**FAKEYE TAIWO 13/MHS06/016 MLS 514 ASSIGNMENT 2020**

**1(a) HORMONES OF THE ANTERIOR PITUITARY GLAND**

**Growth hormone** (**GH**) or **somatotropin**, also known as **human growth hormones** (**hGH** or **HGH**) in its human form, is a [peptide hormone](https://en.wikipedia.org/wiki/Peptide_hormone) that stimulates growth, [cell](https://en.wikipedia.org/wiki/Cell_%28biology%29) reproduction, and cell regeneration in humans and other animals. It is thus important in [human development](https://en.wikipedia.org/wiki/Human_development_%28biology%29). GH also stimulates production of [IGF-1](https://en.wikipedia.org/wiki/IGF-1) and increases the concentration of [glucose](https://en.wikipedia.org/wiki/Glucose) and [free fatty acids](https://en.wikipedia.org/wiki/Free_fatty_acid). It is a type of [mitogen](https://en.wikipedia.org/wiki/Mitogen%22%20%5Co%20%22Mitogen) which is specific only to the [receptors](https://en.wikipedia.org/wiki/Receptor_%28biochemistry%29) on certain types of cells. GH is a 191-[amino acid](https://en.wikipedia.org/wiki/Amino_acid), single-chain [polypeptide](https://en.wikipedia.org/wiki/Polypeptide) that is synthesized, stored and secreted by [somatotropic cells](https://en.wikipedia.org/wiki/Somatotropic_cell%22%20%5Co%20%22Somatotropic%20cell) within the lateral wings of the [anterior pituitary](https://en.wikipedia.org/wiki/Anterior_pituitary) gland.

A [recombinant](https://en.wikipedia.org/wiki/Recombinant_DNA) form of hGH called **somatreopleopin** ([INN](https://en.wikipedia.org/wiki/International_Nonproprietary_Name)) is used as a [prescription drug](https://en.wikipedia.org/wiki/Prescription_drug) to treat children's growth disorders and adult [growth hormone deficiency](https://en.wikipedia.org/wiki/Growth_hormone_deficiency). In the United States, it is only available legally from pharmacies by prescription from a licensed health care provider. In recent years in the United States, some health care providers are prescribing growth hormone in the elderly to increase [vitality](https://en.wikipedia.org/wiki/Vitality). While legal, the efficacy and safety of this use for HGH has not been tested in a clinical trial. Many of the functions of hGH remain unknown.

In its role as an [anabolic](https://en.wikipedia.org/wiki/Anabolic) agent, HGH has been used by competitors in sports since at least 1982, and has been banned by the [IOC](https://en.wikipedia.org/wiki/IOC) and [NCAA](https://en.wikipedia.org/wiki/NCAA). Traditional [urine](https://en.wikipedia.org/wiki/Urine) analysis does not detect [doping](https://en.wikipedia.org/wiki/Use_of_performance-enhancing_drugs_in_sport) with HGH, so the ban was not enforced until the early 2000s, when [blood tests](https://en.wikipedia.org/wiki/Blood_test) that could distinguish between natural and artificial HGH were starting to be developed. Blood tests conducted by [WADA](https://en.wikipedia.org/wiki/World_Anti-Doping_Agency) at the [2004 Olympic Games](https://en.wikipedia.org/wiki/2004_Olympic_Games) in [Athens, Greece](https://en.wikipedia.org/wiki/Athens%2C_Greece) targeted primarily HGH. Use of the drug for performance enhancement is not currently approved by the [FDA](https://en.wikipedia.org/wiki/FDA).

GH has been studied for use in raising livestock more efficiently in [industrial agriculture](https://en.wikipedia.org/wiki/Industrial_agriculture) and several efforts have been made to obtain governmental approval to use GH in livestock production. These uses have been controversial. In the United States, the only FDA-approved use of GH for livestock is the use of a cow-specific form of GH called [bovine somatotropin](https://en.wikipedia.org/wiki/Bovine_somatotropin) for increasing milk production in dairy cows. Retailers are permitted to label containers of milk as produced with or without bovine somatotropin.

