Infectious Diseases and Our Planet

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1 Introduction

The bio-diversity of our planet is incredibly rich and complex in its nature, comprising a huge collection of bio-organisms. We use the term bio-organism to refer to any biological entity that possess some or all the properties of life. The definition of life (or being alive) in biology is mostly descriptive, however, by life, we shall mean a characteristic for any entity on our planet that would enable preservation of that entity and reinforce its existence. In this light we can consider a bio-organism as having life if such an organism has the attribute of being organized (in cells, unicellular or multicellular) and in addition, the ability to perform some or all of the following functions: homeostasis (the ability to regulate its internal environment to maintain a constant state), metabolism (being able to feed and so can transform energy by decomposing organic matter into cellular components and vice versa), growth (being able to maintain a higher rate of anabolism/catabolism and be able to increase in size in all of its parts, rather than simply accumulate matter), adaptation (having the ability to change over time in response to the environment on our planet), responsive to stimuli (ability to react, say via motion, when exposed to external chemicals or others probes), and reproduction (the ability to produce new individual organisms, either asexually from a single parent organism or sexually from two parent organisms). These complex processes, called physiological functions, have underlying physical and chemical bases, as well as signaling and control mechanisms that are essential to maintaining
life. A bio-entity that is capable of performing only one of the listed functions may not be classified as having life. We now discuss a few bio-entities under consideration here: A virus (giant molecule of nucleic acid coated with protein), for example, can replicate itself within a given environment, but because it neither metabolizes nor grows, it does not qualify as being alive. However, being a bio-entity, we would also consider virus in the discussion here. A fungus is another bio-entity that will be considered and could fall in either the living or non-living category, depending on the type. The last bio-entity to be considered is parasites, considered living.

Given the broad range of possibilities available to biological entities, we note that each bio-organism has its niche; the range of environmental conditions wherein each member of the given species can survive and reproduce. Survivorship and reproduction being key existential priorities for each of the bio-organisms that make up our planet’s biosphere. Since there are several bio-organisms sharing the same limited-sized biosphere, issues of co-existence come into play. We can think of the many organisms co-existing in the same biosphere as being in some kind of equilibrium whereby the domain of dependence, and/or range of influence, of the activities of each bio-organism, even if overlapping, engenders some stability in the population numbers of the particular organism that the biosphere can sustainably sustain. Thus, this kind of equilibrium can be thought of as a scenario whereby each living organism will live in tandem with its kind and with members of other species in a co-existence framework. However, in some instances, there is a flare up of the population numbers of one species breaking the equilibrium and causing undesirable effects. For example, a parasite can conveniently survive and grow in a host if the parasite’s numbers are within the host’s carrying capacity.\footnote{The environmental carrying capacity, for any given biological species, is the maximum population size of that species that the specific environment can sustainably sustain. It is the limiting population size imposed by the available life sustaining determinants such as food, water, space, etc., attributable to the particular biological niche or environment.} It is important to think of sustainability in the sense that the environment sustains the life of the organism, without itself being destroyed. In the case where the parasite numbers become larger than the host’s carrying capacity, the host then becomes sick (environmental degradation) and in the extreme situation dies because of the parasite burden. Incidentally, a dead host often goes down with the parasite, hence terminating the parasites’ life cycle, such as in the case of malaria. In order for the parasite to survive and maintain a continuous lineage, it must adapt to its hosts system and find ways to function within the hosts evading the hosts’ fighting mechanisms, such as its immune system, and live in tandem with the host, without killing the host, sometimes changing forms in the process. On the other hand, however, a virus that kills its hosts may terminate the transmission process in due time. However, even with the death of the host, the virus’ potential to transmit may not be diminished but be quite high, as, for example, in the case of the Ebola virus disease (EVD) [1, 9], which is the disease discussed in the chapter entitled: Modeling Ebola.
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Transmission Dynamics with Media Effects on Disease and Isolation Rates, by the authors Oduniyi, Gibbons, Oh, and Agusto, in [14]. In some cases, though an organism’s biological make-up may be suitable for the growth of another, that is, it can serve as a host for the second organism, the carrying capacity of the host may be such that it cannot support the presence of the second invading organism. In this situation, inclusion of any quantity of the second organism will serve as a disease to the host. Since invading parasites can kill their hosts, to ensure continued survival of the species, the parasites have to find a way of transferring themselves from one host to another in a process that we now understand as disease transmission.

