HOST PARASITE RELATIONSHIP

PARASITOLOGY DEFINED: Parasitology is defined as the study of parasites and their relationship to their host. It is one of the most fascinating and rewarding phase of biology. This discipline actually involves several approaches to the study of parasitic organisms.

PARASITISM DEFINED: Parasitism is one of those aspects of biology and in particular ecology, which deals with the relationship of organisms to one another and to their habitat.

Parasitism involves an association between animals of different species where one, the host, is indispensable to the other, the parasite; while the host can quite well do without the parasite. In other words Parasitism is a heterospecific type of an association between two individuals in which one of the partners called parasite is metabolically dependent on another referred to as host.

The relationship may be permanent as in the case of tapeworms found in the intestines of mammals, or temporary, as during the feeding of mosquitoes, leeches, and ticks on their host's blood.

HOST PARASITE RELATIONSHIP INTRODUCTION: When a parasite gains access to a host, the host has to compromise, and the parasite has to adopt itself in host environment. In this way host and parasite establish a sort of relationship which effects each others growth, metabolism, etc.

In general the series of events that constitutes the relation of host and parasite may be considered as beginning with the transmission of parasite from one host to another, then follows the distribution and localization of parasite on or within the host, then growth or multiplication of parasite, the resistance of host to the parasite and the parasite to the host. The method of attack of parasite, changes in host brought about by parasite and those in parasite due to residence in host. Host parasite adjustments during the infection, the escape of infective stages of the parasite from the host and then the recovery or death of host.

HOST-PARASITE RELATIONSHIP STRATEGY

In the host-parasite relationship, we can identify two categories of bio-physiological function. These are:

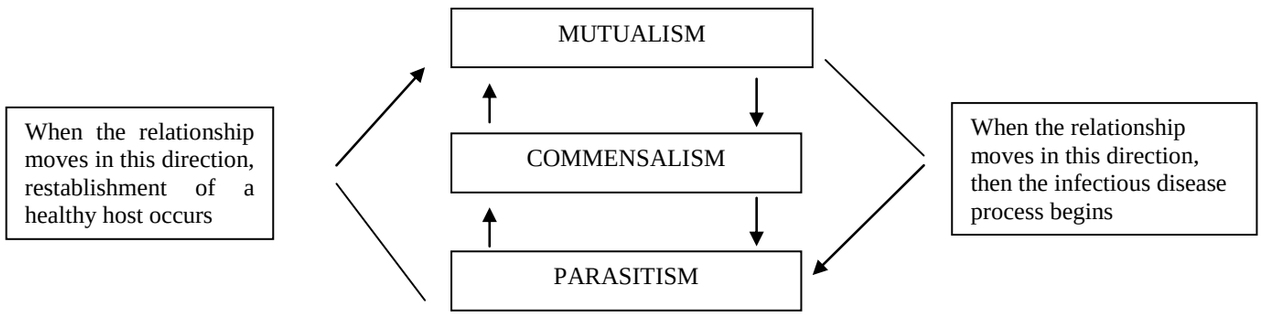
1. Parasite invasiveness which is aimed to obtain entry into the host and continue its life within the host, and
2. Host resistance which tends to prevent the invasion of parasite and its colonization.

In a host-parasite relation we can see that both these functions counter each other thereby acting as a check to maintain balance in the host parasite relationship.

- When a parasite is growing and multiplying within or in a host, the host is said to have an infection.

SYMBIOTIC RELATIONSHIP BETWEEN HOST & PARASITE

Because by definition, parasitic organisms are dependent on their hosts, the symbiotic relationship between the host and parasite is as follows:



HOST PARASITE RELATIONSHIP DEFINED: From the definition of parasitism it is clear that it involves 2 partners, a parasite and a host and also that parasitism effects both the partners. Host Parasite relationship is defined as the influence of each partner by the activities of the other. In general the host-parasite relationship can be studied under two heads:

A. EFFECTS ON THE PARASITE

B. EFFECTS ON THE HOST

A. EFFECTS ON THE PARASITE: Effects of parasite on the host are more obvious than those which operate in the reverse direction, but the later are nonetheless important. The general constitution of the host may profoundly influence the host-parasite relationship. The parasite besides undergoing several modifications called parasitic adaptations to survive in the hostile atmosphere in the host has several specific effects on it as:

