, some arctic animals develop darkened fur in the summer and yet have entirely white fur in the winter.
The posterior pituitary gland, also called the **neurohypophysis**, contains **terminal nerve endings** from nerve tracts that originate in the **supraoptic and paraventricular nuclei** of the hypothalamus. These tracts pass to the neurohypophysis through the **pituitary stalk** (hypophysial stalk). The nerve endings are bulbous knobs that contain many secretory granules. These endings lie on the surfaces of capillaries, where they secrete two posterior pituitary hormones: (1) **antidiuretic hormone (ADH)**, also called **vasopressin**, and (2) **oxytocin**.

If the pituitary stalk is cut above the pituitary gland but the entire hypothalamus is left intact, the posterior pituitary hormones continue to be secreted normally, **after a transient decrease** for a few days; they are then secreted by the cut ends of the fibers within the hypothalamus and not by the nerve endings in the posterior pituitary. The reason for this is that the hormones are initially synthesized in the cell bodies of the supraoptic and paraventricular nuclei and are then transported in combination with **“carrier” proteins called neurophysins** down to the nerve endings in the posterior pituitary gland, requiring several days to reach the gland.
ADH is formed primarily in the supraoptic nuclei, oxytocin is formed primarily in the paraventricular nuclei. Each of these nuclei can synthesize about one sixth as much of the second hormone as of its primary hormone. When nerve impulses are transmitted downward along the fibers from the supraoptic or paraventricular nuclei, the hormone is immediately released from the secretory granules in the nerve endings by the usual secretory mechanism of exocytosis and is absorbed into adjacent capillaries. Both the neurophysin and the hormone are secreted together, but because they are only loosely bound to each other, the hormone separates almost immediately. The neurophysin has no known function after leaving the nerve terminals.

Both ADH and oxytocin are considered as a type of neuroendocrine hormones.

Chemical Structures of Antidiuretic Hormone and Oxytocin

Both oxytocin and ADH (vasopressin) are polypeptides, each containing nine amino acids. Their amino acid sequences are the following:

- **Arginine Vasopressin (Human)**
  
  Cyst - Tyr - Phe - Gln - Asn - Cyst - Pro - Arg - Gly - NH₂

- **Oxytocin (Human)**
  
  Cyst - Tyr - Ile - Gln - Asn - Cyst - Pro - Leu - Gly - NH₂

Note that these two hormones are almost identical except that in vasopressin, phenylalanine (3) and arginine (8) replace isoleucine and leucine of the oxytocin molecule. The similarity of the molecules explains their partial functional similarities.
Physiological Functions of Antidiuretic Hormone

The injection of extremely minute quantities of ADH (as small as 2 nano-grams) can cause decreased excretion of water by the kidneys (anti-diuresis: decrease urine volume, or reducing urination).

Factors stimulate the release of vasopressin are:

1. Angiotensin II,
2. Sympathetic stimulation,
3. Hyper-osmolarity (through osmo-receptors in the hypothalamus)

An increase in plasma osmolality is the principal physiological stimulus for vasopressin secretion.

4. Hypovolemia, Hypotension

Severe hypovolemia/hypotension also is a powerful stimulus for vasopressin release.

5. In addition, Hypoglycemia, pain, nausea, Nicotine, opiates, antineoplastic drugs and hypoxia can stimulate vasopressin secretion.

Factors inhibits the release of vasopressin are:

1. Decrease serum osmolarity
2. Ethanol
3. Alpha-agonists
4. Atrial natriuretic peptide

Atrial receptors respond to a decrease in blood volume or blood pressure and osmo-receptors in hypothalamus respond to a decrease in blood osmolarity both cause the release of vasopressin from the posterior pituitary.
Although some researchers place these osmo-receptors in the hypothalamus itself (possibly even in the supra-optic nuclei), others believe that they are located in the *organum vasculosum* a highly vascular structure in the antero-ventral wall of the third ventricle and lacks a blood brain barrier.
For this reason, ADH has another name, vasopressin (vaso: vasoconstriction; pressin: pressure). Changes in plasma osmolarity is more potent than changes in blood volume to affect ADH secretion.
**Oxytocin Hormone**

Oxytocin is now believed to be involved in a wide variety of physiological and pathological functions such as ① sexual activity, ② penile erection, ③ ejaculation, ④ pregnancy, ⑤ uterine contraction, ⑥ milk ejection, ⑦ maternal behavior, ⑧ social bonding, stress and probably many more, this why sometimes is call “Love hormone” or "cuddle hormone"

Regulation of oxytocin secretion:

1) Suckling
   - is the major stimulus for oxytocin secretion.
   - Afferent fibers carry impulses from the nipple to the spinal cord. Relays in the hypothalamus trigger the release of

2) Dilation of the cervix and orgasm
   - increase the secretion of oxytocin.
Effects of oxytocine

The hormone *oxytocin*, in accordance with its name, powerfully stimulates contraction of the pregnant uterus, especially toward the end of gestation. Therefore, many obstetricians believe that this hormone is at least partially responsible for causing birth of the baby.

The sensitivity of the uterine musculature to oxytocin is enhanced by estrogen and inhibited by progesterone.

In late pregnancy, the uterus becomes very sensitive to oxytocin coincident with a marked increase in the number of oxytocin receptors.

Oxytocin secretion is increased during labor.

After dilation of the cervix, descent of the fetus down the birth canal initiates impulses in the afferent nerves that are relayed to the supraoptic and paraventricular nuclei, causing secretion of sufficient oxytocin to enhance labor.

Oxytocin may also act on the non-pregnant uterus to facilitate sperm transport.

Circulating oxytocin increases at the time of ejaculation in males, and it is possible that this increase causes increased contraction of the smooth muscle of the vas deferens, propelling sperm toward the urethra.
Oxytocin Aids in Milk Ejection by the Breasts.

In lactation, oxytocin causes milk to be expressed from the alveoli into the ducts of the breast so that the baby can obtain it by suckling. This mechanism works as follows: The **suckling stimulus on the nipple of the breast** causes signals to be transmitted through sensory nerves to the oxytocin neurons in the paraventricular and supraoptic nuclei in the hypothalamus, which causes release of oxytocin by the posterior pituitary gland. The oxytocin is then carried by the blood to the breasts, where it causes contraction of **myoepithelial cells** that lie outside of and form a latticework surrounding the alveoli of the mammary glands. In less than a minute after the beginning of suckling, milk begins to flow.

- Oxytocin can be used to
  1. induce labor and
  2. reduce postpartum bleeding.