Preface

Dear student,

Before you lies the booklet for Theme 4 of Semester 1 of Year 1 Medicine at the University of Manchester. Slim Academy has summarized the most important study material for you, so that you can study as pleasantly as possible. We wish you the best of luck with your studies!

Quality
Because we want to keep the quality of our summaries as high as possible, we are very interested in your feedback. For this reason, we will send you an email two weeks after you receive the summary, asking you to evaluate it. You would really help us by filling it out. If at any other time you have comments, tips, or suggestions for improvement, please email us at **s.dejong@slimacademy.nl** — we'll get to work on it right away!

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We wish you lots of success with your studies and your exams!

Team Slim Academy

P.S. This summary has been written based on the author's own interpretation. It remains a summary and should be seen as a supplement to the required study materials — not a replacement

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Post-natal growth and Puberty

Each chapter is based on the respective ILO. The ILO's can be found on Canvas. Because of copyright protection we will no longer copy the ILO's in this booklet. We recommend you find yourself the ILO's and keep them next to the summary.

Chapter 1 - Steroid and peptide hormones

Introduction

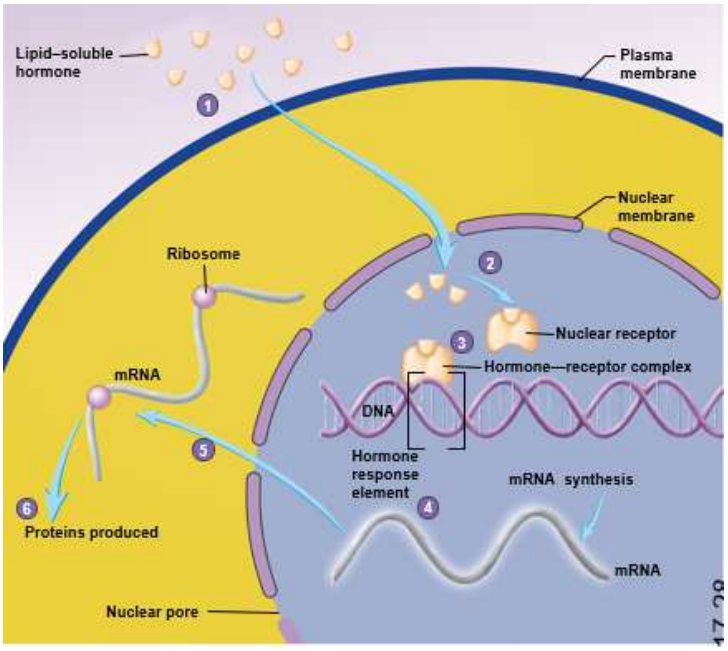
This chapter contrasts the mechanisms of action of steroid and peptide hormones.

Steroid hormones

Steroid hormones are derived from cholesterol, which is a lipid, and are hydrophobic. This means that they require a **transport protein** to move through the bloodstream but they can directly enter cells by passing through the phospholipid bilayer and they bind to intracellular nuclear receptors.

Mechanism

The steroid hormone crosses the phospholipid bilayer to enter the cell. It then binds to nuclear receptors inside the cell. Once bound, the chaperone proteins dissociate. A **dimer** forms and moves to the **nucleus** to enter it via a pore. The dimer binds to the hormone response element of the DNA in the nucleus, and the transcription rate of the **target gene** is altered.



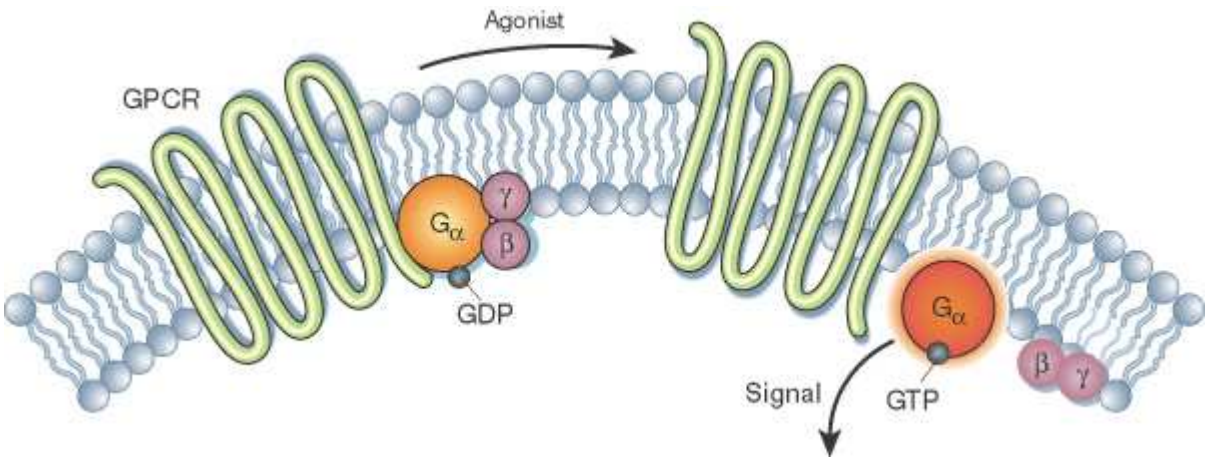
Mechanism of lipid-soluble hormones. *Source: https://quizlet.com*

Peptide hormones

In contrast to steroid hormones, protein-based hormones are hydrophilic and cannot cross the phospholipid bilayer. They must bind to extracellular receptors and usually need a **second messenger** in the cell to bring about its effects. Examples of protein-based hormones include FSH and LH which bind to GPCRs (G-protein coupled receptors) on granulosa and thecal cells respectively. These GPCRs use cAMP as a second messenger.

Mechanism

The hormone binds to the GPCR and activates it. It interacts with the **G_s protein of the receptor** and activates it by replacing GDP with GTP. The **alpha subunit** of the GPCR dissociates and binds to **adenylate cyclase** to activate it. Adenylate cyclase causes more cAMP to be produced. cAMP phosphorylates and activates protein kinase A which activates target genes in the DNA.



Mechanism of GPCRs. *Source: https://www.nature.com/scitable/topicpage/gpcr-14047471/*

Slim Summary!

- Steroid hormones are hydrophobic and bind to intracellular nuclear receptors, which allows a dimer to form and bind to the hormone response element of DNA to alter the transcription rate of the target gene;
- Peptide hormones are hydrophilic and bind to extracellular GPCRs which triggers a series of reactions where cAMP acts as a second messenger to cause the target gene in the DNA to be activated.

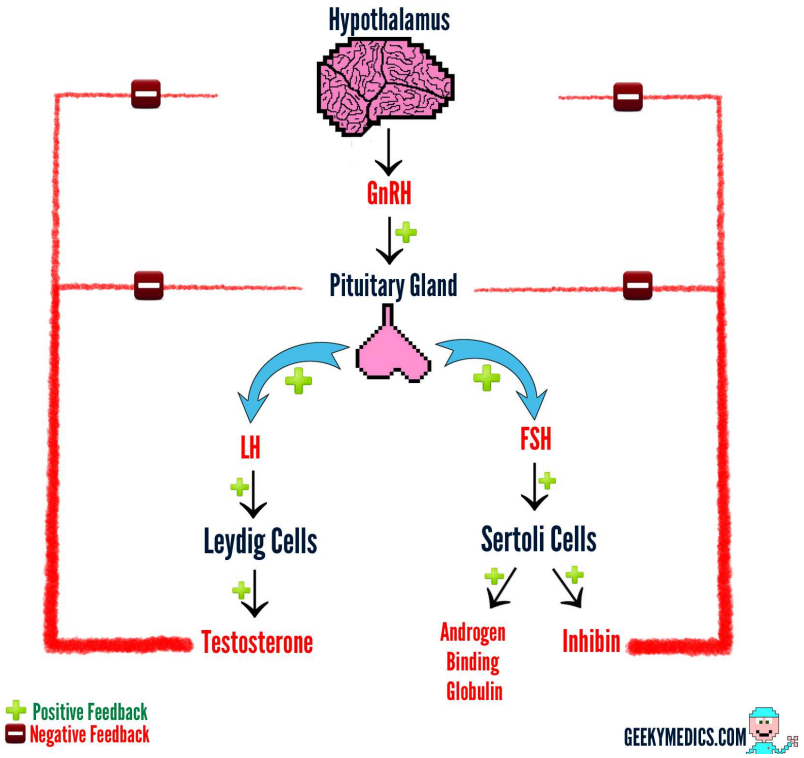
Chapter 2 - Release and effects of testosterone

Introduction
This chapter builds on the prior knowledge of steroid hormone action covered in previous themes and describes the sequence of events that allow testosterone to perform its function. This includes the trigger for its release, the cellular events that result from its binding to its cognate receptor, and its effects on specific body tissues. Testosterone is a steroid hormone and is the primary androgen which is produced by Leydig cells in the testes.