**ADRENOCORTICOTROPIC HORMONE**

**Adrenocorticotropichormone** (**ACTH**;also **adrenocorticotropin**, **corticotropin**) is a [polypeptide](https://en.wikipedia.org/wiki/Peptide) [tropic hormone](https://en.wikipedia.org/wiki/Tropic_hormone) produced by and secreted by the [anterior pituitary gland](https://en.wikipedia.org/wiki/Anterior_pituitary). It is also used as a [medication and diagnostic agent](https://en.wikipedia.org/wiki/Adrenocorticotropic_hormone_%28medication%29). ACTH is an important component of the [hypothalamic-pituitary-adrenal axis](https://en.wikipedia.org/wiki/Hypothalamic-pituitary-adrenal_axis) and is often produced in response to biological stress (along with its precursor [corticotropin-releasing hormone](https://en.wikipedia.org/wiki/Corticotropin-releasing_hormone) from the [hypothalamus](https://en.wikipedia.org/wiki/Hypothalamus)). Its principal effects are increased production and release of [cortisol](https://en.wikipedia.org/wiki/Cortisol) by the [cortex](https://en.wikipedia.org/wiki/Adrenal_cortex) of the [adrenal gland](https://en.wikipedia.org/wiki/Adrenal_gland). ACTH is also related to the [circadian rhythm](https://en.wikipedia.org/wiki/Circadian_rhythm) in many organisms.

Deficiency of ACTH is a sign of secondary [adrenal insufficiency](https://en.wikipedia.org/wiki/Adrenal_insufficiency) (suppressed production of ACTH due to an impairment of the [pituitary gland](https://en.wikipedia.org/wiki/Pituitary_gland) or [hypothalamus](https://en.wikipedia.org/wiki/Hypothalamus), cf. [hypopituitarism](https://en.wikipedia.org/wiki/Hypopituitarism)) or tertiary adrenal insufficiency (disease of the hypothalamus, with a decrease in the release of [corticotropin releasing hormone (CRH)](https://en.wikipedia.org/wiki/Corticotropin_releasing_hormone)). Conversely, chronically elevated ACTH levels occur in primary adrenal insufficiency (e.g. [Addison's disease](https://en.wikipedia.org/wiki/Addison%27s_disease)) when adrenal gland production of [cortisol](https://en.wikipedia.org/wiki/Cortisol) is chronically deficient. In Cushing's disease a pituitary tumor is the cause of elevated ACTH (from the anterior pituitary) and an excess of cortisol (hypercortisolism) – this constellation of signs and symptoms is known as [Cushing's syndrome](https://en.wikipedia.org/wiki/Cushing%27s_syndrome).

**LUTEINIZING HORMONE**

Luteinizing hormone (LH) is produced and released in the anterior pituitary gland. This hormone is considered a gonadotrophic hormone because of its role in controlling the function of ovaries in females and testes in males, which are known as the gonads.

**What does luteinizing hormone do?**

In women, the hormone stimulates the ovaries to produce [**oestradiol**](https://www.hormone.org/your-health-and-hormones/glands-and-hormones-a-to-z/hormones/estradiol). Two weeks into a woman's cycle, a surge in luteinizing hormone causes the ovaries to release an egg during ovulation. If fertilization occurs, luteinizing hormone will stimulate the corpus luteum, which produces progesterone to sustain the pregnancy.

For men, luteinizing hormone stimulates the production of [**testosterone**](https://www.hormone.org/your-health-and-hormones/glands-and-hormones-a-to-z/hormones/testosterone) from Leydig cells in the testes. Testosterone, in turn, stimulates sperm production and helps accentuate male characteristics — like a deep voice or growth of facial hair.

**THYROID STIMULATING HORMONE**

Thyroid stimulating hormone is produced by the pituitary gland. Its role is to regulate the production of hormones by the thyroid gland. Alternative names for thyroid stimulating hormone TSH; thyrotropin, thyrotrophin

Thyroid stimulating hormone is produced and released into the bloodstream by the [pituitary gland](https://www.yourhormones.info/glands/pituitary-gland/). It controls production of the thyroid hormones, [thyroxine](https://www.yourhormones.info/hormones/thyroxine/) and [triiodothyronine](https://www.yourhormones.info/hormones/triiodothyronine/), by the [thyroid gland](https://www.yourhormones.info/glands/thyroid-gland/) by binding to [receptors](https://www.yourhormones.info/glossary/r#receptors) located on cells in the thyroid gland. Thyroxine and triiodothyronine are essential to maintaining the body’s [metabolic rate](https://www.yourhormones.info/glossary/m#metabolic-rate), heart and digestive functions, muscle control, brain development and maintenance of bones.

