S.Y.B.Sc. Life Sciences Paper I Sem IV April 2019
Answer Key: Q.P.Code : 78813

Q. 1. Do as directed:

A): Define / Explain the following terms: (07)

a. Haematopoiesis:- The production of all types of blood cells including formation, development, and differentiation of blood cells. Prenatally, hematopoiesis occurs in the yolk sack, then in the liver, and lastly in the bone marrow. In the normal situation, hematopoiesis in adults occurs in the bone marrow and lymphatic tissues. All types of blood cells are derived from primitive cells (stem cells) that are pluripotent (they have the potential to develop into all types of blood cells).

b. Pathogencity:- **Pathogenicity** pertains to the ability of a **pathogenic** agent to cause disease. Examples of **pathogenic** agents are infectious bacteria, viruses, prions, fungi, viroids, and parasites causing disease.

c. Virulence:- **Virulence** is a pathogen's or microbe's ability to infect or damage a host. In most other contexts, especially in animal systems, **virulence** refers to the degree of damage caused by a microbe to its host.

d. Mutualism:- Mutualisms are defined as interactions between organisms of two different species, in which each organism benefits from the interaction in some way. Eg. A mutualism in which one mutualistic partner removes parasites, as well as dead or diseased skin from another, in return receiving a steady supply of food, is called a cleaning mutualism.

e. Constitutive plant defence:- Each type of **defense** can be either **constitutive** (always present in the plant), or induced (produced in reaction to damage or stress caused by herbivores).

f. Inflammation:- A localized physical condition in which part of the body becomes reddened, swollen, hot, and often painful, especially as a reaction to injury or infection.

g. Endotoxin:- A toxic heat-stable lipopolysaccharide substance present in the outer membrane of gram-negative bacteria that is released from the cell upon lysis

B) Match the columns: (07)

Column A	Column B
a) Vivotif	i) Tobacco Plant (g)
b) Macro conidiae	ii) Agrobacterium (h)
c) Vector Borne	iii) AIDS (e)
d) Filariasis	iv) Vaccine (a)
e) HIV	v) Fungi Imperfectii (b)
f) Crown Gall	vi) Malaria (c)
g) TMV	vii) Wuchereria bancrofti (d)

Q.1. C) True/ False (06)

1. True
2. True
3. True
4. False
5. False
6. False

Q. 2. A. Answer <u>Any one</u> of the following: (10)

1. Homeotherms are animals which are able maintain their body temperature within narrow range, even when there are changes in external temperature.
They have highly developed thermoregulatory centres, situated in hypothalamus.
They have mechanism to generate heat when the external temperature falls and to lose heat when the external temperature rises.
In cold, loss of heat is reduced by lowering the temperature of body surface, which results in conservation of heat in the body. The metabolic rate also changes in response to external temperature changes. Exoskeleton in the form of feathers and hair, and fat layer beneath the skin also help in thermoregulation.
Thermorecepters in the skin, Blood circulation, reflexes all are helpful in thermoregulation.
One example each, for cold temperature and high temperature is expected.

2. Definition of obesity with formula and Information about Leptin is expected.

Detailed explanation about Leptin and obesity.
When the food intake is enough fat stored in adipocytes, Leptin is secreted by adipocytes, as a satiety hormone. It gives signal to stop eating.
Experimental research evidence on mice shows that the OB gene, is involved in production of Leptin. If this gene is mutated the leptin production is hindered and then there is no control over eating. This results in overweight and obesity.

Q. 2. B) Answer <u>any two</u> of the following: (10)

1. Heat exhaustion and heat stroke
During conditions of prolonged exposure to high temperature, when the thermoregulatory mechanism is unable to function, a person may show heat exhaustion. If a person is out of protected environment and without sunglasses, cap to protect eyes and head and is exposed to high temperature for a long time. There is redness of skin, excessive water loss due toprofuse sweating, the hypothalamus thermoregulation can go off control. The person may experience giddiness and faint. This is heat exhaustion.
Heat stroke is a condition when the heat exhaustion is not treated. The physiological functions are not proper by further rise in temperature, dehydration and person may become unconscious,if not taken care this may even lead to death.
2. Anorexia nervosa is an eating disorder characterized by immoderate food restriction and irrational fear of gaining weight as well as distorted body self perception. The constant worry about one's weight and body structure. I t typically involves excessive weight loss and is found usually more in females than in males. Due to the fear of gaining weight, people with this disorder restrict the amount of food they consume. This causes metabolic and hormonal disorders.
Anorexia in medical terms is loss of appetite. People with Anorexia nervosa do not, in fact lose their appetite. May complicated implications, dizziness, headaches, drowsiness, lack of energy. Discuss symptoms and treatment to reverse the condition.

3. Causes of Diabetes Mellitus.
Explanation of what is Diabetes Mellitus
Genetic disposition, sedentary life style, overweight and obesity, wrong eating habits etc. dicuss.

4. **Kisspeptin** (formerly known as metastin) is a protein that is encoded by the KISS1 gene in humans. **Kisspeptin** is a G-protein coupled receptor ligand for GPR54. Kiss1 was originally identified as a human metastasis suppressor gene that has the ability to suppress melanoma and breast cancer metastasis.
Functions of Kisspeptin.
1. In the brain it is released within the hippocampal dentate gyrus. Responsible for pulsatile release of GnRH/LH at puberty in mammals.
2. It s is also transcrined in the vascular endothelium.
3. It is released within the neocortex of the adrenal gland. It can stimulate secretion of Aldesterone.
4. In the pancreas, in the islet cells, it can stimulate secretion of Insulin.
It is expressed in brain, placenta and many other organs.
Kisspeptin is regulated via Leptin signaling.