Hypothalamic-Pituitary-Gonadal Axis
This describes the sequence of events which leads to the release of testosterone. Firstly, fat cells in adipose tissue secrete the hormone leptin. Leptin stimulates the arcuate nucleus of the hypothalamus to secrete another hormone called kisspeptin which acts on the pre-optic nucleus of the hypothalamus. It stimulates the hypothalamus to produce and release GnRH (gonadotrophin releasing hormone) in a pulsatile manner.

GnRH acts on the anterior pituitary gland and stimulates it to release FSH (follicle stimulating hormone) and LH (luteinising hormone). FSH and LH move in the bloodstream to the testes. FSH acts on Sertoli cells in the testes, which allows the process of spermatogenesis to be facilitated, and this will be described further in Chapter 4. FSH is involved in the enlargement of the testes as it stimulates spermatogenesis. The Sertoli cells also secrete the protein hormone, inhibin, as a result of stimulation by FSH. LH acts on Leydig cells and stimulates steroidogenesis in them to produce testosterone. Cholesterol is first converted into pregnenolone, then into DHEA. DHEA is converted into androstenedione, and this is finally converted into testosterone.

The HPG axis also involves a negative feedback system in response to the release of testosterone and inhibin. Testosterone and inhibin act on the hypothalamus to inhibit the secretion of GnRH and consequently inhibit the release of LH and FSH by the anterior pituitary gland.



Male HPG-axis. *Source: https://geekymedics.com/how-the-gonadal-axis-works/*

Effects of testosterone and DHT
As testosterone is a steroid hormone, when it reaches target tissues it crosses the phospholipid bilayer to enter cells and binds to complementary nuclear receptors. Once bound, the chaperone proteins of the receptor dissociate. A dimer then forms and moves to the nucleus to enter it via a pore. The dimer binds to the steroid response element of the DNA in the nucleus, and the transcription rate of the target gene is altered.

Testosterone is produced by Leydig cells and can act on Sertoli cells in the testes to facilitate spermatogenesis. It can also act on distant organs to produce the common changes during puberty in males. It enhances libido (sex drive), increases sebum production (triggering acne breakouts), deepens the voice and also stimulates muscle and bone growth.

Testosterone can also be converted into another hormone, dihydrotestosterone (DHT), by the action of 5-alpha reductase. DHT is mainly synthesised in the liver, but it is produced in the Sertoli cells as well. It is a very potent androgen and cannot be converted into other hormones such as oestrogen. It has an important role in the differentiation of genitalia and also produces some of the secondary sex characteristics. For example, it is involved in the growth of hair post-puberty, and its levels can influence the development of androgenic alopecia. It is also involved in the enlargement of the prostate gland. DHT is also important for the maturation of sperm in the epididymis as they mature and become motile.

Slim Summary!

- The HPG axis regulates the release of testosterone and related hormones in a negative feedback system;
- Testosterone is a steroid hormone and is the primary androgen which is produced by Leydig cells in the testes which produces several sexual characteristics observed in puberty;
- DHT also is involved in stimulating the development of secondary sex characteristics and genitalia differentiation.

Chapter 3 - Oestrogen and testosterone

Introduction
This chapter explains the role of oestrogen and testosterone in the maturation of the male reproductive system.

Role of oestrogen/oestradiol
Testosterone can be converted to oestradiol by the action of aromatase enzymes in the testes. In males, the amount of oestradiol produced is approximately 20% of the oestradiol found in a non-pregnant woman. Although it is in smaller amounts, it still has important roles. It regulates libido and erectile function and stimulates the creation of an ideal environment for spermatogenesis. For example, it stimulates the reabsorption of luminal fluid in the epididymis and maintains the health of the seminiferous tubules. Oestrogen also has a major role in maintaining bone health and density by inhibiting bone resorption, so it is important that its levels are sufficient even in males. Oestrogen is also involved in a negative feedback system affecting the HPG axis. Therefore, it indirectly regulates testosterone production as it suppresses LH release from the anterior pituitary gland. If there is too much oestrogen, it may cause erectile dysfunction, infertility and enlarged breasts (gynecomastia).

Role of testosterone
As mentioned in the previous chapter, testosterone has several reproductive functions in males. It is produced by Leydig cells and can act on Sertoli cells in the testes to facilitate spermatogenesis. It can also act on distant organs to produce the common changes during puberty in males. It enhances libido (sex drive), increases sebum production (triggering acne breakouts), deepens the voice and also stimulates muscle and bone growth. During fetal development, testosterone aids in the development of reproductive organs, such as the prostate gland and seminal vesicle.

Slim Summary!

- Oestradiol still has important functions in males, such as regulating erectile function and the environment for spermatogenesis, and maintaining bone health, although it is present in much smaller amounts than in females;
- Testosterone is responsible for many primary and secondary sexual characteristics in males and also aids in the development of male reproductive organs in the male fetus.

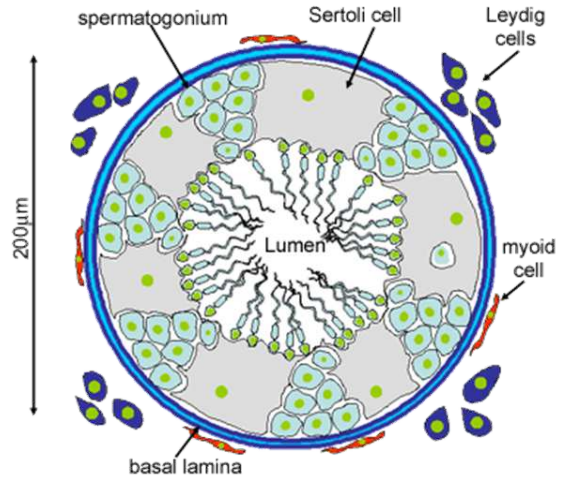
Chapter 4 - Spermatogenesis

Introduction
This chapter outlines the series of events that initiate and maintain spermatogenesis.

Pituitary hormone effects
As LH and FSH are released by the anterior pituitary gland, they act on Leydig and Sertoli cells respectively to stimulate testosterone production and spermatogenesis. Spermatogenesis occurs in the seminiferous tubules and is greatly facilitated by Sertoli cells. The action of FSH on Sertoli cells is particularly important for spermiogenesis (final stage of spermatogenesis where spermatids mature into spermatozoa) and the action of LH on Leydig cells to produce testosterone also aids spermatogenesis. This is because testosterone also helps to maintain the process of spermatogenesis.