**How is thyroid stimulating hormone controlled?**

When thyroid stimulating hormone binds to the receptor on the thyroid cells, this causes these cells to produce thyroxine and triiodothyronine and release them into the bloodstream. These hormones have a negative effect on the pituitary gland and stop the production of thyroid stimulating hormone if the levels of thyroxine and triiodothyronine are too high. They also switch off production of a hormone called [thyrotropin-releasing hormone](https://www.yourhormones.info/hormones/thyrotropin-releasing-hormone/). This hormone is produced by the [hypothalamus](https://www.yourhormones.info/glands/hypothalamus/) and it also stimulates the pituitary gland to make thyroid stimulating hormone.

**FOLLICLE STIMULATING HORMONE**

Follicle stimulating hormone is produced by the pituitary gland. It regulates the functions of both the ovaries and testes. Lack or insufficiency of it can cause infertility or sub fertility both in men and women. Alternative names for follicle stimulating hormone is FSH; follitropin (pharmaceutical preparations).

Follicle stimulating hormone is one of the gonadotrophic hormones, the other being [luteinising hormone](https://www.yourhormones.info/hormones/luteinising-hormone/). Both are released by the [pituitary gland](https://www.yourhormones.info/glands/pituitary-gland/) into the bloodstream. Follicle stimulating hormone is one of the hormones essential to pubertal development and the function of women’s [ovaries](https://www.yourhormones.info/glands/ovaries/) and men’s [testes](https://www.yourhormones.info/glands/testes/). In women, this hormone stimulates the growth of ovarian follicles in the [ovary](https://www.yourhormones.info/glands/ovaries/) before the release of an egg from one follicle at [ovulation](https://www.yourhormones.info/glossary/o#ovulation). It also increases [oestradiol](https://www.yourhormones.info/hormones/oestradiol/) production. In men, follicle stimulating hormone acts on the Sertoli cells of the testes to stimulate sperm production (spermatogenesis).

**How is follicle stimulating hormone controlled?**

The production and release of follicle stimulating hormone is regulated by the levels of a number of circulating hormones released by the ovaries and testes. This system is called the hypothalamic–pituitary–gonadal axis. [Gonadotrophin-releasing hormone](https://www.yourhormones.info/hormones/gonadotrophin-releasing-hormone/) is released from the [hypothalamus](https://www.yourhormones.info/glands/hypothalamus/) and binds to [receptors](https://www.yourhormones.info/glossary/r#receptors) in the [anterior](https://www.yourhormones.info/glossary/a#anterior) pituitary gland to stimulate both the synthesis and release of follicle stimulating hormone and luteinising hormone. The released follicle stimulating hormone is carried in the bloodstream where it binds to receptors in the testes and ovaries. Using this mechanism follicle stimulating hormone, along with luteinising hormone, can control the functions of the testes and ovaries.

In women, when hormone levels fall towards the end of the [menstrual cycle](https://www.yourhormones.info/glossary/m#menstrual-cycle), this is sensed by nerve cells in the hypothalamus. These cells produce more gonadotrophin-releasing hormone, which in turn stimulates the pituitary gland to produce more follicle stimulating hormone and luteinising hormone, and release these into the bloodstream. The rise in follicle stimulating hormone stimulates the growth of the follicle in the ovary. With this growth, the cells of the follicles produce increasing amounts of oestradiol and inhibin. In turn, the production of these hormones is sensed by the hypothalamus and pituitary gland and less gonadotrophin-releasing hormone and follicle stimulating hormone will be released. However, as the follicle grows, and more and more [oestrogen](https://www.yourhormones.info/glossary/o%22%20%5Cl%20%22oestrogen) is produced from the follicles, it simulates a surge in luteinising hormone and follicle stimulating hormone, which stimulates the release of an egg from a mature follicle – ovulation.

Thus, during each menstrual cycle, there is a rise in follicle stimulating hormone secretion in the first half of the cycle that stimulates follicular growth in the ovary. After ovulation the ruptured follicle forms a [corpus luteum](https://www.yourhormones.info/glossary/c#corpus-luteum) that produces high levels of [progesterone](https://www.yourhormones.info/hormones/progesterone/). This inhibits the release of follicle stimulating hormone. Towards the end of the cycle the corpus luteum breaks down, progesterone production decreases and the next menstrual cycle begins when follicle stimulating hormone starts to rise again.

In men, the production of follicle stimulating hormone is regulated by the circulating levels of [testosterone](https://www.yourhormones.info/hormones/testosterone/) and inhibin, both produced by the testes. Follicle stimulating hormone regulates testosterone levels and when these rise they are sensed by nerve cells in the hypothalamus so that gonadotrophin-releasing hormone secretion and consequently follicle stimulating hormone is decreased. The opposite occurs when testosterone levels decrease. This is known as a '[negative feedback](https://www.yourhormones.info/glossary/n#negative-feedback)' control so that the production of testosterone remains steady. The production of inhibin is also controlled in a similar way but this is sensed by cells in the anterior pituitary gland rather than the hypothalamus.