2 Transmittable Infectious Disease Classification

Parasitic bio-entities that have attained the status of disease agents often possess mechanism that enables their transfer from host to host. The process of transfer from one host to another is known as disease spread, and a disease agent that has the ability to spread within a given host population is said to be an infectious disease agent. Infectious diseases may be classified into two broad categories: Directly transmitted and indirectly transmitted infectious diseases. Directly transmitted infectious diseases will not require a secondary medium to transmit the infection from one host to another host. In this case it is sufficient that the susceptible and infected hosts have a physical contact that is long enough for the infection to pass from the infected host to the uninfected one. In this category, we can mention human diseases such as HIV/AIDS, Ebola, etc., among directly transmitted infectious diseases of humans (see [1, 9], as well as the chapters entitled: Modeling Ebola Transmission Dynamics with Media Effects on Disease and Isolation Rates, by Oduniyi, Gibbons, Oh, and Agusto, citation [14] and Reducing the global HIV burden: The importance of uneven exposure to the results of HIV prevention trials, by Moore, Boily, Donnell, and Dimitrov, as discussed in [7]). Some examples of directly transmitted diseases of animals are Pseudorabies as discussed in the chapter [6], entitled Evidence for Multiple Transmission Routes for Pseudorabies in Wild Hogs, by Levy, Lenhart, Collins, and Stiver, and the fungal pathogen Batrachochytrium Salamandrivorans as discussed in the chapter [5], entitled, Identifying the dominant transmission pathway in a multi-stage infection model of the emerging fungal pathogen Batrachochytrium Salamandrivorans on the Eastern Newt, by Islam, Gray, and Peace.

On the other hand, indirectly transmitted infectious diseases will require a secondary medium over which the infection can pass to the next host. These, that is, indirectly transmitted infectious diseases, can again be divided into two broad categories: Those that require a living bio-entity to help transmit the infection from host to host and those that required non-biological entities to help transmit the infectious pathogen from host to host. This classification becomes fuzzy when the infectious disease can be picked up by another host because of a visitation to a location that has been previously contaminated by an infectious host by leaving
behind infectious diseases agent, and the very disease can be transmitted from host to host simply because the infected and susceptible individuals are sharing a common space at the same time. Examples may include Ebola virus disease (as discussed in [14]), cholera, typhoid fever, and the recent Sars-Cov-2 virus, among infectious diseases of humans, and as discussed in [5] and [6], *Batrachochytrium Salamandrivorans* and Pseudorabies, among infectious diseases of animals. The latter disease, *Batrachochytrium Salamandrivorans*, is discussed in the chapter, entitled: *Identifying the dominant transmission pathway in a multi-stage infection model of the emerging fungal pathogen Batrachochytrium Salamandrivorans on the Eastern Newt*, by Islam, Gray, and Peace. It is worth mentioning that, in the context of our discussions, some of the directly transmittable diseases are also indirectly transmittable, transmitted via non-biological entity because they share same physical space in which contacts can occur, such as the Ebola virus disease (EVD) or the *Batrachochytrium Salamandrivorans* fungal infection, to name a few.

