Collegedunia NCERT Notes

The Ultimate 12th NCERT Revision Guide for Class 12 Biology (2024–26 / New NCERT)

Full-colour diagrams, formula boxes, NEET-ready summary tables

Chapter 7: Human Health and Disease

How to use these notes

This chapter blends the entire NCERT text with NEET-ready depth: pathogen tables you can quote verbatim, immunity flow diagrams, the HIV replication cycle, cancer biology and the four drug classes. Skim once for structure, then revisit each **Quick Tip**, **Common Mistake** and **Memory Aid** box before the exam — those are the high-yield checkpoints. Every NCERT figure cited in prose is reproduced from the source, with extra TikZ diagrams to make process flows visual.

Contents

1 Health and Disease: Setting the Stage

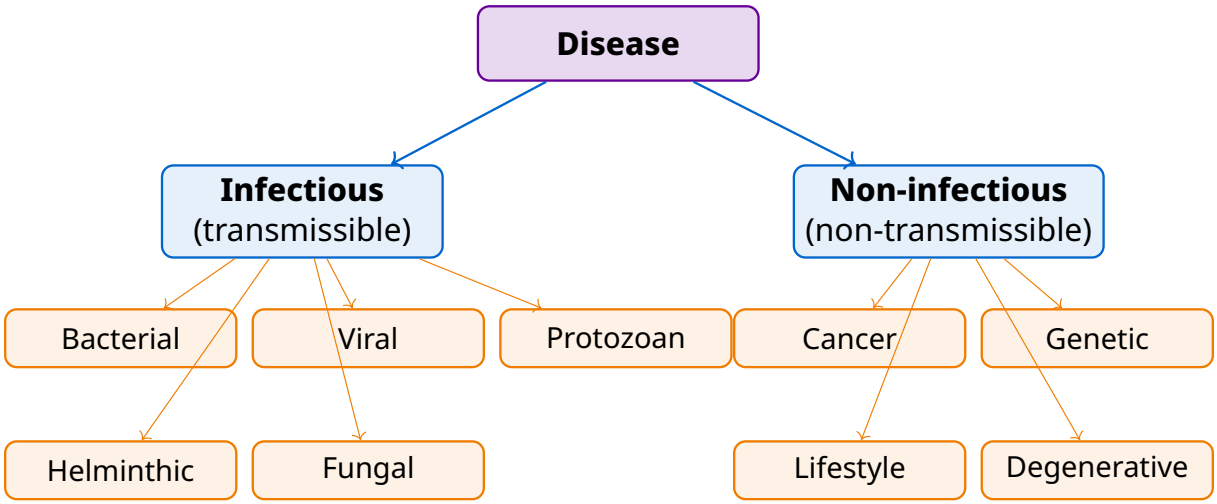
Health is more than "not being sick." The NCERT defines **health** as a state of complete physical, mental and social well-being. It is influenced by:

- **Genetic disorders** — defects inherited from parents (e.g. haemophilia, sickle-cell anaemia).
- **Infections** — caused by pathogens that invade the body.
- **Lifestyle** — diet, water, rest, exercise, hygiene, substance use.

Mind and body are connected. The nervous and endocrine systems modulate the immune system; chronic stress can therefore lower immunity. The classical Greek and Ayurvedic "humors" hypothesis was disproved by William Harvey (blood circulation) and the thermometer.

Disease in one line

A **disease** is any deviation from normal physical, mental or social well-being. Diseases are broadly classified as **infectious** (transmissible, caused by pathogens) and **non-infectious** (non-transmissible, e.g. cancer, diabetes, genetic disorders).



Real-World Application

WHO estimates more than 70% of global mortality now comes from **non-communicable diseases** — cardiovascular disease, cancer, chronic respiratory disease and diabetes — driven heavily by lifestyle. This is exactly the territory of Sections 7.4 and 7.5 of this chapter.

2 Common Diseases in Humans

Pathogens are disease-causing organisms drawn from bacteria, viruses, protozoa, helminths and fungi. To survive in the host, every pathogen must adapt to the host's environment (e.g. resisting stomach pH, evading immune cells). The same skeleton recurs for every disease: *causative agent* $\rightarrow$ *site of infection / mode of transmission* $\rightarrow$ *symptoms* $\rightarrow$ *diagnosis* $\rightarrow$ *prevention/treatment*.

2.1 Bacterial Diseases

Typhoid is caused by *Salmonella typhi*. It enters the small intestine through contaminated food/water and spreads to other organs via the blood. Symptoms include a sustained high fever of 39–40 °C, weakness, stomach pain, constipation, headache and loss of appetite. **Intestinal perforation** (and death) may occur in severe cases. Diagnosis is by the **Widal test**.

Mary Mallon ("Typhoid Mary"), a cook by profession in early 20th century New York, was a healthy *carrier* of *S. typhi* who spread typhoid through the food she prepared for many years — a classic example of a chronic asymptomatic carrier.

Pneumonia is caused by *Streptococcus pneumoniae* and *Haemophilus influenzae*, which infect the alveoli of the lungs. The alveoli fill with fluid, severely impairing respiration. Symptoms: fever, chills, cough and headache; lips and fingernails may turn gray to bluish in severe cases. Transmitted by inhaled droplets or shared utensils.

> **Quick Tip**
>
> Memorise both pneumonia organisms together — NEET often gives *Haemophilus influenzae* as a distractor, expecting students to wrongly choose "influenza virus".

Other notable bacterial diseases mentioned by NCERT: **dysentery, plague, diphtheria**.

2.2 Viral Diseases

Common cold is caused by **Rhinoviruses** (over 100 serotypes). They infect the nose and respiratory passage but *not the lungs*. Symptoms: nasal congestion and discharge, sore throat, hoarseness, cough, headache, tiredness — lasting 3–7 days. Transmission is by droplets and contaminated objects (pens, doorknobs, keyboards, etc.).

> **Common Mistake**
>
> Common cold is **not** caused by influenza virus. NCERT specifies Rhinovirus. Also, the cold virus does **not** infect the lungs — that statement is wrong in MCQs.

2.3 Protozoan Diseases — Malaria

Malaria is caused by *Plasmodium*. Different species cause different types:

- *P. vivax* — benign tertian malaria (every 48 h).
- *P. malariae* — quartan malaria (every 72 h).
- *P. falciparum* — **malignant malaria**, the most serious and often fatal.
- (NEET extra) *P. ovale* — mild tertian.

Vector: female *Anopheles* mosquito (males feed on plant juice). *Plasmodium* has two hosts: human (asexual cycle) and mosquito (sexual cycle).

Life cycle (Figure 7.1). Sporozoites injected by an infected female *Anopheles* bite enter the liver, multiply asexually, then invade red blood cells. When RBCs rupture, they release the toxin **haemozoin** which causes chills and high fever every 3–4 days. Gametocytes are picked up by another mosquito; in the mosquito gut, fertilisation produces zygotes which form sporozoites that migrate to the salivary glands, ready to infect the next human.