**MELANOCYTE STIMULATING HORMONE**

Melanocyte-stimulating hormone describes a group of hormones produced by the pituitary gland, hypothalamus and skin cells. It is important for protecting the skin from UV rays, development of pigmentation and control of appetite. Alternative names for melanocyte-stimulating hormone are MSH; α-melanocyte-stimulating hormone; alpha-MSH; α-MSH; alpha-melanotropin; alpha-melanocortin; alpha-intermedin; melanophore-stimulating hormone.

Melanocyte-stimulating hormone is a collective name for a group of peptide hormones produced by the skin, [pituitary gland](https://www.yourhormones.info/glands/pituitary-gland/) and [hypothalamus](https://www.yourhormones.info/glands/hypothalamus/). In response to ultraviolet (UV) [radiation](https://www.yourhormones.info/glossary/r#radiation) its production by the skin and pituitary is enhanced, and this plays a key role in producing coloured [pigmentation](https://www.yourhormones.info/glossary/p#pigmentation) found in the skin, hair and eyes. It does this by inducing specialised skin cells called melanocytes to produce a pigment called melanin; melanin protects cells from [DNA](https://www.yourhormones.info/glossary/d#%3Ca%20href=)-(1)'>DNA damage, which can lead to skin cancer (melanoma).

Melanocyte-stimulating hormone is produced from the same precursor molecule as [adrenocorticotropic hormone](https://www.yourhormones.info/hormones/adrenocorticotropic-hormone/) called pro-opiomelanocortin (POMC).

Although named for its stimulatory effect on pigment cells, melanocyte-stimulating hormone produced in the hypothalamus can also suppress appetite by acting on [receptors](https://www.yourhormones.info/glossary/r#receptors) in the hypothalamus in the brain. This effect is enhanced by [leptin](https://www.yourhormones.info/hormones/leptin/), a hormone released from [fat](https://www.yourhormones.info/glands/adipose-tissue/) cells.

Melanocyte-stimulating hormone also affects a range of other processes in the body; it has anti-inflammatory effects, can influence the release of the hormone [aldosterone](https://www.yourhormones.info/hormones/aldosterone/), which controls salt and water balance in the body, and also has an effect on sexual behaviour.

**How is melanocyte-stimulating hormone controlled?**

Melanocyte-stimulating hormone secretion from the pituitary is increased by exposure to UV light. Unlike most hormones, melanocyte-stimulating hormone release is not thought to be controlled by a direct feedback mechanism.

**LIPOTROPIN**

**Lipotropin** is the name for two [hormones](https://en.wikipedia.org/wiki/Hormone) produced by the cleavage of [pro-opiomelanocortin](https://en.wikipedia.org/wiki/Pro-opiomelanocortin) (POMC). The [anterior pituitary gland](https://en.wikipedia.org/wiki/Anterior_pituitary) produces the [pro-hormone](https://en.wikipedia.org/wiki/Pro-hormone) POMC, which is then cleaved again to form [adrenocorticotropin](https://en.wikipedia.org/wiki/Adrenocorticotropic_hormone) (ACTH) and β-lipotropin (β-LPH).

β-Lipotropin is a 90-[amino acid](https://en.wikipedia.org/wiki/Amino_acid) [polypeptide](https://en.wikipedia.org/wiki/Polypeptide) that is the [carboxy-terminal](https://en.wikipedia.org/wiki/C-terminus%22%20%5Co%20%22C-terminus) fragment of POMC. It was initially reported to stimulate [melanocytes](https://en.wikipedia.org/wiki/Melanocytes%22%20%5Co%20%22Melanocytes) to produce [melanin](https://en.wikipedia.org/wiki/Melanin). It was also reported to perform lipid-mobilizing functions such as [lipolysis](https://en.wikipedia.org/wiki/Lipolysis%22%20%5Co%20%22Lipolysis) and [steroidogenesis](https://en.wikipedia.org/wiki/Steroidogenesis%22%20%5Co%20%22Steroidogenesis). However, no subsequent studies have been published that support these early findings and no receptor has been identified for β-lipotropin.

