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Constraints faced by parasites during symbiosis – a review

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Although parasitic infections remained major obstacles to economic progress and a better life in developing countries due to their ability to damage the host's tissues directly through metabolism, there are many constraints that parasites faced in the course of their interaction with the host. The present review was conducted to document the various challenges faced by the parasites while forming a symbiotic association with their host. Over sixty (60) articles were reviewed to get information on the constraints faced by the parasites of some organs such as skin, blood, gut, and liver. Examples of parasites living on the skin are lice, fleas, mites, ticks, *Chilodonella*, and *Trichodinids,* while identified constraints faced by parasites on the skin are: nutrients, life cycle stages, desiccation, water*, and temperature; the* Blood parasites include *Trypanosomes, Schistosomes,* and *Plasmodium,* among others*.* Some difficulties in the blood habitats faced by the parasites include host immune responses, life cycle stages, and drainage issues. Similarly*,* the gut parasites include *E. histolytica, S. mansoni, A. lumbricoides,* etc; and the challenges faced by parasites living in the gut include; pH, peristalsis, enzyme digestion, life cycle stages, and others. The present review highlighted and suggested that the hosts should take other necessary efforts that will prevent becoming exposed to parasites through busting their immunity, personal hygiene, and environmental sanitation, because lack of such preventive and control measures may favor parasites from always wine host using some mechanisms to overcome the mentioned constraints.

Keywords: Constraints, Parasites, Skin, Blood, Gut, and Liver.

1. Introduction

A non-mutual symbiotic connection, or parasitism, occurs when one species (the parasite) benefits at the expense of the other (host) (Brown and Neva, 2005). The interactions of tapeworms, flukes, Plasmodium species, and fleas with vertebrate hosts are well-known examples of parasitism that can lead to parasitic diseases (Crewe and Blacklock, 2001). An organism is considered a parasite if it survives by inhabiting or feeding off of another organism (the host) (Despommier *et al.,* 2013). In contrast to predators, parasites usually do not kill their hosts, are considerably smaller than their hosts, and frequently stay inside or on them for long periods. They also exhibit a high degree of specialization and reproduce more quickly than their hosts do (Frank, 2013).

A habitat is an environment where an organism lives; it comprised all biotic and abiotic factors that influence the activities of living organisms (Hatcher and Dunn, 2011). The habitat of the Parasite refers to the place in which the parasite lives and where it can find food, shelter, protection, and mates for reproduction (Lively and Dybdahl, 2010). According to habitat, parasites are categorized into two namely ecto and endo parasites. Ecto-parasites are those parasites that are found on the host while endo-parasites are those parasites found inside the host (Maizels, 2019). The place or position that particularly suits organisms to carry out their life activities is called a "niche" (Hatcher and Dunn, 2010). An environment of parasitic organisms consists of a Macro and Microhabitat, the macro habitat is a place where the host lives while a place where parasites live is called a microhabitat (Manson-Bahr and Bell, 2011).

Parasitic infections of one kind or the other have been estimated to affect more than 3 billion people in the world. Compared to the developed world, where there has been considerable progress in combating major diseases, parasitic infections have remained a major obstacle to economic progress and a better life in developing countries (Muller and Baker, 2013). The most serious parasitic infections are acquired in poor tropical and subtropical areas, but some also occur in the developed world; other, less serious, infections are worldwide in distribution (Peters and Gilles, 2012). Exposure to infection is influenced by climate, hygiene, food preferences, and contact with vectors (Price, 2010).

Many potential infections are eliminated by host defences; others become established and may persist for prolonged periods, even years. Although infections are often asymptomatic, severe pathology can occur (Rook, 2017). Because many parasites are large and often migrate through the body, they can damage the host's tissues directly by their activity or metabolism; almost all organ systems can be affected (Rózsa *et al.,* 2017).

The host provides habitat and a vast amount of physiological and biochemical requirements to the parasites for protection, reproduction, and other necessary life activities and parasites caused different pathogenicity to the host directly, in some cases, damage also occurs indirectly as a result of the host defence mechanisms (Werren and John, 2018).

Interaction with a host, which makes up a biotic environment, is necessary for parasitism. The parasites create a wide range of defence mechanisms while the host tries to drive them out. Such interactions may be useful to comprehend and take advantage of in the battle against parasites. This review's goal is to identify and discuss the many restrictions that parasitic habitats face while interacting with hosts.

