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Step-by-step Coloured PDF solutions for the 2026-27 NCERT (Latest Edition) Class 12th Biology

Chapter 7: Human Health and Disease

About this Chapter

How **pathogens** cause infectious diseases and how the body defends itself through **innate and acquired immunity**. Also covers antibody structure, vaccination, AIDS retrovirus mechanism, cancer (metastasis), and the effects of drug & alcohol abuse.

Topics covered: Common Diseases • Immunity (Innate & Acquired) • Antibody Structure • Vaccination • AIDS • Cancer • Drugs & Alcohol Abuse

Quick Formula Sheet

Antibody:
H_2L_2 (2 heavy + 2 light chains)

Acquired immunity:
Primary $\rightarrow$ Secondary (memory)

HIV:
RNA $\xrightarrow{\text{reverse}}$ DNA $\rightarrow$ host genome

Tumour types:
Benign vs. Malignant (metastasis)

Exercises

Q 7.1 **What are the various public health measures, which you would suggest as safeguard against infectious diseases?**

SOLUTION

Concept used. Infectious diseases are illnesses caused by **pathogens** (disease-causing microorganisms such as bacteria, viruses, protozoans, fungi and helminths) that spread from an infected host to a healthy one. **Public health measures** are organised actions, taken at the level of the community by the government, by health agencies and by individuals, to prevent the entry, the multiplication and the spread of these pathogens. They break the chain of transmission at one or more of three points: the source, the route and the susceptible host.

☞ **Three breakpoints in disease transmission**

Source (sick person or carrier or vector) → Route (air, water, food, vector, contact) → Host (healthy individual). A good public-health measure attacks at least one of these.

Step 1. **Personal hygiene.** Keeping the body clean, taking a regular bath, washing hands with soap before eating and after using the toilet, brushing teeth, trimming nails, and wearing clean clothes. Personal hygiene checks the entry of pathogens of typhoid, amoebiasis, ascariasis, ringworm and many other infections that travel by the faecal-oral route or by skin contact.

Step 2. **Public sanitation.** Proper disposal of garbage and excreta, periodic cleaning and disinfection of water reservoirs and tanks, swimming pools and cisterns, and the use of clean and covered drains. These measures cut off the route of water-borne and food-borne diseases such as cholera, typhoid, amoebiasis and ascariasis.

Step 3. **Safe and clean drinking water.** Drinking only boiled, filtered or chlorinated water; protecting tube-wells and hand-pumps from contamination; and avoiding open street beverages prevents water-borne diseases like typhoid, cholera, amoebiasis and hepatitis A.

Step 4. **Control of vectors and their breeding grounds.** Eradication of mosquito-breeding sources by draining stagnant water from coolers, tyres, pots and gutters, spraying insecticides (DDT in older programmes, today pyrethroids), introducing larvivorous fish such as *Gambusia* into ponds, and using mosquito nets and repellents controls malaria, dengue, chikungunya, filariasis. Similarly, controlling rats, flies and cockroaches checks plague and many enteric infections.

Step 5. **Vaccination and immunisation.** Routine immunisation of infants and children against polio, diphtheria, tetanus, pertussis, measles, mumps, hepatitis-B and tuberculosis builds active immunity in the population and gives **herd immunity**, which protects even the un-immunised.

Step 6. **Health education.** Awareness drives through school programmes, posters, electronic and print media on safe sex, safe food, hand-washing, oral rehydration, breast-feeding, and the dangers of self-medication empower people to protect themselves.

Step 7. **Early diagnosis and treatment.** Screening, easy access to government dispensaries, free essential drugs (DOTS for tuberculosis, ART for HIV), and the timely isolation of cases prevent secondary transmission.

> **Final Answer:** Personal hygiene, public sanitation, safe drinking water, vector control, vaccination, health education and prompt diagnosis-treatment together form the core public-health shield against infectious diseases.

☞ Exam Tip

In Boards and NEET, an answer that lists *seven* clearly named measures (each with a one-line reason and one disease example) typically scores full marks. Bullet your answer.

EXPERT'S SOLUTION *: Aanya Iyer, M.Sc Microbiology, JNU*

Strategic angle: attack the chain. Every infectious disease needs a *source*, a *route* and a *susceptible host*. Public-health measures are easiest to remember if you tie each one to the link of the chain it breaks.

Step 1. **Attack the source.** Isolation of patients (TB ward, COVID quarantine), prompt treatment of confirmed cases with the right antibiotics or antivirals, and notification of infectious diseases to the local health authority dry up the source of pathogens.

Step 2. **Attack the route.** (a) Water-borne: chlorination, boiling, sand filtration. (b) Air-borne: ventilation, masks in crowded settings, covering the mouth while sneezing. (c) Vector-borne: removal of stagnant water, insecticide spraying, biological control with *Gambusia* fish, use of mosquito nets. (d) Food-borne: hygienic kitchens, covered food, washing fruits and vegetables, pasteurisation. (e) Sexual: safe sex, screening of donor blood for HIV and HBV.

Step 3. **Strengthen the host.** Universal Immunisation Programme (UIP) vaccines from birth through adolescence; balanced nutrition that maintains the body's barriers and immune cells; breast-feeding of infants for passive antibody transfer; and treatment of co-morbidities like diabetes that weaken immunity.

Step 4. **Surveillance and education.** Disease registries, epidemic-alert systems, school awareness on hand-washing and safe sex, posters in clinics on the danger of self-medication and indiscriminate antibiotic use.

Why this matters. Most of India's gains in life expectancy since 1950 come not from new drugs but from these simple public-health interventions. Smallpox was wiped out by vaccination alone; polio is on the verge of eradication by the same means.

> **Final Answer:** Break the source, block the route, protect the host: this three-pronged public-health approach is the safest, cheapest and most durable safeguard against infectious diseases.

Q 7.2 In which way has the study of biology helped us to control infectious diseases?

SOLUTION

Concept used. The control of an infectious disease rests on three pillars: (i) knowing the **pathogen** that causes it, (ii) knowing the **life cycle and mode of transmission** of that pathogen, and (iii) designing tools (drugs, vaccines, vector controls, diagnostic tests) that strike specifically at the pathogen or its weak link. Each of these pillars rests on biology, that is, on what microbiology, immunology, biochemistry, molecular biology and biotechnology have discovered.

Step 1. **Discovery and identification of pathogens.** Microbiology has shown that infectious diseases are caused by specific organisms: malaria by *Plasmodium*, typhoid by *Salmonella typhi*, cholera by *Vibrio cholerae*, TB by *Mycobacterium tuberculosis*, AIDS by HIV, amoebiasis by *Entamoeba histolytica*, ringworm by *Microsporum, Trichophyton, Epidermophyton*. Without this knowledge no targeted control is possible.

Step 2. **Understanding life cycles and transmission.** Parasitology and ecology have mapped the life cycles, like the *Plasmodium* cycle between female *Anopheles* and humans, and the *Ascaris* faecal-oral cycle. This knowledge tells us *where* to break the cycle (e.g. kill mosquito larvae, treat drinking water).

Step 3. **Development of drugs and antibiotics.** Pharmacology and biotechnology have given us specific antimicrobials: chloroquine and artemisinin for malaria, penicillin and cephalosporins for bacteria, fluconazole for fungal infections, antiretrovirals (ART) for HIV. Each kills the pathogen with minimum damage to the host.

Step 4. **Vaccines and immunisation.** Immunology has revealed the antigen-antibody system, allowing scientists to design vaccines (live attenuated, killed, sub-unit, recombinant DNA vaccines like Hepatitis B, mRNA vaccines like COVID-19). Vaccines have eradicated smallpox and have brought polio, measles and diphtheria under tight control.

Step 5. **Diagnostic technology.** Molecular biology has given rapid, sensitive tests: ELISA for HIV and dengue, Widal for typhoid, PCR for tuberculosis and COVID-19, microscopy for malaria. Early diagnosis means early treatment and less secondary spread.

Step 6. **Vector and reservoir control.** Entomology and ecology have identified vectors and their breeding habits, leading to integrated vector management: insecticides, larvivorous fish, biocontrol, and habitat modification.

Step 7. **Genetic engineering and biotechnology.** Recombinant vaccines (Hep B), monoclonal antibodies (for cancer, COVID), gene therapy and DNA vaccines are direct fruits of modern biology.

> **Final Answer:** Biology, through microbiology, immunology, pharmacology, biotechnology and ecology, has given us the pathogens' identity, their life cycles, the drugs to kill them, the vaccines to prevent them, the tests to detect them and the controls to interrupt their transmission. That is how we control infectious diseases today.

♥ Twin gains from biology

The eradication of smallpox (1980) and the near-elimination of polio in India (last wild case 2011) are direct trophies of biology: the disease was understood, a vaccine was designed, and a delivery network rolled it out across hundreds of millions of people.

EXPERT'S SOLUTION *: Aarav Sharma, Ph.D Molecular Biology, NCBS Bangalore*

Strategic angle: from microscope to molecule. Trace the arc of biology in three eras and the picture becomes clear.

Step 1. **The microscope era.** Leeuwenhoek, Pasteur and Koch proved that microorganisms cause disease (germ theory). Each infectious illness was tied to a specific microbe. This single insight ended centuries of mysticism and made targeted control even thinkable.

Step 2. **The antibiotic and vaccine era.** Fleming's penicillin (1928) opened the antibiotic era; Jenner's cowpox vaccine (1796) and the later vaccines for polio, measles, mumps, rubella and Hep B opened the immunisation era. India's Universal Immunisation Programme now covers >90% of children against six killer diseases.

Step 3. **The molecular era.** Recombinant DNA technology has given recombinant Hep B vaccine; monoclonal antibodies treat cancer and severe COVID-19; PCR delivers diagnoses in hours; gene therapy is reaching the clinic. Bioinformatics tracks outbreak strains in near real-time.

Step 4. **Public-health applications.** Knowledge of life cycles (Plasmodium needs water for mosquito breeding; Ascaris spreads via faecal contamination of soil and water; HIV needs blood, semen or breast-milk for transmission) lets us design specific public-health drives: drain stagnant water, chlorinate the supply, screen blood, distribute condoms.

Why this matters. A century ago, infectious diseases caused about 40% of all deaths in India. Today they cause less than 15%. That drop is entirely the work of biology applied to public health.