Q.3.A. Answer any one of the following:- (10)

1. What is innate immunity? Give an account on physical and chemical barriers.
Innate immunity refers to nonspecific defense mechanisms that come into play immediately or within hours of an antigen's appearance in the body. These mechanisms include physical barriers such as skin, chemicals in the blood, and **immune** system cells that attack foreign cells in the body.

Physical – Skin and Mucous membranes

Chemical – Acidity of stomach contents and specialized soluble molecules that possess anti microbial activity

The most obvious components of Innate immunity are the external barriers to microbial invasion –Skin and Mucous membranes, which include the mucosal epithelia that line the respiratory, gastrointestinal and urogenital tracts and insulate the body's interior from the pathogens of the exterior world.

The skin consists of two distinct layers-

A thin outer layer, epidermis and a thicker layer, dermis

The epidermis contains several tiers of tightly packed epithelial cells. The outer epidermal layer cnsists of mostly dead cells filled with a waterproofing called keratin.

The dermis is composed of connective tissue and contains blood vessels, hair follicles, sebaceous glands and sweat glands.

Skin and epithelial encases and protects the inner domains of the body from outer world.

But these anatomical barriers are more than just passive wrappers. They also mount active biochemical defenses by synthesizing and deploying peptides and proteins with antimicrobial activity.

The alimentary, respiratory, urogenital tracts and eyes are lined by mucous membranes that consists of an outer epithelial layer and an underlying layer of connective tissue. Many

pathogens enter the body by penetrating these membranes, opposing this entry are a number of non-specific defense mechanisms. For e.g. Saliva, tears and mucous secretions wash away potntial invaders and also contain antibacterial or antiviral substances. The viscous fluid called mucus secreted by epithelial cells of mucous membranes, entraps foreign microorganisms. In the lower respiratory tract, the mucous membrane is covered by cilia, hair like protrusions of the epithelial cell membrane. The synchronous movement of cilia propels mucous entrapped microorganisms from these tracts. With every meal, we ingest huge numbers of mos, but they must run a lot of defenses that begins with the antimicrobial compounds in saliva and in the epithelial of the mouth and includes the hostile mixture of acid and digestive enzymes found in the stomach. In addition to the biochemical and anatomical defenses, pathogenic microbes must compete for body's resources with many non – pathogenic organisms that colonize the mucosal surfaces.

These normal flora, highly adapted to their internal environment, generally outcompete pathogens for attachment sites and necessary nutrients on epithelial surfaces.

Adherence of bacteria to mucous membranes is generally mediated by hair-like protrusions on the bacterium called fimbria /pili, which interact with certain glycoprotein or glycolipid only expressed by epithelial cells of the mucous membrane of particular tissues.

For these reasons and others, some tissues are susceptible to invasion by particular pathogens, despite the general effectiveness of protective epithelial barriers.When this happens, the receptors of innate immunity play the essential roles of detecting the infection and triggering an effective defense against it.

2. Explain the various factors controlling growth of microorganisms.

Microorganisms are similar to more complex organisms in that they need a variety of materials from their environment to function and accomplish two primary goals--supply enough energy to manage their processes and extract building blocks to repair themselves or procreate. In addition to what they take in, microorganisms also thrive in particular environments. These environments vary as much as the organisms do themselves, and even the amount and distribution of elements in any particular environment can be very important. Scientists use this information to grow microorganisms in laboratories for experimentation.

Nutrients

All microorganisms need food. The food sources can vary, but the organisms primarily extract carbon and nitrogen from substances such as proteins, fats and carbohydrates. Some microorganisms seek out and absorb such particles. Others may perform chemical reactions with surrounding elements such as carbon dioxide to gain what they need, while still others can produce their own simple sugars through photosynthesis similar to plants. Nitrogen, which is used to synthesize proteins, can be taken from the surrounding atmosphere or from other organic matter.

Temperature

In general, the higher the temperature, the more easily microorganisms can grow up to a certain point. Very high and very low temperatures both obstruct the enzyme processes microorganisms depend on to survive, but individual species of microorganisms have grown to prefer different levels of temperature. Scientists usually divide them into three different groups: psychrophiles, mesophiles and thermophiles. Psychrophiles prefer temperatures from 0 to 5 degrees Celsius; mesophiles like it in the middle, 20-45 degrees Celsius; and thermophiles like it hot, thriving in temperatures around or above 55 degrees.

pH Levels

Microorganisms also prefer a certain pH level in the substance or environment in which they grow--that is, they prefer to have particular acidic qualities in their surroundings. Most microorganisms, including most human pathogens, are neutriphils, organisms that prefer a neutral pH level. Some like high pH levels, but most often, if conditions are too acidic, then the organism's enzymes break down.

Moisture

The free flow of water is vital to microorganisms for their cells to exchange materials and for their metabolic processes. All microorganisms require some level of water, but a few can survive in low-moisture conditions by conserving all the water they find and by staying in a moisture-rich environment. As a general rule, though, the more moisture, the more microorganisms there will be found.

Elements Present

In addition to water, microorganisms usually require the presence of certain elements in the air--gases that they absorb to produce needed nutrients. Nitrogen is one necessary element, as is oxygen. There are many microorganisms that require an oxygen-rich environment to survive, but others actually flourish in low-oxygen surroundings. Between these two extremes is a wide variety that may prefer more or less oxygen and that will be able to flourish equally no matter how much oxygen is present.

Q.3.B. Answer any two of the following: (10)

1. Describe the structural modification in plants that are useful in its defence

Many plants have external structural defenses that discourage herbivory. Depending on the herbivore's physical characteristics (i.e. size and defensive armor), plant structural defenses on stems and leaves can deter, injure, or kill the grazer. Some defensive compounds are produced internally but are released onto the plant's surface; for example, resins, lignins, silica, and wax cover the epidermis of terrestrial plants and alter the texture of the plant tissue. The leaves of holly plants, for instance, are very smooth and slippery making feeding difficult. Some plants produce gummosis or sap that traps insects.