1.1 Mode of Entry into Habitats by Parasites

Endoparasites can infect a host in a variety of ways. The most straightforward is caused by accidentally ingesting a parasite's infective stage; several protozoan parasites, as well as worms, possess infective stages that aggressively enter the skin (for example as seen in *Trypanosomes*, *Schistosomes*, *Strongyloides*, etc) through the bite of intermediates host. For example, the arthropod vectors of plasmodium, trypanosomes, and filarial worms can transmit infectious stages when they bite the host to take a blood meal; in other instances, the larvae are contained in the tissues of the intermediate host and are consumed when a human eats that host (Clonorchis in fish, tapeworms in meat and fish, and *Trichinella* in meat) (Donelson, 2016).

Therefore, the degree of infection in the host depends on hygienic standards (since cyst, trophozoite, egg, and larvae are frequently transmitted in urine or faeces), the climate (which may favour the survival of infectious stages), the preparation of food, and the level of exposure to insect vector (Despommier, 2013).

1.2 Factors Influencing Host Susceptibility

A significant aspect affecting susceptibility to infection is human behaviour. Certain behaviours, especially those related to diet and cleanliness, will increase exposure if the infective stages of parasites are present in the environment. *Strongyloides*, *Trichinella*, and some tapeworm larvae are a few exceptions to the rule that most parasites do not multiply within the same host, hence the number of infective stages seen strongly correlates with the severity of the illness (Desjardins and Descoteaux, 2017).

Not all exposures result in the growth of an infection that is fully developed. The host's general defence mechanisms eliminate a large number of infectious germs. Of those who do survive and establish themselves, many are wiped out or annihilated by certain defences. The amount of parasites that are present at any given time consequently shows a dynamic balance between the pace of infection and the effectiveness of defence (Degrave *et al.,* 2018). Changes in the host's behaviours and capacity to express defence mechanisms affect this balance, which represents the host's total susceptibility (Colley, 2010).

Children are usually the much more extensively afflicted individuals of society since they are commonly more sensitive to various parasites than adults. Increased infection levels could potentially be caused by aging's effects on immune function. It is well recognized that in sick groups, certain individuals are predisposed to greater outbreaks than others. People are different genetically in their ability to resist infection. The hormonal and immunological changes that occur during pregnancy and lactation as well as dietary changes may have an impact on susceptibility. The immune suppression that results from concurrent infections with some other diseases and the growth of specific malignancies is a significant factor in increased susceptibility. Similar to radiation therapy, immunosuppressive medications may make people more vulnerable to parasite infection. The emergence of disseminated Strongyloidiasis, in which a high number of larvae develop in the body via autoinfection from relatively few adult *S. stercoralis*, poses a special risk to immunocompromised people. Intriguingly, the human immunodeficiency virus does not raise people's vulnerability to contracting certain parasite infections generally (Donelson *et al.,* 2019).

1.3 Factors Influencing Parasite Susceptibility

The capacity of parasites to evade host defence mechanisms and increase their chances of survival counteracts the capacity of hosts to control infection. Many parasites are unaffected by the host's attempts to restrict their activity or eliminate them simply because they are huge, mobile can hide in a cryptic location, and can infiltrate particular immune responses. Many significant species (*Ascaris*, hookworms, hydatid cysts, and *Trichuris*) are several centimetres in length or diameter, while some are tiny (Dowdle, 2018).

Many defence mechanisms become ineffective by size alone, as well as the mature roundworms' thick cuticle. Because they can actively travel through tissues, parasites can get away from inflammatory foci (Dowdle and Hopkins, 2019). Since the host is exposed to long-term harm, immunological stimulation, as well as the obvious physical effects of being inhabited by big foreign entities, many of the pathogenic effects of parasite infections are connected to the size, mobility, and longevity of the parasites (Ferreira *et al.,* 2000).

2. Living on the Skin

The skin serves as the initial line of protection against encroaching parasites. Dead cell layers that make up skin serve as a physical barrier to encroaching pathogens. The protein keratin found in these dead cells gives skin its resilience and water resistance. The majority of microbes require moisture to survive. It keeps the skin dry, which hinders the colonization of invasive organisms. Additionally, sebum, which is produced by human skin and is harmful to the majority of bacteria, provides a habitat for parasites (Finkelman *et al.,* 2011). Lice, fleas, mites, ticks, *Chilodonella*, *Trichodinids*, and Monogeneanes (Gyrodactylus and Capsalids) are a few examples of parasites that live on the skin (Reiner, 2014).