β-Lipotropin can be cleaved into smaller peptides. In humans, γ-lipotropin, [β-MSH](https://en.wikipedia.org/wiki/%CE%92-MSH), and [β-endorphin](https://en.wikipedia.org/wiki/%CE%92-endorphin), are all possible fragments of β-lipotropin.β-Lipotropin is the predominant opioid of the anterior human and rat pituitary gland. It is found in essentially equimolar concentrations to that of [corticotropin](https://en.wikipedia.org/wiki/Corticotropin). Evidence shows that β-Lipotropin is metabolized into [endorphins](https://en.wikipedia.org/wiki/Endorphins) that can greatly affect mood and behavior and is thus regarded as a [prohormone](https://en.wikipedia.org/wiki/Prohormone%22%20%5Co%20%22Prohormone).[[3]](https://en.wikipedia.org/wiki/Lipotropin#cite_note-3)

γ-Lipotropin

γ-lipotropin is the [amino-terminal](https://en.wikipedia.org/wiki/N-terminus) peptide fragment of β-lipotropin. In humans, it has 56 amino acids. Gamma lipotropin is identical to the first 56 amino acid sequences of β-lipotropin. It can be cleaved to [β-melanocyte stimulating hormone](https://en.wikipedia.org/wiki/Melanocyte-stimulating_hormone).

Use in sport

Lipotropin has also, under its alternate name AOD-9604 (Anti-Obesity Drug-9604),been connected with controversies in [Australian Rules Football](https://en.wikipedia.org/wiki/Australian_Rules_Football). Allegations have arisen around the use of the drug and its administration to players of the [Essendon Football Club](https://en.wikipedia.org/wiki/Essendon_Football_Club%22%20%5Co%20%22Essendon%20Football%20Club) as a supplement, including weekly administration to players in the 2012 season. The matters are currently under investigation due to the relationship between Lipotropin and growth hormones, as noted by club medical staff.

**1(b)**

Gonadotropin has been used to stimulate ovulation in clomiphene-resistant infertile women with polycystic ovary syndrome (PCOS), but it is associated with over stimulated cycles with the development of many follicles. The aim of the study was to evaluate the effectiveness and efficacy of letrozole and clomiphene citrate (CC) combined with human menopausal gonadotropin (HMG) in CC-resistant infertile women with PCOS.

Ovulation induction is the method for treating anovulatory infertility. For patients with hypogonadotrophic hypogonadism, the treatment involves administration of both FSH and LH, while HCG is injected for follicle rupture. Pulsatile GnRH has the same effectiveness as gonadotrophins and the advantage of the low multiple pregnancy rates. In polycystic ovary syndrome (PCOS), the first treatment choice is clomiphene citrate. With this drug, in properly selected patients, the cumulative pregnancy rate approaches that of normal women. Low-dose protocols of FSH are the second line of treatment, effective in inducing monofollicular development. Laparoscopic ovarian drilling can be an alternative but not as a first choice treatment in clomiphene-resistant patients. Other treatments, such as pulsatile GnRH and GnRH agonists, are hardly used today in PCOS. However, in obese women with PCOS, weight loss and exercise should be recommended as the first line of therapy. Newer agents including aromatase inhibitors and insulin sensitizers, although promising, need further evaluation.

**2)** **Causes of absent menstruation**

Natural causes most likely to cause amenorrhea include pregnancy, breast-feeding, and menopause. Lifestyle factors may include excessive exercise and stress. Also, having too little body fat or too much body fat may also delay or stop menstruation. Hormonal imbalances may cause amenorrhea.

Amenorrhea is defined as the absence of menstrual flow and is classified as either primary or secondary. After pregnancy is excluded by hCG testing, initial evaluation includes thyroid stimulating hormone (TSH), prolactin, follicle stimulating hormone (FSH), and luteinizing hormone (LH) tests.