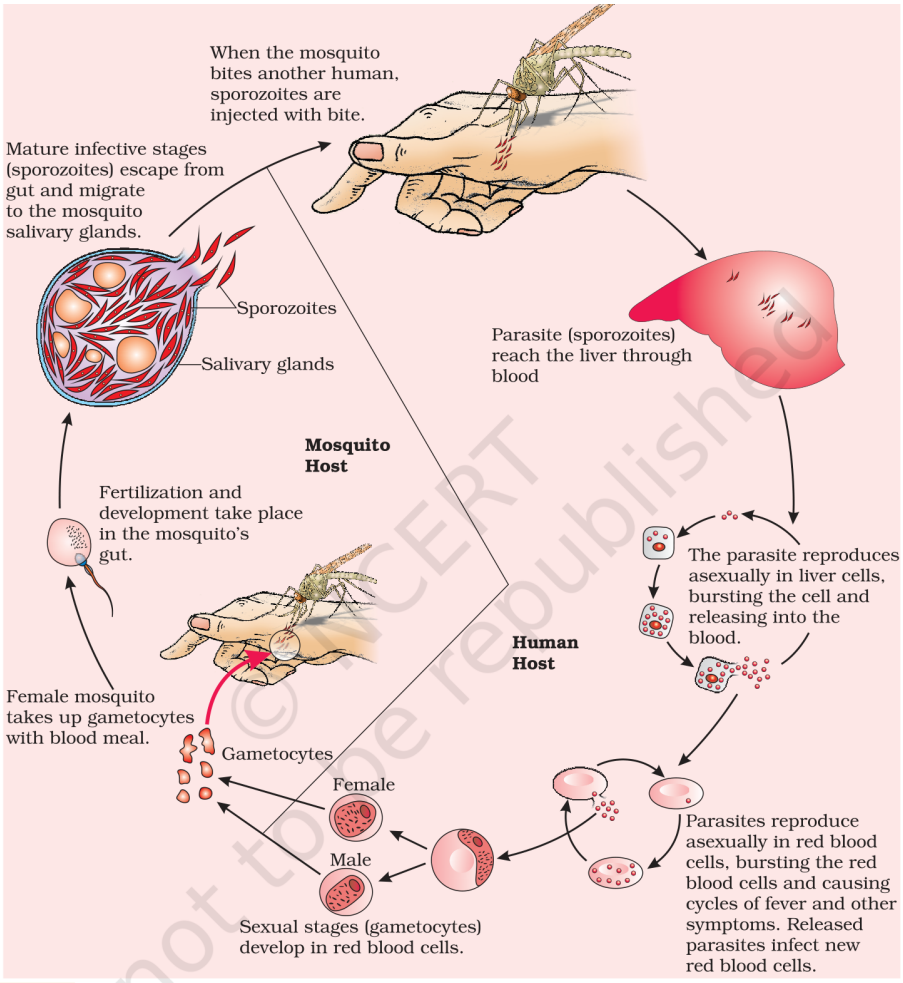


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Figure 7.1 Stages in the life cycle of *Plasmodium*

Source: NCERT Class 12 Biology, Chapter 7 — Fig. 7.1: Stages in the life cycle of Plasmodium — alternating between the human host (asexual cycle in liver and RBCs) and the mosquito

host (sexual cycle, sporogony in the gut).

Malaria at a glance

Pathogen: *Plasmodium* (P. vivax, P. malariae, P. falciparum, P. ovale)
Vector: Female *Anopheles* mosquito
Symptoms: Chills + recurrent high fever (haemozoin toxin); anaemia from RBC lysis
Hosts: Human (intermediate, asexual) + Mosquito (definitive, sexual)

Hosts of *Plasmodium*

"**Mosquito does Sex, Man does Math**" — **S**exual reproduction occurs in the **S**ipping mosquito; the asexual schizogony (multiplication) occurs in **M**an.

2.4 Other Protozoan and Helminthic Diseases

Amoebiasis (amoebic dysentery): *Entamoeba histolytica* parasitises the large intestine. Symptoms: constipation, abdominal pain and cramps, stools with excess mucus and blood clots. Transmission: contaminated food/water; **houseflies are mechanical carriers**.

Ascariasis: *Ascaris lumbricoides* (common round worm), an intestinal helminth. Symptoms: internal bleeding, muscular pain, fever, anaemia, blockage of the intestinal passage. Eggs pass in faeces and contaminate water, soil and vegetables.

Filariasis (Elephantiasis): *Wuchereria bancrofti* and *W. malayi*, filarial worms that cause chronic inflammation of the lymphatic vessels of the lower limbs (and sometimes the genital organs). Transmitted by female mosquito bites (*Culex* is the common vector — NEET extra). See Figure 7.2 below.

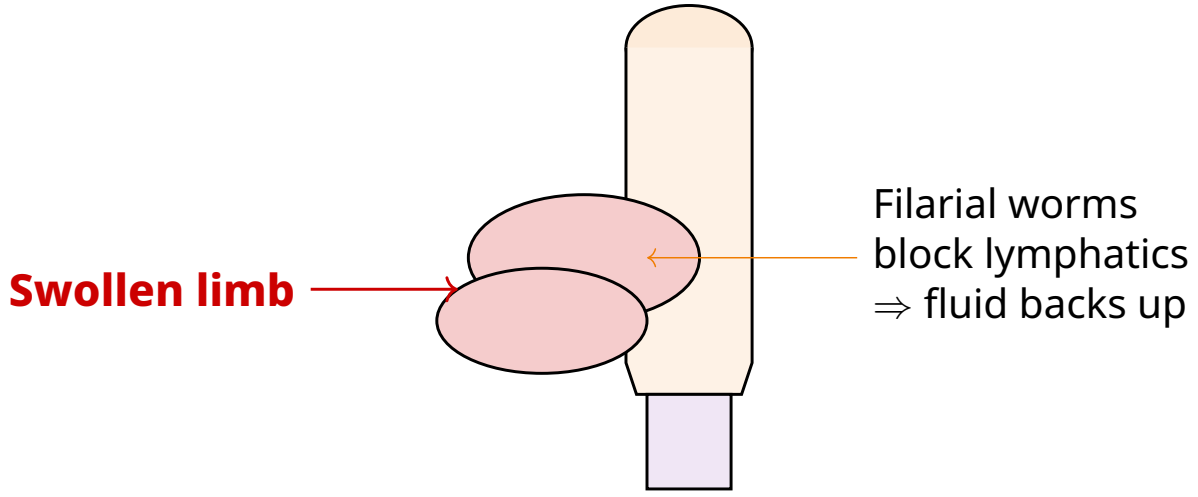


Fig. Elephantiasis: lower-limb lymphatic blockage

Ringworm (fungal, despite the name — no worm!): *Microsporum*, *Trichophyton* and *Epidermophyton*. Symptoms: dry, scaly lesions on skin, nails and scalp, with intense itching. The fungi thrive in heat and moisture (groin, between toes). Transmitted by soil or shared towels, clothes, combs.

Common Mistake

"**Ringworm**" is a **fungal** disease — not caused by a worm. The name describes the ring-shaped lesion, not the pathogen. Three fungal genera cause it; NEET routinely asks all three.