Sertoli cells
These are the major support cells for spermatogenesis, found within the seminiferous tubules, and are often termed “nurse cells”. They form the blood testis barrier to protect the developing sperm from immune attack. This is a physical barrier of tight junctions between the Sertoli cells, which prevents the antigens on sperm from touching the lymphatics in the testes so that the immune system does not attack the developing sperm. They also transfer nutrients to developing sperm from the capillary system and remove waste (for example degenerating germ cells) by phagocytosis.

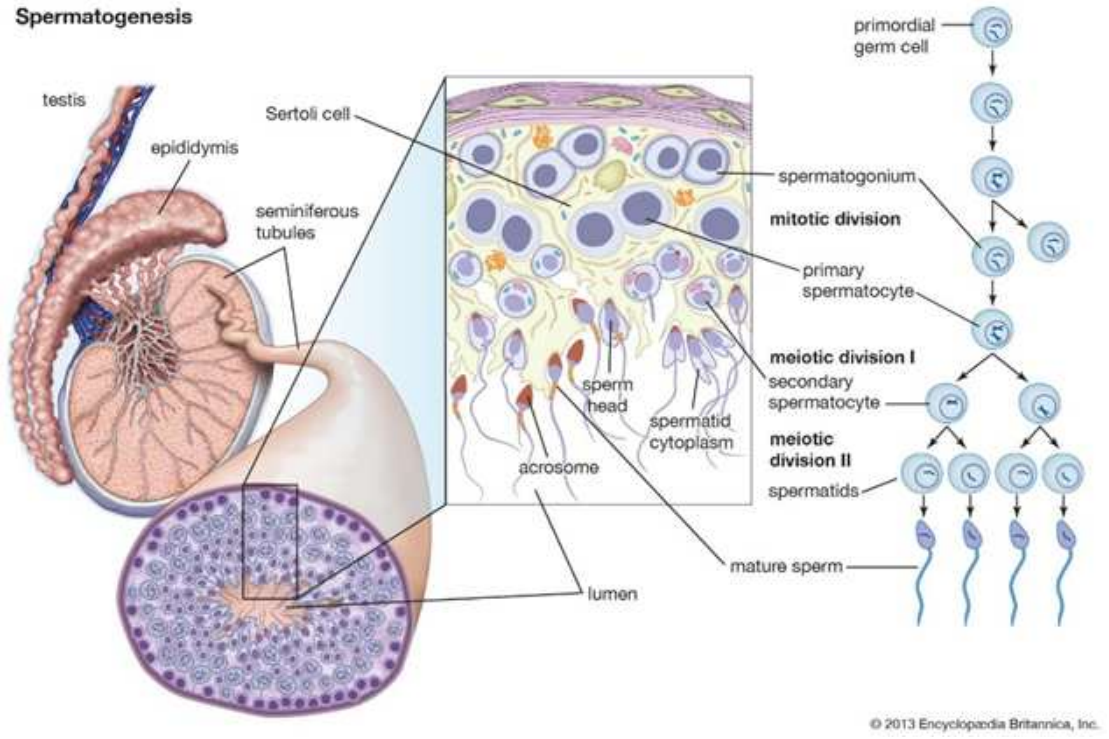
Other supporting cells
The Leydig cells, which are the interstitial cells between seminiferous tubules, produce testosterone, which binds to receptors on Sertoli cells to support spermatogenesis. There are also myoid cells found in the testes, which help to move maturing sperm through the seminiferous tubules to reach the rete testis via peristaltic contractions. This is required as the sperm are not yet motile. They mature and become motile in the epididymis.



Structures in and surrounding a seminiferous tubule. *Source: www.histology.leeds.ac.uk*

Spermatogenesis
During puberty, primordial germ cells migrate into the Sertoli cells of seminiferous tubules and become spermatogonia, which are stem cells. Type A spermatogonia are recycled and act as the reserve of stem cells. Type B spermatogonia are the ones that proliferate and undergo meiosis

to form primary spermatocytes under the influence of testosterone. Primary spermatocytes (diploid) undergo meiosis I to form secondary spermatocytes (haploid), then secondary spermatocytes undergo meiosis II to form spermatids (also haploid). Spermiogenesis is the final stage where spermatids mature into spermatozoa, which is facilitated by FSH. The mature spermatozoa are released into the seminiferous tubule lumen, and this process is called spermiation.



Spermatogenesis. *Source: www.britannica.com*

Slim Summary!

- The actions of hormones such as LH, FSH and testosterone stimulate and maintain spermatogenesis as they act on cells in the seminiferous tubules such as Leydig and Sertoli cells;
- Spermatogenesis is the process of spermatogonia undergoing meiosis and becoming spermatozoa.

Chapter 5 - Phases of linear growth and hormonal regulation

Introduction
This chapter will address the three phases of linear growth → infancy, childhood, and puberty which are distinct stages of physical development, each including unique hormonal regulation and growth patterns.

Infancy Phase
The infancy phase occurs from birth to 2–3 years of age and includes rapid growth → infants typically grow around 10 inches in the first year and 5 inches in the second. Growth during this stage is influenced by nutritional factors and by several hormones. Gonadotropin-releasing hormone (GnRH) is produced by the hypothalamus, and is low during infancy, leading to low activity of the reproductive system. Growth hormone (GH) is secreted by the anterior pituitary and stimulates overall body growth, including bones, muscles, and tissues. GH acts by signalling to the liver and other tissues to produce insulin-like growth factor 1 (IGF-1), which promotes bone elongation, especially in the epiphyseal (growth) plates, and supports cellular development in soft tissues. Thyroid hormones (T3 and T4) are produced by the thyroid gland and are important for regulating metabolism as well as increasing brain and central nervous system (CNS) development. They ensure that there is energy available for growth and play a crucial role in myelination and cognitive development. Thyroid hormone secretion is regulated by thyroid-stimulating hormone (TSH) from the anterior pituitary. During this phase, insulin also facilitates nutrient uptake and anabolic metabolism, ensuring that there is energy available for rapid growth.

Childhood Phase
The childhood phase begins from about 2–3 years until the start of puberty (approximately ages 10–12 in females and 9–14 in males). Growth during childhood is steady, averaging about 2–3 inches per year. GnRH secretion remains low during this stage, and there is low gonadal activity leading to a period of non-reproductive growth and development. Growth hormone is the dominant growth-regulating hormone, and maintains stable secretion levels to promote linear growth. GH stimulates hepatic and local production of IGF-1, which acts on growth plates to stimulate bone elongation, and on soft tissues to promote protein synthesis and muscle development. Thyroid hormones are essential during this phase, as they regulate metabolism and enhance the effect of GH and IGF-1 on bones and other tissues. Proper thyroid function ensures that there is proper skeletal development, CNS maturation, and energy balance. Nutritional factors play a key role in determining growth rates, as malnutrition can reduce GH and IGF-1 activity. Throughout childhood, the hypothalamic-pituitary-thyroid and hypothalamic-pituitary-growth hormone axes function to sustain growth, maintain normal metabolism, and prepare the body for the hormonal changes of puberty.

Puberty Phase
The puberty phase begins around ages 10–12 in girls and 12–14 in boys and continues until approximately 18 years of age. It marks the transition to physical and sexual maturity. This phase is characterized by growth spurt with individuals growing 3–5 inches per year at the peak. GnRH is released by the hypothalamus in a pulsatile manner, which stimulates the anterior pituitary to secrete luteinizing hormone and follicle- stimulating hormone which acts on the gonads. The release of these hormones in females will trigger the production of estrogen and progesterone, and in males they stimulate testosterone production. The increase in sex hormones will lead to the development of secondary sexual characteristics such as breast development, menstruation, facial hair growth, and voice deepening. The secretion of GH rises significantly during puberty and works in coordination with sex hormones to increase bone and muscle growth. This increase in GH stimulates greater production of IGF-1 from the liver which triggers further bone elongation at the epiphyseal plates, accelerating linear growth. Thyroid hormone activity also rises, increasing the metabolic rate and supporting the anabolic processes required for rapid growth, bone maturation, and energy metabolism. Together, GH, IGF-1, thyroid hormones, and

sex steroids produce the characteristic growth spurt of puberty. Growth plates gradually ossify and close under the influence of sex hormones, making the end of linear growth and final adult height

Slim Summary!