A.1. EFFECT OF NUTRITION
A.2. EFFECT OF HORMONES
A.3. EFFECT OF AGE
A.4. EFFECT OF IMMUNITY
A.5. EFFECT OF HOST SPECIFICITY
A.6. EFECT OF PARASITIC DENSITY
A.7. EFFECT OF HOST SEX

A.1. EFFECT OF NUTRITION: The kind of nutritive material ingested by parasites effects their development. A diet consisting largely of milk has an adverse effect on intestinal helminths or protozoan fauna, because it lacks p-aminobenzoic acid which is necessary for the parasite growth. A high protein diet has been found to be unfavourable for the development of many intestinal Protozoa. On the other hand, a diet low in protein favours the appearance of symptoms of amoebiasis.

It has also been shown that carbohydrate rich diet favours the development of certain tapeworms. In fact the presence of carbohydrate in the diet is known to be essential for some of the worms.

The nutritional status is of an increased importance both in determining whether or not a particular infection will be accompanied by symptoms and in influencing their severity if present.

Nutritional disturbances may also influence resistance through its effects upon the immune mechanisms of the host.

A.2. EFFECT OF HORMONES: Hosts hormones have direct effect on the growth and in many cases sexual maturity of parasites *e.g*., *Ascaridia galli* attains greater lengths in hyperthyroid chickens whereas *Heterakis gallinae* attains greater length in hypothyroid host, the two worms apparently respond differently to the hormone thyroxin.

The dog nematode *Toxocara canis* develops into adult only in the female dogs *i.e*., bitches, during their pregnancy as hosts sex hormones are necessary for its maturity and growth.

A.3. EFFECT OF HOST AGE: (Table at end) Human schistosomes usually infect young persons, and adults over thirty generally do not become infected on exposure. Age resistance does not appear to depend on immune reactions but rather to changes in the host tissues that render them as unsuitable environment for the parasite.

A.4. EFFECT OF IMMUNITY: The host produces one or more substances known as antibodies that are chemically antagonistic to the parasite or its products. These antibodies may stunt the growth of the parasite or kill it or prevent its attachment to the host tissues or they may precipitate or neutralize parasitic products.

Primary infection with *Leishmania* seems to confer a degree of immunity to reinfection while many protozoal and helminthic infections confer no long lasting immunity to reinfection. They do seem to stimulate resistance during the time that the parasites are still in the body. This resistance to hyperinfection is known as premonition.

A.5. EFFECT OF HOST SPECIFICITY: The host specificity varies greatly among helminths. Even closely related helminths may exhibit great differences in host requirements. It is usually supposed that a helminth requires a very specific environment complex for its development and this is found only in proper hosts.

A.6. EFFECT OF PARASITE DENSITY: When a number of helminths of one species is present in one host, the worms are usually stunted and of low reproductive capacity. This stunting effect seems to result not from insufficient food supply but from some action of the parasites on each other.

A.7. EFFECT OF HOST SEX: An influence of host sex is evidenced in the development of some helminths *e.g*., *Cysticercus fasciolaris* is more frequent in male than in female rats, as a consequence of the action of sex hormones; gonadectomy lowers the resistance of females and increases that of males to infection, and injection of female hormones into males also increases the resistance of the latter, whereas injection of females with male hormones lowers their resistance to the Cysticercus.

Also *Toxocara canis* develops only in pregnant bitch and not in males and others.

B. EFFECTS ON THE HOST: Sometimes the parasites bring about some changes within their hosts that may be interpreted as affecting the host's welfare. It is necessary to give some consideration to types and degrees of changes caused by the parasitic animals.

In classifying the various types of effects, one should remember that in a number of cases multiple effects may be present and it is often not possible to state that a given parasite causes only one specific type of effect. Furthermore, the types of effects often merge into each other so that sharp lines of demarcation between types cannot always be recognised.