2.5 Pathogen Summary Table (NCERT + NEET)

Disease	Causative organism	Type / Vector	Key symptom / Diagnosis
Typhoid	*Salmonella typhi*	Bacterium	Sustained 39–40 °C fever, intestinal perforation; **Widal test**
Pneumonia	*Streptococcus pneumoniae*, *Haemophilus influenzae*	Bacterium	Alveoli fill with fluid; fever, chills, bluish lips/nails
Common cold	Rhinoviruses	Virus	Nasal discharge, sore throat; lasts 3–7 days; *lungs not infected*
Malaria	*Plasmodium falciparum* (and others)	Protozoan / female *Anopheles*	Chill + high fever every 3–4 days; haemozoin toxin
Amoebiasis	*Entamoeba histolytica*	Protozoan / housefly carrier	Constipation, mucus + blood in stool
Ascariasis	*Ascaris lumbricoides*	Helminth (round worm)	Internal bleeding, anaemia, gut blockage
Filariasis	*Wuchereria bancrofti*, *W. malayi*	Helminth / female *Culex*	Elephantiasis: chronic inflammation, lymphatic blockage
Ringworm	*Microsporum*, *Trichophyton*, *Epidermophyton*	Fungus	Dry scaly itchy lesions; warm moist body folds

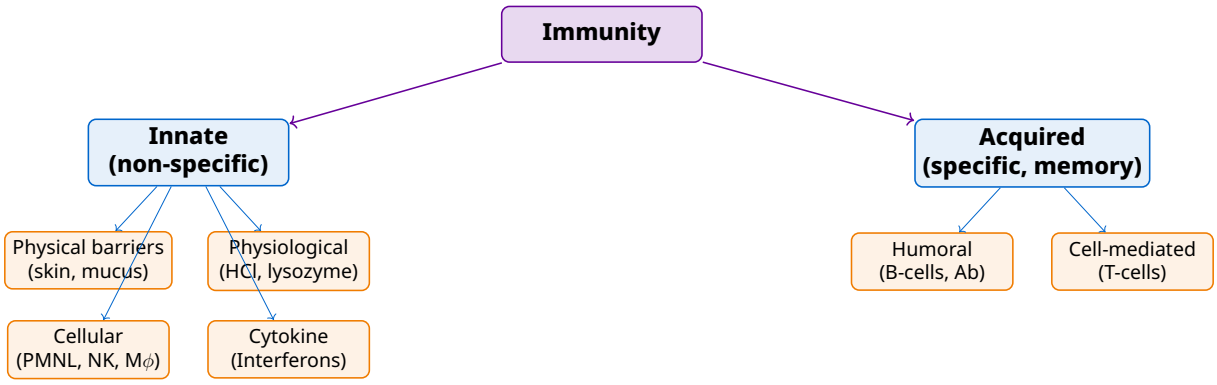
2.6 Prevention and Control

- **Personal hygiene** — clean body, clean drinking water, washed fruits and vegetables.
- **Public hygiene** — proper disposal of waste/excreta, periodic disinfection of water reservoirs, hygienic public catering.
- **Air-borne diseases** (cold, pneumonia) — avoid close contact with infected persons and their belongings.
- **Vector-borne diseases** (malaria, filariasis, dengue, chikungunya) — eliminate stagnant water, use mosquito nets, introduce larvivorous fish like *Gambusia*, spray insecticides, install wire mesh on doors/windows.
- **Vaccines and immunisation** — have eradicated smallpox and controlled polio, diphtheria, pneumonia and tetanus.
- **Antibiotics** — effective against bacterial infections (*not* viral ones).

> **Quick Tip**
>
> *Gambusia* is the **larvivorous fish** used to eat mosquito larvae — NEET asks this every 2–3 years. Don't confuse it with *Spirulina* (a food supplement, Chapter 8 territory).

3 Immunity

Immunity is the overall ability of the host to fight disease-causing organisms, conferred by the **immune system**. NCERT classifies immunity into two broad types — **innate** (non-specific, present at birth) and **acquired** (specific, develops after exposure).



3.1 Innate Immunity — Four Barriers

Innate immunity is **non-specific**, present from birth and acts through four lines of barriers:

1. **Physical barriers**: skin (main barrier); mucus coating of respiratory, GI and urogenital tracts traps microbes.
2. **Physiological barriers**: stomach acid (HCl), saliva (lysozyme), tears (lysozyme) prevent microbial growth.
3. **Cellular barriers**: **PMNL / neutrophils**, **monocytes** in blood; **macrophages** in tissues; **natural killer (NK) lymphocytes**. All phagocytose and destroy microbes.
4. **Cytokine barriers**: virus-infected cells secrete **interferons** that protect nearby uninfected cells from viral infection.

Four innate barriers

"Please Protect Cells from Cytokines"
Physical → **P**hysiological → **C**ellular → **C**ytokine

Innate vs Acquired in one line

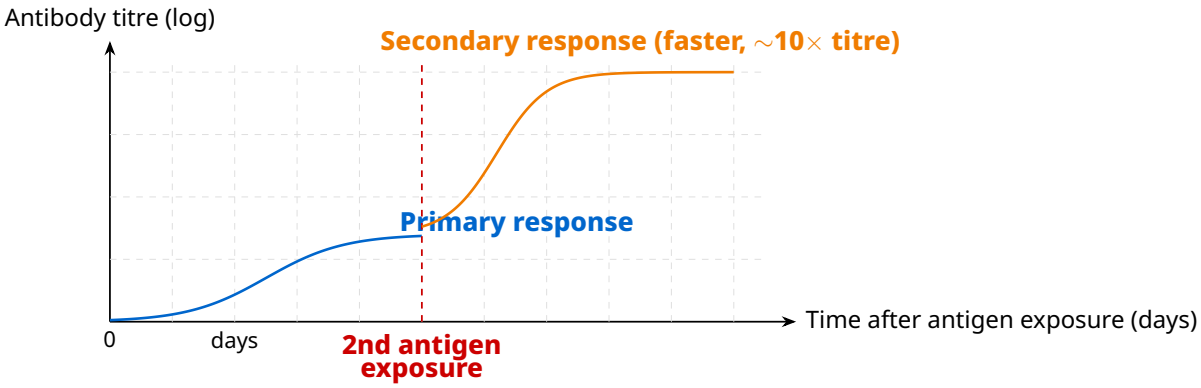
Innate is fast, broad, non-specific, no memory. **Acquired** is slow on first exposure, highly specific, has memory — so the second exposure is much faster and stronger.

3.2 Acquired Immunity — Primary and Secondary Response

Acquired immunity is **pathogen-specific** and shows **memory**. The first encounter produces a low-intensity **primary response**; the second encounter with the same pathogen triggers a much more intense **secondary (anamnestic) response**.

Two specialised lymphocytes carry this out:

- **B-lymphocytes** — produce **antibodies** (proteins in blood). They mediate **humoral** (antibody-mediated) immunity.
- **T-lymphocytes** — do not secrete antibodies; they help B-cells and directly mediate **cell-mediated immunity (CMI)**.



Antibody structure. Each antibody is a protein with four polypeptide chains: two

light (L) chains and two **heavy (H)** chains, written as H_2L_2. The variable region at the N-terminus of each arm forms the antigen-binding site (Fab); the constant region (Fc) is the stem.