We shall also be interested in those infectious diseases that cannot be transmitted from host to host by mere touch or sharing of the same physical space but must require a second biological host within which the pathogen must grow and develop before it is in a form that can be transmitted to the next human, animal, or plant. In this category, we have malaria, yellow fever, leishmaniasis, etc., among infectious diseases of humans, which will require a secondary bio-entity called the disease vector, to help transmit the infection from one host to the other. See the chapters: [16] entitled, *A Multistage Mosquito-Centered Mathematical Model for Malaria Dynamics that Captures Mosquito Gonotrophic Cycle Contributions to its Population Abundance and Malaria Transmission*, by Teboh-Ewungkem, Ngwa, and Fomboh-Nforba; [15] entitled, *Charles Darwin meets Ronald Ross—A population-genetic framework for the evolutionary dynamics of malaria*, by Schneider; [12], entitled *Dynamic Regulation of T cell activation by coupled feedforward loops*, by Buri, Zelleke, and Ndifon. We also have the cassava mosaic virus diseases as plant diseases, as discussed in the chapter [3], entitled *Application of Mathematical Epidemiology to Crop Vector-Borne Diseases. The Cassava Mosaic Virus Disease Case*, by Chapwanya and Dumont. Because this form of transmission requires the interaction of at least two living organisms, say humans and mosquitoes, and development of the pathogen within these interacting living organisms, we will devote Sect. 4 to discuss further on vector-borne infections.

The pattern for disease transmission can be influenced by the spatial location/state of the organism to be infected. So the pattern would be different depending on whether we have a spatially distributed collection of host to be infected or we have a spatially distributed collection of hosts and diseases vectors to be infected and whether or not the infectious agent is spreading through the distributed hosts. Thus there are several categories of host–pathogen–disease vector mediated interactive mechanisms: In the one category, the host may be stationary over large time scales and the vector distributes the infectious disease agent from host to host as in most plants diseases, as discussed in the chapter [3]. In another category, both the host
and the vector are mobile and the pathogen is transferred from host to host by the vector as it follows the host from spatial location to spatial location, as is the case with human diseases such as malaria. In yet another instance, it is the infected host that carries the disease agent from one spatial location to other hosts in other spatial locations as could be the case with infectious diseases of humans such as cholera, HIV/AIDS, and Ebola virus disease to name but a few. Some of these classes of disease shall be examined in the chapters that lay ahead cited as [2, 3, 5–7, 12, 14–16]. As shall be seen, each type of transmission would require a specified type of modeling assumptions to get the transmission dynamics approximately correct.

Irrespective of the mode of transmission of infectious disease agents from one host to the other, it is important to note that each living organism has a natural defense mechanism that is hard-wired into its genetic make-up that allows it to fight invasion by parasites or death through exposure to toxins. These natural defense mechanism for single cellular organisms, including most parasites, is achieved via genetic mutations. Mutations, when they occur can be advantageous or disadvantageous to the particular organism whose genetic make-up has mutated. Mutations are advantageous to the organism if such a mutation renders the bio-entity fitter and better to survive in the environment. This is probably the situation that has been observed for most pathogens that develop mutations that make them resistant to certain chemical substances, such as medicines that are usually useful for the elimination of the parasite within the host system. An example is the development of drug resistance by the malaria parasite as well as the development of resistance of insecticides by some mosquito species. This phenomenon of development of resistance through mutations is an important evolutionary survival pathway and therefore constitutes aspects to be considered when studying human diseases and their potential control mechanism through use of drugs. Thus understanding genetic mechanism, as discussed in the chapter [15], that can serve as plausible pathways towards the onset or development of drug resistance by parasites is an important idea in infectious disease modeling. Mutations that are detrimental to the particular living organism render such mutated organism less competitive in their biological niche, and in most extreme cases the mutated variants of the organism do not survive. This is Darwinism whereby the concept of survival of the fittest gets manifested through genetic mutations. We shall also consider at least one such model (see [15]) where Darwinism is considered towards a better understanding of the behavior of the malaria parasite.

\[2\] A mutation is a change that occurs in the DNA sequence of a living organism, either due to mistakes when the DNA is copied or as the result of environmental factors. In general, a mutation is recognizable as having occurred if there is an alteration of the nucleotide sequence in the genome of an organism, virus, or extrachromosomal DNA.
3 Defense Mechanisms Against Infection