Final Answer: Biology's progress, from germ theory through antibiotics and vaccines to recombinant DNA and PCR, has armed humanity with the specific tools needed to identify, prevent, diagnose and treat every major infectious disease.

Q 7.3 **How does the transmission of each of the following diseases take place?**
(a) Amoebiasis (b) Malaria (c) Ascariasis (d) Pneumonia

SOLUTION

Concept used. **Transmission** is the route by which a pathogen passes from an infected source (a sick person, a carrier or a contaminated reservoir) to a healthy susceptible host. The four diseases asked here run on four distinct routes: faecal-oral (water/food), vector-borne (mosquito bite), faecal-oral (soil/water contaminated with eggs), and respiratory droplet.

☞ **Pathogen + Route, side-by-side**

Disease	Pathogen	Transmission route
Amoebiasis	*Entamoeba histolytica* (protozoan)	Cysts in food/water contaminated by faeces; carried by houseflies.
Malaria	*Plasmodium* (4 species; *P. falciparum* most dangerous)	Bite of infected female *Anopheles* mosquito.
Ascariasis	*Ascaris lumbricoides* (roundworm)	Ingestion of embryonated eggs from soil/water/vegetables polluted by faeces.
Pneumonia	*Streptococcus pneumoniae*, *Haemophilus influenzae* (bacteria)	Inhalation of droplets/aerosols from a sick person; shared utensils.

Step 1. **(a) Amoebiasis (amoebic dysentery).** Caused by the protozoan *Entamoeba histolytica*. The pathogen lives as a cyst in the large intestine of infected people. Cysts are released in their faeces. When faeces contaminate food or drinking water, or when houseflies (mechanical vectors) carry the cysts onto exposed food, a healthy person who eats or drinks the contaminated material picks up the infection. Hence **faecal-oral transmission** through contaminated food and water; flies act as mechanical carriers.

Step 2. **(b) Malaria.** Caused by *Plasmodium* species (*P. vivax*, *P. malariae*, *P. ovale* and the deadliest *P. falciparum*). It needs two hosts: a female *Anopheles* mosquito and a human. When an infected female *Anopheles* bites a healthy human, it injects *sporozoites* (the infective form) into the blood; they enter liver cells, multiply, then infect RBCs. When a fresh *Anopheles* bites the patient, it picks up

gametocytes, which fuse and develop in the mosquito gut. Hence **vector transmission** through the bite of an infected female *Anopheles* mosquito.

Step 3. **(c) Ascariasis.** Caused by the intestinal round-worm *Ascaris lumbricoides*. Adult worms in the intestine of an infected person release thousands of eggs that pass out in the faeces and pollute soil, water, plants and vegetables. A healthy person who eats raw or poorly washed vegetables, drinks contaminated water, or puts dirty hands in the mouth swallows the embryonated eggs and develops the disease. Hence **faecal-oral transmission** via soil, water and vegetables contaminated with eggs.

Step 4. **(d) Pneumonia.** Caused chiefly by the bacteria *Streptococcus pneumoniae* and *Haemophilus influenzae*. The pathogen lodges in the alveoli of the infected person's lungs and is released in the tiny droplets that come out when the patient coughs, sneezes or talks. A healthy person inhales these droplets, or comes into contact with the patient's utensils, glass or used tissue, and picks up the bacteria. Hence **droplet (air-borne) transmission** through coughing/sneezing and through shared utensils.

Final Answer: Amoebiasis and ascariasis spread by the faecal-oral route through contaminated food and water; malaria spreads by the bite of an infected female *Anopheles* mosquito; pneumonia spreads by inhaled respiratory droplets from a sick person.

✗ Common Mistake

A frequent slip in board answers is to say malaria is caused by the *Anopheles* mosquito. The mosquito is only the vector; the *cause* (pathogen) is *Plasmodium*.

EXPERT'S SOLUTION *: Riya Kapoor, M.Sc Zoology, Banaras Hindu University*

Strategic angle: name pathogen and route together. Pair each disease with its pathogen first, then state the route. This structure scores cleanly and never confuses cause with vehicle.

Step 1. **Amoebiasis** – pathogen *Entamoeba histolytica* (protozoan). Cysts shed in faeces of an infected person contaminate drinking water and uncooked food (salads, fruits); houseflies act as mechanical vectors. The healthy person ingests the cysts and develops amoebic dysentery.

Step 2. **Malaria** – pathogen *Plasmodium* (protozoan). Transmitted biologically by an infected female *Anopheles* mosquito. When the mosquito bites, it injects sporozoites in saliva. Mosquito control breaks transmission.

Step 3. **Ascariasis** – pathogen *Ascaris lumbricoides* (round-worm). Embryonated eggs from faeces contaminate soil, water and vegetables grown in such soil; the healthy person eats them. Hence faecal-oral. Adequate sanitation and washing of vegetables stops the cycle.

Step 4. **Pneumonia** – pathogens *Streptococcus pneumoniae* and *Haemophilus influenzae* (bacteria). Spread by droplets when an infected person coughs/sneezes, or through shared utensils; inhalation by a healthy person seeds the alveoli. Covering the mouth while coughing and not sharing utensils breaks transmission.

Why this matters. Two of the four (amoebiasis, ascariasis) need clean water and sanitation; one (malaria) needs vector control; one (pneumonia) needs personal respiratory hygiene and vaccination. The control strategy follows directly from the route.

> **Final Answer:** Faecal-oral for amoebiasis and ascariasis, female *Anopheles* bite for malaria, droplet inhalation for pneumonia.

Q 7.4 What measure would you take to prevent water-borne diseases?

SOLUTION

Concept used. Water-borne diseases are diseases whose pathogens travel from sick to healthy people in contaminated drinking water. Typical examples are typhoid, cholera, amoebiasis, hepatitis A, polio, ascariasis and several diarrhoeal illnesses. Prevention works at two ends: (i) stop the pathogen from getting into the water, and (ii) purify the water before drinking.

Step 1. **Provide clean, treated drinking water.** Use municipal treated water; if unsure, boil drinking water for at least $10\,\text{minutes}$ to kill bacteria, viruses, protozoan cysts and worm eggs. Alternatively, treat water with chlorine tablets, ultraviolet (UV) light, or pass it through a tested ceramic/RO filter.

Step 2. **Protect water sources from contamination.** Keep wells, tube-wells and hand-pumps covered. Place pit latrines and soak-pits at least $15\,\text{m}$ away from any drinking-water source. Do not allow sewage, animal waste or industrial effluent to drain into ponds, rivers and reservoirs used for drinking.

Step 3. **Proper disposal of faeces and sewage.** Use proper toilets connected to septic tanks or a covered drainage system. Open defecation contaminates soil and rainwater run-off, which then enters surface water bodies.

Step 4. **Personal hygiene.** Wash hands thoroughly with soap and clean water before

preparing or eating food and after using the toilet. Avoid drinking from open glasses at street stalls; avoid ice made from untreated water.

Step 5. **Food and kitchen hygiene.** Wash fruits and vegetables with treated water; do not eat cut/peeled fruits sold on the roadside; cover cooked food to keep flies off; do not store drinking water in open vessels.

Step 6. **Public sanitation drives.** Periodic cleaning and chlorination of community water tanks; control of houseflies (which carry pathogens from faeces to food); regular inspection of water-pipes for leaks (sewage seeps into them through fractures and creates outbreaks).

Step 7. **Vaccination and prompt treatment.** Vaccines for typhoid, cholera and hepatitis A protect specifically against water-borne pathogens. Patients of cholera or dysentery should be treated quickly with oral rehydration solution (ORS) and appropriate antibiotics to stop further shedding.

> **Final Answer:** Drink only treated/boiled/filtered water; protect drinking sources from sewage; use covered toilets; wash hands with soap; keep food and kitchen clean; chlorinate community tanks; and take available vaccines for typhoid, cholera and hepatitis A.

☞ Exam Tip

For 3-mark questions, group your answer into source → distribution → point-of-use protection. Examiners love this 3-step structure because it covers every angle.

EXPERT'S SOLUTION *: Pooja Banerjee, M.Sc Biotechnology, AIIMS Delhi*

Strategic angle: kill the cycle at every step from toilet to glass. A water-borne pathogen travels: infected gut → faeces → environment (soil, water source) → piped water/well → glass → healthy gut. Block *any* step and you prevent the disease.

Step 1. **Block at the toilet.** Provide closed toilets connected to septic tanks/STP; stop open defecation. Treat the patient's faeces with bleaching powder before disposal in outbreak settings.

Step 2. **Block at the source.** Locate wells, tube-wells and rainwater catchments well away from soak-pits and drains. Maintain a buffer zone around ponds and rivers used for drinking.

Step 3. **Block at distribution.** Repair leaking water pipes, especially where they cross sewer lines. Maintain residual chlorine in the piped supply.

Step 4. **Block at point of use.** Boil for 10 minutes, or chlorinate with one halazone

tablet per litre, or use a certified RO/UV filter. Store treated water in a covered steel pot with a long-handled ladle to avoid hand contact.

Step 5. **Block at the host.** Hand-washing with soap, especially before eating and after the toilet, removes any residual pathogen at the gateway to the gut. Vaccinate against typhoid, cholera and hepatitis A in high-risk areas.

Why this matters. Diarrhoeal disease is still a top-three killer of Indian children. Studies show that supplying chlorinated piped water plus universal hand-washing alone cut its incidence by more than half. The measures are cheap, the gains are huge.

> **Final Answer:** Cut the faecal-oral chain at the toilet, at the source, in distribution, at the point of use and at the host. Boiling/filtering, clean toilets, hand-washing with soap and selective vaccination together prevent virtually all water-borne diseases.

Q 7.5 **Discuss with your teacher what does 'a suitable gene' means, in the context of DNA vaccines.**

SOLUTION

Concept used. A **DNA vaccine** (also called a third-generation vaccine) is a small circular piece of DNA (a plasmid) carrying the gene that codes for one or more **antigenic proteins** of the pathogen we want to vaccinate against. When the plasmid is injected into a host, the host's own cells transcribe and translate the gene, the antigen protein appears on the cell surface, and the host's immune system makes antibodies and memory cells against it. Hence a *suitable gene* is the specific piece of pathogen DNA that, when expressed in the host, produces a protein that triggers a strong, lasting, protective immune response without causing disease.