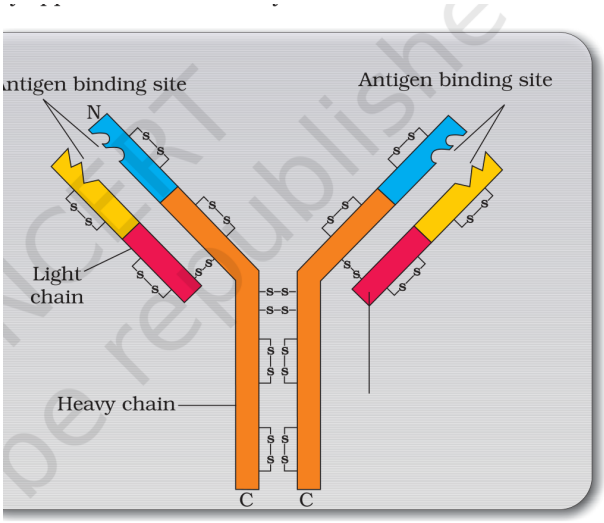


Figure 7.4 Structure of an antibody molecule

Source: NCERT Class 12 Biology, Chapter 7 — Fig. 7.4: Structure of an antibody molecule. Two light chains and two heavy chains (Y-shape, H_2L_2) joined by disulphide (S–S) bonds. The N-terminal tips (variable regions) form two antigen-binding sites.

NCERT names five immunoglobulin classes: **IgA, IgM, IgE, IgG** — and IgD is implied (NEET-relevant). Their roles are summarised below.

Class	Location / Form	Function (NEET extra)
IgG	Most abundant (~75%); blood, lymph	Crosses placenta; secondary response
IgM	Pentamer; blood (largest Ab)	First antibody made in primary response
IgA	Dimer; **colostrum**, saliva, tears, mucus	Passive immunity to newborn; mucosal defence
IgE	Trace; bound to mast cells	Allergy and parasitic infection
IgD	Surface of mature B-cells	Antigen receptor on B-cells

Antibody classes

"GAMED" — **G**eneral (IgG, most common), **A**irway/colostrum (IgA), **M**assive primary (IgM), **E**osinophilic/allergy (IgE), **D**etector on B-cell (IgD).

Acquired immunity: two arms

Humoral (AMI) — B-cells secrete antibodies into body fluids; deals with extracellular pathogens.
Cell-mediated (CMI) — T-cells kill infected/abnormal cells directly; deals with intracellular pathogens, cancer cells, and **transplant graft rejection**.

Graft rejection is the work of CMI. The body distinguishes "self" from "non-self" MHC markers; non-matching grafts are rejected. Hence tissue and blood group matching are essential before transplant, and recipients take **immunosuppressants** for life.

3.3 Active and Passive Immunity

Active immunity: the host produces its own antibodies after exposure to live, dead or antigenic material. Slow to develop but long-lasting. Induced by natural infection or by vaccination.

Passive immunity: ready-made antibodies are transferred to the host. Quick but short-lived (no memory).

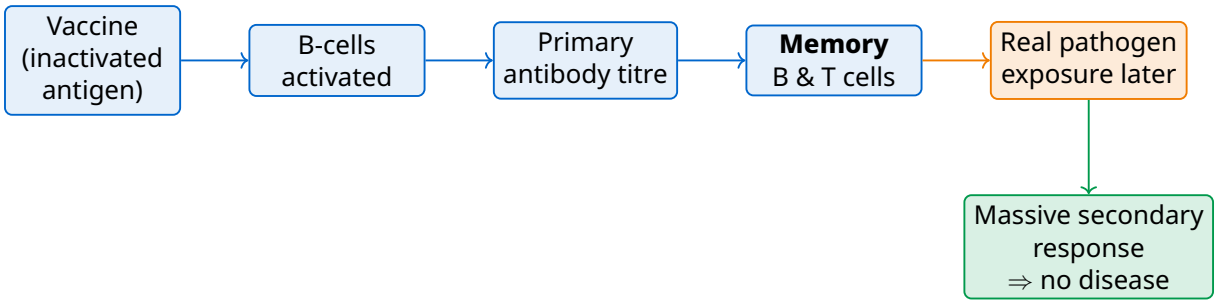
- **Natural passive**: IgG crosses the placenta (mother $\rightarrow$ foetus); IgA in colostrum (mother $\rightarrow$ newborn).
- **Artificial passive**: antitoxin injection for tetanus, anti-snake venom, monoclonal antibodies.

Active immunity	Passive immunity
Host produces its own antibodies	Ready-made antibodies supplied to host
Develops slowly (days–weeks)	Acts immediately
Long-lasting (memory cells formed)	Short-lived (no memory)
Examples: vaccination, recovery from infection	Examples: colostrum, antitoxin, anti-venom

3.4 Vaccination and Immunisation

Vaccination relies on the **memory** property of the immune system. A vaccine — antigenic proteins, or inactivated/weakened pathogen — is introduced. The body mounts a primary response and forms **memory B and T cells**. On real infection, these memory cells unleash a massive, fast secondary response that neutralises the pathogen before disease develops.

- For deadly infections needing rapid action (**tetanus, snakebite, rabies post-exposure**), preformed antibodies (antitoxin / antiserum) are injected. This is **passive immunisation**.
- **Recombinant DNA technology** produces large-scale, safe vaccines, e.g. **Hepatitis B vaccine from yeast**.



Real-World Application

The Hepatitis B vaccine produced from yeast (*Saccharomyces cerevisiae*) was the first commercially successful recombinant vaccine. It is now part of India's Universal Immunisation Programme (UIP).

3.5 Allergies

An **allergy** is an exaggerated response of the immune system to certain environmental antigens called **allergens** (dust mites, pollens, animal dander, certain foods). The antibodies involved are **IgE**. On exposure, allergens cross-link IgE bound to **mast cells**, releasing **histamine and serotonin** — producing sneezing, watery eyes, running nose, wheezing.

- **Diagnosis**: skin prick test — a small dose of suspected allergen is injected and the response watched.
- **Treatment**: anti-histamines, adrenaline, steroids.

Quick Tip

Hygiene hypothesis: children raised in too-clean "protected" environments don't train their immune system on the usual harmless microbes, and so overreact later to common allergens. This is why allergy and asthma are rising in metro cities of India (NCERT mentions this explicitly).

3.6 Auto-immunity

Auto-immune disease arises when the body fails to distinguish self from non-self and attacks its own cells, damaging tissues. The classic NCERT example is **rheumatoid arthritis** (joint synovial membrane attacked). Other examples (NEET extras): **Type 1 diabetes mellitus** (pancreatic β-cells), **multiple sclerosis** (myelin), **lupus erythematosus** (multi-organ), **myasthenia gravis** (acetylcholine receptors).

Common Mistake

Don't confuse **auto-immunity** (immune attacks self) with **immuno-deficiency** (immune system too weak — like AIDS). They are opposites: hyperactive vs deficient.

3.7 Immune System in the Body

The immune system consists of **lymphoid organs, tissues, cells** and **soluble molecules** (antibodies, cytokines, complement).