With our advanced knowledge of host-parasite relations it is increasingly apparent that in many instances it is extremely difficult if not impossible to distinguish between a true parasite and a commensal, because the effect of parasite in some cases may be so minute that it can hardly be considered injurious. However, some classical types of parasite inflicted effects on the host can be sighted:

B.1. UTILIZATION OF HOST'S FOOD
B.2. UTILIZATION OF HOST'S NON-NUTRITIONAL MATERIALS.
B.3. DAMAGE TO HOST TISSUE.
B.4. ABNORMAL GROWTH.
B.5. EFFECT OF TOXINS, SECRETIONS, EXCRETION, POISONS ETC.
B.6. MECHANICAL INTERFERENCE.
B.7. BIOLOGICAL EFFECTS IN THE HOST.
B.8. HOST-TISSUE REACTION.
B.9. IMMUNITY TO THE PARASITE.
B.10. WEIGHT AND SIZE GAIN.

B.1. UTILIZATION OF HOST'S FOOD: Utilization of host's food to a detrimental point by a parasite is probably the first kind of damage that comes one's mind. Although in the past some biologists had doubts as the impact of parasites in this regard, since the amount of food microscopic parasites can utilize seems to be negligible. But recent physiological studies of nutritional requirements especially endoparasites have indicated that they robe the host of a good amount of nutrition resulting in serious consequences. *Diphyllobothrium* latum (*Dibothriocephalus latus*) in man has been known to cause an anemia similar to pernicious anemia, because of the affinity this tapeworm has for Vitamin B_{12}. This tapeworm can absorb 10-50 times as much vit. B12 as other tapeworms. Since B12 plays an important role in blood formation, its uptake by *D. latus* causes anemia. Studies of cestode nutritional requirements show that tapeworms not only simple sugars but also nitrogen containing amino acids from the host.

B.2. UTILIZATION OF HOST'S NON-NUTRITIONAL MATERIALS: In some cases parasites also feed on host's substances other than nutrients. The endo- and ecto-parasites that feed on the hosts' blood are examples. The exact amount of blood utilized by these parasites is difficult to measure during the survival in the host. Approximately e.g., due to blood feeding parasitic hookworms, the haemoglobin may drop 30% below normal.

It is obvious that the blood lost through parasitic infections can become an appreciable amount over a period of time. Lepage (1958) estimated that 500 hookworms can remove 250 cc of blood ($1/24^{th}$ of the total volume of blood) each day. This estimate may be too high, for others estimate that no more than 50 cc of blood is removed. Nevertheless, the loss of even 50 cc of blood per day constitutes a serious drainage of blood cells, haemoglobin and serum.

Table showing Blood intake of certain blood-feeding parasites

Parasite	***Host***	***No. of Parasites***	***Amount of blood taken/day***
1. Larvae of *Ixodes recinus*	Sheep	1,000	5 cc

2. Adult *Ixodes recinus*	Sheep	1,000	1 cc
3. *Ancylostoma duodenale*	Man	500	250 cc
4. Necator americanus	Man	500	250 cc

B.3. DESTRUCTION OF HOST TISSUE: Not all parasites are capable of destroying the host's tissue, and even among those that do so; the gradation in the degree of damage is large. The parasites destroy the host's tissue in two ways:

(a) Some parasites destroy (injure) the host's tissue when they enter the host, and

(b) Others inflict tissue damage after they have successfully entered.

A combination of these two types of injury may occur.

The hookworms – *Necator americanus* & *Ancylostoma duodenale* exemplify the first instance, for the infective larva of the nematodes do extensive damage to cells and underlying connective tissue during penetration of the host's skin.

The cercariae of certain Schistosomes that cause "Swimmers Itch" while penetrating the host skin cause inflammation and damage to the surrounding host tissues. Although cercaria caused dermatitis is extremely irritating, fortunately as the result of host incompatibility these worms do not become established in the host's skin by schistosome cercariae not only involve slight tissue damage but more important is that it involves an allergic reaction.

Various armed helminths such as Acanthocephala, certain flukes and tapeworms, initially damage the cells lining the lumen of their host's intestine which they are holding. In most cases the damage is minute, but repeated irritation over long periods can result in appreciable damage. Furthermore microscopic lesions resulting from such irritation can become sites for secondary infections by bacteria.