Step 1. **It must code for an antigenic protein of the pathogen.** The gene must encode a protein the immune system recognises as 'non-self', so that B-cells and T-cells respond. Surface proteins, capsid proteins or envelope glycoproteins are usual choices (for example the spike-protein gene for SARS-CoV-2, the HBsAg gene for Hepatitis B virus, the circumsporozoite-protein gene for *Plasmodium*).

Step 2. **The protein must elicit protective immunity.** Some antigens make antibodies that bind but do not block the pathogen; the chosen gene must give a *neutralising* response, i.e. the antibodies should actually stop infection or kill the pathogen.

Step 3. **It must be safe.** The gene must not code for any toxin or any protein that lets the pathogen multiply inside the host. It is only the antigen, never the whole

pathogen.

Step 4. **It must be conserved across strains.** The gene's product should not change easily between strains and seasons, otherwise the vaccine quickly becomes useless (a problem with the influenza virus's surface antigens).

Step 5. **It must be efficiently expressed in human cells.** The gene is placed under a strong human-cell promoter (typically the CMV promoter) so that the host cell makes plenty of antigen. The gene's sequence is sometimes codon- optimised for human ribosomes.

Step 6. **It must be small enough to clone into a plasmid.** Practical plasmids carry roughly ≤ 10 kb of insert; a good vaccine gene is usually 1–3 kb.

Final Answer: A 'suitable gene' for a DNA vaccine is a small, conserved, non-toxic piece of pathogen DNA that codes for an antigenic surface protein, is efficiently expressed in human cells, and elicits a strong, lasting and protective (neutralising) immune response.

♥ The new vaccine generation

DNA vaccines are easy to design, fast to make, stable at room temperature and protect by triggering both antibody and cell-mediated responses. India's ZyCoV-D (against COVID-19) was the world's first approved DNA vaccine for human use.

EXPERT'S SOLUTION *: Karan Mehta, M.Sc Biotechnology, AIIMS Delhi*

Strategic angle: think like a vaccine engineer. You are shopping for a gene in the pathogen's genome. What do you check?

Step 1. **Antigenicity check.** Run the candidate protein through immunoinformatic prediction or pull data from infected-patient sera. If their immune system has already targeted the protein, your vaccine will too. Spike, capsid, envelope proteins normally pass.

Step 2. **Neutralisation check.** Does the antibody to this protein actually stop infection in cell culture? If not, the gene is unsuitable, however antigenic. The HIV envelope is antigenic but most antibodies fail to neutralise; that is why an HIV DNA vaccine has been hard to make.

Step 3. **Conservation check.** Compare the gene across strains and species. A region conserved across all variants gives a long-lived vaccine (universal flu vaccines target conserved stalk regions).

Step 4. **Safety check.** The gene must not encode a toxin, an oncoprotein, an integrase

or any virulence factor. It must not provoke autoimmunity by mimicking a host protein.

Step 5. **Expression check.** Add a strong promoter (CMV) and a polyA tail; codon-optimise; verify protein expression in a cell line. Without expression, no antigen, no vaccine.

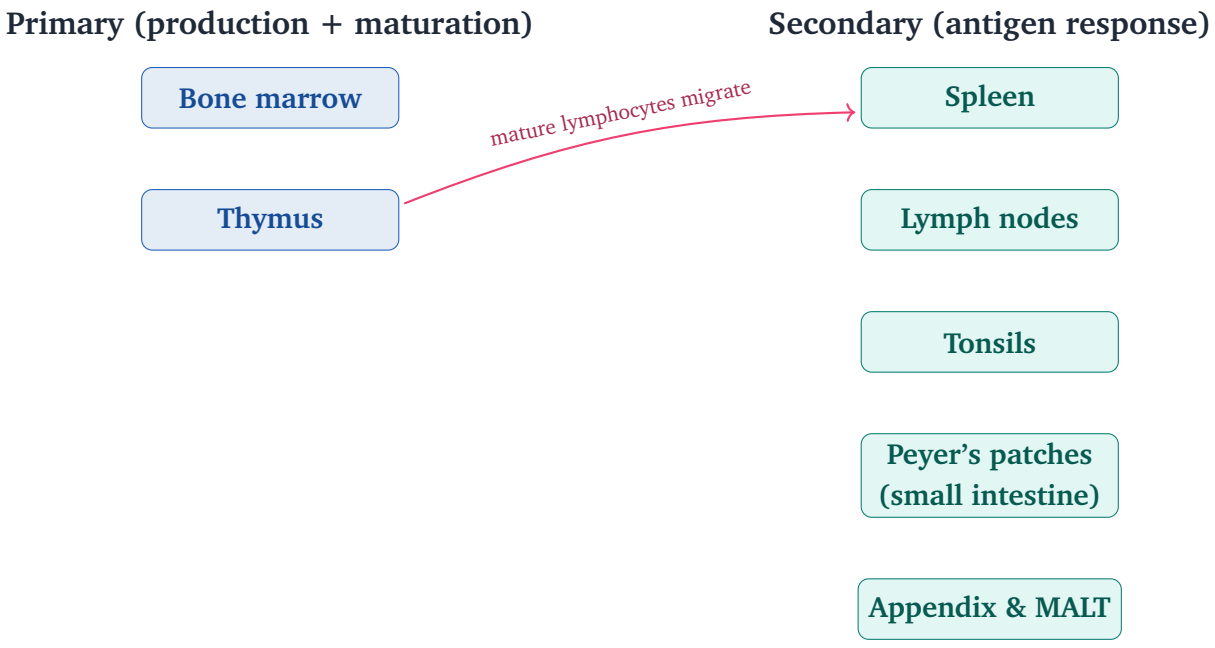
Why this matters. The right gene is the difference between a life-saving vaccine and a useless plasmid. The criteria above turn 'which gene?' from a guess into a checklist.

> **Final Answer:** The suitable gene is the antigenic, neutralising, conserved, safe and well-expressed piece of pathogen DNA that, when made in the host's cells, gives lasting protection without causing disease.

Q 7.6 Name the primary and secondary lymphoid organs.

SOLUTION

Concept used. The **lymphoid system** is a network of organs and tissues where the cells of the immune system (lymphocytes: B and T cells) are produced, mature and act. **Primary lymphoid organs** are the sites where immature lymphocytes are produced and undergo *maturation* into immuno- competent B and T cells (i.e. cells able to recognise self from non-self). **Secondary lymphoid organs** are the sites where the matured lymphocytes meet antigens, get activated, multiply clonally and mount the actual immune response.



Step 1. **Primary lymphoid organs.** (i) **Bone marrow** is the main site where all blood cells, including lymphocytes, are produced. B-lymphocytes mature here in mammals. (ii) **Thymus** is a lobed organ near the heart, beneath the breast-bone. It is large in babies but shrinks with age (involution). T-lymphocytes are formed in the bone marrow and then migrate to the thymus, where they mature. Both bone marrow and thymus also provide the microenvironment for self-tolerance: lymphocytes that react against the body's own tissues are eliminated here.

Step 2. **Secondary lymphoid organs.** (i) **Spleen**: a bean-shaped organ in the upper left abdomen; filters blood-borne microbes and stores lymphocytes. (ii) **Lymph nodes**: bean-shaped, scattered through the body along lymphatic vessels; trap micro-organisms and antigens that enter the lymph and tissue fluid. (iii) **Tonsils**: at the back of the throat; guard against pathogens entering through the mouth and nose. (iv) **Peyer's patches** of the small intestine. (v) **Appendix**. (vi) **Mucosa-associated lymphoid tissue (MALT)**: located within the lining of the respiratory, digestive and urogenital tracts; about 50% of the body's lymphoid tissue lies in the MALT.

> **Final Answer:** Primary lymphoid organs: **bone marrow** and **thymus**. Secondary lymphoid organs: **spleen, lymph nodes, tonsils, Peyer's patches, appendix and MALT**.

☞ Exam Tip

A common 2-mark question. Always state the function (production + maturation vs. antigen interaction) along with the names. A bare list often loses one mark.

EXPERT'S SOLUTION *: Vivaan Reddy, M.Sc Biotechnology, AIIMS Delhi*

Strategic angle: factory vs. battlefield. Primary organs are the factory (make and train soldiers); secondary organs are the battlefield (where the soldiers meet the enemy antigen).

Step 1. **Factory.** Bone marrow makes all lymphocytes and matures B-cells. Thymus matures T-cells (T = thymus). Lymphocytes that pass quality control here become *immunocompetent*; the rest are killed (negative selection).

Step 2. **Battlefield.** Spleen filters blood; lymph nodes filter lymph; tonsils, Peyer's patches, appendix and MALT guard the body's mucosal openings (mouth, gut, airways, urogenital tract).

Step 3. **Self-test.** If asked 'where do B-cells mature?' $\rightarrow$ bone marrow. 'Where do T-cells mature?' $\rightarrow$ thymus. 'Where do lymphocytes meet antigen?' $\rightarrow$

secondary organs.

Why this matters. Removing the spleen (after trauma) leaves the patient vulnerable to encapsulated bacteria (pneumococcus, meningococcus). Thymic dysfunction (DiGeorge syndrome) causes T-cell deficiency. Each organ's role is a clinical reality.

> **Final Answer:** Bone marrow and thymus are primary; spleen, lymph nodes, tonsils, Peyer's patches, appendix and MALT are secondary.

Q 7.7 The following are some well-known abbreviations, which have been used in this chapter. Expand each one to its full form:
(a) MALT (b) CMI (c) AIDS (d) NACO (e) HIV

SOLUTION

Concept used. The chapter introduces several biomedical acronyms that recur in immunology and public health. Memorising both the full form and a one-line meaning is enough for board and NEET marks.

Step 1. **(a) MALT = Mucosa-Associated Lymphoid Tissue.** The diffuse lymphoid tissue embedded in the mucous lining of the respiratory, digestive and urogenital tracts. It is a secondary lymphoid tissue and constitutes about *50%* of the body's total lymphoid tissue.

Step 2. **(b) CMI = Cell-Mediated Immunity.** The arm of acquired immunity executed by activated *T-lymphocytes* (rather than circulating antibodies). It is responsible for defence against intracellular pathogens (viruses, *Mycobacterium*) and for the rejection of grafts.

Step 3. **(c) AIDS = Acquired Immuno-Deficiency Syndrome.** A deadly disease caused by HIV, marked by a progressive loss of helper T-cells, repeated opportunistic infections, weight loss, swollen lymph glands and ultimately death if untreated.