Primary lymphoid organs: lymphocytes originate *and mature into antigen-sensitive cells* here.

- **Bone marrow** — main lymphoid organ; produces all blood cells. Site of B-cell maturation.
- **Thymus** — lobed organ near the heart beneath the breastbone. *Large at birth, shrinks with age* (involutes after puberty). Site of T-cell maturation.

Secondary lymphoid organs — sites where mature lymphocytes meet antigens and proliferate:

- **Spleen** — bean-shaped; filters blood-borne microbes; reservoir of erythrocytes.
- **Lymph nodes** — trap antigens carried in lymph and tissue fluid.
- **Tonsils, Peyer's patches** (small intestine), **appendix**.
- **Mucosa-associated lymphoid tissue (MALT)** — lines respiratory, digestive and urogenital tracts; constitutes **about 50%** of the body's lymphoid tissue.

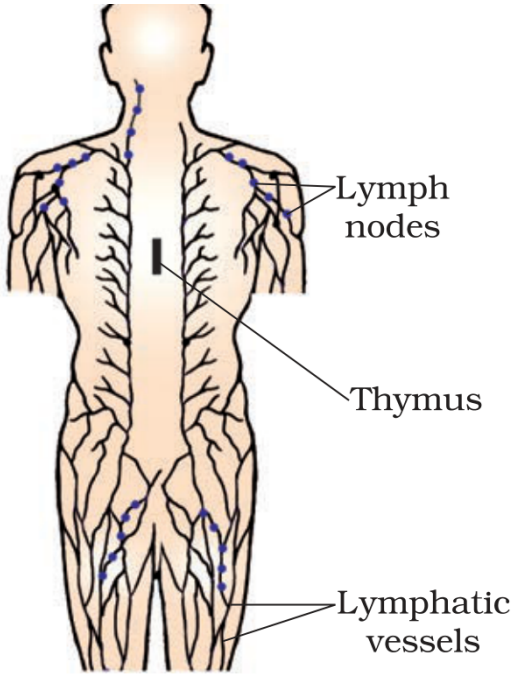


Figure 7.5 Diagrammatic representation of Lymph nodes

Source: NCERT Class 12 Biology, Chapter 7 — Fig. 7.5: Diagrammatic representation of the human lymphoid system — showing the lymph nodes, thymus and the network of lymphatic vessels.

Primary vs Secondary lymphoid organs

Primary = lymphocytes *originate / mature* here (bone marrow, thymus).
Secondary = mature lymphocytes *encounter antigens and proliferate* (spleen, lymph nodes, MALT, tonsils, Peyer's patches, appendix).

Primary lymphoid organs

"**BoTh**" = **Bo**ne marrow + **Th**ymus. Just two organs are primary — everything else is secondary.

4 AIDS

AIDS = **A**cquired **I**mmuno-**D**eficiency **S**yndrome. "Acquired" indicates that immunity is lost during the lifetime — it is not congenital. "Syndrome" means a group of symptoms (not a single disease). First reported in **1981**; has killed more than 25 million people worldwide.

4.1 The Pathogen — HIV

Causative agent: **Human Immunodeficiency Virus (HIV)**, a member of the **retrovirus** group. The virion has a lipid envelope enclosing two copies of single-stranded **RNA genome** plus the enzyme **reverse transcriptase**. Two types (NEET extra): **HIV-1** (worldwide, more virulent) and **HIV-2** (mainly West Africa).

HIV at a glance

Family: Retrovirus (envelope + ssRNA)
Key enzyme: Reverse transcriptase (RNA $\rightarrow$ DNA)
Target cells: Helper T-lymphocytes (T_H, CD4$^+$) and macrophages
Diagnostic test: **ELISA** (Enzyme-Linked Immuno-Sorbent Assay)

4.2 Modes of Transmission

NCERT lists four routes:

1. Sexual contact with an infected person.
2. Transfusion of contaminated blood or blood products.
3. Sharing infected needles (IV drug abuse, unsterilised tattoo needles).
4. Infected mother $\rightarrow$ child through the placenta (vertical transmission).

HIV is **not** transmitted by mere touch, sharing meals, mosquito bites, or casual contact. The window between infection and onset of symptoms (the **latency period**) is typically 5–10 years.

Common Mistake

HIV is **not** an air-borne or vector-borne disease. Mosquito bites do not transmit HIV. NEET frequently throws this as a distractor.

4.3 Replication Cycle (Figure 7.6)

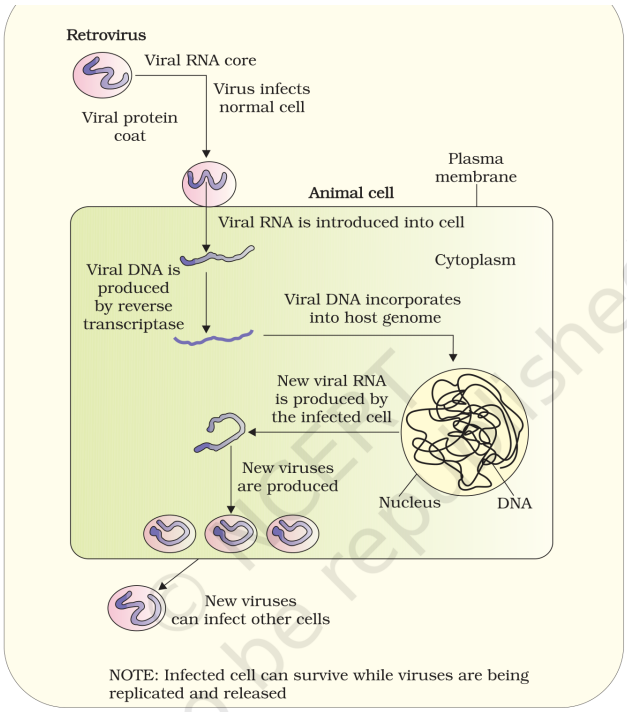
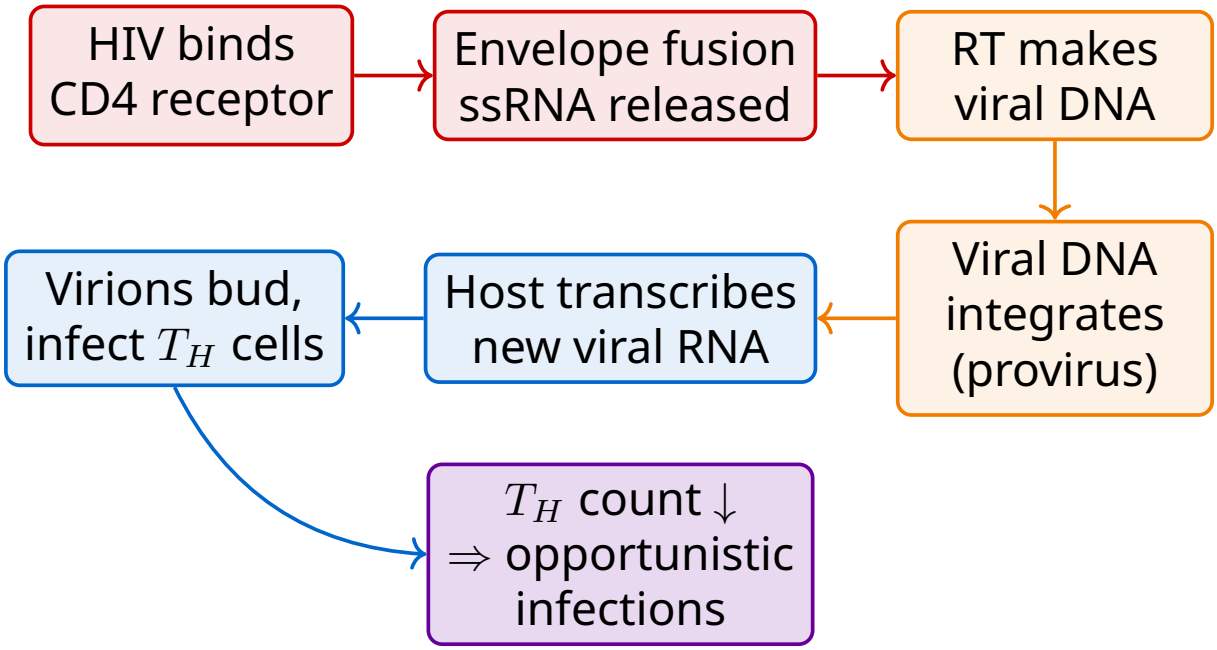