Amoebic dysentery, caused by *Entamoeba histolytica*, actively ingest the epithelial cells lining host's large intestine, causing large ulcerations that are not only damaging in themselves but also serve as sites for secondary infections. The same amoeba is also known to cause large ulcers in the host's liver. Partial or total destruction of the hepatopancreatic cells of molluscan intermediate hosts harbouring larvae (redae and sporocyst) of digenetic Trematodes is also known. Cheng and James (1960) reported one such case in which the liver of the freshwater bivalve *Spliaerium striatinum* was completely destroyed through ingestion by the rediae of *Crepidostomum cernutum*, a fluke that as an adult is parasite in the intestine of various species of bass. In some cases the molluscan hosts were killed by the severe damage. In addition to direct ingestion, disruption of hepatopancreatic cells caused by the parasites movements and ectolysis caused by the parasites excreta, both contributed to the destruction.

During migration of larvae of the large nematode *Ascaris lumbricoides* within its host, these larvae pass through the lungs of the host, as a result of the migration of a large number of worms, damage sometimes is due to the lung tissue.

Ancylostoma duodenale, one of the hookworms is a good example of a parasite that causes both internal and external tissue damage. The external damage phase has been discussed. Once established within the host's intestine, this roundworm causes considerable damage to the gut wall. It may actually engulf small pieces of tissue, thus producing small lesions.

Histopathological studies of parasite damaged tissues reveal that cell damage other than removal by ingestion or from mechanical disruption is of three major types:

1. Parenchymatous or albuminous degeneration occurs when the cells become swollen and packed albuminous or fatty granules, the nucleus, becomes indistinct, and the cytoplasm becomes pale. This type of damage is characteristic of liver, cardiac muscles and kidney cells.

2. Fatty degenerations, describes the conditions in which the cells become filled with an abnormal amount of fat deposits that give them a yellowish appearance. Hepatic cells display this type of degeneration which are in contact with the parasites.

3. Necrosis occurs when any type of cell degeneration persists. The cells finally die giving the tissue an opaque appearance. In the encystment of *Trichinella spiralis* in striated muscles, necrosis of the surrounding tissue is followed by calcification.

B.4. ABNORMAL GROWTH: One of the possible consequences of parasitism associated with cell and tissue parasites is a change in the growth pattern of the affected tissue. Some of these are serious changes, whereas others are structural and have no serious systemic importance to the whole organism. Such changes can be broadly divided into four main types:

(a) Hyperplasia (Increase in the rate of cell division): It is an accelerated rate of cell division resulting from an increased level of cell metabolism. This results to a greater total number of cells, but not necessarily an increase in their absolute size, *e.g.*, the presence of trematode *Fasciola hepatica* in bile ducts is known to effect rapid division of the lining epithelial cells. The eggs of *Schistosoma haematobium* with their spiny projections are known to irritate the transitional epithelium of human urinary bladder causing hyperplasia. The presence of the protozoan *Eimeria steidae* is known to cause hyperplasia in the hepatic cells of rabbit.

(b) Hypertrophy (Increase in cell size): This condition is commonly associated with intracellular parasites *e.g.*, during the erythrocytic phase of *Plasmodium vivax*, the parasitized red blood cells are commonly enlarged.

(c) Metaplasia (Transformation of one type of tissue into another): Metaplasia describes the changing of one type of tissue into another without the intervention of embryonic tissue. When the lung fluke – *Paragonimus westermani* parasitizes man and carnivores, the normal cuboidal cells lining the bronchioles commonly undergo both hyperplasia and metaplasia and are transferred to stratified epithelium.

(d) Neoplasia (Cancer or tumour formation): It is the growth of cells in a tissue to form a new structure, for example, a tumour. The neoplastic tumour: (i) is not inflammatory, (ii) is not required for the repair of organs, & (iii) does not conform to a normal growth pattern. Neoplasms may be benign or malignant.

Hyperplasia resulting from parasitic infections may result in neoplastic reactions *i.e.*, the development of tumors from existing tissues, other known instances of neoplasm development include adenoma formation and papilloma which is caused by *Schsitosoma mansoni* eggs in the colon of man.