Complex and higher order organisms will require more sophisticated defense mechanisms to fight pathogens that can invade them, be it viruses, bacteria, or parasites, for example. This type of defense mechanism is triggered when the cells in the organism have the capacity to detect foreign objects in its system and/or activate or trigger mechanisms to produce other types of cells that can mitigate the invasion of a foreign object. The production of system cleansing cells (immune cells) that target invading pathogen cells in order to destroy them, falls broadly under the category of immune response. A properly functioning immune response system must have the ability to kick into action once a foreign organism invades the system (activation), the ability to produce killer cells that would target and kill the invading organism (active immunization), and the ability to switch off and go quiescent when the invading substance has been removed (deactivation). The understanding of the proper mechanisms for the functioning of the immune system in humans is still evolving. However, several hypotheses have been proposed for the better understanding of the action of the immune system. One plausible mathematical characterization would be to consider that the activation of the immune system happens in a two-step process: production of activator precursors followed by production of effector cells that then trigger the active immunization response. We shall consider some models that have this characteristic as well as in [2].

In some cases, the degree of infestation is so severe that the human immune system is overwhelmed such that in the absence of external assistance, be it pharmaceutically-related or just supportive management of the infected system(s), death is expected. For example, in malaria parasite infestation, if the parasite load in the human host is at manageable levels, the human’s immune system (innate and adaptive) will effectively control the infection [10], however, in the case of high parasitemia, massive destruction of the red blood cells by the parasite would mean that if external interventions are not employed, the human would die of anemia. When it has been diagnosed that a malaria infection is present, anti-malarial drugs are normally prescribed to help reduce the parasite load in the patient to levels where the natural immune system can continue to offer protection to the human. For some viruses, for example, the Ebola virus disease, help is in the form of supportive care to help alleviate symptoms while allowing the body’s natural mechanisms to fight. These supportive care, which may involve increased intake of electrolytes, can help boost the immune system, giving the affected system an opportunity to recover. Other supportive mechanisms involve oxygen therapy, treatment/management of secondary disease-related infections.
4 Vector-Borne Diseases: Vectors, Pathogen, and Transmission

Any living organism that facilitates the transfer of a pathogen from one living host to another living host (perhaps of the same species) will constitute a disease vector. By disease transmission we shall mean an effective transfer of the disease pathogen from one host to another. The disease pathogen is the infectious agent, usually another living organism, that when present within the living tissue of another host can cause sickness and possibly leading to the death of the infected host.

4.1 The Disease Vector

A living organism can serve as a possible (efficient or effective) disease vector if it has a complete biological and genetic make-up that requires that, from time to time, the organism will seek contact with the particular host (perhaps humans). This contact can be for several biological reasons including the need to derive determinants that it will require for growth and development. For example, the Anopheles sp. mosquito’s biological make-up requires that the female interacts with vertebrate host to draw blood which she needs for the maturation of her eggs. The interaction between the disease vector and the host may therefore be driven by a physiological and biological need for growth and development. Though such interaction can also be to seek shelter, it can be safely assumed that where the interaction is successful, the disease vector’s chances of survival as a species are improved as it may live to reproduce. A step towards the study of indirectly transmitted diseases could be to seek to understand the disease vectors themselves (as in [8, 11, 13], for example), and as such we may seek to understand and quantify the developmental and reproductive gains that would accrue to the vector after a successful interaction with the particular host. For example, a female Anopheles sp. mosquito that successfully acquires a blood meal from a vertebrate host (such as a human or animal) and also survives the resting period that she needs after this blood meal, will lay a batch of eggs that will hatch and eventually mature into more adult mosquitoes in the future. The reproductive gain in foraging for blood meals, against when weighed with risk of being killed by predators, is the successful maturation and subsequent laying of eggs. It is true that in the absence of disease, bio-organisms that have the appropriate biological and genetic make-up to feed on other organism will always seek to interact with those organisms. Those that require plants as a place to develop their next generation will also continue to interact with that plant.