Step 4. **(d) NACO = National AIDS Control Organisation.** A body of the Ministry of Health and Family Welfare, Government of India, set up in 1992 to oversee HIV/AIDS prevention, diagnosis, treatment (ART) and awareness in India.

Step 5. **(e) HIV = Human Immuno-deficiency Virus.** The retrovirus that causes AIDS. It is an RNA virus with the enzyme reverse transcriptase; it infects helper T-cells ($CD4^+$) and macrophages of the human immune system.

Final Answer: (a) MALT = Mucosa-Associated Lymphoid Tissue; (b) CMI = Cell-Mediated Immunity; (c) AIDS = Acquired Immuno-Deficiency Syndrome; (d) NACO = National AIDS Control Organisation; (e) HIV = Human Immuno-deficiency Virus.

☞ **Exam Tip**

Pure full-form questions like this are easy 5-marks – but only if you also add one short defining line per acronym, as shown above. A naked full form (e.g. "AIDS = Acquired Immuno-Deficiency Syndrome") typically scores only half the mark.

EXPERT'S SOLUTION *: Diya Joshi, M.Sc Microbiology, JNU*

Strategic angle: pair acronym with picture. For each acronym, hold a one-image mental note: MALT → gut lining patrolled by lymphocytes; CMI → T-cells punching holes in an infected cell; AIDS → a depleted immune-cell graph; NACO → red-ribbon awareness drives; HIV → spiky envelope virus budding from a T-cell. Pictures stick better than letters.

Step 1. **MALT.** Mucosa-Associated Lymphoid Tissue. 50% of total body lymphoid tissue; first responders at the gut, airway and urogenital mucosae.

Step 2. **CMI.** Cell-Mediated Immunity. Mediated by T-lymphocytes; targets intracellular pathogens and rejects foreign tissue grafts.

Step 3. **AIDS.** Acquired Immuno-Deficiency Syndrome. Caused by HIV; defined by $CD4^+$ count < 200 cells/μL plus opportunistic infections.

Step 4. **NACO.** National AIDS Control Organisation, the apex body of the Government of India's HIV/AIDS response since 1992.

Step 5. **HIV.** Human Immuno-deficiency Virus. Family Retroviridae, genus Lentivirus; RNA genome reverse- transcribed and integrated into host DNA.

Why this matters. In NEET and Boards, full-form questions are easy marks. In the clinic, knowing the acronym is shorthand for knowing the disease.

Final Answer: MALT, CMI, AIDS, NACO, HIV expanded and defined as above.

Q 7.8 **Differentiate the following and give examples of each:**
(a) Innate and acquired immunity (b) Active and passive immunity

SOLUTION

Concept used. Immunity is the overall ability of the body to fight disease-causing organisms. It has two broad divisions based on *how* the protection is acquired: *innate* (present at birth) vs. *acquired* (developed during life). The acquired arm further splits into *active* (the body itself makes antibodies) vs. *passive* (ready-made antibodies are received from outside).

Step 1. **(a) Innate vs. Acquired immunity.**

Innate (non-specific) immunity	**Acquired (specific) immunity**
Present from birth; inherited from parents.	Develops over the lifetime in response to specific pathogens.
Non-specific: acts on every type of microbe.	Specific: responds to a particular pathogen.
Acts immediately; no time-lag.	Slower (5–14 days for primary response).
No immunological memory; same response every time.	Memory cells remember the pathogen; faster, stronger *secondary response* on re-exposure.
Four barriers: physical (skin, mucous), physiological (HCl in stomach, lysozyme in tears/saliva), cellular (PMN-leucocytes, monocytes, NK cells, macrophages), cytokine (interferons).	Two arms: humoral (B-cells make antibodies in blood) and cell-mediated (T-cells attack infected cells/grafts).
Example: skin keeps microbes out; stomach acid kills swallowed bacteria; lysozyme in tears destroys bacteria in the eye.	*Example:* antibodies produced after measles infection or after the MMR vaccine; T-cell killing of virus-infected cells; rejection of an unmatched organ graft.

Step 2. **(b) Active vs. Passive immunity.**

Active immunity	Passive immunity
The host's own immune system makes antibodies on exposure to the antigen.	Ready-made antibodies (antiserum) are received from outside.
Slow to develop (days to weeks) but long-lasting; memory cells form.	Acts at once but is short-lived (a few weeks); no memory.
Examples of *natural* active immunity: recovery from measles, mumps, chicken-pox. Examples of *artificial* active immunity: vaccination with polio, MMR, hepatitis-B, COVID-19 vaccine.	Examples of *natural* passive immunity: IgG antibodies transferred from mother to foetus through the placenta; IgA antibodies in colostrum to a newborn. Examples of *artificial* passive immunity: anti-tetanus serum (ATS), anti-snake-venom serum, monoclonal antibodies for COVID-19.

Final Answer: (a) **Innate**: inborn, non-specific, immediate, no memory (skin, stomach HCl, lysozyme, phagocytes). **Acquired**: develops during life, pathogen-specific, slower, has memory (antibodies after vaccination/recovery, T-cell graft rejection). (b) **Active**: body itself makes antibodies, slow, long-lasting (vaccination, natural recovery). **Passive**: ready-made antibodies given, quick, short-lived (anti-tetanus serum, mother's milk antibodies).

✗ Common Mistake

Students often write that 'vaccination is passive immunity'. Vaccines contain killed/weakened pathogens or antigens, so the body itself makes the antibodies. Vaccination is therefore *active* artificial immunity. Anti-tetanus serum, in contrast, is passive.

EXPERT'S SOLUTION *: Ananya Kumar, M.Sc Microbiology, JNU*

Strategic angle: who makes the antibody? If the body itself makes the antibody, the immunity is active. If the antibody comes ready-made from outside, the immunity is passive. The same logic, applied across natural and artificial routes, gives every example.

Step 1. **Innate vs. acquired in one line.** Innate is the bodyguard you are born with; acquired is the bodyguard you train through experience.

Step 2. **Examples, innate.** Skin, mucous, gastric HCl, tear lysozyme, ciliated airway, phagocytic neutrophils and macrophages, NK cells, interferons.

Step 3. **Examples, acquired.** Antibody response after measles or mumps, response to a tetanus toxoid booster, T-cell rejection of an unmatched kidney transplant.

Step 4. **Active immunity (body makes).** Natural: recovery from measles, chicken-pox, mumps. Artificial: every vaccine – polio, BCG, DPT, MMR, Hep B, COVID-19.

Step 5. **Passive immunity (body receives).** Natural: maternal IgG across the placenta, IgA in colostrum. Artificial: anti-tetanus serum, anti-rabies serum, anti-snake-venom serum, monoclonal antibodies (e.g. Casirivimab) for severe COVID-19.

Why this matters. A new-born is protected for ~6 months by maternal antibodies (passive). After that, vaccines (active) take over. Active and passive immunity, working together, keep a baby alive through the first vulnerable year.

Final Answer: Innate = inborn, non-specific, immediate. Acquired = learned, specific, slower, memory. Active = body makes the antibody (vaccine, infection). Passive = body receives the antibody (mother's milk, anti-tetanus serum).

Q 7.9 Draw a well-labelled diagram of an antibody molecule.

SOLUTION

Concept used. An **antibody (immunoglobulin, Ig)** is a Y-shaped protein produced by B-lymphocytes in response to a specific antigen. Each antibody molecule has *four polypeptide chains*: two identical **light (L) chains** and two identical **heavy (H) chains**. Because of this H_2L_2 composition, the molecule is symbolically written as $\mathrm{H_2L_2}$. The two arms of the Y end in identical **antigen-binding sites**, which recognise and bind the specific antigen. The chains are held together by disulphide ($-\mathrm{S}-\mathrm{S}-$) bonds. Each chain has a *variable* (V) region (the antigen-binding end) and a *constant* (C) region (the stem). The five major immunoglobulin classes are IgA, IgD, IgE, IgG and IgM, distinguished by their heavy-chain constant regions.

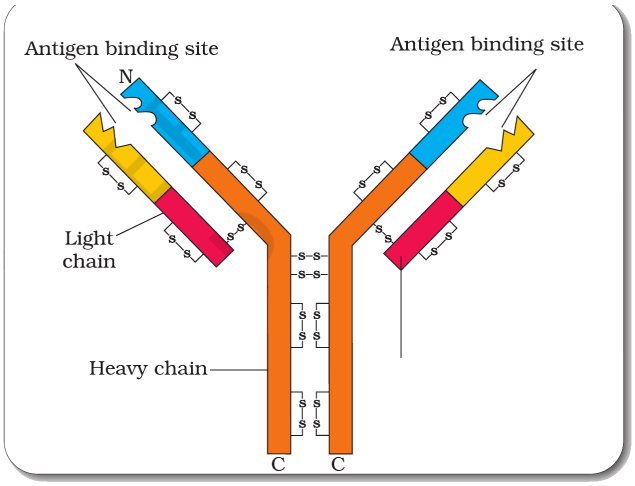


Figure 7.4 Structure of an antibody molecule

Fig. 7.4, NCERT Class 12 Biology, Chapter 7 (Human Health and Disease): structure of an antibody molecule, showing the two heavy chains, two light chains, the disulphide bonds and the two antigen-binding sites.

Step 1. **Identify the four chains.** Two long *heavy chains* run as the trunk and inner arm of the Y. Two short *light chains* sit on the outside, one alongside each heavy arm.

Step 2. **Mark the antigen-binding sites.** The two upper tips of the Y, where one light and one heavy chain meet, are the identical antigen-binding sites. Each binds one antigen molecule, so a single antibody can bind two antigens.

Step 3. **Mark the disulphide bonds.** Several $-\mathrm{S}-\mathrm{S}-$ bridges hold the heavy chains to each other (in the hinge region) and each light chain to its heavy chain. They keep the four-chain assembly together.

Step 4. **Mark the variable (V) and constant (C) regions.** The V region (the upper part of each arm) is highly variable across antibodies and gives them their specificity. The C region (the lower part of the stem) is conserved within each Ig class and decides effector function. Label the N-terminus and C-terminus accordingly.

Step 5. **State the formula.** Because there are two H and two L chains, the formula is $\mathbf{H_2L_2}$.