Infancy:
- Rapid growth and CNS development occur;
- Driven primarily by insulin, growth hormone (GH), insulin-like growth factor 1 (IGF-1), and thyroid hormones.

Childhood:
- GH and thyroid hormones maintain steady growth and regulate metabolism;
- GnRH (Gonadotropin-Releasing Hormone) levels remain low.

Puberty:
- Activation of the hypothalamic-pituitary-gonadal axis occurs through increased GnRH release;
- Leads to elevated levels of sex hormones, GH, IGF-1, and thyroid hormones;
- Results in accelerated growth and the appearance of secondary sexual characteristics.

Adulthood / End of Growth:
- Epiphyseal plate closure marks the end of linear growth, signaling that height increase has ceased.

Chapter 6 - Physiological changes during puberty

Introduction
This chapter will discuss the physiological events and physical changes that occur in puberty in males and females.

Puberty Overview
Puberty is the transition phase between childhood and adulthood, which is marked by sexual maturation and rapid physical growth. The onset of puberty is typically from ages, 9-14 in males and 8-13 in females → boys begin puberty a year later than girls. The first physical sign of puberty in females is breast budding, and in males, a testicular volume of 4mL. The hypothalamic-pituitary-gonadal (HPG) axis is the complex interaction between the hypothalamus, pituitary glands, and gonads which is central in puberty. Leptin which is released by adipose tissue within the periphery activates kisspeptin neurons within the arcuate and periventricular nuclei in the thalamus, stimulating GnRH release. GnRH is then released onto the anterior pituitary, causing the secretion of LH and FSH. In males, LH acts on leydig cells to produce testosterone and FSH acts on sertoli cells to begin spermatogenesis. The release of testosterone creates a negative feedback on the hypothalamus and pituitary while the sertoli cells produce inhibin which suppresses FSH release. In females, LH and FSH regulate ovarian follicle development and estrogen secretion, leading to ovulation and menstrual cycles.

Physical and Psychological Changes in Males
In males, puberty occurs between 9 and 14 years, and height increases most rapidly around age 12. Major physical changes include: growth of the testes and penis, appearance of pubic, axillary, and facial hair, broadening of the shoulders, increased muscle mass, deepening of the voice due to laryngeal enlargement, and increased oil and sweat gland activity (may cause acne). As spermatogenesis begins → spontaneous erections and nocturnal emissions can occur. Testosterone, which is produced by Leydig cells, is responsible for libido (sexual drive), voice deepening, muscle and bone growth, and sebum production. A by-product of testosterone called Dihydrotestosterone (DHT) is formed by the enzyme 5-alpha reductase. It is potent androgen that promotes facial and body hair growth, prostate enlargement, and external genital differentiation. Low DHT levels can lead to hypogonadism or underdeveloped genitalia. An enzyme, aromatase also produces Estradiol within the testes which is essential for spermatogenesis.

Physical and Psychological Changes in Females
In females, puberty typically occurs between ages 8 and 13, with growth spurts between 10 and 14 years. Key changes: breast development (thelarche), enlargement of the areola, growth of pubic and axillary hair, and activation of oil and apocrine sweat glands. The onset of menstruation or menarche occurs around age 12 to 13, which marks the start of reproductive capability. Estrogen promotes breast development, uterine growth, fat deposition, and bone maturation, while progesterone prepares and maintains the uterus for pregnancy. Psychological changes include heightened self-awareness, mood fluctuations, and the development of sexual identity.

Tanner Staging and Growth Assessment
Tanner staging describes the physical progression of puberty. In males, Stage 1 is prepubertal, Stage 2 involves scrotal and testicular enlargement with skin reddening, Stage 3 sees penile lengthening and testicular growth, Stage 4 involves further genital development and glans enlargement, and Stage 5 represents adult genitalia. In females, Stage 1 is prepubertal, Stage 2 involves breast budding, Stage 3 further enlargement of the breast and areola, Stage 4 formation of a secondary mound, and Stage 5 projection of the papilla with recession of the areola to

mature contours. Pubertal progression is often assessed using an orchidometer, which measures testicular volume: puberty begins at about 4 mL, a growth spurt occurs around 10 mL, and adult testicular size reaches approximately 25 mL. Bone age assessment can help determine pubertal timing and predict adult height.

Hormonal Regulation of Puberty
The hypothalamus releases GnRH in a pulsatile manner, stimulating the anterior pituitary to secrete LH and FSH. In males, LH stimulates Leydig cells to produce testosterone, while FSH acts on Sertoli cells to support spermatogenesis. Testosterone diffuses to Sertoli cells and is converted to DHT for stronger local androgenic effects. Both LH and FSH act through specific membrane receptors (LHR and FSHR). In females, LH and FSH regulate follicular maturation and estrogen production. Estrogens promote female secondary sexual characteristics, while progesterone supports endometrial preparation for pregnancy. Three major sex steroid hormone families are involved: estrogens, progestins (progesterone derivatives), and androgens.

Growth Hormone and Related Disorders
Growth hormone (GH), released from the anterior pituitary, plays a vital role in growth during puberty. GH secretion is stimulated by exercise, deep sleep, and ghrelin, and regulated by GHRH and somatostatin. GH promotes bone and soft tissue growth, acting synergistically with sex steroids. Excess GH before epiphyseal plate closure causes gigantism (excessive linear growth), while excess GH after closure results in acromegaly, characterized by enlargement of hands, feet, and facial features without increased height. GH stimulation tests evaluate pituitary function, and disorders such as Laron syndrome result from GH receptor insensitivity, leading to short stature despite normal GH levels.

Causes of Late or Absent Puberty
Delayed or absent puberty can arise from constitutional delay, GnRH deficiency (resulting in low or absent hypothalamic drive), or primary gonadal failure. Precocious puberty occurs before age 9 in boys and 8 in girls, while delayed puberty is diagnosed if no signs appear after age 14 in boys or 13 in girls. Evaluation includes hormonal assays, bone age assessment, and imaging to determine underlying endocrine or structural causes

Slim Summary!

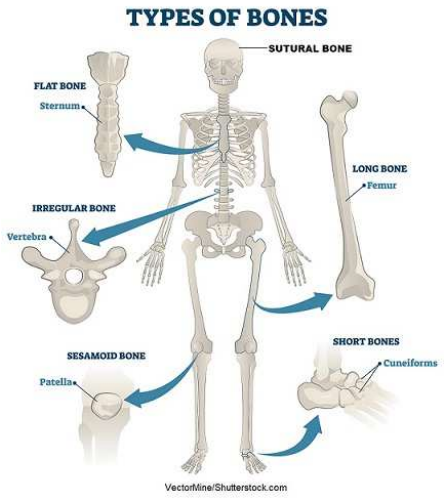
- Puberty is the transition from childhood to adulthood, driven by activation of the hypothalamic-pituitary-gonadal axis, leading to sex hormone production;
- Males develop under testosterone and DHT (genital growth, muscle mass, voice deepening), while females develop under estrogen and progesterone (breast development, menstruation, fat distribution);
- Growth hormone and sex steroids drive rapid bone and soft tissue growth, with assessment via Tanner staging, orchidometry, and bone age;
- Disorders include precocious or delayed puberty, requiring evaluation of hormonal and structural causes.

Chapter 7 – Structure of a Long Bone

Introduction
This chapter discusses the function, classification and microscopic structure of bones.

Function of Bone
Bone is a tissue that provides structural support, protection for vital organs, facilitates movement, mineral homeostasis (especially of calcium and phosphate), storage of triglycerides in marrow, and blood cell production through hematopoiesis.