Figure 7.6 Replication of retrovirus

Source: NCERT Class 12 Biology, Chapter 7 — Fig. 7.6: Replication of retrovirus (HIV). The virus binds the cell, releases its ssRNA, reverse transcriptase makes viral DNA, which integrates into host DNA. The host machinery then produces fresh viral RNA and proteins, releasing new virions that infect other cells.

Step-by-step:

1. HIV enters a **macrophage**; the viral envelope fuses with the cell membrane.
2. Inside, **reverse transcriptase** synthesises **viral DNA** from the viral RNA template.
3. The viral DNA integrates into the host genome (now called a **provirus**).
4. Host machinery transcribes the provirus, producing new viral RNA and viral proteins.
5. New virions are assembled and bud out, infecting fresh **helper T-lymphocytes (T_H)**.
6. Progressive destruction of T_H cells $\Rightarrow$ collapse of cell-mediated and humoral responses $\Rightarrow$ susceptibility to *opportunistic infections* (*Mycobacterium*, fungi, *Toxoplasma*, etc.).



4.4 Diagnosis, Treatment and Prevention

Diagnostic test: **ELISA** — detects anti-HIV antibodies in patient serum. Confirmatory tests: Western blot, PCR (NEET extra).

Treatment: **Anti-retroviral therapy (ART)** — combinations of RT, protease and integrase inhibitors. Partially effective: prolongs life but cannot eradicate the integrated provirus.

Prevention (the only cure is prevention):

- Safe sex; **free distribution of condoms** (NACO).
- Use only disposable, sterile needles and syringes.
- Screen all donor blood for HIV before transfusion.
- Education and counselling — "*don't die of ignorance*".
- **NACO** (National AIDS Control Organisation) and many NGOs run awareness drives; WHO coordinates the global programme.

Real-World Application

HIV-infected people deserve sympathy, not stigma. Hiding the infection lets it spread; isolating patients harms their psychological health without helping anyone. The chapter explicitly stresses this social dimension.

Quick Tip

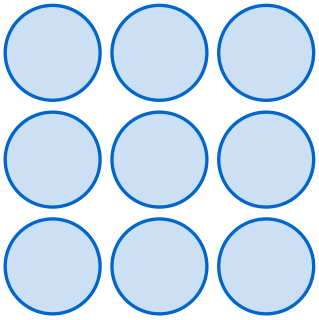
NEET often asks why anti-HIV drugs "don't cure" HIV. Answer: the integrated proviral DNA sits dormant inside the host genome where drugs cannot reach it; only the actively replicating virus is targeted.

5 Cancer

Cancer is one of the most dreaded human diseases and a leading cause of death worldwide. More than a million Indians have cancer, with a large fraction dying annually. In a healthy body, cell growth and differentiation are tightly regulated. In cancer, the regulatory brakes fail.

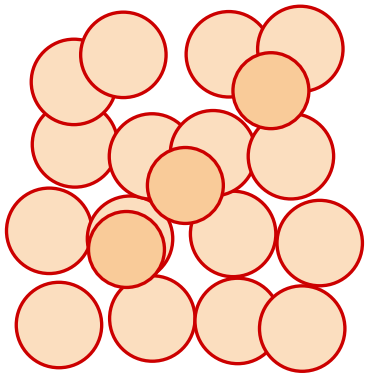
5.1 Cancerous Transformation — Loss of Contact Inhibition

Normal cells show **contact inhibition**: physical contact with neighbouring cells stops further division. Cancer cells lose this property and divide endlessly, piling into masses called **tumours**.



(a) Normal cells

Single layer
contact inhibition



(b) Cancer cells

Pile up, invade
no contact inhibition

Tumour types:

- **Benign** — confined to original location, do not spread, generally cause little damage.
- **Malignant** (neoplastic) — mass of rapidly proliferating cells. Invade and damage surrounding tissues, starve normal cells of nutrients, and most dangerously, undergo **metastasis** — cells slough off, travel via blood/lymph and start fresh tumours at distant sites.

Metastasis — the killer property

Metastasis is the spread of malignant cells from the primary tumour to distant organs through blood and lymph, where they seed new tumours. It is the single most feared property of cancer because it makes surgical removal of the primary tumour insufficient.

Benign tumour	Malignant tumour
Confined to original location	Invasive; metastasises via blood/lymph
Slow growth	Rapid uncontrolled growth
Cells well-differentiated	Cells poorly differentiated (neoplastic)
Little damage; rarely fatal	Damages tissues, often fatal
Surgery is usually curative	Surgery alone may not cure

5.2 Causes of Cancer — Carcinogens

Transformation of a normal cell into a cancer cell can be triggered by **physical, chemical or biological** agents called **carcinogens**:

- **Physical** — ionising radiation (X-rays, γ-rays) and non-ionising radiation (UV) cause DNA damage.
- **Chemical** — carcinogens in tobacco smoke (benzopyrene, nicotine-derived nitrosamines) are a major cause of **lung cancer**; asbestos $\rightarrow$ mesothelioma; aflatoxins $\rightarrow$ liver cancer (NEET extras).
- **Biological** — **oncogenic viruses** carry **viral oncogenes (v-onc)**. Examples: Human Papilloma Virus (cervical cancer), Hepatitis B/C (liver), EBV (Burkitt's lymphoma).

Even normal cells carry **cellular oncogenes (c-onc)** or **proto-oncogenes**. When mutated or aberrantly activated, these drive cancerous transformation. Tumour-suppressor genes (e.g. **p53**, **Rb**) normally restrain growth; losing them removes the brakes.

Cancer genetics in two genes

Proto-oncogenes (e.g. *ras*, *myc*) — gain-of-function mutation $\Rightarrow$ oncogene $\Rightarrow$ accelerator stuck on.
Tumour-suppressor genes (e.g. *p53*, *Rb*) — loss-of-function $\Rightarrow$ brakes removed.
Cancer typically requires multiple "hits" in both categories.