Final Answer: An antibody is a Y-shaped protein with $\mathbf{H_2L_2}$ four-chain composition: 2 heavy + 2 light chains linked by $-\mathrm{S}-\mathrm{S}-$ bonds, ending in two identical antigen-binding sites at the variable (V) tips of the Y, with the constant (C) stem deciding the immunoglobulin class (IgA, IgD, IgE, IgG, IgM).

♥ Why the Y-shape matters

The Y-shape lets one antibody crosslink two antigens, clumping microbes together so

phagocytes can engulf them more easily. This *agglutination* is the visible end-point of many lab tests, for example the Widal test for typhoid and routine blood-group typing.

EXPERT'S SOLUTION *: Ishaan Bhat, Ph.D Molecular Biology, NCBS Bangalore*

Strategic angle: dissect by chain, by region, by function. First name the chains, then the bonds, then the regions, then the function. The same logic gives a clean labelled drawing every time.

Step 1. **Chains.** 2 light (L) chains $\approx$ 25 kDa each; 2 heavy (H) chains $\approx$ 50 kDa each. Total mass $\approx$ 150 kDa. Symbol H_2L_2.

Step 2. **Bonds.** Inter-chain disulphide bonds link L–H and H–H in the hinge region. Intra-chain disulphide loops give each chain its globular domain structure.

Step 3. **Regions.** V_L + V_H at the tip = antigen- binding site (Fab end). C_L + C_H at the stem = Fc end, which binds Fc receptors on macrophages and complement proteins.

Step 4. **Classes.** The C_H heavy-chain isotype defines the class: μ in IgM, γ in IgG, α in IgA, ε in IgE, δ in IgD. Each has a characteristic effector role (IgG crosses placenta, IgA in mucosa, IgE in allergy, IgM is the first responder).

Step 5. **Specificity.** The V regions form a 3-D pocket ($\sim$ 10 kDa wide) shaped to fit one antigen epitope. One clone of B-cell makes only one specificity.

Why this matters. Knowing the parts of an antibody is the basis of every immunological technique: ELISA, Western blot, immunofluorescence, antibody therapy for cancer (Rituximab, Trastuzumab) and for COVID-19 (Casirivimab, Imdevimab).

Final Answer: Y-shaped, four-chain $\mathbf{H_2L_2}$ protein with two identical antigen-binding sites at the Fab tips and an Fc effector stem.

Q 7.10 What are the various routes by which transmission of human immuno-deficiency virus takes place?

SOLUTION

Concept used. HIV is a retrovirus that lives inside cells of the human immune system and inside body fluids (blood, semen, vaginal secretions, breast-milk). It is fragile outside the body and *cannot* spread through air, food, water, casual contact, mosquitoes, sharing toilets or insect bites. It can only spread when one of these contaminated body fluids of an infected person enters the bloodstream of a healthy person. Hence there are four main routes.

Step 1. **Sexual contact.** Unprotected sexual intercourse (vaginal, anal or oral) with an HIV-infected person is the commonest route. Semen and vaginal secretions of the infected partner carry the virus; small abrasions in the mucosa of the healthy partner let it enter the blood.

Step 2. **Blood and blood products.** Transfusion of infected blood or blood products (plasma, platelets, clotting factors in haemophiliacs) was a major route before donor screening became universal. Today, mandatory ELISA screening has largely closed this route in regulated blood banks.

Step 3. **Sharing of infected needles or syringes.** Common among intravenous drug users who share needles; also a risk during unsafe medical injections, ear-piercing, tattooing and acupuncture with poorly sterilised equipment.

Step 4. **Mother-to-child (vertical) transmission.** An HIV-positive mother can transmit the virus to her child: (i) across the placenta during pregnancy, (ii) through blood contact during childbirth, or (iii) through breast-milk during nursing.

☞ **HIV does NOT spread by**

Mosquito bites, sharing utensils, hugging, shaking hands, swimming pools, toilet seats, sweat, sneezing, or eating together. Knowing this is as important as knowing the real routes, because the social stigma is what kills patients alongside the disease.

Final Answer: HIV spreads through four established routes only: (i) unprotected sexual contact with an infected partner; (ii) transfusion of infected blood/blood products; (iii) sharing of infected needles, syringes or sharp instruments; (iv) from an infected mother to her child during pregnancy, delivery or breast-feeding.

✗ **Common Mistake**

Writing ‘HIV spreads through mosquito bites’ or ‘HIV spreads by sharing food’ is wrong and loses marks. The virus survives only in specific body fluids and not in the mosquito’s salivary glands.

EXPERT’S SOLUTION *: Tara Singh, M.Sc Microbiology, JNU*

Strategic angle: fluid carries virus into blood. HIV needs a contaminated body fluid (blood, semen, vaginal fluid, breast-milk) of an infected person to physically reach the blood of a healthy person. The four routes are exactly the four ways such a contact happens.

Step 1. **Sex.** Unprotected vaginal, anal or oral intercourse with an HIV-positive partner. Use of latex condoms blocks this route.

Step 2. **Blood.** Transfusion of unscreened blood or blood products; sharing of needles among injecting drug users; unsafe medical and cosmetic procedures with unsterile equipment.

Step 3. **Vertical.** An infected mother passes the virus to her baby in utero, during delivery, or through breast-milk. Antiretroviral therapy of the mother during pregnancy plus formula feeding (where safe) cuts the risk to below 1%.

Step 4. **Occupational, rare.** Needle-stick injuries in healthcare workers; the risk is small (about 0.3% per injury) and is reduced further by post-exposure prophylaxis (PEP).

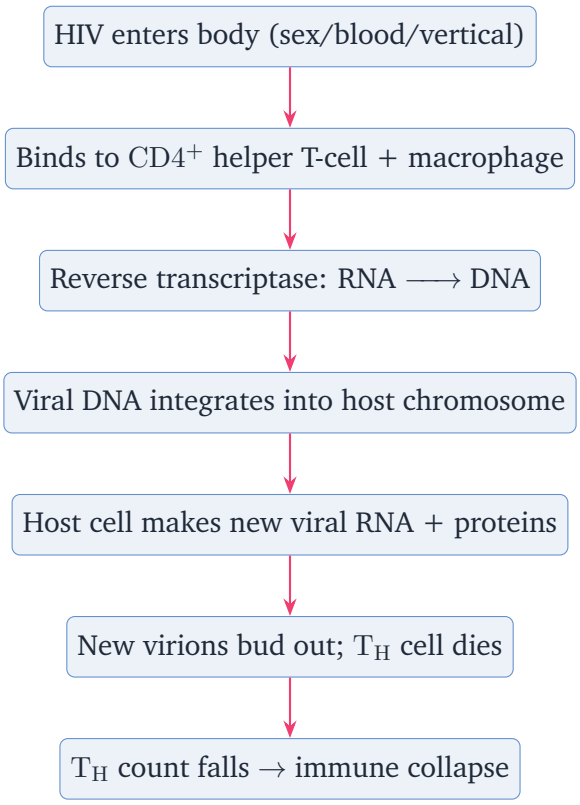
Why this matters. Each route has a specific block: condom for sex, screening + needle-exchange for blood, ART + safe delivery + formula feeding for mother-to-child. India's NACO programme runs all three blocks together.

> **Final Answer:** Sexual contact, infected blood/needles and infected mother to child: the four established HIV transmission routes.

Q 7.11 What is the mechanism by which the AIDS virus causes deficiency of immune system of the infected person?

SOLUTION

Concept used. The **AIDS virus** (HIV) is a **retrovirus**: its genome is single-stranded RNA enclosed in a protein coat surrounded by a lipid envelope studded with glycoprotein spikes. Inside its capsid it also carries the enzyme **reverse transcriptase**. The virus specifically targets the **helper T-lymphocytes (T_H cells, also called $CD4^+$ T-cells)** and macrophages of the human immune system. Repeated cycles of infection, replication and killing of T_H cells progressively destroy this central co-ordinator of the immune response, leaving the patient unable to defend against secondary infections. That progressive loss is the *immunodeficiency* of AIDS.



Step 1. **Entry.** After transmission, HIV enters macrophages. Its envelope glycoprotein (gp120) binds specifically to the $CD4$ receptor on macrophages and helper T-cells, and the virus is taken into the cell.

Step 2. **Reverse transcription.** Inside the cell, the viral enzyme *reverse transcriptase* copies the single- stranded RNA genome into a double-stranded DNA. This step gives the retrovirus its name (the reverse of the usual $\mathrm{DNA} \longrightarrow \mathrm{RNA}$ flow).

Step 3. **Integration.** The newly synthesised viral DNA enters the nucleus of the host cell and integrates into the host chromosome (catalysed by viral integrase). The virus now lives as a permanent passenger of the cell.

Step 4. **Replication and budding.** The infected cell is directed by the integrated viral DNA to produce new viral RNA genomes and viral proteins. These assemble at the cell membrane and *bud off* as new virions, which infect more macrophages and helper T-cells.

Step 5. **Destruction of helper T-cells.** Each round of replication destroys the infected T_H cell. Because T_H cells are the central co-ordinators of the immune response (they activate B-cells for antibody production and activate cytotoxic T-cells), a progressive fall in their number cripples both humoral and cell-mediated immunity. Normal $CD4^+$ count is ~ 1000 cells/μL; in AIDS it drops below 200.

Step 6. **Opportunistic infections.** With the immune defence gone, organisms that the body normally easily controls (*Mycobacterium tuberculosis*, *Pneumocystis jirovecii*, *Cryptococcus*, *Toxoplasma*, cytomegalovirus, *Candida*) cause severe and

recurrent infections, and certain cancers (Kaposi's sarcoma) develop. These are the clinical symptoms of AIDS.

> **Final Answer:** HIV binds the $\mathrm{CD4}$ receptor of helper T-cells/macrophages, reverse-transcribes its RNA into DNA, integrates into the host chromosome, replicates and buds out, killing the $\mathrm{T_H}$ cell. The progressive loss of $\mathrm{T_H}$ cells cripples the coordination of both humoral and cell-mediated immunity, leaving the patient open to opportunistic infections and cancers – the immunodeficiency we call AIDS.

☞ Exam Tip

A 5-mark question. Answer in two parts: (i) the molecular cycle (entry → RT → integration → replication → budding) and (ii) the immunological consequence ($\mathrm{T_H}$ loss → opportunistic infections). A clean cycle diagram earns a bonus.