Spines and thorns

A plant's leaves and stem may be covered with sharp prickles, spines, thorns, or trichomes-hairs on the leaf often with barbs, sometimes containing irritants or poisons. Plant structural features like spines and thorns reduce feeding by large ungulate herbivores (e.g. kudu, impala, and goats) by restricting the herbivores' feeding rate, or by wearing down the molars.Raphides are sharp needles of calcium oxalate or calcium carbonate in plant tissues, making ingestion painful, damaging a herbivore's mouth and gullet and causing more efficient delivery of the plant's toxins. The structure of a plant, its branching and leaf arrangement may also be evolved to reduce herbivore impact. The shrubs of New Zealand have evolved special wide branching adaptations believed to be a response to browsing birds such as the moas. Similarly, African Acacias have long spines low in the canopy, but very short spines high in the canopy, which is comparatively safe from herbivores such as giraffes.

Coconut palms protect their fruit by surrounding it with multiple layers of armor.

Trees such as palms protect their fruit by multiple layers of armor, needing efficient tools to break through to the seed contents. Some plants, notably the grasses, use indigestible silica(and many plants use other relatively indigestible materials such as lignin) to defend themselves against vertebrate and invertebrate herbivores.Plants take up silicon from the soil and deposit it in their tissues in the form of solid silica phytoliths. These mechanically reduce the digestibility of plant tissue, causing rapid wear to vertebrate teeth and insect mandibles, and are effective against herbivores above and below ground. The mechanism may offer future sustainable pest control strategies.

Thigmonastic movements

Thigmonastic movements, those that occur in response to touch, are used as a defense in some plants. The leaves of the sensitive plant, *Mimosa pudica*, close up rapidly in response to

direct touch, vibration, or even electrical and thermal stimuli. The proximate cause of this mechanical response is an abrupt change in the turgor pressure in the pulvini at the base of leaves resulting from osmotic phenomena. This is then spread via both electrical and chemical means through the plant; only a single leaflet need be disturbed. This response lowers the surface area available to herbivores, which are presented with the underside of each leaflet, and results in a wilted appearance. It may also physically dislodge small herbivores, such as insects.

Mimicry and camouflage

Some plants mimic the presence of insect eggs on their leaves, dissuading insect species from laying their eggs there. Because female butterflies are less likely to lay their eggs on plants that already have butterfly eggs, some species of neotropical vines of the genus *Passiflora* (Passion flowers) contain physical structures resembling the yellow eggs of *Heliconius* butterflies on their leaves, which discourage oviposition by butterflies.

2. Explain Koch's postulate.

Four criteria that were established by Robert Koch to identify the **causative agent of a particular disease**, these include:

1. the microorganism (pathogen) must be **present in all cases of the disease**
2. the pathogen can be **isolated** from the diseased host **and grown in pure culture**
3. the pathogen from the pure culture must cause the **same disease when inoculated** into a healthy, susceptible laboratory animal
4. the pathogen must be **reisolated** from the new host and **shown to be the same** as the originally inoculated pathogen

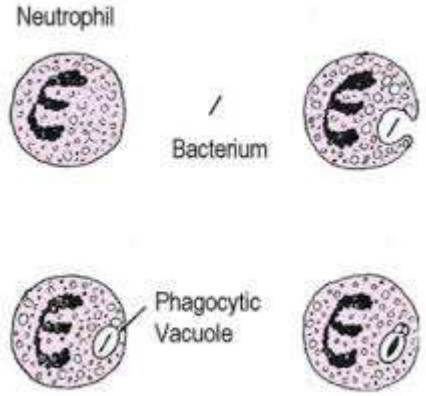
3. With neat diagram of Neutrophils, explain its function.

Neutrophils are **white blood cells** that play some very important roles in our innate **immune system**. They circulate around our **body** in the bloodstream, and when they sense signals that an infection is present, they are the first cells to migrate to the site of the infection to begin killing the invading microbes

Neutrophils participate in antimicrobial host defense both as the first line of innate immune defense and as effectors of adaptive immunity. Neutrophils are short-lived cells that usually die while performing their antimicrobial function.

- The neutrophil is one of the body's main cellular mediators of the destruction of microorganisms, and inevitably damages cells and tissues of the host. Neutrophil-mediated tissue destruction is most often a life-saving process, and the host relies on tissue injury as one of the main sources of information that launches inflammation and immunity.
- Large numbers of immature forms of neutrophils, called neutrophilic band cells, are produced by the bone marrow when the demand is high.
- Neutrophils make important contributions to the recruitment, activation and programming of dendritic cells and macrophages. In turn, the adaptive immune system controls the rate of neutrophil production in the bone marrow.
- Neutrophils have important roles in healing wounds, including sterilization of microorganisms, generation of signals that slow the rate of accumulation of more neutrophils, and instigation of a macrophage-based programme that switches the state of damaged epithelium from pro-inflammatory and nonreplicative, to anti-inflammatory and replicative.

- Neutrophil production is coordinated through cytokine production by adaptive immune cells.
- Neutrophil recruitment to sites of infection involves unique molecular interactions in different tissues.
- Recognition of pathogens by neutrophils involves coordination between a repertoire of cellular receptors.
- Killing of pathogens is achieved through the production of toxic metabolites and the release of nuclear contents.
- Heritable disorders of neutrophils provide key insights into molecular mechanisms of neutrophil function.
- Neutrophils play a central role in coordinating the response of other immune effector cells.
- Pathologic interactions between adaptive immune cells and neutrophils are a major contributor to many autoimmune and inflammatory disease states.
- Neutrophils play both positive and negative roles in cancer progression.
- Neutrophils are short-lived cells that die within a limited time after entering the circulation . In the absence of infection or inflammation, they die by a spontaneous apoptosis program , likely within 1 day (although some investigators propose this time to be up to 5 days) . Inflammatory signals are capable of prolonging the lifespan of the cells by several days, during which they release inflammatory mediators and contribute to the orchestration of the inflammatory response.