5.3 Detection and Diagnosis

Early detection is essential for cure. Techniques used:

- **Biopsy + histopathology** — gold standard; tissue is sectioned, stained, examined microscopically.
- **Blood / bone-marrow tests** — elevated WBC counts in **leukaemia**.
- **Radiography** (X-rays), **CT scan** (3-D X-ray reconstruction), **MRI** (strong magnetic fields, non-ionising) — detect internal-organ tumours.
- **Antibody-based assays** — detect cancer-specific antigens (PSA, CEA; NEET extras).

- **Molecular biology** — detects genes that pre-dispose individuals to certain cancers (BRCA1/BRCA2 for breast cancer).

> **Quick Tip**
>
> NCERT distinguishes **CT** (uses ionising X-rays) from **MRI** (uses strong magnetic fields and *non-ionising* radio waves — safer). NEET asks this every other year.

5.4 Treatment of Cancer

Three main approaches, usually combined:

- **Surgery** — excision of the tumour.
- **Radiotherapy** — lethal irradiation of tumour cells, sparing normal tissue.
- **Chemotherapy** — drugs that kill rapidly dividing cells. Side-effects: hair loss, anaemia, nausea (because other rapidly dividing cells like hair-follicle and gut-lining cells are also hit).
- **Immunotherapy** — **biological response modifiers** like α**-interferon** boost the patient's immune system to recognise and destroy tumour cells.

> **Real-World Application**
>
> Cancer cells normally evade immune surveillance. The 2018 Nobel Prize in Medicine was awarded for immune-checkpoint blockade (CTLA-4 and PD-1 inhibitors) — a modern extension of the "biological response modifier" principle NCERT mentions.

6 Drugs and Alcohol Abuse

The drugs commonly abused are **opioids, cannabinoids and coca alkaloids** — most are obtained from flowering plants, some from fungi. Tobacco and alcohol round out the major substances of abuse covered in NCERT.

6.1 The Three Main Drug Classes

1. Opioids. Bind specific **opioid receptors** in the central nervous system and gastrointestinal tract.

- **Morphine** — extracted from the **latex of opium poppy** (*Papaver somniferum*). Powerful sedative and painkiller, used clinically for post-surgical pain.
- **Heroin (smack)** — chemically **diacetylmorphine**; a white, odourless, bitter crystalline compound made by **acetylation of morphine**. Taken by snorting or injection; **depressant** that slows down body functions.

2. Cannabinoids. Interact with **cannabinoid receptors** principally in the brain. Source: inflorescences of *Cannabis sativa*.

- Products: **marijuana, hashish, charas, ganja** (varying preparations of flower tops, leaves and resin).
- Route: inhalation or oral ingestion.
- Effects: chiefly on the **cardiovascular system** (raised heart rate, blood pressure).
- Misuse by some sportspersons — listed as a banned substance in athletics.

3. Coca alkaloids (Cocaine). Source: *Erythroxylum coca* (native to South America). Mechanism: interferes with the transport of the neurotransmitter **dopamine**. Commonly called "coke" or "crack"; usually snorted. **Potent CNS stimulant** producing euphoria and increased energy; excessive doses cause hallucinations.

Other hallucinogens (NCERT): *Atropa belladonna* (deadly nightshade) and *Datura*.

Drug class	Source plant	CNS effect	Key example / Action
Opioids	*Papaver somniferum* (poppy)	**Depressant**	Heroin (diacetylmorphine); morphine = strong painkiller
Cannabinoids	*Cannabis sativa*	Variable; CV effects	Marijuana, hashish, charas, ganja
Coca alkaloids	*Erythroxylum coca*	**Stimulant**	Cocaine: blocks dopamine reuptake; euphoria
Hallucinogens	*Atropa belladonna*, *Datura*	Hallucination	Used historically in folk medicine, rituals
Tobacco	*Nicotiana tabacum*	Stimulant (autonomic)	Nicotine activates adrenal → adrenaline release

Drug class effects

"Op Depresses, Coke Excites, Cannabis Confuses" — opioids depress the CNS, cocaine stimulates it, cannabinoids alter perception. Tobacco's nicotine? Stimulates the adrenal axis.

6.2 Tobacco

Tobacco has been used by humans for more than 400 years — smoked, chewed or used as snuff. It contains many chemicals, including the alkaloid **nicotine**.

Mechanism: nicotine stimulates the **adrenal gland** to release **adrenaline and nor-adrenaline**, raising blood pressure and heart rate.

Adverse effects (smoking):

- Cancers — lung, urinary bladder, throat.
- Bronchitis, emphysema (chronic obstructive pulmonary disease).
- Coronary heart disease.
- Gastric ulcers.
- Increased blood **CO**, which binds haemoglobin and reduces oxygen-carrying capacity (*carboxyhaemoglobin*) $\Rightarrow$ tissue oxygen deficiency.

Adverse effects (chewing tobacco): increased risk of **oral cancer**.

> **Common Mistake**
>
> Don't write that nicotine *is* a stimulant of the central nervous system in the same way amphetamines are. NCERT specifies that nicotine acts on the **adrenal gland** to release adrenaline / noradrenaline, indirectly raising BP and HR.

6.3 Adolescence and Drug/Alcohol Abuse

Adolescence (NCERT definition): the period *and* process between ages **12–18 years** during which a child becomes mature in attitudes and beliefs. It is the bridge from childhood to adulthood, marked by biological and behavioural changes — making it a vulnerable phase.

Common triggers for drug/alcohol use among adolescents:

- Curiosity, need for adventure, experimentation.
- Escape from problems; academic stress.
- Perception that it is "cool" or modern — amplified by TV, films, internet.
- Unstable family structures; peer pressure.

> **Quick Tip**
>
> NEET frequently quotes the **12–18 years** adolescence range verbatim. Memorise the upper and lower bounds.

6.4 Addiction and Dependence

Addiction is a *psychological* attachment to the effects (euphoria, well-being) of drugs/alcohol. With repeated use, the body's receptors become tolerant — they respond only to higher doses, fuelling escalation.

Dependence is the body's tendency to manifest a characteristic, unpleasant **withdrawal syndrome** when regular dosing is abruptly stopped: anxiety, shakiness,

nausea, sweating. In severe cases withdrawal can be life-threatening (requires medical supervision).

Tolerance, addiction and dependence — the vicious circle

Tolerance (receptors need more drug) → higher doses → **addiction** (psychological need) → **dependence** (physical withdrawal if stopped). The user is pulled into a vicious cycle of escalating use.

6.5 Effects of Drug/Alcohol Abuse

Immediate effects: reckless behaviour, vandalism, violence; coma or death from respiratory failure, heart failure or cerebral haemorrhage; overdose deaths often from combining drugs or mixing with alcohol.

Warning signs in youth: drop in academic performance, unexplained absence from school/college, lack of personal hygiene, withdrawal, isolation, depression, fatigue, aggression, deteriorating relationships, change in sleeping and eating habits, fluctuating weight and appetite.