EXPERT'S SOLUTION *: Krishna Nair, Ph.D Molecular Biology, NCBS Bangalore*

Strategic angle: a retrovirus that hijacks the conductor. The helper T-cell is the conductor of the immune orchestra; HIV is a parasite that lives in the conductor and slowly silences him, after which the orchestra goes to pieces.

Step 1. **Receptor.** HIV's gp120 envelope spike fits the $\mathrm{CD4}$ receptor on helper T-cells and macrophages (and the coreceptor CCR5/CXCR4). This is the specificity that makes the virus immune-tropic.

Step 2. **Genome flow.** A retrovirus flips the central dogma: RNA $\xrightarrow{\text{reverse transcriptase}}$ DNA inside the host. That DNA integrates into the host genome as a *provirus*, becoming a permanent part of the cell.

Step 3. **Slow burn.** For months to years the provirus lies relatively quiet (the asymptomatic phase). Stress signals and cytokines turn on transcription, and the cell starts making viral particles, which kill it.

Step 4. **Numbers.** The body initially replaces lost $\mathrm{T_H}$ cells, but the bone marrow cannot keep pace with the rate of destruction. Over 5–10 years the $\mathrm{CD4^+}$ count crashes from ~ 1000 to $< 200/\mu$L. Below 200, AIDS is declared.

Step 5. **Consequence.** Without $\mathrm{T_H}$ cells, the immune orchestra cannot mount an effective response. Common microbes become deadly: *Pneumocystis* pneumonia, TB, cryptococcal meningitis, candidiasis, CMV retinitis, Kaposi's sarcoma.

Why this matters. Knowing the mechanism gave us antiretroviral therapy: reverse-transcriptase inhibitors (zidovudine, tenofovir), protease inhibitors and integrase inhibitors. Triple-drug ART now turns AIDS from a death-sentence into a

chronic illness with near-normal life expectancy.

> **Final Answer:** HIV binds $CD4^+$ T-cells, reverse-transcribes its RNA to DNA, integrates as provirus, replicates, and kills the T_H cell. Progressive loss of T_H cells cripples both arms of immunity $\rightarrow$ opportunistic infections $\rightarrow$ AIDS.

Q 7.12 How is a cancerous cell different from a normal cell?

SOLUTION

Concept used. A **normal cell** grows and divides under the strict control of two systems: *contact inhibition* (cells stop dividing once they touch their neighbours) and *cell-cycle checkpoints* (the cell only divides when growth signals and DNA repair allow it). A **cancerous cell** (neoplastic cell) is one in which these controls have broken down: it divides repeatedly, ignores neighbour signals, refuses to die when damaged, and may break free of its original site.

Step 1. **Loss of contact inhibition.** Normal cells stop dividing on touching neighbours, so a culture of normal cells forms a single layer (monolayer). Cancer cells ignore this contact signal and pile up on each other, forming multilayered, disorganised masses.

Step 2. **Uncontrolled, rapid division.** Normal cells divide a limited number of times and only when needed (replacement, repair). Cancer cells divide continuously and rapidly, producing a mass of cells called a **tumour**.

Step 3. **Loss of differentiation.** Normal cells differentiate into specialised cell types (e.g. liver cells, skin cells). Cancer cells lose their normal shape, size and function; they look immature (anaplastic) under the microscope.

Step 4. **Failure to die (apoptosis).** Normal cells with damaged DNA trigger programmed death (apoptosis). Cancer cells escape apoptosis because regulatory genes such as $p53$ are mutated; damaged cells survive and accumulate.

Step 5. **Invasion and metastasis (in malignant tumours).** Normal cells stay in their organ of origin. Malignant cancer cells break out of the primary site, invade surrounding tissues, enter blood and lymph, and seed distant organs — this is metastasis. Benign tumours, by contrast, stay encapsulated.

Step 6. **Angiogenesis.** Malignant tumours secrete factors (VEGF) that recruit new blood vessels to feed themselves.

Step 7. **Genetic changes.** Cancer cells carry mutations in **proto-oncogenes** (converted

to oncogenes that drive growth) and in **tumour-suppressor genes** (e.g. $p53$, RB, which normally restrain growth). Normal cells have intact copies.

Step 8. **Other features.** Cancer cells often have abnormal chromosome numbers (aneuploidy), high telomerase activity (so they do not senesce), altered metabolism (Warburg effect), and elevated tumour markers (CEA, PSA, AFP).

Normal cell	Cancerous cell
Shows contact inhibition; divides only when needed.	No contact inhibition; divides continuously.
Limited number of divisions; senesces.	Almost unlimited divisions; immortal.
Specialised (differentiated).	Poorly differentiated (anaplastic).
Stays in its tissue of origin.	Can invade and metastasise.
Undergoes apoptosis if damaged.	Escapes apoptosis ($p53$ inactive).
Normal chromosome number.	Aneuploidy, chromosomal rearrangements.
Normal blood supply.	Stimulates new blood vessels (angiogenesis).
Genome intact.	Mutated proto-oncogenes and tumour-suppressor genes.

Final Answer: Cancer cells differ from normal cells in losing contact inhibition, dividing uncontrollably, losing differentiation, escaping apoptosis, sustaining their own blood supply, harbouring mutated oncogenes and tumour-suppressor genes, and (in malignant tumours) invading neighbouring tissues and metastasising to distant organs.

♥ The eight hallmarks of cancer

Hanahan and Weinberg's classic 2000 paper grouped these differences into eight hallmarks: sustained growth signalling, escape from growth suppressors, escape from cell death, immortality, induced angiogenesis, invasion-metastasis, altered metabolism, and immune evasion. Modern cancer therapies aim at one or more of these hallmarks (anti-VEGF for angiogenesis; checkpoint inhibitors for immune evasion).

EXPERT'S SOLUTION *: Yash Verma, M.Sc Biotechnology, AIIMS Delhi*

Strategic angle: rules-followed vs. rules-broken. A normal cell follows the rules of the tissue community: divide only when asked, stop when neighbours say so, die if damaged, stay home. A cancer cell breaks all of them.

Step 1. **Rule 1: Divide only when asked.** Normal cells need growth signals (growth factors). Cancer cells synthesise their own or have constitutively active

receptors (Ras mutations).

Step 2. **Rule 2: Stop when neighbours say so.** Normal cells feel contact inhibition; cancer cells ignore it.

Step 3. **Rule 3: Die if damaged.** Normal cells with damaged DNA undergo $p53$-mediated apoptosis. Cancer cells have $p53$ mutations and survive.

Step 4. **Rule 4: Stay home.** Normal epithelial cells stay within the basement membrane. Cancer cells secrete proteases (MMP9), break out and seed distant organs (metastasis).

Step 5. **Rule 5: Run out of divisions.** Normal cells exhaust their telomeres after ~ 50 divisions. Cancer cells reactivate *telomerase* and become immortal.

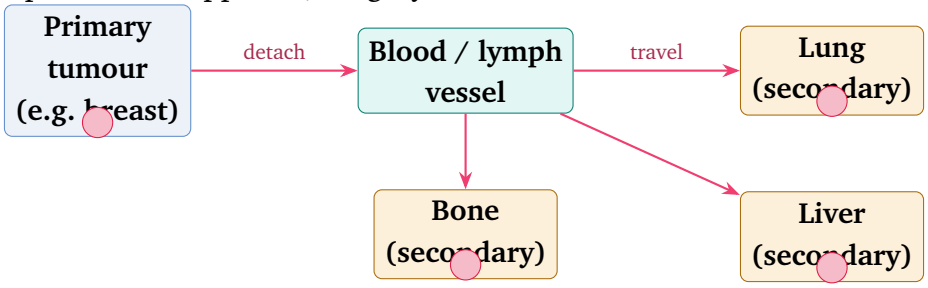
Why this matters. Every modern cancer drug, from imatinib to nivolumab, targets a specific broken rule. Diagnosis (biopsy + histopathology + molecular markers) and therapy follow directly from these differences.

> **Final Answer:** Cancer cells lose contact inhibition, divide without limit, lose differentiation, escape apoptosis, build their own blood supply and (in malignancy) invade and metastasise to distant organs. Normal cells obey all these rules; cancer cells break them all.

Q 7.13 Explain what is meant by metastasis.

SOLUTION

Concept used. Metastasis is the most dangerous property of malignant tumours. It is the process by which cancer cells of a primary (original) tumour break away, travel through the *bloodstream* and/or *lymphatic system* to distant parts of the body, settle there and grow into new (secondary) tumours. The word literally means *a change of place*. It is the single most important reason why cancer is hard to treat – once metastatic spread has happened, surgery alone cannot remove all the disease.



Step 1. **Detachment.** Cancer cells in a malignant primary tumour lose cell-to-cell adhesion (down-regulated E-cadherin) and break out of their tissue of origin.

Step 2. **Invasion.** They secrete proteolytic enzymes (matrix metalloproteinases) that digest the basement membrane and surrounding extracellular matrix, allowing the cells to push into nearby tissues.

Step 3. **Intravasation.** The cells enter the lumen of blood capillaries or lymphatic vessels.

Step 4. **Circulation.** Tumour cells travel as circulating tumour cells (CTCs) through blood or lymph. Most are destroyed by the immune system; a few survive.

Step 5. **Extravasation.** A surviving cell adheres to the endothelium of a distant capillary, squeezes between endothelial cells, and enters the new tissue.

Step 6. **Colonisation.** The cell multiplies at the new site, recruits new blood vessels (angiogenesis), and grows into a *secondary* (metastatic) tumour. Common sites of metastasis are the liver, lungs, bones and brain.

> **Final Answer:** Metastasis is the spread of cancer cells from a malignant primary tumour, through the blood or lymphatic system, to distant organs, where they form new (secondary) tumours. It is the property that makes malignant cancer especially dangerous, because the disease is no longer confined to one site.

✗ Common Mistake

A common error is to confuse metastasis with invasion. *Invasion* is local spread into neighbouring tissues; *metastasis* is distant spread via blood or lymph. Benign tumours may invade locally but do *not* metastasise.

EXPERT'S SOLUTION *: Sneha Desai, M.Sc Microbiology, JNU*

Strategic angle: travel itinerary of a cancer cell. Think of a malignant cell as an unwanted traveller. It packs (loses adhesion), exits the building (basement membrane), boards transport (blood/lymph), gets off at a far station (extravasation) and rents a new flat (colonisation).

Step 1. **Pack.** Loss of E-cadherin lets cells separate.

Step 2. **Exit.** MMP-secretion dissolves the basement membrane.