4. Describe Parasitism.

Parasitism, relationship between two species of plants or animals in which one benefits at the expense of the other, sometimes without killing the host organism.

Parasites may be characterized as ectoparasites—including ticks, fleas, leeches, and lice—which live on the body surface of the host and do not themselves commonly cause disease in the host; or endoparasites, which may be either intercellular (inhabiting spaces in the host's body) or intracellular (inhabiting cells in the host's body). Intracellular parasites—such as bacteria or viruses—often rely on a third organism, known as the carrier, or vector, to transmit them to the host. Malaria, which is caused by a protozoan of the genus *Plasmodium* transmitted to humans by the bite of an anopheline mosquito, is an example of this interaction. The plant ailment known as Dutch elm disease (caused by the fungus *Ceratocystis ulmi*) can be spread by the European elm bark beetle.

Q.4. A) Answer <u>any one</u> of the following: (10)

1. Explain the Helminthic Disease that you study w.r.t its aetiology, diagnosis and treatment.

Filariasis – Also known as Elephantiasis/Lymphatic filariasis.

Causative agent : Wuchehreria bancrofti,Brugia malayi and Brugia timori.

nfection occurs when filarial parasites are transmitted to humans through mosquitoes. Infection is usually acquired in childhood causing hidden damage to the lymphatic system.

The painful and profoundly disfiguring visible manifestations of the disease, lymphoedema, elephantiasis and scrotal swelling occur later in life and can lead to permanent disability. These patients are not only physically disabled, but suffer mental, social and financial losses contributing to stigma and poverty.

Currently, 886 million people in 52 countries are living in areas that require preventive chemotherapy to stop the spread of infection.

The global baseline estimate of people affected by lymphatic filariasis was 25 million men with hydrocele and over 15 million people with lymphoedema. At least 36 million people remain with these chronic disease manifestations. Eliminating lymphatic filariasis can prevent unnecessary suffering and contribute to the reduction of poverty.

Cause and transmission

Lymphatic filariasis is caused by infection with parasites classified as nematodes (roundworms) of the family Filariodidea. There are 3 types of these thread-like filarial worms:

- *Wuchereria bancrofti*, which is responsible for 90% of the cases
- *Brugia malayi*, which causes most of the remainder of the cases
- *Brugia timori*, which also causes the disease.

Adult worms nest in the lymphatic vessels and disrupt the normal function of the lymphatic system. The worms can live for approximately 6–8 years and, during their life time, produce millions of microfilariae (immature larvae) that circulate in the blood.

Mosquitoes are infected with microfilariae by ingesting blood when biting an infected host. Microfilariae mature into infective larvae within the mosquito. When infected mosquitoes bite people, mature parasite larvae are deposited on the skin from where they can enter the body. The larvae then migrate to the lymphatic vessels where they develop into adult worms, thus continuing a cycle of transmission.

Lymphatic filariasis is transmitted by different types of mosquitoes for example by the *Culex* mosquito, widespread across urban and semi-urban areas, *Anopheles*, mainly found in rural areas, and *Aedes*, mainly in endemic islands in the Pacific.

Symptoms

Lymphatic filariasis infection involves asymptomatic, acute, and chronic conditions. The majority of infections are asymptomatic, showing no external signs of infection while contributing to transmission of the parasite. These asymptomatic infections still cause damage to the lymphatic system and the kidneys, and alter the body's immune system.

When lymphatic filariasis develops into chronic conditions it leads to lymphoedema (tissue swelling) or elephantiasis (skin/tissue thickening) of limbs and hydrocele (scrotal swelling). Involvement of breasts and genital organs is common. Such body deformities often lead to social stigma and sub-optimal mental health, loss of income-earning opportunities and increased medical expenses for patients and their caretakers. The socioeconomic burdens of isolation and poverty are immense.

Acute episodes of local inflammation involving skin, lymph nodes and lymphatic vessels often accompany chronic lymphoedema or elephantiasis. Some of these episodes are caused

by the body's immune response to the parasite. Most are the result of secondary bacterial skin infection where normal defences have been partially lost due to underlying lymphatic damage. These acute attacks are debilitating, may last for weeks and are the primary cause of lost wages among people suffering with lymphatic filariasis.

WHO response

World Health Assembly resolution WHA50.29 encourages Member States to eliminate lymphatic filariasis as a public health problem. In response, WHO launched its Global Programme to Eliminate Lymphatic Filariasis (GPELF) in 2000. In 2012, the WHO neglected tropical diseases roadmap reconfirmed the target date for achieving elimination by 2020.

WHO's strategy is based on 2 key components:

- stopping the spread of infection through large-scale annual treatment of all eligible people in an area or region where infection is present; and
- alleviating the suffering caused by lymphatic filariasis through provision of the recommended basic package of care.

Large-scale treatment (preventive chemotherapy)

Elimination of lymphatic filariasis is possible by stopping the spread of the infection through preventive chemotherapy. The WHO recommended preventive chemotherapy strategy for lymphatic filariasis elimination is mass drug administration (MDA). MDA involves administering an annual dose of medicines to the entire at-risk population. The medicines used have a limited effect on adult parasites but effectively reduce the density of microfilariae in the bloodstream and prevent the spread of parasites to mosquitoes.