2.1 Constraints on Skin Habitat

The constraints faced by parasites on the skin are: Nutrients, Life cycle stages, Desiccation, Water and Temperature (Sasa, 2016)

2.2 How they Overcome Skin Constraints

2.2.1 Nutrients

Parasites must connect with hosts since nutrition is a hallmark of living things. However, because the host's skin is strong and selectively semi-permeable, it is challenging for parasites to obtain nutrition from the skin. The toughness and permeability of skin make it difficult for parasites to absorb nutrition; to get over this limitation, they employ a variety of techniques to extract food or nutrients from the skin (Schofield and Dias, 2019).

As an illustration, many ectoparasites are capable of sucking blood with their proboscis while injecting saliva that contains an anticoagulant (vasodilator) and utilizing the nutrients from the blood for growth and other life processes. These ectoparasites include, for instance: Lice and ticks (Roos *et al.,* 2019). Certain skin parasites possess hooks that enable them to firmly cling to the host's skin and feed on using a structure called prohaptor, as is the case of fish skin parasites known as monogeneans (Foley and Tilley, 2019).

2.2.2 Life Cycle Stages

Skin parasites struggle to complete the phases of their life cycle on the skin habitat. These parasites have created several strategies to get around this. For instance: *Tunga penetrans* only requires the host to produce eggs; it completes the rest of its life cycle without it (Rosmaninho *et al.,* 2010) The ability of female lice to discharge glue from their reproductive organ and cover their eggs' tiny living spaces, where the baby can breathe, is a second example (Leung *et al.,* 2010).

Additionally, as monogeneans contain both male and female reproductive organs and are viviparous, they do not need a partner to procreate with them. Instead, an embryo can develop inside the mother (Bakke *et al.,* 2007)

2.2.3 Desiccation

Not only did the glue released by *Pediculus humanuscorporis*' reproductive system aid in the attachment of the eggs for future development, but it also helped prevent the desiccation of the parasites' embryos (Rosmaninho et al., 2010). Ticks' delicate, leathery integument protects them from desiccation (Werren and John, 2003). To prevent desiccation, several monogeneans that live in terrestrial habitats often stay away from the host's most exposed exterior surface (Chaudhary *et al.,* 2013).

2.2.4 Water

In addition to having the ability to obtain water from their macro environment, facultative skin parasites such as *Gyrodactylus* and ***Capsalids*** may move around in quest of water wherever it may be found (Hunter and Barker, 2013). According to reports, many obligate ectoparasites, including ticks and lice, may obtain water from the blood's plasma (liquid component) (Downs *et al.,* 2012)

2.2.5 Temperature

There is little microclimatic improvement for parasites in aquatic environments near the surface of the host animals. The environment adjacent to the host skin may be much less thermally changeable for terrestrial ectoparasites, notably birds and mammals, and the parasites can use this temperature for their metabolic activities (Mumcuoglu *et al.,* 2018).

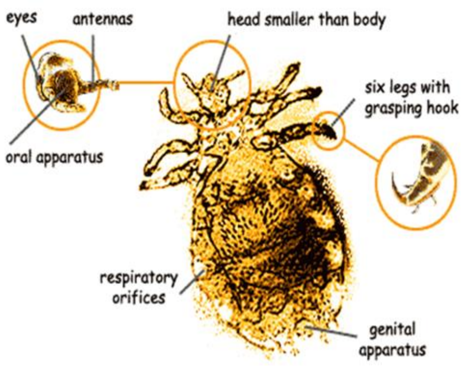

Figure 1. Lice shows some structures such as hooks, and respiratory orifices that help the parasite to overcome constraints on skin habitats (Orion *et al.,* 2014)

3. Living in Blood

Schistosomes, Trypanosomes, and Plasmodium are a few examples of blood parasites (Nogueria and Cohn, 2017). Blood parasites are protected against physical removal, but they are unaffected by temperature, desiccation, oxygen, nutrition, or other factors. However, the blood habitat has significant limitations (Pearce and Sher, 2018).

3.1 Constraints in Blood Habitat

The constraints faced by the parasite living in blood habitat include: Host immune responses, life cycle stages and problem of drainage (Mordue *et al.,* 2019).