Classification of Bones
Bones are classified according to shape and structure. Long bones, such as the femur, tibia, fibula, radius, and humerus, include a shaft and two ends and function as levers for movement. Short bones, including carpals and tarsals, provide stability with limited motion. Flat bones, like the skull, sternum, ribs, and scapulae, are thin and flattened, serving protective and attachment roles. Irregular bones, such as vertebrae, facial bones, and parts of the pelvis, have complex shapes for specific functions. Sesamoid bones, like the patella, are small, round bones embedded within tendons to reduce friction. Sutural bones are small bones located between cranial sutures.



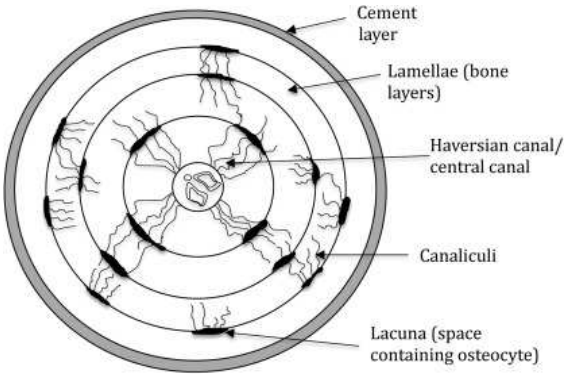
Types of bones. *Source: registerednursern.com*

Structure of a Long Bone
A long bone consists of the diaphysis (shaft), metaphysis, and epiphysis. The epiphyses are the distal and proximal ends, containing spongy bone with red marrow, and are covered by articular cartilage to reduce friction and absorb shock. The metaphysis connects the diaphysis and epiphysis and contains the epiphyseal plate, which allows longitudinal growth before becoming the epiphyseal line after maturation. The diaphysis is composed of compact bone surrounding the medullary cavity, which contains yellow marrow for fat storage. Compact bone provides rigidity and strength through Haversian systems that contain blood vessels and nerves. Spongy bone, composed of trabeculae, supports marrow and distributes forces efficiently. The periosteum is a tough connective tissue sheath covering the bone (except at joints) with an outer fibrous layer and an inner cellular layer containing osteoprogenitor cells. The endosteum lines the medullary cavity and contains osteoclasts involved in bone resorption.

Bone Matrix Composition and Cellular Components

The bone matrix is composed of organic and inorganic components. The inorganic portion, mainly hydroxyapatite (calcium phosphate crystals), provides hardness and rigidity, while type I collagen (accounting for 90% of bone collagen) is for flexibility and tensile strength. Bone cells are derived from mesenchymal and hematopoietic stem cells. Osteoprogenitor cells, found in the periosteum and endosteum, differentiate into osteoblasts, chondrocytes, fibroblasts, or adipocytes. Osteoblasts synthesize osteoid (unmineralized bone matrix) and are responsible for bone formation. Once osteoblasts are trapped in the matrix, they become osteocytes, mature cells residing in lacunae, which maintain bone tissue and regulate calcium homeostasis via canaliculi connections. Osteoclasts, derived from monocyte-macrophage lineages, are large multinucleated cells responsible for bone resorption and remodeling, playing a role in calcium balance.

Haversian System and Bone Organization
Compact bone is organized into structural units called osteons or Haversian systems. Each osteon consists of concentric lamellae (layers of bone matrix) surrounding a central canal containing blood vessels and nerves. Lacunae, which house osteocytes, are connected by canaliculi that facilitate nutrient and waste exchange. Spongy bone, found mainly in epiphyses and lining the medullary cavity, contains trabeculae that provide structural support and house bone marrow.



Haversian Systems. *Source: sciencedirect.com*

Cartilage and Its Role in Bone Development
Cartilage is an avascular connective tissue that cushions joints and provides a framework for bone formation. Chondrocytes, the main cartilage cells, are embedded in lacunae within the extracellular matrix. There are three main types: hyaline cartilage (found in the nose, trachea, and ends of long bones), elastic cartilage (in the ear and epiglottis), and fibrocartilage (in intervertebral discs and pubic symphysis). Cartilage acts as a precursor in endochondral ossification and contributes to smooth joint articulation as articular cartilage.

Slim Summary!

- Bone Functions: Support, protection, movement, mineral storage, fat storage, and blood cell production;
- Bone Types: Long, short, flat, irregular, sesamoid, and sutural bones;
- Long Bone Structure: Diaphysis, metaphysis (growth plate), epiphysis; periosteum and endosteum contain bone-forming/resorbing cells;
- Matrix & Cells: Hydroxyapatite + collagen; osteoblasts build, osteocytes maintain, osteoclasts resorb; cartilage cushions joints and guides bone formation.

Chapter 8 - Cellular Mechanisms Underpinning Growth

Introduction
This chapter discusses the cellular mechanisms underpinning growth and the role of different hormones which contribute to it.

Cellular Mechanisms Underpinning Growth
Growth is defined as an increase in size due to tissue increase, organ enlargement, and soft tissue expansion. Cellular growth occurs via hypertrophy (increase in cell size) and hyperplasia (increase in cell number), while differentiation allows cells to assume specialized functions. Cells respond to external signals through three mechanisms: paracrine (local cell-to-cell communication), endocrine (hormones acting on distant targets via the bloodstream), and autocrine (self-regulation through self-secreted signals). The hypothalamus plays a central role by releasing growth hormone–releasing hormone (GHRH), which stimulates somatotrophs in the anterior pituitary to release growth hormone (GH). GH then travels through the bloodstream to target tissues, promoting protein synthesis, lipolysis, bone growth, and soft tissue development.

Role of Growth Hormone (GH) and Insulin-like Growth Factor 1 (IGF-1)
In the liver, GH binds to growth hormone receptors (GHR), stimulating the production of IGF-1, which acts through tyrosine kinase receptors on bones, muscles, and adipose tissue to promote protein synthesis, cell proliferation, and tissue repair. IGF-1 also has paracrine effects on bone, it stimulates chondrocyte differentiation, osteoblast activity, bone growth, and maturation. GH secretion is pulsatile, with peak levels during deep sleep. Together, GH and IGF-1 promote longitudinal bone growth by ensuring chondrocyte proliferation within the epiphyseal plate. GH enhances lipolysis in adipocytes, increasing circulating fatty acids for energy, stimulates amino acid uptake for protein synthesis, and increases hepatic glucose output while decreasing glucose uptake in peripheral tissues(ensuring glucose is available for uptake). IGF-1 complements these actions by increasing protein synthesis, promoting lipogenesis, and facilitating glucose uptake into tissues, exerting insulin-like effects.

GH and IGF-1 Signaling Pathways
The GH receptor (GHR) is a cytokine receptor requiring activation of an associated enzyme to initiate signaling. Upon GH binding, the receptor dimerizes, binding first to a high-affinity and then to a low-affinity site, activating JAK-2). JAK-2 phosphorylates itself and tyrosine residues on the receptor, creating docking sites for the transcription factor STAT-5. STAT-5 becomes phosphorylated, dimerizes, and translocates to the nucleus, where it binds DNA response elements to regulate gene expression involved in growth and metabolism.

IGF-1 binds to its intrinsic tyrosine kinase receptor, which undergoes autophosphorylation and activates downstream pathways including PI3K–AKT (cell survival, metabolism, and protein synthesis) and RAS–RAF–MEK–MAPK (cell proliferation). IGF-1 within the circulation that must remain dormant is regulated by IGF-binding proteins (IGFBPs). When IGF-1 is ready to be in its active form, proteases cleave IGFBPs to release active IGF-1. The hypothalamus controls GH secretion through GHRH stimulation and somatostatin inhibition, with IGF-1 providing negative feedback to suppress GH and GHRH production.