The MDA regimen recommended depends on the co-endemicity of lymphatic filariasis with other filarial diseases. WHO recommends the following MDA regimens:

- albendazole (400 mg) alone twice per year for areas co-endemic with loiasis
- ivermectin (200 mcg/kg) with albendazole (400 mg) in countries with onchocerciasis
- diethylcarbamazine citrate (DEC) (6 mg/kg) and albendazole (400 mg) in countries without onchocerciasis

Recent evidence indicates that the combination of all three medicines can safely clear almost all microfilariae from the blood of infected people within a few weeks, as opposed to years using the routine two-medicine combination.

WHO now recommends the following MDA regimen in countries without onchocerciasis:

- ivermectin (200 mcg/kg) together with diethylcarbamazine citrate (DEC) (6 mg/kg) and albendazole (400 mg) in certain setting.

2. Explain the Bacterial Infection caused in Plants

Crown gall is a common plant disease caused by the soil-borne bacterium *Agrobacterium tumefaciens*. It is found throughout the world and occurs on woody shrubs and herbaceous plants including grapes, raspberries, blackberries and roses.

Crown gall symptoms include round, wart-like growths — 2 inches or larger in diameter — that appear at or just above the soil line, or on lower branches and stems. Plants with several galls may be unable to move water and nutrients up the trunk and become weakened, stunted and unproductive. Young plants can be killed by developing gall tissue.

The bacteria responsible for crown gall can persist in the soil for many years and are released when galls become saturated with moisture or as older galls decompose. Susceptible plants are infected through fresh wounds or abrasions, many of which are a result of pruning, freeze injury, soil insects, cultivation and other factors that may damage plants. Nursery stock is often infected through grafting and budding scars. Thousands of plant species are susceptible. They include especially grape, members of the rose family (Rosaceae), shade and nut trees, many shrubs and vines, and perennial garden plants. Symptoms include roundish rough-surfaced galls (woody tumourlike growths), several centimetres or more in diameter, usually

at or near the soil line, on a graft site or bud union, or on roots and lower stems. The galls are at first cream-coloured or greenish and later turn brown or black. As the disease progresses, plants lose vigour and may eventually die.Crown gall can be avoided by using nursery stock free of suspicious bumps near the crown, former soil line, or graft union; practicing five-year rotation or avoiding replanting for that period; removing severely infected plants (including as many roots as possible); protecting against injury; keeping down weeds; controlling root-chewing insects and nematodes; cutting away large galls on trees; and disinfecting wounds.
Treatment

1. Select resistant cultivars when possible and purchase plants from a reputable nursery.
2. Do not buy plants that shows signs of swelling or galling.
3. When caring for susceptible plants, avoid injury or pruning wounds that may come in contact with the soil.
4. Use Tree Wrap to protect against string trimmer damage and keep your garden tools clean.
5. Provide winter protection with natural burlap so bark does not crack.
6. In many cases, existing galls can be removed with a sharp pruning knife. Destroy the infected plant tissue and treat the wound with pruning sealer. If the plant does not recover, remove and destroy it.

Tip: To get rid of this problem on roses, remove the infested plant and prune out gall tissue. Soak the entire root system and damaged areas for 15 minutes in a solution of 2 level Tbsp of Actinovate per 2-1/2 gallons of water. Replant in healthy soil and apply 1/2 Tbsp per 2-1/2 gallons of water as a foliar spray at weekly intervals.

Q. 4. B) Answer <u>any two</u> of the following: (10)

1. Give an account of signs and symptoms of the air borne infection given in your curriculum.

TB is an abbreviation of the word Tuberculosis and is a air borne infection spread through droplet nuclei when a infected person sneezes or coughs.
Latent TB
The bacteria that usually cause the disease in humans, usually affect the lungs, but can affect other parts of the body. If you are infected with the bacteria you won't necessarily become sick, because you can have either <u>latent TB</u> or TB disease.[1] People with latent TB do not feel sick and do not have any symptoms.
TB disease is what happens when a person has latent TB and then becomes sick. Sometimes this is known as having active TB. Overall about 5 to 10% of people with latent TB, who do not receive treatment for it, will become sick at some time in their lives.[2]
Some people become sick soon after they have become infected, before their immune system (the part of the body that fights diseases) can fight the bacteria. Other people don't get sick at first but they get sick years later when their immune system becomes weak for another reason. This can be because they have an infection, such as HIV, or some other health problem.
Some people are known to have a higher risk of becoming ill.[3]
These include

- Infants and children aged less than 4 years
- People infected within the previous two years
- People infected with HIV
- People who have certain illnesses or conditions which affect their immune system, such as people with diabetes, and people with chronic renal failure.

Symptoms of TB:

The symptoms depend on which area of the body has been infected. If someone has pulmonary disease, which is TB in the lungs, then they may have a bad cough that lasts longer than two weeks. They may also have pain in their chest and they may cough up blood or phlegm from deep inside their lungs. Other symptoms of TB include weakness or fatigue, weight loss, lack of appetite, chills, fever and night sweats.[4]

Diagnosing TB:

It is very difficult to diagnose TB by a person's symptoms on their own. This is because some other diseases have the same symptoms.

A diagnosis is usually only certain when there is definite evidence of TB bacteria. Some of the TB tests used for diagnosis look directly for the bacteria. Others such as the chest X-ray look for the effect of the bacteria on the person suspected of having TB. Tests for diagnosis include the TB skin test, sputum microscopy, the culture test as well as the new Genexpert test.

Major problems with the older tests are the lack of accuracy as well as the time they take. With newer tests a major issue is the cost.

Treatment

TB can usually be cured and more than twenty drugs have been developed for treating TB. But most of the drugs were developed many years ago. The treatment usually consists of a combination of TB drugs that must be taken for at least six months. But the treatment will only be successful if the drugs are taken exactly as required for the entire length of time.

The drugs are used in different combinations in different circumstances. For example, the five "first line" drugs are given to people who have never had treatment before. If people have had treatment before they may need to take second line drugs.