## Diagnosis

### Initial Evaluation and Testing for Primary Amenorrhea

* Urinary or serum beta [human chorionic gonadotropin](https://arupconsult.com/content/hcg-testing) (hCG) to exclude pregnancy – if negative, proceed with physical and pelvic examination to rule out uterine absence (may require ultrasonography to confirm)
	+ Anatomic abnormality
		- Uterus present – consider transverse vaginal septum, imperforate hymen, abnormal cervical os, or other vaginal abnormality
		- Uterus absent – order free testosterone testing
			* Normal – consider chromosome analysis
			* High – androgen insensitivity confirmed
	+ Normal pelvic examination – order thyroid stimulating hormone (TSH), prolactin, follicle stimulating hormone (FSH), and luteinizing hormone (LH)
		- Elevated prolactin – MRI of head
		- Abnormal TSH – [thyroid disease](https://arupconsult.com/content/thyroid-disease)
		- Normal prolactin, TSH
			* LH and FSH elevated – primary ovarian failure confirmed
				+ Consider [X-chromosome abnormality](https://arupconsult.com/content/developmental-delay-dd-or-intellectual-disability-id-testing), Turner syndrome, FSH receptor deficiency, autoimmune gonadal destruction, [fragile X syndrome](https://arupconsult.com/content/developmental-delay-dd-or-intellectual-disability-id-testing)
			* LH and FSH normal – functional hypothalamic amenorrhea confirmed
				+ Consider eating disorder, stress/chronic illness, delayed puberty, gonadotropin-releasing hormone (GnRH) deficiency, [pituitary disorders](https://arupconsult.com/content/hypopituitarism), medication-induced
				+ If hypertensive, consider [17-hydroxylase deficiency](https://arupconsult.com/content/congenital-adrenal-hyperplasia)
				+ If [virilization](https://arupconsult.com/content/polycystic-ovarian-syndrome) present, order free testosterone

Elevated – order serum dehydroepiandrosterone sulfate (DHEA-S)

Elevated – consider androgen-secreting tumor

Not elevated – consider [polycystic ovarian syndrome](https://arupconsult.com/content/polycystic-ovarian-syndrome) (PCOS)

### Initial Evaluation and Testing for Secondary Amenorrhea

* Urinary or serum beta hCG to exclude pregnancy
	+ If negative pregnancy test, measure prolactin, LH/FSH, TSH
	+ Abnormal TSH – thyroid disease
	+ Normal prolactin, low/normal LH/FSH, normal TSH, no hirsutism
		- Order serum estradiol
			* Normal – hypothalamic dysfunction; consider testing for fragile X syndrome
			* Low – pituitary or hypothalamic abnormality
		- Consider eating disorder, excessive exercise
	+ Normal prolactin, high LH, normal/low FSH, hirsutism, virilization, acne
		- Order free testosterone, DHEA-S
			* Elevated free testosterone (high) – rule out tumor with pelvic ultrasound or abdominal CT
			* Elevated free testosterone (moderate) – ovarian hyperandrogenism (PCOS) confirmed
			* Elevated DHEA-S (high) – rule out adrenal tumor with adrenal CT
			* Elevated DHEA-S (moderate) – adrenal hyperandrogenism or PCOS
	+ Normal prolactin, high LH/FSH – ovarian failure (may represent menopause); consider chromosome analysis for X chromosome abnormalities
	+ High prolactin, normal LH/FSH
		- Order TSH
			* Normal – evaluate medication history
				+ Negative – CT/MRI, sella turcica
				+ Positive – discontinue medication
			* High TSH – primary hypothyroidism confirmed

2(b) **Menopause** is when a woman stops having periods and is no longer able to get pregnant naturally. Periods usually start to become less frequent over a few months or years before they stop altogether. Sometimes they can stop suddenly. Menopause occurs when a woman hasn’t menstruated in 12 consecutive months and can no longer become pregnant naturally. It usually begins between the ages of 45 and 55, but can develop before or after this age range. Menopause can cause uncomfortable [symptoms](https://www.healthline.com/health/menopause/symptoms-signs), such as hot flashes and weight gain. For most women, medical treatment isn’t needed for menopause.

**When does menopause begin and how long does it last?**

Most women first begin developing menopause symptoms about four years before their last period. Symptoms often continue until about four years after a woman’s last period. A small number of women experience menopause symptoms for up to a decade before menopause actually occurs, and [1 in 10](https://www.nhs.uk/conditions/menopause/symptoms/) women experience menopausal symptoms for 12 years following their last period. The median age for menopause is 51, though it may occur on average up to two years earlier for African-American and Latina women. More studies are needed to understand the onset of menopause for non-Caucasian women.

There are many factors that help determine when you’ll begin menopause, including genetics and ovary health. [Perimenopause](https://www.healthline.com/health/menopause/difference-perimenopause) occurs before menopause. Perimenopause is a time when your hormones begin to change in preparation for menopause. It can last anywhere from a few months to several years. Many women begin perimenopause some point after their mid-40s. Other women skip perimenopause and enter menopause suddenly.

About [1 percent](http://www.menopause.org/docs/default-source/2014/nams-recomm-for-clinical-care) of women begin menopause before the age of 40, which is called premature menopause or primary ovarian insufficiency. About [5 percent](http://www.menopause.org/docs/default-source/2014/nams-recomm-for-clinical-care) of women undergo menopause between the ages of 40 and 45. This is referred to as [early menopause](https://www.healthline.com/health/menopause/causes-early).