Another step towards the study of indirectly transmitted diseases could be to seek to understand disease transmission process and possibly quantify the role of the vector in transmission and seek ways to prevent their success. The chapters by Teboh-Ewungkem et al. [16], Chapwanya and Dumont [3] and [12], address these. While [16] focuses on the malaria transmitting vectors’ interaction with humans,
Table 1  Examples of some disease vectors and the diseases they carry. Note that other interacting populations may exist, even though not mentioned

| Disease vectors | Species                        | Diseases transmitted       | Pathogen transmitted          | Example Interacting population |
|-----------------|--------------------------------|----------------------------|-------------------------------|--------------------------------|
| Mosquito        | Anopheles sp.                  | Malaria                    | Plasmodium sp.                | Humans and animals             |
|                 | *Aedes aegypti*                | Dengue                     | Dengue virus                  | Humans                         |
|                 | *Aedes aegypti*                | Zika                       | Zika virus                    | Humans                         |
|                 | *Aedes sp.* & *Mansonia sp.*   | Lymphatic filariasis (elephantiasis) | Parasitic filarial worms (Wuchereria bancrofti) | Humans                         |
| Tse-Tse fly     | *Glossina sp.*                 | Sleeping sickness          | Trypanosoma brucei            | Humans                         |
| Cockroach       | *Periplaneta sp.*              | Bacteria and viruses       | Salmonella, Polio virus       | Humans                         |
| Sand fly        | *Phlebotominae sp.*            | Leishmaniasis              | Leishmania parasites          | Humans                         |
| White fly       | *Bemisia tabaci*               | Cassava mosaic disease     | Cassava mosaic virus          | Cassava plants                 |

[3] looks at that of the white-flies, their interaction and disease transmission with the cassava plants. Nipa and Allen in [12] study a general vector–host interaction model. Table 1 summarizes some disease transmitting vectors and the diseases they can transmit.

4.2 The Disease Transmission

Disease transmission for indirectly transmitted infections occurs when the pathogen is passed from the host to the disease vector and again when it is passed from the disease vector to the host. So, for these kinds of diseases, infection occurs twice: In the first instance, the vector gets infected by the human host and the disease then has time to mature within the vector and in the second case a second human is infected by the vector. For such an infection to occur, there must be a successful interaction between the disease vector and the host, as well as a successful transfer of the pathogen from host to vector or from vector to host. Such double infections would have to occur over distinct points in time that are separated by the length of time equivalent to the maturation period of the infection in both organisms. In this regard, one may regard the pathogen as an opportunist that only exploits the life style of the disease vector, and see the vector as the active conduit that helps transfer the infection from one human to another. The second step then, towards a proper understanding of indirectly transmitted diseases of humans would then
be to seek to understand the indices of transmissibility; that is the quantifiable measurements of quantities that can be used to ascertain whether or not the disease will spread between and within the different populations upon introduction of at least one index case and factors that may facilitate this process, be it environmental and/or evolutionary, as in the chapters [3, 12] and [16].

4.3 The Disease Pathogen

The disease pathogens in some instances are living organisms while others are not, and, normally could take advantage of the life style of the disease vector and then could divide its life cycle and developmental pathway so that part of it is in the host and the other part is in the disease vector. This type of division of life cycle of the organism can, from an evolutionary standpoint, be beneficial to the pathogen since at each time it has part of its progeny in two different biological entities, thereby making extinction difficult. For example, the pathogen that causes the disease malaria in humans has divided its life cycle such that one part of the life cycle is in the human and the other part in the mosquito. One major characteristic of a disease pathogen that has divided its life cycle to be able to survive in different organisms is that only certain forms of the pathogen can grow in the different hosts. So there is a need to develop in essentially different ways, so as to produce the forms of the organism that can be transferred from host to the disease vector and vice versa. For example, the form of the malaria parasite that can be transmitted from humans to mosquitoes and begin development within the mosquito are called gametocytes and they are produced in the human. On the other hand, the form that can be transmitted to humans by mosquitoes and begin development in the human are called sporozoites and these are produced in the mosquito. Each of the hosts, humans and vectors, offers a different biological niche to the disease pathogen and the different parasite forms within these hosts introduce delays in the development and maturation times of the infection both in the human and in the vector. A second step, therefore, in the study of indirectly transmitted diseases would be to seek to understand how the disease pathogen weaves its own life style into the life style of its vector. In this case we may wish to understand how the disease agent affects the behavior of both the disease vector and the host as well as factors that allow them to survive and thrive, as in the chapters [2] and [15].