Step 3. **Transport.** Entry into capillaries or lymph vessels.

Step 4. **Survive the trip.** Resist immune attack and haemodynamic shear stress.

Step 5. **Disembark.** Adhere to vessel wall at a distant organ, transmigrate through the endothelium.

Step 6. **Settle.** Colonise the new tissue; trigger angiogenesis to feed the new colony;

grow into a secondary tumour.

Why this matters. 90% of cancer deaths are from metastasis, not from the primary tumour. Treatments that block any of the six steps (e.g. anti-angiogenics, MMP inhibitors, checkpoint inhibitors) are an active area of research.

> **Final Answer:** Metastasis is the journey of malignant cells from the primary tumour, through blood/lymph, to distant organs, where they form secondary tumours — the property that defines malignancy.

Q 7.14 List the harmful effects caused by alcohol/drug abuse.

SOLUTION

Concept used. Drug/alcohol abuse is the use of drugs (opioids, cannabinoids, cocaine, hallucinogens, tobacco) and alcohol for purposes *other* than medical, in amounts and frequencies that harm the user, the family and society. The harm acts at three levels: (i) the body of the user, (ii) the user's mind and behaviour, (iii) the user's family and society.

Step 1. **Immediate physical effects.** Reckless behaviour, impaired judgement, accidents on the road, vandalism, violence. Heroin and alcohol overdose can cause respiratory depression and death. Loss of inhibition leads to unsafe sex, raising the risk of HIV and hepatitis B/C.

Step 2. **Long-term effects on the body.**

- *Liver:* chronic alcohol use causes fatty liver, alcoholic hepatitis and cirrhosis; long-term opioid use damages the liver too.
- *Heart and lungs:* tobacco causes hypertension, atherosclerosis, coronary artery disease, chronic bronchitis, emphysema and lung cancer.
- *Nervous system:* chronic abuse causes memory loss, dementia, peripheral neuropathy and Wernicke– Korsakoff syndrome (in alcoholics).
- *Reproductive system:* reduced fertility, impotence in males, foetal alcohol syndrome in babies of alcoholic mothers (low birth weight, mental retardation, facial anomalies).
- *Infectious diseases:* HIV, hepatitis B and C spread through shared needles among intravenous drug users.

Step 3. **Psychological effects.** Dependence and addiction, depression, anxiety, paranoia, hallucinations (with cannabinoids), suicidal tendency, and a sharp drop in academic and work performance.

Step 4. **Withdrawal effects.** Once dependent, stopping the drug suddenly produces severe nausea, sweating, tremors, muscle pain, hallucinations and seizures; this withdrawal can be life-threatening.

Step 5. **Tolerance and escalation.** The body adapts so that the same dose stops giving the same 'kick'; the user takes progressively larger doses, increasing the risk of overdose and toxicity.

Step 6. **Family and social effects.** Disturbed family relationships, financial ruin, child neglect, domestic violence, school drop-out, and loss of employment. Generations of 'addicts of addicts' if the cycle is not broken.

Step 7. **Crime and societal cost.** To finance the habit, an addict may steal or sell stolen goods, sell illegal drugs, engage in prostitution. Drug trafficking and gang violence weaken the social fabric.

Step 8. **Use of performance-enhancing drugs in sport.** Anabolic steroids cause acne, mood swings, depression, liver damage, kidney failure, masculinisation in females (facial hair, deepened voice, menstrual irregularities), feminisation in males (breast enlargement, shrunken testes), aggression and premature heart disease.

> **Final Answer:** Drug and alcohol abuse harm the body (liver cirrhosis, heart disease, lung damage, brain damage, HIV/hepatitis from shared needles), the mind (addiction, depression, hallucinations, suicide), the family (financial ruin, neglect, violence) and society (crime, loss of productivity). They also raise the risk of overdose, foetal damage and premature death.

☞ Exam Tip

Group your answer into *physical*, *mental*, *family/social* and *economic* effects. This 4-bucket structure earns full marks in 5-mark questions.

EXPERT'S SOLUTION *: Aditya Chatterjee, M.Sc Biotechnology, AIIMS Delhi*

Strategic angle: four concentric circles. Imagine four circles widening around the user – self, family, peers, society. Each gets damaged by drug abuse in characteristic ways.

Step 1. **Self.** Acute effects (loss of inhibition, accidents, overdose). Chronic effects (cirrhosis, lung cancer, brain damage, infertility, HIV/HCV from shared needles). Psychological dependence, depression, suicide.

Step 2. **Family.** Disturbed relationships, child neglect, domestic violence, financial bankruptcy, loss of trust. Foetal alcohol syndrome in unborn children of addicted mothers.

Step 3. **Peers.** Spread of the habit; pressure to share needles; involvement in trafficking; school drop-outs.

Step 4. **Society.** Crime to finance the habit (theft, prostitution); reduced productivity; healthcare burden; organised drug trafficking and gang violence.

Why this matters. The total social cost of substance abuse is enormous (lost lives, lost work, healthcare bills). Prevention through early education, family support and timely de-addiction is cheaper and far more effective than treatment of long-standing addiction.

> **Final Answer:** Drug/alcohol abuse damages the user's body (liver, heart, lungs, brain, reproductive system), the user's mind (addiction, depression, suicide), the family (neglect, violence, foetal damage) and society (crime, productivity loss, healthcare burden).

Q 7.15 **Do you think that friends can influence one to take alcohol/drugs? If yes, how may one protect himself/herself from such an influence?**

SOLUTION

Concept used. Adolescence is a stage where the urge to belong is strong and the brain's risk-reward circuitry is still maturing. **Peer pressure**, the influence of friends and classmates, is one of the leading reasons that teenagers *first* try a drug or a drink. Studies and the NCERT text itself note that *the company we keep* is the single most important external factor in adolescent drug initiation. Hence the honest answer is yes, friends can strongly influence one to take alcohol or drugs, especially during early teenage.

Step 1. **How peer pressure operates.** An adolescent wants to fit in, be accepted, be seen as 'cool' or grown-up. When the peer group experiments with cigarettes, alcohol or drugs, the urge not to be left out, the fear of being teased, and the temptation of 'just trying once' often win over good sense.

Step 2. **Curiosity and adventure-seeking.** Teenagers seek novelty, take risks and like to challenge rules. Friends amplify this curiosity: 'try it once and see, nothing will happen'.

Step 3. **Imitation of role-models.** Older siblings, popular seniors and celebrities glamorised in films or social media are imitated. A friend who looks up to such a figure passes the influence on.

☞ **Recognise the moment of choice**

Almost every addiction begins with one moment when a friend says, "Try this". Knowing in advance that this moment will come and deciding the answer in advance is half the protection.

How to protect oneself.

Step 1. **Choose friends with care.** Friendships built around sports, reading, music, dance, debate or community service give the same sense of belonging without the drug. Avoid groups where substance use is a routine 'fun'.

Step 2. **Be assertive without being aggressive.** A firm, polite 'No, thanks' practised in advance is usually enough. One does not have to argue or apologise. True friends will accept the refusal.

Step 3. **Build self-esteem and self-identity.** A teenager with strong self-respect, a sport or hobby and clear goals does not need a drug to feel adult. Parents, teachers and counsellors can help by acknowledging the student's achievements and giving responsibility.

Step 4. **Channel curiosity and energy into healthy outlets.** Sports, music, art, NCC, NSS, scouts, debate, trekking, environmental clubs. These give a real 'high' of achievement and friendship.

Step 5. **Talk to parents and teachers without fear.** Open communication with adults at home and at school is a strong safety net. If something has already been tried, admitting it early is much safer than hiding it.

Step 6. **Seek help early.** Counsellors, NACO helpline, de-addiction centres, family physicians. The earlier one seeks help, the easier the way back.

Step 7. **Learn the facts about drugs.** Knowing what cigarettes, alcohol, cannabis and opioids actually do to the body and mind (the harmful effects listed in Q14) replaces glamour with realism.

Final Answer: Yes, friends – especially during adolescence – are a strong influence on drug or alcohol initiation. The way to protect oneself is to choose friends with care, learn to say a firm 'No', build self-esteem and goals, channel energy into sports/hobbies, talk openly with parents and teachers, learn the facts about drugs, and seek counselling early if needed.

♥ **The window of first use**

Most lifelong addicts trace their first use to age 13–17. Protecting the adolescent for these few years often protects them for life.

EXPERT'S SOLUTION *: Meera Pillai, M.Sc Microbiology, JNU*

Strategic angle: yes, but plan the defence. Accept the risk plainly and then list the seven defences. This direct structure is what examiners want.

Step 1. **Acceptance.** Yes, peer pressure is a leading cause of teenage substance use, recognised by WHO, NCERT and every public-health agency.

Step 2. **Choice of company.** Spend time with friends who share constructive interests.

Step 3. **Refusal skills.** 'No' is a complete sentence. Practise it with parents/siblings.

Step 4. **Self-worth.** Build identity through achievement, sport, art, leadership. Self-respect is the strongest immune system against peer pressure.

Step 5. **Healthy outlet.** Channel curiosity, energy and adventure into sport, music, trekking, debating, theatre.

Step 6. **Open communication.** Talk to parents and teachers without fear of judgement.

Step 7. **Information.** Read about what drugs actually do (Q14 effects).

Step 8. **Professional help.** Counsellors, NACO helpline, de-addiction centres are available and confidential.

Why this matters. Schools and parents who teach refusal skills, build self-esteem and keep dialogue open cut substance-use rates among their teens by more than half. Prevention is teachable.

> **Final Answer:** Yes, peers influence first use. Defence: good company, firm 'No', strong self-esteem, healthy hobbies, open talk with parents/teachers, factual knowledge of drug harms, early professional help.

Q 7.16 **Why is that once a person starts taking alcohol or drugs, it is difficult to get rid of this habit? Discuss it with your teacher.**

SOLUTION

Concept used. Alcohol and drugs of abuse act on the **reward pathway** of the brain (the meso-limbic dopaminergic pathway, including the nucleus accumbens and the ventral tegmental area). They flood it with the neurotransmitter *dopamine*, far above the levels released by natural rewards like food, music or friendship. With repeated use the brain adapts to this artificial surge, producing two stubborn phenomena: **tolerance** (progressively larger doses are needed to feel the same kick) and **dependence** (the body and mind *need* the drug to feel normal). Together they make stopping extremely hard.