**Perimenopause vs. menopause vs. postmenopause**

During perimenopause, menstrual periods become irregular. Your periods may be late, or you may completely skip one or more periods. [Menstrual flow](https://www.healthline.com/health/menopause/perimenopause-periods) may also become heavier or lighter.

Menopause is defined as a lack of menstruation for one full year.

[Postmenopause](https://www.healthline.com/health/menopause/postmenopausal-health) refers to the years after menopause has occurred.

## What are the symptoms of menopause?

Every woman’s menopause experience is unique. Symptoms are usually more severe when menopause occurs suddenly or over a shorter period of time. Conditions that impact the health of the ovary, like [cancer](https://www.healthline.com/health/cancer/ovarian-cancer-early-signs) or [hysterectomy](https://www.healthline.com/health/hysterectomy), or certain lifestyle choices, like [smoking](https://www.healthline.com/health/smoking/effects-on-body), tend to increase the severity and duration of symptoms. Aside from menstruation changes, the symptoms of perimenopause, menopause, and postmenopause are generally the same. The most common early signs of perimenopause are:

* less frequent menstruation
* [heavier or lighter periods](https://www.healthline.com/health/menopause/perimenopause-periods) than you normally experience
* vasomotor symptoms, including [hot flashes](https://www.healthline.com/health/menopause/understanding-hot-flashes), [night sweats](https://www.healthline.com/health/menopause/hot-flashes-at-night), and [flushing](https://www.healthline.com/health/skin-blushing-flushing)

An estimated [75 percent](http://www.menopause.org/docs/default-source/2014/nams-recomm-for-clinical-care) of women experience hot flashes with menopause.

Other common symptoms of menopause include:

* [insomnia](https://www.healthline.com/health/menopause/menopause-and-insomnia)
* [vaginal dryness](https://www.healthline.com/symptom/vaginal-dryness)
* [weight gain](https://www.healthline.com/health/menopause/weight-gain)
* [depression](https://www.healthline.com/health/menopause/mental-health)
* [anxiety](https://www.healthline.com/health/anxiety/effects-on-body)
* [difficulty concentrating](https://www.healthline.com/health/menopause/menopause-brain-fog)
* memory problems
* [reduced libido](https://www.healthline.com/health/menopause/menopause-libido), or sex drive
* [dry skin](https://www.healthline.com/health/menopause/menopause-itching), mouth, and [eyes](https://www.healthline.com/health/menopause/menopause-and-dry-eyes)
* [increased urination](https://www.healthline.com/health/menopause/overactive-bladder-menopause)
* [sore or tender breasts](https://www.healthline.com/health/menopause/sore-breasts-menopause)
* [headaches](https://www.healthline.com/health/menopause-headaches)
* [racing heart](https://www.healthline.com/health/menopause/menopause-and-heart-palpitations)
* [urinary tract infections (UTIs)](https://www.healthline.com/health/urinary-tract-infection-adults)
* reduced muscle mass
* painful or [stiff joints](https://www.healthline.com/health/stiff-joints)
* [reduced bone mass](https://www.healthline.com/health/menopause/osteoporosis)
* [less full breasts](https://www.healthline.com/health/aging-changes-in-the-breast)
* hair thinning or [loss](https://www.healthline.com/health/menopause/hair-loss)
* [increased hair growth](https://www.healthline.com/health/excessive-or-unwanted-hair-in-women) on other areas of the body, such as the face, neck, chest, and upper back

**3(a) TESTS FOR MEN**

Male fertility requires that the testicles produce enough healthy sperm, and that the sperm is ejaculated effectively into the vagina and travels to the egg. Tests for male infertility attempt to determine whether any of these processes are impaired.

You may have a general physical exam, including examination of your genitals. Specific fertility tests may include:

* **Semen analysis.** Your doctor may ask for one or more semen specimens. Semen is generally obtained by masturbating or by interrupting intercourse and ejaculating your semen into a clean container. A lab analyzes your semen specimen. In some cases, urine may be tested for the presence of sperm.
* **Hormone testing.** You may have a blood test to determine your level of testosterone and other male hormones.
* **Genetic testing.** Genetic testing may be done to determine whether there's a genetic defect causing infertility.
* **Testicular biopsy.** In select cases, a testicular biopsy may be performed to identify abnormalities contributing to infertility or to retrieve sperm for assisted reproductive techniques, such as IVF.
* **Imaging.** In certain situations, imaging studies such as a brain MRI, transrectal or scrotal ultrasound, or a test of the vas deferens (vasography) may be performed.
* **Other specialty testing.** In rare cases, other tests to evaluate the quality of the sperm may be performed, such as evaluating a semen specimen for DNA abnormalities.