5 Discussion and Conclusion

The manuscripts published, herein, address different aspects of the diseases discussed. The results highlight the intricate interconnection between our planet and infectious diseases. Mother nature plays a role as it can drive seasonal fluctuations of diseases pathogens, as well as human factors. In [12], Nipa et al. used a stochastic vector–host indirectly transmissible framework to show how seasonal patterns and
variability in the demography of the vectors and host affect disease outbreaks for a
general vector-borne disease.

With malaria as a specific example, Teboh-Ewungkem et al. in [16] highlighted
one aspect of indirectly transmitted diseases, which has often been neglected in
mathematical models. They highlighted the fact that for an indirectly transmitted
infectious disease to spread within human populations each disease vector must
interact with two different humans at two different time periods in the following
way: In the first instance, the vector interacts with human $A$ and may pick up the
infection. Then the vector, if it successfully picked the infection, survives through
the incubation period and becomes infectious and then interacts with human $B \neq A$,
it can then pass on the infection to the human $B$. So, since it is the vector that
actively seeks the humans, the infected vector must interact with at least two
different humans at two different points in time (the length of this time difference
will be compared with the length of the incubation period in the vector to infer
infectiousness of the vector) to propagate the infection into the human population.
On the other hand, many vectors can pick up the infection from one human
simultaneously, which is an important aspect to consider when writing down models
for indirectly transmitted diseases. The authors noted that any realistic mathematical
model should take this aspect into consideration, as well as the fact that transmission
of the infection is contingent on effective interactive contact. Thus, the reasons
why disease vectors of humans are efficient vectors for human diseases are partly
because the need to interact with humans is hard-wired into the physiological make
of the disease vector as the interaction is tied to the survivability of the vectors next
generation. For example, the mosquito, the vector that transmits malaria interacts
with humans because the female adult needs to harvest blood from humans that she
needs for the maturation of her eggs. Understanding and capturing these aspects as
malaria continues to affect millions and kill many, especially children.

Instead of the human malaria disease, Chapwanya and Dumont [3] investigated
the cassava mosaic virus disease that is also transmitted by a vector, in this case
the white fly. The disease affects the cassava plant, a plant that forms an integral
perennial crop serving as an important staple food for millions in the African
continent on a regular basis and as food security against famine, since its roots
can last a long time in the ground. Thus, understanding the interactions between the
vectors that transmit the disease, the disease causing virus and the cassava plants are
essential to the health and survival status of a significant population on our planet,
those that rely on the cassava plant for nutrition. Thus controlling the disease and
minimizing infection transmission is important.

Diseases with multiple transmission pathways can introduce challenges espe-
cially with control. Thus questions about which pathway might be dominant are
important because they will impact how control measures are applied. Moreover,
how these transmission pathways are adapted to the environment, climate, and
our planet is important because a non-dominant pathway may potentially still
serve as a source allowing for the potentials of increased epidemic frequencies
and posing a challenge to disease control. In the chapter [6], where Levy et al.
studied the viral disease, Pseudorabies, in Wild Hog populations in the Smokey
Mountains, they illustrated that the disease has multiple transmission routes: In particular, they highlighted four transmission routes, namely: direct route though density dependent contacts between wild hogs, or in the process of mating or during nursing, from mothers to piglets, and the fourth being as a result of stress, with carriers becoming re-infected. Notice that the first three routes are tied to the life style and evolutionary need for survival and protection of the next generation. While the fourth route, linked to stress factors, can be attributed to both natural causing and man-made factors since stress could be generated as a result of many factors including overcrowding, raised water levels, food shortage, etc. Regardless of the transmission route, pseudorabies in wild hogs impacts other domestic and wild animals in the regions inhabited by the diseased animals and has implications to our planet. Hence finding ways to curtail the spread of this disease is important for the ecosystems that interact with these wild hog populations.