3.2 How Blood Parasites Overcome the Constraints

3.2.1 Host Immune Responses

The skin parasites can create several escape mechanisms to defeat the host's immune response (Millar *et al.,* 2015). Parasites use escape mechanisms to circumvent the immune system's virulent effects in an immune-competent host. The following are some examples of blood parasites' escape mechanisms:

a) Antigenic Masking: A parasite's capacity to evade immune identification by covering itself with host antigens is known as antigenic masking (Montesano *et al.,* 2015). The cell surfaces of numerous *Trypanosome* species are covered in host immunoglobulins. According to various investigations, these antibodies are most likely attached to the *Trypanosomes* through the Fc region of their molecules rather than through their variable sections. These antibodies may conceal the parasite, preventing the host's immune system from recognizing it (Millar *et al.,* 2015).

b) Blocking of Serum Factors: Many parasites develop a layer of non-cytotoxic antibodies or antigen-antibody complexes that sterically prevent specific antibodies or lymphocytes from adhering to the parasite surface antigens (Marchesini *et al.,* 2010). Antigen-antibody complexes in the serum of infected animals have been theorized to occasionally bind to the surface of the parasite, physically impeding the actions of cytotoxic antibodies or lymphocytes and directly limiting the actions of lymphocytes. Both tumour cells and parasitic helminths have been suggested to have this type of immune escape mechanism (Liew *et al.,* 2019).

c) Intracellular Location: Some parasites are shielded from the host's immune response by their internal environment. This tactic also delays the immune system's identification of the parasite antigens (Lingelbach and Joiner, 2018).

Numerous protozoan parasites develop and multiply inside host cells. For instance, Plasmodium parasites develop in red blood cells after first developing in hepatocytes. One genus of parasitic protozoa, *Theilera*, not only multiplies in lymphocytes but also seems to boost the proliferation of the infected lymphocytes. *Leishmania* and *Toxoplasma* organisms are capable of developing in macrophages. While some parasites, like *Plasmodium*, are bound to specific types of host cells, parasites like *T. cruzi* and *Toxoplasma* appear to be able to multiply and flourish in a wide range of host cells (Lingelbach and Joiner, 2018).

A parasite may be shielded from the damaging or fatal effects of antibodies or cellular defence mechanisms by an intracellular sanctuary. Plasmodium, for instance, might only be vulnerable to the effects of an antibody during the brief extracellular phases of its life cycle (the sporozoite and merozoite stages). It is important to keep in mind that Plasmodium actually lives inside a host cell vacuole that is membrane-bound. Thus, at least two host membranes protect plasmodia from their surroundings (the outer cell membrane and an inner vacuole membrane) (Lauer *et al.,* 2017).

Although early on in their growth, intracellular plasmodia are very effectively protected from the host's immune response, this tactic does cause physiologic issues for the parasite. For instance, the parasite must pass three membranes (two hosts and one parasite) to absorb nutrients for growth and pass the same three membranes to pass waste products. This issue is resolved by plasmodia by correctly altering host cell membranes. The outer membrane of red blood cells contains parasitic proteins. These antigens eventually elicit a response from the host, and this response ultimately causes an increase in the elimination of infected host cells (La Flamme *et al.,* 2019).

d) Antigenic Variation: During an infection, certain parasites modify their surface antigens. The immune response to the original antigens is bypassed by parasites carrying the new antigens (Kane and Mosser, 2010). It is known that three significant kinds of parasitic protozoa can alter the antigenic characteristics of their surface coat. Every time the host displays a fresh humoral reaction, the African trypanosomes can totally replace the antigens in their glycocalyx. The African trypanosomes escape their host's defensive mechanism in part thanks to these changes in serotype, and less well-known, comparable modifications are also reportedly present in *Plasmodium*, Babesia, and Giardia (Johnson *et al*., 2015).

e) Immunosuppression: Host immunosuppression is typically brought on by parasitic infections. This diminished immune response could postpone the discovery of antigenic variations. Additionally, it can lessen the immune system's capacity to stop parasite growth and/or eradicate it (Hayward *et al.,* 2010).

Nearly every parasitic organism that has been extensively researched to date has been associated with the immunosuppression of the host. In some circumstances, the suppression is particular and only affects the host's reaction to the parasite. Other times, the inhibition affects a wide range of heterologous and non-parasite antigen responses. That this immunosuppression enables the parasites to live in a usually immune-competent host has not yet been established (Zhang *et al.,* 2011).