Thyroid Hormone and Growth Regulation
Thyroid hormones, produced via the hypothalamic-pituitary-thyroid (HPT) axis, are essential for normal bone growth and maturation. The hypothalamus releases TRH, stimulating TSH release from the anterior pituitary, which acts on the thyroid gland to produce T3 (triiodothyronine) and T4 (thyroxine). Iodine is incorporated into tyrosine residues on thyroglobulin by thyroid

peroxidase, forming monoiodotyrosine (MIT) and diiodotyrosine (DIT), which combine to produce T3 and T4. These hormones are released into circulation after proteolysis of thyroglobulin. T3 and T4 enhance chondrocyte proliferation, hypertrophy, and differentiation, promoting ossification and mineralization of cartilage. They work synergistically with GH and IGF-1 to maximize growth plate activity and influence the timing of epiphyseal plate closure.

Cortisol and Growth Inhibition
Elevated cortisol levels, such as during chronic stress or Cushing's syndrome, suppress growth by reducing GH production from the anterior pituitary and inhibiting epiphyseal plate activity. Cortisol decreases chondrocyte proliferation and lessens tissue sensitivity to GH and IGF-1, impairing both cartilage and bone growth. It also inhibits hepatic production of IGF-1, resulting in stunted growth and decreased bone density.

Stages of Bone Remodelling
Osteoblasts secrete osteoid, an unmineralized organic matrix composed of collagen and proteins, which is the precursor of bone mineralization. Osteoid facilitates the deposition of calcium and phosphate, leading to osteogenesis and the calcification of bone from the osteoid precursor. Osteoclasts, derived from monocyte progenitor cells, are multinucleated cells responsible for bone resorption. In a process called osteolysis, osteoclasts secrete acids and lysosomal enzymes to dissolve mineralized bone and degrade the organic matrix. Bone resorption and formation are tightly coupled, ensuring the replacement of bone with equivalent strength and maintaining mineral homeostasis. When bones experience increased mechanical load, osteoblast activity increases, leading to bone thickening (Wolff's law). Conversely, lack of mechanical stress enhances osteoclast activity, resulting in bone resorption and reduced density.

Intramembranous Ossification
Intramembranous ossification is the direct transformation of mesenchymal tissue into bone without a cartilage intermediary, forming flat bones such as the skull and sternum. The process involves mesenchymal cells differentiating into osteoprogenitor cells and then osteoblasts, which secrete osteoid. The osteoid becomes mineralized, trapping osteoblasts as osteocytes. The calcified matrix forms trabeculae, giving rise to spongy bone, while surrounding mesenchyme develops into the periosteum. Subsequent remodeling converts portions of the spongy bone into compact bone.

Endochondral Ossification
Endochondral ossification is the process by which long bones develop from a preexisting cartilage model. Mesenchymal cells differentiate into chondroblasts that secrete a cartilage matrix, forming a hyaline cartilage model surrounded by the perichondrium. In response to bone morphogenetic proteins (BMPs) and growth factors, perichondrial cells differentiate into osteoblasts, forming a bone collar around the mid-diaphysis. Chondrocytes in the center hypertrophy, secrete alkaline phosphatase to calcify the matrix, and die, creating a central cavity. Blood vessels invade the space, bringing osteoprogenitor cells that form the primary ossification center. Osteoblasts deposit bone matrix on calcified cartilage remnants, forming spongy bone, while osteoclasts remodel it into a medullary cavity containing yellow marrow. Secondary ossification centers develop in the epiphyses, and articular cartilage remains on joint surfaces. The epiphyseal plate between the diaphysis and epiphysis allows longitudinal bone growth until it ossifies into the epiphyseal line after puberty.

Regulatory Molecules in Ossification
Key regulators of ossification include bone morphogenetic proteins (BMPs), which induce mesenchymal differentiation into chondrocytes and osteoblasts; Indian hedgehog (IHH), which regulates chondrocyte proliferation and differentiation; and alkaline phosphatase, which promotes matrix calcification. These signaling molecules coordinate chondrocyte maturation, matrix mineralization, and vascular invasion.

Epiphyseal Plate Structure and Function
The epiphyseal (growth) plate lies between the epiphysis and diaphysis and consists of several distinct zones. The reserve cartilage zone contains inactive chondrocytes serving as a reservoir. The proliferative zone features rapidly dividing chondrocytes producing type II collagen-rich matrix. The hypertrophic zone contains enlarged chondrocytes secreting enzymes to prepare the matrix for calcification. The calcification zone involves chondrocyte death and matrix mineralization, providing a scaffold for new bone formation. Finally, in the zone of reabsorption, near the diaphysis, blood vessels introduce osteoprogenitor cells, osteoblasts, and osteoclasts that replace calcified cartilage with bone. This coordinated process enables longitudinal growth until the epiphyseal plates close, marking skeletal maturity.

Estrogen and Epiphyseal Plate Closure
Estrogen, derived from the aromatization of testosterone, plays a crucial role in growth plate closure at the end of puberty. Estrogen diffuses into chondrocytes of the reserve zone, binding to cytoplasmic estrogen receptors that are initially complexed with chaperone proteins. Upon hormone binding, these complexes dissociate, allowing estrogen-receptor complexes to enter the nucleus and bind to DNA response elements, regulating genes involved in apoptosis of progenitor chondrocytes. This stops chondrocyte proliferation and closes the epiphyseal plates, marking the end of longitudinal bone growth.

Slim Summary!

- **Bone remodelling** continuously replaces old bone with new tissue via coordinated osteoblast and osteoclast activity, maintaining strength, mineral balance, and adapting to mechanical stress;
- **Ossification and growth** occur through intramembranous (flat bones) and endochondral (long bones) processes, regulated by BMPs, IHH, and the epiphyseal plate zones;
- **GH and IGF-1** stimulate longitudinal bone growth, protein synthesis, and tissue repair via JAK-STAT and PI3K-AKT/MAPK pathways, with thyroid hormones enhancing growth plate activity;
- **Hormonal regulation of growth cessation** involves estrogen-induced epiphyseal plate closure and cortisol-mediated growth inhibition through suppression of GH, IGF-1, and chondrocyte proliferation.

Chapter 9 - Predicted Adult Height

Introduction
This chapter discusses how predicting adult height helps assess a child's growth potential based on genetics, hormones, and environmental factors. The Mid-Parental Height Formula offers a simple way to estimate expected adult stature.

Predicting Adult Height
A common method used to estimate a child's eventual adult height is the Mid-Parental Height Formula, which accounts for parental stature and average sex-based growth differences. This approach provides an approximate genetic potential for height, assuming normal health, nutrition, and hormonal development.

Mid-Parental Height Formula

- For boys:
  Predicted Adult Height = (Father's Height + Mother's Height + 13 cm) ÷ 2
- For girls:
  Predicted Adult Height = (Father's Height + Mother's Height – 13 cm) ÷ 2

The addition or subtraction of 13 cm (approximately 5 inches) adjusts for the average difference in height between adult males and females.

Example Calculation
If the father is 180 cm and the mother is 165 cm:

- For a boy:
  Predicted height = (180 + 165 + 13) ÷ 2 = 358 ÷ 2 = 179 cm
- For a girl:
  Predicted height = (180 + 165 – 13) ÷ 2 = 332 ÷ 2 = 166 cm

Target Centile Range
Because environmental and genetic factors influence growth, the predicted height should be interpreted as a range rather than an exact figure. The target centile range is typically calculated by adding and subtracting about 8.5 cm (3.5 inches) from the predicted adult height.

For example, if a boy's predicted adult height is 179 cm:

- Lower bound: 179 – 8.5 = 170.5 cm
- Upper bound: 179 + 8.5 = 187.5 cm
  Thus, his expected height range would be 170.5–187.5 cm, representing his genetic growth potential under normal conditions.