**TESTS FOR WOMEN**

Fertility for women relies on the ovaries releasing healthy eggs. The reproductive tract must allow an egg to pass into the fallopian tubes and join with sperm for fertilization. The fertilized egg must travel to the uterus and implant in the lining. Tests for female infertility try to find out if any of these processes are impaired.

A general physical exam, including a regular gynecological exam. Specific fertility tests may include:

* **Ovulation testing.** A blood test measures hormone levels to determine whether there is ovulation.
* **Hysterosalpingography.** Hysterosalpingography (his-tur-o-sal-ping-GOG-ruh-fee) evaluates the condition of the uterus and fallopian tubes and looks for blockages or other problems. X-ray contrast is injected into the uterus, and an X-ray is taken to determine if the cavity is normal and to see if the fluid spills out of the fallopian tubes.
* **Ovarian reserve testing.** This testing helps determine the quantity of the eggs available for ovulation. This approach often begins with hormone testing early in the menstrual cycle.
* **Other hormone testing.** Other hormone tests check levels of ovulatory hormones, as well as pituitary hormones that control reproductive processes.
* **Imaging tests.** Pelvic ultrasound looks for uterine or ovarian disease. Sometimes a sonohysterogram, also called a saline infusion sonogram, is used to see details inside the uterus that are not seen on a regular ultrasound.

Depending on the situation, rarely testing may include:

* **Hysteroscopy.** Depending on the symptoms, the doctor may request a hysteroscopy to look for uterine disease. During the procedure, the doctor inserts a thin, lighted device through your cervix into your uterus to view any potential abnormalities.
* **Laparoscopy.** This minimally invasive surgery involves making a small incision beneath the navel and inserting a thin viewing device to examine the fallopian tubes, ovaries and uterus. A laparoscopy may identify endometriosis, scarring, blockages or irregularities of the fallopian tubes, and problems with the ovaries and uterus.

**3(b) Treatment**

Infertility treatment depends on:

* What's causing the infertility
* How long you've been infertile
* Your age and your partner's age
* Personal preferences

Some causes of infertility can't be corrected.

In cases where spontaneous pregnancy doesn't happen, couples can often still achieve a pregnancy through use of assisted reproductive technology. Infertility treatment may involve significant financial, physical, psychological and time commitments.

### Treatment for men

Men's treatment for general sexual problems or lack of healthy sperm may include:

* **Changing lifestyle factors.** Improving lifestyle and certain behaviors can improve chances for pregnancy, including discontinuing select medications, reducing or eliminating harmful substances, improving frequency and timing of intercourse, exercising regularly, and optimizing other factors that may otherwise impair fertility.
* **Medications.** Certain medications may improve sperm count and likelihood for achieving a successful pregnancy. These medicines may increase testicular function, including sperm production and quality.
* **Surgery.** For some conditions, surgery may be able to reverse a sperm blockage and restore fertility. In other cases, surgically repairing a varicocele may improve overall chances for pregnancy.
* **Sperm retrieval.** These techniques obtain sperm when ejaculation is a problem or when no sperm are present in the ejaculated fluid. They may also be used in cases in which assisted reproductive techniques are planned and sperm counts are low or otherwise abnormal.

### Treatment for women

Some women need only one or two therapies to improve fertility. Other women may need several different types of treatment to achieve pregnancy.

* **Stimulating ovulation with fertility drugs.** Fertility drugs are the main treatment for women who are infertile due to ovulation disorders. These medications regulate or induce ovulation. Talk with your doctor about fertility drug options — including the benefits and risks of each type.
* **Intrauterine insemination (IUI).** During IUI, healthy sperm are placed directly in the uterus around the time the ovary releases one or more eggs to be fertilized. Depending on the reasons for infertility, the timing of IUI can be coordinated with your normal cycle or with fertility medications.
* **Surgery to restore fertility.** Uterine problems such as endometrial polyps, a uterine septum, intrauterine scar tissue and some fibroids can be treated with hysteroscopic surgery. Endometriosis, pelvic adhesions, and larger fibroids may require laparoscopic surgery or surgery with a larger incision of the abdomen.