3.2.2 Life Cycle Stages

Some blood parasites have the potential to proliferate by fission at a higher rate in the blood and are capable of escaping into intermediate hosts because there is no direct aperture in the blood through which the parasites can release eggs. Examples include *Plasmodium* and *Leishmania* (Zhang *et al.,* 2011).

3.2.3 Drainage

Due to their dorsoventrally flattened bodies, many blood parasites, like *Schistosomes*, can escape being washed out by blood circulation in the blood arteries (Wiser *et al.,* 2017).

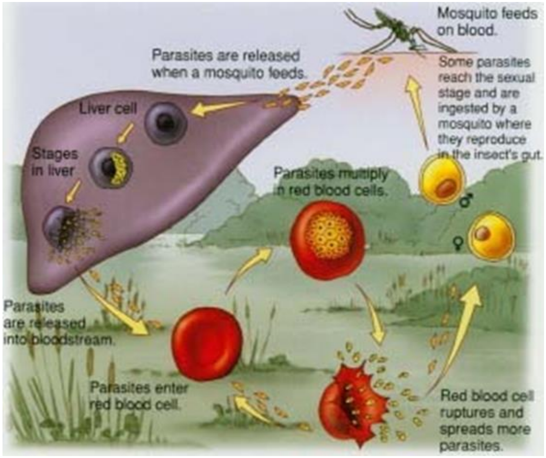

Figure 2: Shows how blood parasites (Plasmodium) reproduce in liver red blood cells (Hayward *et al.,* 2010)

4. Living in Gut

The intestinal parasites include *E. histolytica, S. mansoni,* and *A. lumbricoides* among others. Saliva in the mouth serves as a barrier to saliva, preventing foreign organisms from entering the body orally. Lysozyme, an enzyme present in saliva and tears, is also present in the mouth. This enzyme dissolves the cell walls of invasive microbes (White *et al.,* 2017).

The stomach is the body's second line of defence after the mouth, where the organism passes. Since gastric acids and hydrochloric acid are present in the stomach, their pH is close to 2. In this situation, the stomach's acidity aids in the killing of the majority of bacteria that attempt to enter the body through the gastric intestinal tract (Okhuysen and White, 2019).

4.1 Constraints in Gut Habitat

These showed that constraints faced by parasites in the gut include: pH, Peristalsis, Enzyme digestion, Life cycle stages (Petri *et al.,* 2014)

4.2 How Gut Parasites Overcome Such Constraints

4.2.1 pH

This is the negative logarithm of hydrogen ion concentration; it measures the acidity and alkalinity of a substance. The pH of the gut ranges from 1.5 to 8.4 (Haque *et al.,* 2013). Eggs or larvae of parasitic worms and other categories of parasites must pass through the mouth and stomach before being established in the small or large intestine, they must be able to withstand drastic changes in pH along the gut as they pass and takes advantage of being established (Tanaka and Tsuji, 2017).

For example, *Ascaris* eggs, hatch requires pH (7.3) with carbon dioxide at 370C which is available in the small intestine; Secondly, Infective larvae of *Haemencus contortus* are excited at 370C, pH 7.0 with high carbon dioxide (Su *et al.,* 2019).

4.2.2 Peristalsis

This is the muscle motion used to move food that has been consumed from one area of the gut to another. The parasites in the intestines could be killed by this movement or moved to a more dangerous environment (Okhuysen *et al.,* 2019).

To get over these restrictions, the gut parasites had attachment mechanisms like suckers in trematodes and hooks and chitinous teeth in nematodes, which allow them to firmly connect to the gut wall (White *et al.,* 2017). Additionally, intestinal parasites have a larger capacity for reproduction. In the intestine, protozoa parasites can reproduce quickly by simple binary fission, while nematodes like *A. lumbricoides* can lay more than 200, 000 000 eggs each day (Sinis and Sim, 2011).

4.2.3 Enzyme digestion

The intestinal parasites have created specific strategies to get over the limitations of enzyme digestion in the gut (Mehmet *et al.,* 2015). The majority of parasites that are detected in the gut have a covering that prevents digestive enzymes from breaking them down. This covering may be an impermeable cell membrane (in protozoa), teguments in cestodes, or a thick cuticle (in nematodes) (Sinis and Sim 2011). Some parasites in the stomach can produce inhibitor (anti enzyme) molecules that render the digestive enzyme inactive as seen in*, H. diminuta* (Scott *et al.,* 2010).