Differences in Growth and Growth Potential Between Males and Females
Males and females differ significantly in growth timing, hormonal influences, and final height outcomes, mainly due to variations in pubertal onset and the action of sex steroids on growth plates.

Growth in Females
Females typically begin puberty earlier than males , between ages 10 and 14, and experience a rapid growth spurt soon after onset. Estrogen accelerates chondrocyte maturation and

ossification of the epiphyseal plate, leading to earlier closure of growth plates. Consequently, females usually reach their adult height earlier (around 15–16 years old) and have a lower overall growth potential. Estrogen's strong effect on bone maturation also explains why girls tend to stop growing sooner than boys.

Growth in Males
Males enter puberty later, usually between 12 and 16 years, with their peak growth spurt occurring in mid to late puberty. Testosterone promotes longitudinal bone growth and increases muscle and bone mass, contributing to greater height and more robust skeletal development. The later fusion of growth plates in males allows for a longer growth period, resulting in a higher average adult height compared to females. Testosterone is eventually converted to estrogen via aromatase in growth plate chondrocytes, which triggers closure, but this process occurs later than in females, allowing for extended growth time.

In summary, males generally grow for a longer period and reach greater adult stature due to the combined effects of delayed puberty, prolonged growth plate activity, and androgen-driven skeletal development. Females, under stronger and earlier estrogen influence, mature more quickly and complete growth earlier.

Slim Summary!

- Mid-Parental Height Formula: Estimates genetic growth potential using parental heights, adjusted for sex differences (+13 cm for boys, –13 cm for girls);
- Target Range: Predicted height should be viewed as a range (±8.5 cm) due to genetic and environmental variability;
- Sex Differences: Females mature earlier with shorter growth duration due to estrogen, while males grow longer and taller under delayed testosterone and growth plate fusion.

Chapter 10 - Short Stature

Introduction
This chapter identifies the potential causes of short stature, the treatments for it, and the ethical issues associated with the treatments. Short stature is defined as a height of more than two standard deviations below the mean height for their age, sex, and population group.

Causes of Short Stature
There are several potential causes of short stature which may result from poor growth, late or absent puberty, or chromosomal syndromes. Poor growth may simply result from poor nutrition or being born small. This may lead to a constitutional delay of puberty and growth, which should only be temporary. Chronic illnesses such as coeliac disease or asthma, as well as metabolic bone diseases such as dysplasia can also lead to poor growth.

Hormone deficiencies can cause late or absent puberty. This could involve a gonadotrophin hormone deficiency or gonadal failure (hypogonadisms), meaning that hormones in the HPG axis would be affected, such as GnRH, FSH, LH and steroid hormones including testosterone. These hormones are essential for producing the sexual characteristics that develop in puberty, so if there is a deficiency in them puberty would be delayed, resulting in a short stature. For example, in hypergonadotropic hypogonadism, there is a high level of LH and FSH, but a low level of testosterone due to the gonads being unresponsive to LH and FSH. Contrastingly, in hypogonadotropic hypogonadism, there is low LH and FSH which leads to low testosterone, and this is caused by an issue in the hypothalamus or pituitary gland. FSH and LH levels can be tested to determine which diagnosis it is out of the two.

As well as pubertal hormones, hormones which affect growth directly can also be deficient. These include IGF-I, growth hormone, TSH, and thyroxine. The levels of these hormones would be measured in blood tests and compared to normal levels for a patient's age group. Deficiencies in thyroid hormone production would indicate hypothyroidism. In extreme cases, there may be a tumour near the hypothalamus, pituitary gland or thyroid gland which may cause hormone deficiencies.

There are also genetic causes of short stature. An example of this is **Turner syndrome**, which is the complete monosomy of the X chromosome. Females with Turner syndrome therefore only have a single X chromosome instead of two. Its characteristic features are short stature, a short and wide neck, low hairline and low-set ears. Turner syndrome is the only complete monosomy of a chromosome that is compatible with life.

Treatments and ethical considerations
The most common treatment option for short stature is hormone replacement therapy, including growth hormone therapy, and thyroid hormone replacement. The levels of pubertal and growth hormones can be tested for in blood tests and the relevant hormones which are deficient can be administered as pills, patches, gels, creams and injections. The side effects can vary between hormone therapies, and patients should always be well-informed about these before starting treatment.

In extreme cases, patients may undergo limb-lengthening surgery, which can involve placing metal implants in the legs to gradually lengthen the legs. There are obvious risks with these surgeries, such as nerve and muscle damage and infection.

Nutritional and lifestyle support is also important, especially if there are nutrient deficiencies. Children may face bullying due to their conditions and body acceptance should also be encouraged. With the use of hormone therapies and surgeries, patients may feel encouraged to continue taking these beyond what is needed if they wish to grow further and become taller. Therefore, ethical issues can arise in regard to when to stop treatment and giving the right advice to patients.

Slim Summary!

- Short stature can result from poor nutrition, delayed puberty, growth hormone deficiencies, and genetic conditions;
- Several treatments are available, such as hormone replacement therapy and surgery, which carry ethical implications, as well as nutritional and lifestyle support.

Chapter 11 - Psychosocial development of adolescents

Introduction
This chapter describes how the biopsychosocial model explains the interactions between the psychosocial development of adolescents (including the role of body image) and health and wellbeing.

Biological factors
Body image is very commonly influenced by the biological events which occur during puberty, including growth spurts and hormone changes. Hormone fluctuations also affect the mood of adolescents, and this influences their health and wellbeing. Physical features may cause adolescents to feel insecure, especially if they have a condition that may affect how they develop during puberty. They may be mocked by peers and develop low self-esteem, leading to psychological consequences as well.

Psychological factors
Self-esteem and stress are important psychological factors in the development of adolescents, as negative thoughts about body image can lead to worse consequences such as depression, anxiety and suicidal thoughts. Emotional state can be hard to regulate during puberty due to hormone fluctuations, so effective and healthy coping strategies should be developed to prevent psychological disorders.

Social factors
A person's social background and support network are significant factors in their development. Peers and family may make comments about an adolescent's physical features which can affect their body image, and these thoughts can be exacerbated by time spent on social media. Social context also affects a person's access to money and support and impacts their wellbeing.

All of these factors interact with each other. For example, the stress from negative body image can lead to coping strategies like overeating, which impacts physical health. The development of positive feedback loops is key for healthy psychosocial development, which involves positive interactions and engaging in activities which promote good self-esteem and body image.

Slim Summary!

- Biological, psychological and social factors all interact with each other and affect the body image and mental health of adolescents;
- Developing positive feedback loops is key for healthy psychosocial development and preventing low self-esteem and mental health disorders.

Chapter 12 - Shared decision-making

Introduction
This chapter discusses the continuum of shared decision making and how it can impact patient satisfaction and adherence.

The continuum of shared decision-making describes how responsibility for healthcare decisions shifts between patients and professionals, ranging from doctor-led to patient-led approaches. It exemplifies flexibility, adapting to each patient's needs according to their clinical situation, competence, with the goal of ensuring medical decisions are prioritized with both medical expertise and a patient's autonomy in mind.

At one end is the clinician-led (paternalistic) model, where the doctor makes decisions on a patient's behalf which is appropriate in emergencies or when a patient lacks capacity. However, this approach may limit patient involvement and reduce patient satisfaction of adherence to treatments if the patient feels excluded.

In the middle lies shared decision-making (SDM), where clinicians and patients collaborate. The professional explains diagnoses, options, and risks, while the patient shares their values and preferences. This approach improves satisfaction, trust, adherence, and outcomes by ensuring care aligns with personal goals. However, SDM can be time-consuming, requires effective communication, and depends on the patient's willingness and ability to engage.