Long-term effects:

- Cirrhosis of the liver, nervous system damage.
- IV drug abusers risk **AIDS and Hepatitis B** via shared needles — both fatal chronic infections.
- Foetal damage from drug/alcohol use in pregnancy (foetal alcohol syndrome — NEET extra).
- Financial and emotional distress to family and friends.

Drugs in sport — the misuse of performance enhancers: certain sportspersons (mis)use **narcotic analgesics, anabolic steroids, diuretics** and certain hormones to increase muscle strength and aggressiveness.

Side-effects of anabolic steroids:

In females	In males
Masculinisation (male features)	Acne; mood swings; depression
Increased aggressiveness; mood swings	Reduced testicle size; decreased sperm count
Depression; abnormal menstrual cycles	Kidney and liver dysfunction
Excessive facial / body hair; enlarged clitoris	Breast enlargement
Deepening of voice	Premature baldness; enlarged prostate

In adolescents (both sexes): severe facial and body acne, and **premature closure of the long-bone growth plates**, causing stunted growth.

6.6 Prevention and Control

NCERT lays out a clear strategy: identify the risk situation and intervene early. Parents and teachers have a special responsibility. Five practical measures:

1. **Avoid undue peer pressure** — each child has a unique personality. Don't push beyond limits in studies, sports or activities.
2. **Education and counselling** — channel curiosity, stress and frustration into healthy pursuits (sport, books, music, yoga, meditation).
3. **Seek help from parents and peers** — talk to parents or trusted friends at the first sign of dependence; don't bottle it up.
4. **Look for danger signs** — spot early warning signs in friends; alert their families and seek professional help.
5. **Seek professional and medical help** — psychologists and psychiatrists at de-addiction and rehabilitation centres can break the dependence cycle.

Real-World Application

India's **National Mental Health Programme (NMHP)** and many NGOs run de-addiction centres. The slogan "*prevention is better than cure*" is literal here: once dependence is established, recovery rates fall sharply.

7 Quick Reference Summary

A one-stop revision sheet for the chapter — ideal for the last 24 hours before the board or NEET exam.

7.1 Pathogen ↔ Disease Quick-Recall

Pathogen	Disease	Type / Vector
Salmonella typhi	Typhoid (Widal test)	Bacterium
Streptococcus pneumoniae & *Haemophilus influenzae*	Pneumonia	Bacterium
Rhinoviruses	Common cold	Virus
Plasmodium falciparum	Malignant malaria	Protozoan; *Anopheles*
Entamoeba histolytica	Amoebiasis	Protozoan; housefly carrier
Ascaris lumbricoides	Ascariasis	Helminth
Wuchereria bancrofti / malayi	Filariasis (elephantiasis)	Helminth; *Culex*
Microsporum, *Trichophyton*, *Epidermophyton*	Ringworm	Fungus
HIV (retrovirus)	AIDS	Virus (ELISA test)

7.2 Immunity At-a-Glance

- **Two types**: Innate (4 barriers — physical, physiological, cellular, cytokine) and Acquired (B-cell humoral + T-cell CMI).
- **Antibody**: H_2L_2; classes IgG (most abundant, crosses placenta), IgM (first in primary response, pentamer), IgA (colostrum, mucosa), IgE (allergy, mast cells), IgD (B-cell receptor).
- **Lymphoid organs**: Primary = bone marrow + thymus. Secondary = spleen, lymph nodes, MALT (50% of all lymphoid tissue), tonsils, Peyer's patches, appendix.
- **Active vs Passive**: active = body makes Ab (slow, long-lasting). Passive = preformed Ab supplied (fast, short-lived; e.g. colostrum IgA).
- **Allergy**: IgE + mast cells → histamine & serotonin. Treat with anti-histamines.
- **Auto-immunity**: body attacks self — rheumatoid arthritis is the NCERT example.

7.3 AIDS Key Facts

- Causative agent: HIV (retrovirus, ssRNA + reverse transcriptase).
- Target: helper T-cells (T_H, CD4$^+$); also macrophages.
- Routes: sexual contact, contaminated blood, shared needles, mother→child.
- Diagnostic test: **ELISA**.
- Treatment: anti-retroviral drugs (partially effective; cannot eradicate the integrated provirus).

- Prevention: NACO, free condoms, sterile needles, blood screening, awareness.

7.4 Cancer Key Facts

- Loss of **contact inhibition** $\Rightarrow$ uncontrolled division $\Rightarrow$ tumour.
- **Benign vs malignant**: confined vs invasive; only malignant tumours metastasise.
- Carcinogens: physical (X-, γ-, UV-rays), chemical (tobacco smoke), biological (oncogenic viruses).
- Genes: proto-oncogenes (c-onc), viral oncogenes (v-onc); tumour suppressors p53, Rb (NEET).
- Diagnosis: biopsy + histopathology; CT (ionising); MRI (non-ionising).
- Treatment: surgery + radiotherapy + chemotherapy; immunotherapy uses α-interferon (a biological response modifier).

7.5 Drug & Alcohol Abuse Key Facts

- **Opioids**: *Papaver somniferum* $\rightarrow$ morphine, heroin (smack, diacetylmorphine). **Depressant**.
- **Cannabinoids**: *Cannabis sativa* $\rightarrow$ marijuana, hashish, charas, ganja. Affect cardiovascular system.
- **Cocaine**: *Erythroxylum coca* $\rightarrow$ blocks dopamine reuptake. **Stimulant**.
- **Hallucinogens**: *Atropa belladonna*, *Datura*.
- **Tobacco**: nicotine $\rightarrow$ adrenal gland $\rightarrow$ adrenaline/noradrenaline $\rightarrow \uparrow$BP, $\uparrow$HR; CO binds Hb; cancers (lung, urinary bladder, throat, oral).
- **Adolescence**: 12–18 years (NCERT-specified range).
- **Addiction** = psychological; **Dependence** = withdrawal syndrome on stopping.
- Performance-enhancing drugs in sport: **anabolic steroids**, narcotic analgesics, diuretics, certain hormones.

7.6 High-Yield NEET Pitfalls

- Common cold $=$ Rhinovirus, infects upper airway only, NOT lungs.
- Ringworm $=$ fungus, NOT a worm.
- Vector of malaria $=$ female *Anopheles*; vector of filariasis $=$ female *Culex*.
- *Plasmodium falciparum* $=$ malignant malaria (fatal).
- Diagnostic test of typhoid $=$ Widal; of AIDS $=$ ELISA.
- Most abundant antibody $=$ IgG; first antibody in primary response $=$ IgM.
- Primary lymphoid organs $=$ bone marrow + thymus ONLY.
- Heroin chemistry: diacetylmorphine (acetylation of morphine).
- Cocaine mechanism: blocks dopamine reuptake.

- Adolescence range: 12–18 years.

One-sentence chapter summary

Health is a balance maintained by an immune system (innate + acquired) that fights bacteria, viruses, protozoa, helminths and fungi; when this balance fails — through pathogen overload (AIDS), uncontrolled cell growth (cancer), or chemical dependence (drugs, alcohol, tobacco) — disease results, and prevention through hygiene, vaccination, awareness and counselling is humanity's most reliable defence.