Some of the drugs have very severe side effects and are very difficult to take for such a long period of time. This is why there is an urgent need for new TB drugs to be developed. In addition many people are now resistant to one or more of the drugs.

Drug resistant TB:

If someone has drug resistant TB it means that the bacteria in their body won't be affected by certain drugs that they are resistant to. The drugs just won't work. There are two main reasons why people develop it. It can be because the person doesn't take their drugs properly. It can also be that the bacteria that they are infected with, have come from someone who has already got drug resistant TB. Being drug sensitive is the opposite of being drug resistant.

If someone has drug resistant TB then they must change drugs. But usually they mustn't have just one new drug. They need to have several new drugs and for it to be believed that they will all be effective. Drug susceptibility testing which is available in many countries, and is very important, provides information about which drugs a person is resistant to.

Prevention

A major part of the prevention of TB is to stop the spread of bacteria from one adult to another. This is done by firstly finding the adults who have TB. Then providing them with effective treatment which means that they are no longer infectious, and they will also usually then recover from being sick. There is a vaccine, the BCG vaccine, but it is used for children as it doesn't seem to prevent the disease in adults.

TB infection control is also important to prevent people from getting infected in health care facilities.

Sometimes it is believed that education only needs to involve people who already have the disease. But there is also a need to educate the general public. This is to ensure that people know how the disease is spread, and also to reduce the stigma surrounding the disease. It can also help to ensure that people come forward for testing and treatment as soon as possible.

2. Discuss the treatment for leprosy.

Several drugs are used in combination in multidrug therapy (MDT). (See table) These drugs must never be used alone as monotherapy for leprosy.

Dapsone, which is bacteriostatic or weakly bactericidal against *M. leprae,* was the mainstay treatment for leprosy for many years until widespread resistant strains appeared. Combination therapy has become essential to slow or prevent the development of resistance. **Rifampicin** is now combined with **dapsone** to treat paucibacillary leprosy. **Rifampicin and clofazimine** are now combined with **dapsone** to treat multibacillary leprosy.

A single dose of combination therapy has been used to cure single lesion paucibacillary leprosy: **rifampicin** (600 mg), **ofloxacin** (400 mg), and **minocycline** (100 mg). The child with a single lesion takes half the adult dose of the 3 medications.

WHO has designed blister pack medication kits for both paucibacillary leprosy and for multibacillary leprosy. Each easy-to use kit contains medication for 28 days. The blister pack medication kit for single lesion paucibacillary leprosy contains the necessary medication for the one time administration of the 3 medications.

Any patient with a positive skin smear must be treated with the MDT regimen for multibacillary leprosy. The regimen for paucibacillary leprosy should never be given to a patient with multibacillary leprosy. Therefore, if the diagnosis in a particular patient is uncertain, treat that patient with the MDT regimen for multibacillary leprosy.Ideally, the patient should go to the leprosy clinic once a month so that clinic personnel may supervise administration of the drugs prescribed once a month. However, many countries with leprosy have poor coverage of health services and monthly supervision of drug administration by health care workers may not be possible. In these cases, it may be necessary to designate a responsible third party, such as a family member or a person in the community, to supervise the monthly drug administration. Where health care service coverage is poor and supervision of the monthly administration of drugs by health workers is not possible, the patient may be given more than the 28 days supply of multidrug therapy blister packs. This tactic helps make multidrug therapy easily available, even to those patients who live under difficult conditions or in remote areas. Patients who ask for diagnosis and treatment are often sufficiently motivated to take full responsibility for their own treatment of leprosy. In this situation, it is important to educate the patient regarding the importance of compliance with the regimen and to give the patient responsibility for taking his or her medication correctly and for reporting any untoward signs and symptoms promptly. The patient should be warned about possible lepra reactions.

WHO Recommended treatment regimens

6 month regimen for Paucibacillary (PB) Leprosy

	Dapsone	Rifampicin
Adult	100 mg	600 mg
50 - 70 kg	Given daily	Given once a month under supervision
Child	50 mg	450 mg
10 - 14 years[a]	Given daily	Given once a month under supervision

[a] Adjust dose appropriately for child less than 10 years. For example, dapsone 25 mg daily and rifampicin 300 mg given once a month under supervision

12 month regimen for Multibacillary (MB) Leprosy

	Dapsone	Rifampicin	Clofazimine	
Adult 50 - 70 kg	100 mg Given daily	600 mg Given once a month under supervision	50 mg Given daily	AND 300 mg Given once a month under supervision
Child 10 - 14 years[b]	50 mg Given daily	450 mg Given once a month under supervision	50 mg Given every other day	AND 150 mg Given once a month under supervision

[b] Adjust dose appropriately for child less than 10 years. For example, dapsone 25 mg daily, rifampicin 300 mg given once a month under supervision, clofazimine, 50 mg given twice a week, and clofazimine 100 mg given once a month under supervision.

3. Explain the lifecycle of puccinia in wheat.

Puccinia graminis is long cycled rust (macro cyclic). At the time of reproduction it produces five distinct stages in a regular sequence.

These are as follows:

Stage 0: Spermogonia bearing spermatia and receptive hyphae.

Stage I: Aecia bearing aeciospores.

Stage II: Uredia bearing uredospores.

Stage III: Telia bearing teleutospores.

Stage IV: Promycelia bearing basidiospores.

Out of these five stages, Uredo stage, Teleuto stage are produced on the primary host (wheat) and remaining two stages, (spermogonial and aecial stages) are produced on the secondary host i.e., barberry.

Stages on Primary Host (Wheat):

Uredospore's or Uredo stage:

This stage is formed by the infection of the aeciospores brought from the infected barberry plants or by the uredospore's themselves coming from the neighbouring wheat plants infected earlier. Both the spores are bi-nucleate and on germination, produce a germ tube on wheat leaf.