Step 1. **Tolerance.** On repeated exposure, dopamine receptors in the reward pathway

down-regulate (their number falls). The same dose now produces a smaller effect, so the user takes a larger dose to feel the previous 'kick'. This escalation traps the user in an ever-rising spiral of use.

Step 2. **Physical dependence.** The body alters its own biochemistry to function in the constant presence of the drug. When the drug is stopped, the altered biochemistry produces *withdrawal symptoms* – tremors, sweating, cramping, nausea, vomiting, seizures, severe anxiety, sometimes hallucinations and even death. The simplest way to make these symptoms go away is to take the drug again.

Step 3. **Psychological dependence (craving).** The brain learns to associate the drug with relief from stress, boredom or sadness. The memory of pleasure becomes so strong that even years after the body has detoxed, a familiar place, friend or smell can trigger an intense craving and bring the user back to use (relapse).

Step 4. **Reward pathway rewiring.** Natural rewards (food, music, friendship) stop feeling rewarding because the brain has reset its baseline to the drug. The user feels flat and joyless without the substance, deepening the dependence.

Step 5. **Social factors.** Continued company with using friends, easy availability of the drug, family discord, unemployment, and stigma around seeking help all keep pulling the person back to the substance.

Step 6. **Triggers and relapse.** Even after de-addiction, triggers such as stress, a known location, an old friend, or simply the time of day at which the user once consumed the drug can fire the craving. This is why relapse is common and why long-term support (counselling, family, de-addiction groups) is essential.

> **Final Answer:** Because alcohol and drugs hijack the brain's reward pathway, the user develops tolerance (progressively larger doses needed for the same effect), physical dependence (severe withdrawal on stopping), psychological dependence (deep cravings) and a rewired reward circuit. These changes, combined with social pressures and easy availability, make it extremely hard to break the habit once it has been formed.

♥ Addiction is a brain disease

The WHO and modern neuroscience define addiction as a chronic, relapsing brain disease, not a moral failing. Understanding this removes blame and opens the door to treatment – medical de-addiction, behavioural therapy, group support (Alcoholics Anonymous, Narcotics Anonymous), family counselling and, where indicated, medication.

EXPERT'S SOLUTION *: Rohit Gupta, Ph.D Molecular Biology, NCBS Bangalore*

Strategic angle: three locks on the door. Tolerance, dependence and craving are three locks the drug puts on the way out. Each must be opened to stop using.

Step 1. **Lock 1: Tolerance.** Receptors down-regulate; doses rise. Breaking it needs medically supervised dose tapering.

Step 2. **Lock 2: Physical dependence.** Withdrawal is so unpleasant that users restart the drug just to feel normal. Breaking it needs supportive medical care: rehydration, sedatives for seizures, gradual taper, substitution (methadone, buprenorphine) where indicated.

Step 3. **Lock 3: Psychological dependence.** Cues from the past trigger cravings. Breaking it needs cognitive- behavioural therapy, group support (AA, NA), and a rebuilt social environment far from old triggers.

Step 4. **Add social glue.** Family support, employment, purpose. Recovery rates double when the recovering person has a job, a home and an accepting community.

Step 5. **Prevent relapse.** Identify triggers, plan responses, stay close to a support group long after the last use. Recovery is a lifelong project, not a one-time event.

Why this matters. 'Just stop' is poor medical advice. Recognising addiction as a brain disease with three locks helps both the patient and the family approach recovery scientifically.

> **Final Answer:** Drug abuse rewires the reward pathway, producing tolerance, physical and psychological dependence. The combined neurobiology and social environment make stopping very hard, and recovery needs medical, psychological and social support together.

Q 7.17 In your view what motivates youngsters to take to alcohol or drugs and how can this be avoided?

SOLUTION

Concept used. A youngster's first contact with alcohol or drugs is rarely random. It is driven by a cluster of *push factors* (curiosity, peer pressure, family stress, social glamourisation) and *pull factors* (an immediate thrill of relaxation or excitement). Avoidance therefore needs an equally multi-pronged response.

What motivates youngsters.

Step 1. **Curiosity and thrill-seeking.** Adolescents are biologically wired to seek novelty and adventure. 'What if I try it once?' is a powerful pull.

Step 2. **Peer pressure.** Friends who already use drugs may tease, dare or persuade. Wanting to fit in beats common sense (also covered in Q15).

Step 3. **Family stress.** Frequent quarrels, lack of warmth at home, unrealistic academic expectations, divorce or neglect push the child to look for escape.

Step 4. **Failure to cope with academic and personal pressures.** Heavy syllabus, competition, exam fear and peer comparison drive some adolescents to relaxants (alcohol, cannabinoids) or stimulants (cocaine, amphetamines).

Step 5. **Glamourisation in media.** Films, web series, music videos and social media often depict drinking and drug use as 'cool', adult or rebellious. Young viewers imitate their on-screen idols.

Step 6. **Easy availability.** If alcohol, cigarettes and gateway drugs (cannabis) are easily available near schools and colleges, experimentation is far more likely.

Step 7. **Misinformation.** 'Everyone does it', 'It is harmless', 'I can stop whenever I want'. Such myths, spread by older peers and the internet, lower the barrier to first use.

Step 8. **Need for an identity and rebellion.** Adolescence is a phase of identity formation. Some youngsters use drugs as a way to assert independence from parents and teachers.

How this can be avoided.

Step 1. **Early education about drugs.** School syllabi should include factual information on what each drug does to the body and brain, replacing glamour with realism (the Q14 effects).

Step 2. **Strong, warm and communicative families.** Parents who listen, who set reasonable expectations, who share meals and conversations with their children, who watch for early signs (changing friends, mood swings, falling grades, money missing) and who address them gently — these families have the lowest rates of drug abuse.

Step 3. **Teachers and counsellors as a safety net.** Trained school counsellors, accessible without fear of punishment, can catch trouble early. Anonymous helplines (NACO, de-addiction centres) extend the net.

Step 4. **Avoid peer-pressure traps.** Teach refusal skills, encourage choice of constructive peer groups, build self-esteem (see Q15).

Step 5. **Channel energy positively.** Sports, music, dance, debate, NCC, NSS, scouts, art, social work. Real achievement is a far stronger natural high than any drug.

Step 6. **Address root stress.** Mental-health counselling for anxiety, depression, exam fear or family discord treats the cause before the youngster reaches for a drug.

Step 7. **Regulate availability.** Strict enforcement of laws on the sale of alcohol and tobacco to minors; control of narcotics; quick action on drug peddlers near schools and colleges.

Step 8. **Looking for danger signs.** A sudden drop in grades, withdrawal from family, isolated lifestyle, unkempt appearance, weight loss, mood swings, missing money, changed peer group – any of these calls for a gentle adult conversation, not punishment.

Step 9. **Professional help on time.** Once experimentation has begun, professional de-addiction (medical detox + counselling + family + group support) gives the best chance of recovery. The earlier the intervention, the better.

Step 10. **Role of media.** Responsible portrayal in films and social media; warning labels; campaigns by celebrities; public-service messaging (NACO posters, NSS drives).

> **Final Answer:** Curiosity, peer pressure, family stress, academic pressure, media glamourisation, easy availability and the need for an identity together motivate youngsters to try alcohol or drugs. Avoidance needs early factual education, warm families, accessible school counsellors, refusal skills, channelling of energy into sports and arts, mental-health support, strict regulation of availability, early detection of warning signs, prompt professional help and responsible media.

☞ Exam Tip

A typical 5-mark question. Answer in two sections: causes (physiological + family + peer + media + availability) and prevention (education + family + school + sport + counselling + law + media). A clean two-column layout earns full marks.

EXPERT'S SOLUTION *: Aditi Rao, M.Sc Biotechnology, AIIMS Delhi*

Strategic angle: matched pairs. For every push factor that takes a youngster towards drugs, there is a counter-force that takes them away. Pair them up and the answer becomes both balanced and memorable.

Step 1. **Curiosity** → replace with adventure sports, science clubs, real experiments.

Step 2. **Peer pressure** → choose company well; practise saying 'No' politely.

Step 3. **Family stress** → family counselling; warm, regular dialogue at home.

Step 4. **Academic pressure** → realistic expectations; coaching on time-management; mental-health counselling.

Step 5. **Media glamour** → media literacy in school; warning labels; responsible storytelling by film industry.

Step 6. **Easy availability** → strict enforcement of age-laws on alcohol/tobacco; tighter narcotics control.

Step 7. **Need for identity** → leadership roles in school clubs, social work, sport teams.

Step 8. **Misinformation** → factual NCERT-level education on what drugs really do.

Step 9. **Early signs** → trained school counsellors, accessible helplines, non-judgemental teachers.

Step 10. **Professional help** → NACO, government de-addiction centres, AA/NA groups.

Why this matters. A youngster surrounded by good company, warm parents, alert teachers, channels for energy, and accessible counsellors is at very low risk of substance abuse. Society can build all of these.

> **Final Answer:** Curiosity, peers, family stress, academic pressure, media glamour, easy availability and identity-formation drive youngsters to substance use. Education, families, schools, sport, counselling, regulation and responsible media together prevent it.

Key Takeaways

- Public-health measures (hygiene, sanitation, safe water, vaccination, vector control, health education and prompt treatment) form the front line against infectious diseases.
- Biology has given us pathogens' identity, life cycles, drugs, vaccines, diagnostics and vector controls – the complete toolkit of disease control.
- Amoebiasis and ascariasis spread faecal-orally; malaria by female *Anopheles* mosquito bite; pneumonia by respiratory droplets.
- Innate immunity is non-specific and inborn; acquired immunity is specific and develops with memory. Active immunity (vaccines, infection) is body-made; passive immunity (mother's milk, antiserum) is body-received.
- An antibody is a Y-shaped $\mathbf{H_2L_2}$ molecule with two heavy + two light chains, two antigen-binding sites and disulphide bonds.
- HIV is a retrovirus that targets $CD4^+$ helper T-cells through reverse transcription, integration and budding, leading to progressive immunodeficiency – AIDS.
- Cancer cells lose contact inhibition, divide uncontrollably, escape apoptosis and (when malignant) metastasise to distant organs through blood and lymph.
- Drug/alcohol abuse damages body, mind, family and society; addiction is a brain disease with tolerance and dependence at its core; prevention rests on family, education, counselling and healthy outlets.

End of Exercises