At the opposite end is the patient-led (informed) model, where the clinician provides full information and the patient makes the final decision independently. This maximizes autonomy and empowerment, promoting ownership and motivation in self-care. Yet it may cause anxiety, decision fatigue, or choices that conflict with medical advice, potentially impacting outcomes.

In practice, decision-making moves along this continuum depending on age, capacity, health literacy, and urgency. Shared decision-making is often ideal in chronic care, while clinician-led approaches suit emergencies.

Slim Summary!

- The continuum supports patient-centered care, combining professional expertise with the patient's perspective;
- It promotes flexibility and respect for autonomy, aiming for informed, ethical, and collaborative healthcare decisions that best serve each individual.

Chapter 13 - GMC Guidance: 0-18 years: guidance for all doctors

Introduction
This chapter discusses the framework of The GMC's *0–18 years: guidance for all doctors* which describes a continuum of decision-making that prioritizes both, a child's autonomy and a doctor's duty to act in their patients best interest.

The text addresses parental responsibility from birth to 18 years, allowing parents and legal guardians to make healthcare decisions on behalf of their child. However, as children grow, they should be more involved in decision making according to their maturity and capacity. Doctors must consider both the opinions of the parents as well as the child's emerging independence.

For children under 16, their ability to consent and make an informed choice is assessed through **Gillick Competence**. A child is competent if they understand the purpose and benefits of treatment, as well as recognize potential risks and consequences, and can weigh these options maturely and independently. A Gillick-competent child may consent to treatment without parental approval. If such a child refuses treatment, doctors must carefully balance the harm of overriding that refusal against the benefits of intervention, always acting in the child's best interests.

The **Fraser guidelines** apply specifically to contraceptive advice and treatment for those under 16. A doctor can provide contraception without parental consent if the young person fully understands the implications, cannot be persuaded to involve their parents, and would be at risk of harm to their physical or mental health without it. The doctor must be confident that this action serves the patient's best interests. If there is evidence that a young person is being coerced or influenced, confidentiality can be broken to protect their welfare.

In **emergencies**, decisions must prioritize the child's welfare. If immediate treatment is necessary and the child lacks capacity, consent can be given by a parent, guardian, or authorized agency. If none are available, healthcare professionals have both the authority and duty to act to preserve life or prevent serious harm, following the principle of beneficence.

The GMC guidance is grounded in **NHS core values**. *Working together for patients* emphasizes collaboration among children, families, and healthcare professionals, always placing the patient's needs first. *Respect and dignity* highlights valuing each child as an individual with unique priorities and limits, while *compassion* and *commitment to quality of care* stress providing empathetic, high-quality support that relieves suffering. *Improving lives* reflects the duty to enhance health and wellbeing through both everyday care and innovation, and *everyone counts* ensures fairness, inclusion, and responsible use of healthcare resources for the benefit of all.

Slim Summary!

- The GMC's continuum of decision-making recognizes children's evolving autonomy while ensuring their welfare remains central;
- Gillick competence: Children under 16 can consent to treatment if they fully understand the purpose, benefits, and risks, and can make independent, informed decisions in their best interests;
- Fraser guidelines: Doctors may provide contraception without parental consent if the young person understands the implications, refuses parental involvement, and would face harm without it, ensuring the decision protects their welfare.

Chapter 14 - Gillick competence

Introduction
This chapter explains the concept of Gillick competence and its assessment. This concept first came about in the early 1980s when a mother, Victoria Gillick, challenged the Department of Health guidelines enabling doctors to provide contraceptive advice and treatment to girls under 16 without their parents' knowledge. The criteria for Gillick competence as an assessment to determine if children had capacity to make medical decisions were consequently introduced.

Gillick competence
Gillick competence is a legal term used in England and Wales to determine whether a child under 16 years old can consent to receiving a medical treatment without the consent or permission of their parents. As opposed to adults (who are assumed to have capacity), children under 16 years old are usually under the responsibility of their parents when it comes to making medical decisions. Therefore, if a child wishes to receive a treatment without parental agreement and consent, they must first be assessed for capacity. If the child is deemed to have capacity, they will be able to receive the treatment. Health professionals should still encourage the child to inform their parents or get consent to inform them on their behalf, but if they have capacity and refuse then their decision should be respected. Any safeguarding concerns should also be raised if the child is believed to be in a dangerous situation.

Assessment of capacity
To be Gillick competent, a child under 16 must have sufficient understanding and intelligence to fully understand what is involved in the treatment, including the purpose, nature, effects, chances of success, and availability of other options. In a capacity assessment, they must be able to understand the information, then retain it and weigh it up to make an informed decision. They must also be able to communicate their decision.

Slim Summary!

- Gillick competence determines whether a child under 16 years old can consent to receiving a medical treatment without the consent of their parents;
- Their capacity must be assessed and if they are Gillick competent they are able to consent to a treatment without parental consent.

Chapter 15 - Clinical care for transitioning

Introduction
This chapter outlines the clinical care for a young person who is transitioning under the NHS, including the ethical and legal considerations.

General care structure
When a young person presents to their GP with concerns about gender, they are referred to the children and young person's (CYP) gender identity services, where they may receive appropriate consultations and treatment. Once they turn 18, they are transferred to the adult waiting list and, under the adult gender identity services, they may start or continue treatment after assessment and diagnosis. Depending on the age of the patient at referral and other factors, a patient may not start treatment until their 20s.

Key terms

Gender incongruence – characterised by marked and persistent incongruence between an individual's experienced gender and assigned gender.

Gender dysphoria – Psychological and physiological discomfort or distress caused by a discrepancy between a person's gender identity, their sex assigned at birth , and their primary or secondary sex characteristics.

Gender affirming hormone treatments
Patients may be started on hormone treatments, which would either be feminising or masculinising. Feminising hormone treatment would involve taking oestradiol/oestrogen and masculinising would involve taking testosterone. These could be administered as patches, tablets, gels or injections, and it may be several months or years before the maximum effect is reached. GnRH analogues, which cause a puberty blockade, used to be prescribed, but their use has now been restricted following the Cass Review, and they are only used by patients that were already taking them. GnRH analogues are, however, still used to treat other conditions such as endometriosis and prostate cancer.

Other treatments on NHS
Other treatments apart from hormones include voice therapy, hair reduction by laser or electrolysis and gamete storage. There are also surgeries available, such as chest contouring or mastectomy, phalloplasty (reconstruction of the penis), metoidioplasty (construction of a penis from an enlarged clitoris), and vaginoplasty.

Considerations
There are several ethical considerations when deciding on long term treatment for those experiencing gender dysphoria, especially when the individual is young and still under the responsibility of their parents. Allowing irreversible puberty is not a neutral act and there is also a lack of long-term data on treatments. Adolescents have a right to participate in their own decision making but often parents and guardians also play a role in decision making. It is important to consider the reversibility of treatments, as some, such as surgery, are irreversible and others are reversible. There are also social methods such as changing names and pronouns, which are reversible.

The Cass Review
This was an independent review into the CYP gender identity services, commissioned by NHS England and NHS Improvement in 2020. This report aimed to evaluate the existing services for transgender youth and those experiencing gender dysphoria. It led to closure of the gender identity development service (GIDS) in 2023 due to criticism about its use of interventions based

on poor evidence, which left young people at risk of poor mental health. For example, the use of puberty blockers was found to be poorly evidenced and as a result they were banned from being prescribed to under 18s. The development of regional healthcare centres instead to provide services was planned and these are beginning to open and expand across the country. These centres hope to promote a more holistic care service, addressing both gender dysphoria and mental health concerns.

Slim Summary!

- Clinical care for gender dysphoria and other concerns about gender can involve gender affirming hormone treatment and surgeries, but it may take a long time to receive treatment due to waiting lists;
- Regional healthcare centres are currently expanding across the UK and hope to deliver a more holistic care service following the Cass Review.

Afterword

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