The germ tube grows over the surface of the epidermis of the host and on reaching a stoma its tip swells up and forms a vesicle like structure called appressorium. The protoplasm of the germ tube migrates into the appressorium. Now it is cut off from the germ tube by a septum. The appressorium produces a narrow hypha. It enters inside the sub-stomatal chamber through stoma.

Its tip again swells up and forms a sub-stomatal vesicle. The contents of the appressorium migrate to vesicle through narrow hypha. A hypha of the dikaryon (two nuclei) cells develops from this vesicle. It branches and produces hyphae which spread in between the cell (intercellular) but occasionally produce hausotria.

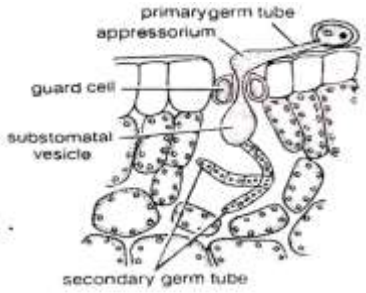


Fig. 3. *Puccinia* : Germination of uredospore on wheat leaf. Note the formation of appressorium

Development of Uredospore's:

Within 5-6 days, the mycelium absorbs sufficient food from the host. It begins to aggregate near the surface of the infected organs and forms a compact mass. These are called uredia. From these uredia arise vertically a layer of bi-nucleate parallel cells known as basal cells. The basal cells elongate vertically and divide transversely into a lower cell (foot cell) and an upper cell (uredospore mother cell).

The upper cell divides again and its upper daughter cell swells to form a single, bi-nucleate, oval, uredospore or uredinospore, while the lower daughter cell matures into a stalk.Thus, the uredospore's are formed in a group and each such group is called as uredospore's or uredinium.The developing uredospore's exert pressure on the over-lying epidermis. By this pressure the epidermis bulges out and later breaks up and the uredospore's get exposed.

The uredospore's are golden brown and oblong, ovate or ellipsoidal in shape. They are double walled, echinulate, binucleate (the two nuclei belonging to opposite strains) and possess four equatorially arranged germ pores.

A uredospore can infect only a wheat plant. After falling on a suitable host it germinates within a few hours and produces a dikaryotic mycelium. The mycelium is capable of producing uredospore's again within 10-12 days after germination. Thus, these spores cause several successive infections during the season, and spread the fungus and the disease from field to field provided the environmental conditions are favourable (sufficient moisture). On the basis of the aforesaid behaviour these uredospore's are also known as repeating spores.

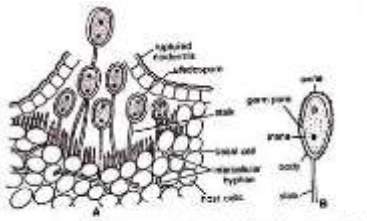


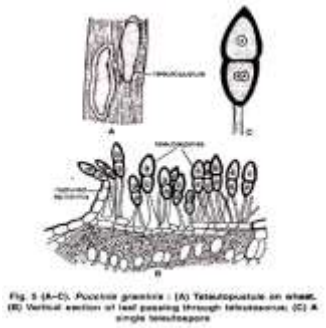
Fig. 4 (A–B). *Puccinia graminis* : T.S. wheat leaf passing through a uredosorus, (B) A uredospore

Teleutospores or Telial Stage:

Towards the end of the growing season of wheat crop, the environmental conditions become unfavorable (hot and dry) for the growth of the uredospore's. Now uredosori produce another kind of spores called teleutospores. First, they develop among the uredospore's within the same sorus, but later they develop in separate sori known as teleutosori or teleutopustule.

As the crop matures, the number of uredospore's is reduced and the sori contain only teleutospores. This stage is known as the black stage and hence the name black rust is given to the disease. The teleutospores are dark brown or black in colour. They are bi-celled and spindle shaped structure with a pointed apex and thick smooth wall.

Each cell of a teleutospore has a single germ pore and two nuclei (one of plus strain and the other of minus strain. As the teleutospores reach towards maturity, karyogamy takes place and the two nuclei fuse to form a diploid nucleus. The development of teleutospores is entirely similar to the uredospore's.



At this stage the teleutospores undergo a period of rest. During resting period they lie on the ground or still attached to the host. These are the dormant cells and are capable of tiding over unfavorable period.

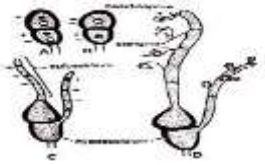
Basidial Stage:

After the renting period, the teleutospores germinate during the early part of spring. They germinate in situ and either one or both of its cells give rise to a germ tube. known as promycelium. The promycelium together with the teleutospore cell is called basidium. However, many authors prefer to call the teleutospore cell as the hypo-basidium and the promycelium as the epibasidium.

The diploid nucleus of the teleutospore migrates into the promycelium and divides meiotically into four haploid nuclei. The septa appear between the nuclei and divide the promycelium into four haploid cells. Each haploid cell of the promycelium produces a slender, short, lateral, tube-like structure known as sterigma. The sterigma swells up at the end to form a spore like cell.

The haploid nucleus from each promycelium cell migrates into this developing spore cell through its respective sterigma. Thus, at the tip of each sterigma, a minute spore is formed.

This spore is called basidiospore. Each cell of promycelium produces a single basidiospore. Thus, from a single cell of teleutospore four haploid, unicellular, uninucleate basidiospores are formed. Two, out of the four telutospore basidiospores are of '+' strain and the other two of strain.



Soon after the basidiospore formation they are forcibly ejected by the 'water droplet method'. (In this method a liquid begins to collect in the form of a droplet at the base of the basidiospore.

This droplet gradually attains a bigger size and suddenly pushes off the basidiospore forcibly into the air to a short distance.) The basidiospores are carried away by wind. They are capable of germinating only on Barberry plants (Berberis vulgaris) available on hills. They perish soon if the alternate host is not available.