Some parasites that live in the stomach can use digestive enzymes to their advantage. *Taenia taenia formis,* for instance (White *et al.,* 2017).

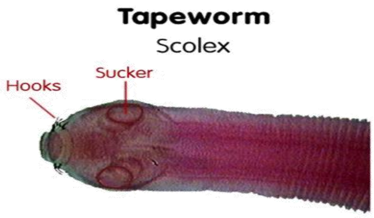

Figure 3: Gut parasite (*Taenia*) shows hooks and suckers which help them overcome some constraints in the gut (Okhuysen *et al.,* 2019)

5. Living in the Liver

Examples of parasites found in the liver are *F. hepatica, F. gingantica, Echinococcus granulosus, and Clonorchis sinensis* (Fawole *et al.,* 2008).

The liver is highly rich in growth factors such as iron, vitamins, and plasma proteins. However, there are some constraints to the parasitic activities in the liver (Happi *et al.,* 2010).

5.1 Constraints in Liver Habitat

These include: Host immune response, Drainage and Barrier (Pantelouris, 2015)

5.2 How Liver Parasites Overcome Such Constraints

5.2.1 Host Immune Response

The liver parasites can create specific escape mechanisms to circumvent the host immune response. For instance, *F. hepatica* can release cathepsin L proteinase, which inhibits eosinophil adhesion to newly excysted juvenile parasites mediated by antibodies (Olsen, 2012).

Secondly, the superoxide dismutase produced by *F. gigantica* neutralizes superoxide radicals that the host secretes to target the juveniles (Charlier *et al.,* 2014). Because they can grow and replicate in liver cells, some liver parasites, including *Plasmodium* sporozoites, might weaken the host immune response (Hickey *et al.,* 2015).

5.2.2 Drainage

Blood circulation in the vessels has become a constraint to the liver parasite but they possessed a flat body which gives them the ability not to be washed out of the liver example as seen in *F. gigantic* (Heppleston, 2012).

5.2.3 Barrier

The liver has several barriers that stop external substances and some microbes from passing through and entering the liver. The Second Barrier for Sporozoites Infections is made up of the Sinusoidal Barrier and Kupffer Cells (Rowclife and Ollerenshaw, 2015). They have created many strategies to combat liver parasites. For instance, plasmodium had an apical complex that allowed it to get through the kupffer cells and sinusoidal barrier, while *F. hepatica* had suckers that allowed it to pass through the barriers while still alive (Bennema *et al.,* 2011).

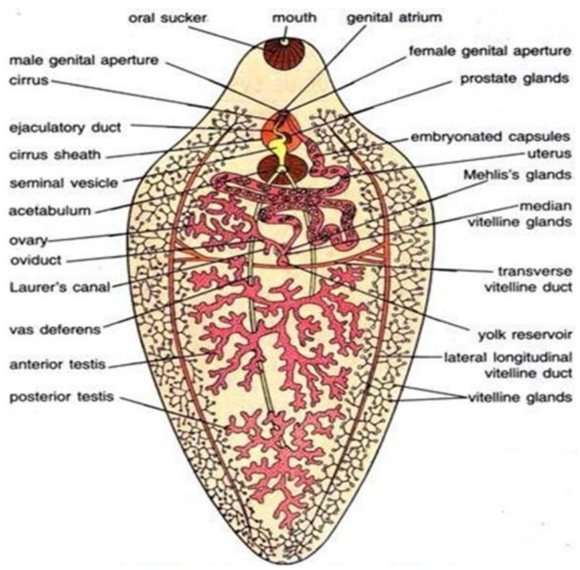

Figure 4: Shows *Fasciola* have a reproductive system, dorsoventrally flattened body, and suckers helped the parasites to overcome constraints in the liver habitat (Fox *et al.,* 2011)

6. Conclusion

Parasites are organisms that inhabit the host community getting benefits and causing damage. In the host, they are exposed to different constraints like host immune response, pH, life cycle stages, peristalsis, etc.

However, the parasites tend to overcome the constraints by the development of certain mechanisms like escaping mechanisms, taking advantage of the constraints, and possessing devices.

7. Recommendation

Further research should be conducted to study how parasites overcome constraints in their habitats as the knowledge may help in the prevention and control of parasites.

Conflict of Interest

The author declares that there is no conflict of interest.

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