Bullock Chris (1920) demonstrated that cysticerca of *Taenia taeniaeformis* in the livers of rats, mice and other rodents can cause the formation of tumours, other known instances of neoplasm development include adenoma (gland cells surrounded by connective

tissue) formation from *Eimeria stiedae* infections in the epithelial lining of bile ducts of rabbits. Papilloma (a core of vascularized connective tissue surrounded by epithelial cells) formation as the result of the presence of *Schistosoma mansoni* eggs in the colon of man, and growths in the stomachs of cats and dogs resulting from infections with the roundworm *Gnathostoma spinigerum*. True cancerous neoplasm has been reported associated with helminth parasites. The liver fluke *Opisthorchis sinensis* has been suspected of initiating cancer in the liver of man, the lung fluke *Paragonimus westermai* has been suspected of contributing to cancer in the lungs of tigers.

B.5. EFFECT OF TOXINS, SECRETIONS, EXCRETIONS, POISONS ETC.: Specific toxins or poisons, egested, secreted or excreted by parasites have been sited in many cases as the cause of irritation and damage to hosts.

Example of irritating parasite that elicits an allergic reaction in the host is that of *Schistosoma* cercaria which causes cercarial dermatitis. The severe inflammatory reaction of the host tissue strongly suggests that the fluke secretes some substances that causes the inflammation and indeed such a secretion is now known to exist.

In case of blood sucking insects such as mosquitoes, the swelling resulting from the bites represent the host's response to the irritating salivary secretions of the insect.

A known parasitic toxin is the perintestinal or coelomic fluid of the nematode *Parascaris equorum*. The irritability of this fluid to the cornea and mucus membranes of the nasopharangeal cavity is well known. Weinburg & Julian (1911, 13) collected a quantity of this fluid under aseptic conditions and injected it into guinea pigs, not surprisingly, they found that 0.5 μmm of this highly toxic fluid kills a guinea pig. Weinberg also placed drops of this fluid in the eyes of horses and found that it generally caused a violent reaction. Some horses, however, were not affected. Further investigations revealed that the unaffected horses were heavily infected with *P. equorum*, thus suggesting that these hosts had developed immunity against the toxins.

B.6. MECHANICAL INTERFERENCE: Less is known about the injuries to the host resulting from mechanical interference by parasites. According to Cheng (1964) they are more common than generally supposed.

The best known case of the type of damage is elephantiasis. In persons infected with the filarial nematode *Wucheraria bancrofti*, the adult worms become lodged in the lymphatic ducts and thus obstruct the lymph into abnormal channels resulting in the swelling of affected parts, thus causing elephantiasis.

Mechanical damage by nematode is also demonstrated by *Ascaris lumbricoides* in the intestine and bile ducts of their host. This intestinal parasite which measures up to 14 inches in length, when person in large numbers, can easily block the normal flow of bile down the bile duct and the passage of chime into the intestine.

Another example of mechanical interference is provided by *Echinococcus granulosus* whose fluid filled hydatid cysts often attain large size and prevent the proper functioning of neighbouring organs due to pressure. These cysts are often found in the liver, lungs or brain where they may prove fatal. These fluid filled cysts can attain a diameter of several inches. The Cysticercus larva of the tapeworm *Multiceps multiceps* is known to exert extreme pressure on the brain and spinal cord of sheep, which serve as intermediate hosts. Infected sheep are said to suffer from staggers or gid because of the struggling movements resulting from pressure on brain.

It is known that erythrocytes of chickens infected with the avian malaria organism *Plasmodium gallineraium* have a tendency to stick together, thus clogging the fine capillaries. Blood vessels damaged up by the infected blood cells often rupture. Those rupturing in the area of the brain permit blood to take into the brain tissue, thus often causing the death of the host. Similar instances have been reported among human victims of malaria.

Strangulation of fishes whose gills are paralyzed by monogenetic Trematodes is another good example of mechanical damage to hosts.

B.7. BIOLOGICAL EFFECTS IN THE HOST (Sex reversal): Other interesting and challenging aspect of host parasite relationship are the biological effects on the host. Among the most important and interesting of these are the secondary manifestations resulting from damage to the specific organs. Giard (1911, 13) and Smith (1910, 11) have shown that in crabs parasitized by *Sacculina*, the host's genital tissue is invaded by this crustacean causing drastic changes in males, but not in females. 70% of the parasitized male crabs undergo degeneration of testes and acquire secondary female characteristics. This was found to be true also of ovarian tissue in parasitized females, thus an infected female crab acquires male characteristics. Hence there is a complete loss of sexual dimorphism. However, if the parasites are removed, female acquires normal ovaries but the males' originally atrophied testis develop into ovotestes capable of producing both the eggs and the sperms. Such an effect on the host due to the parasite is termed as **parasitic castration**. There have been many studies of *Sacculina* because this unique crustacean parasite is known to effect the metabolism and sex-life of its host.