4. What are the preventive measures for Typhoid.

Typhoid is an infection caused by the bacterium *Salmonella typhimurium (S. typhi)*.

The bacterium lives in the intestines and bloodstream of humans. It spreads between individuals by direct contact with the feces of an infected person.

Countries with less access to clean water and washing facilities typically have a higher number of typhoid cases.

Vaccination

If traveling to an area where typhoid is prevalent, vaccination is recommended.
Before traveling to a high-risk area, getting vaccinated against typhoid fever is recommended. This can be achieved by oral medication or a one-off injection:

- Oral: a live, attenuated vaccine. Consists of 4 tablets, one to be taken every second day, the last of which is taken 1 week before travel.
- Shot, an inactivated vaccine, administered 2 weeks before travel.

Vaccines are not 100 percent effective and caution should still be exercised when eating and drinking.
Vaccination should not be started if the individual is currently ill or if they are under 6 years of age. Anyone with HIV should not take the live, oral dose.
The vaccine may have adverse effects. One in 100 people will experience a fever. After the oral vaccine, there may be gastrointestinal problems, nausea, and headache. However, severe side effects are rare with either vaccine.
There are two types of typhoid vaccine available, but a more powerful vaccine is still needed. The live, oral version of the vaccine is the strongest of the two. After 3 years, it still protects individuals from infection 73 percent of the time. However, this vaccine has more side effects.
The current vaccines are not always effective, and because typhoid is so prevalent in poorer countries, more research needs to be done to find better ways of preventing its spread.
Typhoid is spread by contact and ingestion of infected human feces. This can happen through an infected water source or when handling food.
The following are some general rules to follow when traveling to help minimize the chance of typhoid infection:

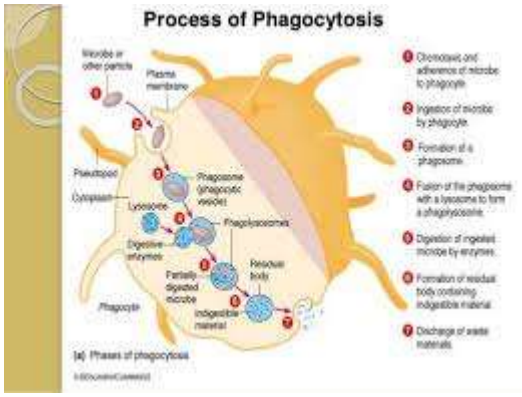
- Drink bottled water, preferably carbonated.
- If bottled water cannot be sourced, ensure water is heated on a rolling boil for at least one minute before consuming.
- Be wary of eating anything that has been handled by someone else.
- Avoid eating at street food stands, and only eat food that is still hot.
- Do not have ice in drinks.
- Avoid raw fruit and vegetables, peel fruit yourself, and do not eat the peel.

Q.5. Write short notes on <u>any four</u> of the following: (20)

1. Poikilotherms. Definition, types, examples, thermoregulation via morphology, behavior, physiology, etc. Any 10 valid points.

2. Plants response to cold temperature.
Cold temperature stress. Tropical and temperate plants have different tolerance to cold temperature. Temperature tolerant, resistant, sensitive plants. Chilling injury, freezing injury, membrane changes, symptoms of injury, antifreeze proteins, examples.
Any 10 valid points.

3. Describe the process of phagocytosis.



4. Differentiate between pathogenicity and virulence
PATHOGENICITY: the quality of producing disease or the ability to produce pathologic changes or disease
VIRULENCE: a measure of pathogenicity; a measurement of the degree of disease-producing ability of a microorganism as indicated by the severity of the disease produced; commonly ascertained by measuring the dosage required to caused a specific degree of pathogenicity; one general standard is the LD_{50} (lethal dose 50%)

5. Enlist the different causative agents of vector borne disease given in your curriculum.
Malaria is caused by single-celled protozoan parasites of the genus Plasmodium. Four species infect humans by entering the bloodstream: Plasmodium falciparum, which is the main cause of severe clinical malaria and death; Plasmodium vivax; Plasmodium ovale; and Plasmodium malariae. Inoculation of parasite sporozoites occurs via the bite of infected blood-feeding female mosquitoes of the genus Anopheles. In humans, the parasites multiply exponentially in the liver, releasing merozoites that develop and multiply in infected red blood cells. With a blood meal, mosquitoes ingest Plasmodium gametocytes, which undergo another reproductive phase inside the mosquito before being transferred to another human host. Current global status. Malaria is responsible for 273 million clinical cases and 1.12 million deaths annually. More than 40% of the global population (>2.1 billion people) is estimated to be at risk.

6.Explain the preventive measures for candidiasis infection.
Candidiasis is caused to the fungus, *Candida albicans*. To prevent the infection, following are a few things to be done:
Maintain good oral health: brush teeth daily, gargle with antiseptic mouthwash (like Listerine) and floss.
Decrease or avoid sugars (corn and maple syrup, glucose, fructose and sucrose). Sugars are food for candida and help it to grow.
Decrease or avoid alcohol. Alcohol converts to sugar and promotes the growth of candida.
Ingest large amounts of garlic (fresh is considered best -- mince and put into empty gelatin capsules, up to six cloves a day). Garlic is believed to have some natural antifungal properties and may help to prevent candidiasis.
Drink milk or eat yogurt that contains acidophilus bacteria. Acidophilus is "friendly" bacteria which helps keep our body in balance and able to fight of "unfriendly" bacteria and fungus, like candida.
Apply yogurt containing "friendly" bacteria directly into the vagina (such as *Lactobacillus bifidus* or *Lactobacillus acidophilus*).
Wear loose fitting clothes to help prevent vaginal candidiasis, since these allow areas of the body ventilate better and dry out.

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