In order to find effective and sustainable control measures for diseases with multiple transmission pathways, we must first understand the pathways and understand what factors are the greatest propagators of the diseases for specific pathways or how these pathways may be intricately tied to the demography of the populations affected. Islam et al., in [5], examined the transmission dynamics *Batrachochytrium salamandrivorans* (Bsal), an emerging fungal pathogen that affects the North American salamander population. As noted in [4], salamanders play a significant role in our ecosystem as they perform various ecological functions and provide ecosystem services that benefit the human race. Noted examples include: their service as pest controllers as they feed on mosquitoes; their service as nutrition for other larger animals, hence maintaining a balance in the ecosystem; their service as health indicators of the ecosystem due to their vulnerability and susceptibility to environmental toxic substances and drought; their role in carbon cycling and hence link to climate change, and their service as pets for humans, to name a few. Thus, maintaining a healthy salamander population is tied to the health of our planet. Thus models to investigate how to prevent and control any potential *Batrachochytrium salamandrivorans* (Bsal) infection is important. In this light, the authors Islam et al., in [5], investigated the invasion potential of the Bsal fungal infection in a population of Eastern Newts and showed that population density was a factor in the form of transmission that was dominant. In particular, for small population densities, they showed that the dominant transmission pathway was the direct host-to-host contact transmissions, meanwhile for larger population densities, environmental transmission was the dominant transmission form. Hence, the form of control should be tied to population density information in order to achieve a greater success of inhibiting invasion.

Control of the diseases that affect the humans and animals in our planets requires an understanding of all aspects tied to the disease. For malaria, for example, one has to view the disease as a three component problem: the parasite component, the human component, and the vector component, all interacting. The chapter in [16] captures all three components, with the vector as diseases drivers and the human serving as components infected by the malaria parasite. The chapters in [2] and [15] focus on the parasite component in the human. When a parasite infects a human,
it transforms and change forms with the end goal being survival in the human, leading to the production of the transmissible forms of the parasites, the mature gametocytes. However, during such an infection the body establishes a defense mechanism, with T cells being part of the defense mechanism cells. For malaria in particular, two types of immune response are reported—innate and adaptive, developed due to repeated exposure to malaria. In [2], Buri et al. studied the principles involved in the activation of T cells after a pathogen invasion. Their focus was on the adaptive immune system T cells and its activation, a process that requires that at least two signals be received before the T cells can be activated.

Within human host malaria parasites can respond positively to effective pharmaceutical control measures, if there is no drug resistance to the parasite. This is a primary method of disease control in a malaria-infected and sick patient. If, however, the parasite form is resistant to the administered drugs, then there is a problem and disease control and or eradication is not achieved. Schneider, in the chapter [15], investigated the dynamics of resistance—conferring mutations and their resulting impacts on genetic mutation. There, the author shows that the multiplicity of infection (MOI), defined as the number of super infections during the course of an infection, mediates the interplay between selection and recombination.

For diseases such as HIV/AIDS and Ebola, human factors are an integral aspects of control, especially with no actual cure (even though for HIV, there are medications that can decrease transmissibility). In [7], the authors highlighted that with the goal to reduce the global HIV burden, uneven exposure between trial participants in randomized controlled trials much be considered as they can affect the effectiveness of the trial. In particular, their results showed that effectiveness decreased with HIV exposure rate and trial length. In [14], Odunyi et al. studied media effects on an Ebola model that incorporates isolated and non-isolated individuals, as well as sexually infectious individuals. They showed that increased media effect is correlated with lower Ebola disease epidemic peak and this peaks lags behind the epidemic peak without media coverage.

In all, several factors contribute to the transmission of diseases within our planet. Some aspects are enhanced by human factors like deforestation, etc., while others by natural factors such as the climatic aspects of the regions affected by the specific disease. Overall, understanding how to control these diseases, as well as the actions we must take, are vital and require an understanding of the underlying process.

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