The parasitic isopod *Entoniscus* begins as an ectoparasite, but it soon enters the body cavity of the host and eventually causes castration without ever touching the hosts' gonads.

Another interesting parasitic caused biological change has to do with metabolic alterations in the host. Wheeler (1910) pointed out that workers of the ant *Pheidole commulata*, when parasitized by the found worm *Mermis*, becomes hypertrophied & the entire body becomes much larger than that of normal ant.

B.8. HOST TISSUE REACTION: In instances of host tissue reaction, certain host cells and cell products aggregate around the invading parasite forming what is commonly known as host cyst, although cysts are not always of host origin *e.g*., when the metacercaria of the yellow grubs, *Clinostomum marginatum* encysts in the skin of fish, two cyst walls are formed around the parasite. The inner one is secreted by the parasite, the outer one is laid down by the host in response to the parasitic invasion. Although the double wall of Trematode metacercarial cysts is a common occurrence, single walled cysts also occur.

Another example of host-tissue reaction is demonstrated by *Trichinella spiralis* encystment in mammalian muscles. Once the nematode larvae reach the musculature, it is surrounded by connective tissue cells and eosinophils. These host cells soon form a capsule around the coiled worm (larvae) hence the cyst is completely of host origin. At times, the capsule becomes calcified, with calcium, coming primarily from the host, although there is some evidence that the parasite also contributes some calcium.

B.9. IMMUNITY TO THE PARASITE: Majority of the hosts build resistance or immunity against the parasite and they show no visible effects. Immunity may be natural or acquired by previous infections.

Parasitism often becomes an elaborate compromise between the parasite and the host. The parasite harmonizes itself to the habitat and the host protects itself by formation of antibodies and by increasing its efficiency for repair of tissues. This delicate adjustment between the host and the parasite is very common but if it is lacking; then either the parasite fails to survive or the host injured and destroyed.

B.10.WEIGHT AND SIZE GAIN: Some authors believe that due to presence of parasite the hosts gain weight and size. Muller (1972) found that male rats gained weight when they ingested scolices of *Sporgana* of *Spirometra mansonoides*. Some species of carpenter ants infected with metacercariae of *Brachyleistrium mosquensis* are more obscure than the uninfected ones.

C.1. HOST – PARASITE SPECIFICITY

This term indicates the relation between hosts and parasites regarding the degree of susceptibility of the host and degree of infectivity or powers of infestation of parasite e.g., man is susceptible to infections or infestations with certain species of Protozoa, worms and arthropods and this degree of susceptibility differs more or less for each species; rats are susceptible to certain of the same species and to different species as well. This is host-parasite specificity viewed from the stand point of the host.

On the other hand host-parasite specificity when applied to the parasite refers to their degree of infectivity or powers of infestations *e.g.*, *Entamoeba histolytica* is known to be able to live in man, monkeys, dogs, rats & cats, but infects certain species of hosts more readily than others and varies with respect to its pathogenicity in different hosts.

C.2. HOST SUSCEPTIBILITY

Parasitologists have long recognized different types of hosts with respect to their susceptibility to various parasites. Thus if a host is easily parasitized by a certain species, it is said to be tolerant, whereas if it is difficult to parasitize, it is called as refractory. A host that is frequently found parasitized by a certain species in nature – Natural host, whereas one that does not become so parasitized is called foreign host.

If a species of parasite that habitually lives in or on a host that is very seldom infected that is spoken as an accidental or casual host. A host may become infected but throw off the infection after a short time is known as provisional transitory host; or it may serve as a host for short stage in life cycle of a parasite – temporary host.

Whether or not a susceptible species of animal becomes infected with a particular species of parasite in nature depends primarily on 3 factors:

(i) Animal and parasite must live in the same geographical region;

(ii) The habits of the animal must be such as to bring it into proper relation with the infective stages of the parasite;

(iii) The life cycle of the parasite must be such that their infective stage is reached when & where the host is available to be parasitized.

C.3. IMMUNOLOGICAL INTERACTION BETWEEN HOST AND PARASITE

As the host has immune system which has efficiency to destruct the parasites by producing antibodies (IgG, IgE, etc.) against the specific parasites or their products. Host's Immune system usually performs Antigen-Antibody reactions, such as toxin neutralization, Agglutination, Precipitation, Lyses, Compliment fixation, Increased Phagocytosis (Opsonification) & allergic sensitization. With the help of these host can resist against the parasite or can destroy it.

Parasites also have evolved mechanisms to evade their host's immune systems. Usually the mechanisms used by parasites for defending them against the host response are: Induce Immunosuppressions, Becoming hypoantigenic, Change their surface antigens rapidly & repeatedly (*e.g.*, *Trypanosoms rhodiense*, *T. gambiense*), Becoming functionally non-antigenic (*T. theileri*, *T. lewis*), Mimicry of host antigens, Adsorption of host antigens (*Schistosoma mansoni*), Antigenic variation and blocking antibodies.

C. CONCLUSION

Host-Parasite relationship is the extreme case of animal association, in which both partners influence each others life by affecting each others metabolism and behaviour using different adaptive mechanisms in order to ensure their survival.

Although the parasite leaves no stone unturned to establish itself fully in its host by utilizing its food, destroying its tissues, secreting poisons and by deceiving host's immune system by molecular mimicry. On the other hand the host tries its best to destroy the parasite by activating its immune system. Yet neither the host nor parasite succeeds to destroy each other completely.

Host tries to avoid unwanted pathogenic, potent, prolific, parasitic guest and parasite tries to have an intimate relationship with its host so that it becomes a permanent guest, however at some point of relationship, one partner or both partners manage to remain independent, resulting in death of either host or parasite mostly of parasite.

So it seems that the relation between host and parasite is eternal and are made for each other.

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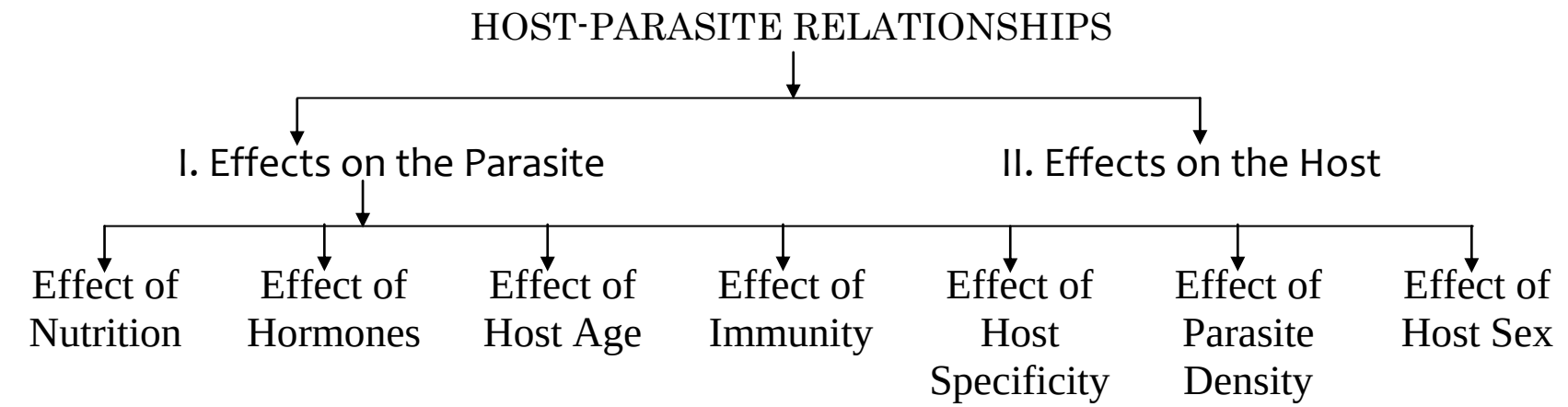
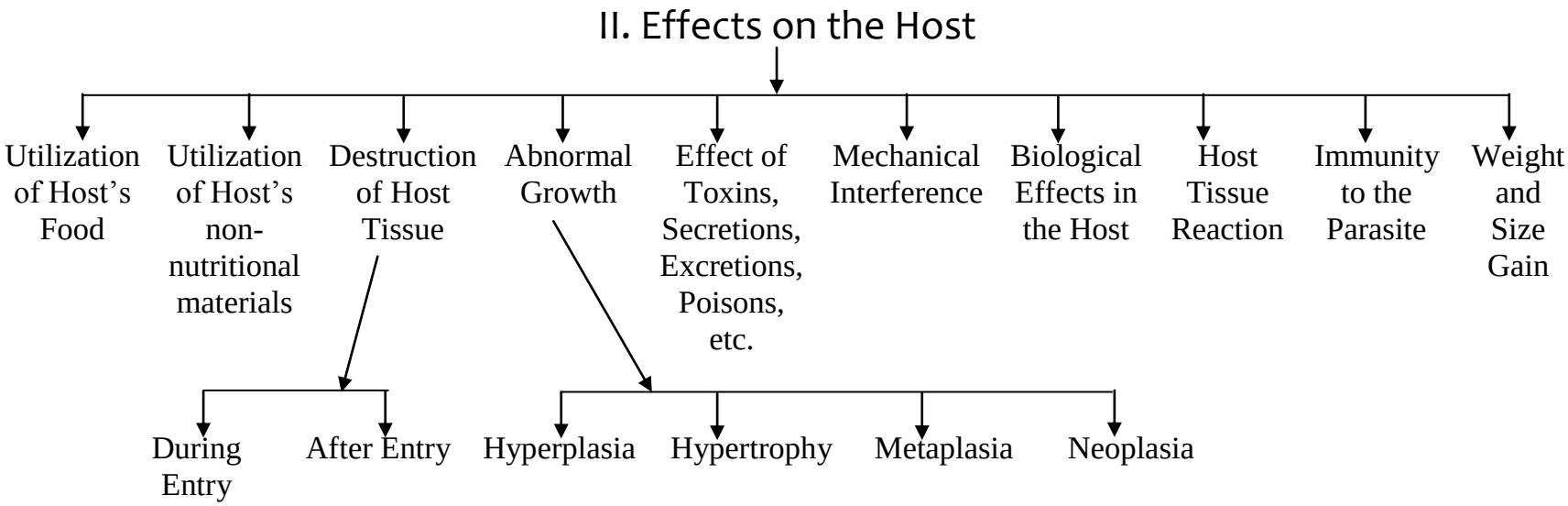
IMMUNITY

a. natural Immunity	**b. Acquired Immunity**	**c. Active Immunity**	**d. Tolerant Immunity**	**e. Passive Immunity**	**f. Premunition**
Most of the parasites can have a specific host only, *e.g.*, human beings are immune to infection by avian and most of the Simian *Plasmodia*. This is because of Natural Immunity.	Antigenic stimulation by the parasite results in acquired immunity against them.	The host acquires it after acquiring a disease. It may be **concomitant**. It means that immunity is present when there is **parasitemia**. It may be residual *i.e.*, it persists after eradication of the parasite.	This immunity decreases the effect of disease on the host for a particular number of parasites. It is because of this that a larger number of parasites are needed to produce infection.	The transfer of antibodies from the mother to the child through the placenta in the first milk causes immunity in infants. Passive immunity can also be conferred by the administration of antisera or gamma globulins isolated from man.	This is an incomplete immunity as it exists as long as the host harbours the parasite. It tends to disappear right after the host is cured of the parasite.


HOST-PARASITE RELATIONSHIPS
I. Effects on the Parasite
II. Effects on the Host
Effect of Nutrition
Effect of Hormones
Effect of Host Age
Effect of Immunity
Effect of Host Specificity
Effect of Parasite Density
Effect of Host Sex


II. Effects on the Host
Utilization of Host's Food
Utilization of Host's non-nutritional materials
Destruction of Host Tissue
Abnormal Growth
Effect of Toxins, Secretions, Excretions, Poisons, etc.
Mechanical Interference
Biological Effects in the Host
Host Tissue Reaction
Immunity to the Parasite
Weight and Size Gain
During Entry
After Entry
Hyperplasia
Hypertrophy
Metaplasia
Neoplasia