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Manipulation of Arthropod Vector by Pathogens

Tigist Ashagrie and Fekadu Massebo

National Animal Health Diagnostic and Investigation Center,
Ministry of Agriculture, P.O. Box: 04, Sebeta, Ethiopia

Abstract: The objective of this review was to discuss concept of vector manipulation by pathogens, arthropod-pathogen interaction and its impact on vector blood feeding, vector longevity and vector reproductive capacity. A vector-borne parasite manipulates phenotypic traits of their vectors and hosts in ways that increase contacts between them and hence increasing transmission of parasite to a new host. Any changes in vector physiology or behavior could thus take place at a local tissue level or at a distance from the infection site and at any time during the infection. The strategies might be increasing the amount of vector – host contact epically at the infective stage, reducing vector reproductive output and consequently altering vector resource management to increase available nutrient reserves and increasing vector longevity. However, the mechanisms by which the parasite manipulate the vector is very complex and need further molecular and field studies as most studies come from the laboratories.

Key words: Phenotypic · Physiology · Behavior

INTRODUCTION

Arthropods are important vectors of the most devastating human diseases such as malaria, leishmaniasis, trypanosomiasis, dengue, plague, yellow fever and others. In most cases vectors are not born with parasites they transmit, but rather they get infected while feeding on blood. Arthropods must blood feed twice transmit pathogen, firs on an infectious host then again on a susceptible host. This necessity for two blood meal to fulfill the parasite's life cycle makes blood – feeding a major component to vector-borne disease transmission [1]. Most pathogens vectored by arthropods, must undergo an extrinsic incubation period (EIP) in the vector, before they can be transmitted to a new host. During this time the pathogens penetrate the vectors mid gut, replicate in various host tissues and infect the salivary glands prior to transmission during subsequent blood feeding [2].

The duration of the EIP is variable and is heavily influenced by ambient temperature. For many pathogens the duration of the EIP consumes a significant proportion of the vector's lifespan. As a consequence, only a small fraction of the vector population that is oldest is of epidemiological importance [3]. Many of the arthropod-borne parasites undergo either development, reproduction or both development and reproduction inside the insect vectors [4]. During this stage of their life cycles, selection pressures favor parasites that can manipulate their vectors to enhance transmission [5]. Strategies may include increasing the amount of contact between vector and host, reducing vector reproductive output and consequently altering vector resource management to increase available nutrient reserves and increasing vector longevity. Manipulation of these life history traits may be more beneficial at some phase of the parasite's developmental process than at others [6].

Concepts of Vector Manipulation by Pathogen: A vector-borne parasite manipulates phenotypic traits of their vectors and hosts in ways that increase contacts between them and hence increasing transmission of parasite to a new host [7]. Interactions between parasites and their vector insects can occur at various points in their development, starting with the arrival of an infected blood meal in the insect mid-gut [5]. For instance, *Trypanosoma cruzi* spends all vector phases of their life cycle in the gut; others penetrate into the hemocoel and

Corresponding Author: Tigist Ashagrie, National Animal Health Diagnostic and Investigation Center, Ministry of Agriculture, P.O. Box: 04, Sebeta, Ethiopia. Tel: +251910207064, E-mail: martiashagrie@gmail.com.

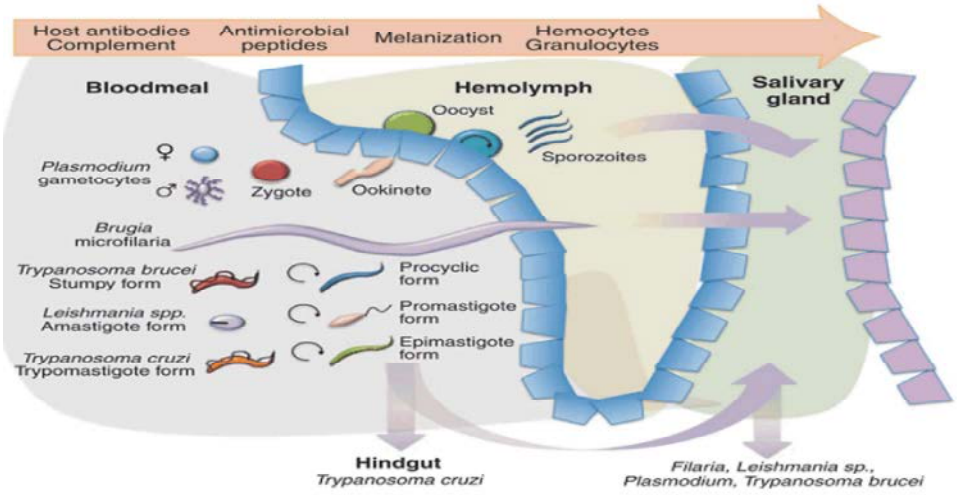

Fig. 1: Routes of transmission of different parasite groups through an arthropod vector [5]

invade other tissues early in the infection, such as *Onchocerca volvulus*, or later, such as *Plasmodium* species [5]. Any changes in vector physiology or behavior could thus take place at a local tissue level or at a distance from the infection site and at any time during the infection. Points of contact between vector and parasites begin with ingestion of the infective blood meal. Once infected, the immune system may be manipulated; circulating metabolite titers changed, vector reproductive success and longevity altered and the process of further blood feeding affected [8]. Behavioral manipulation is adaptive and regarded as a forced collaboration. First, it is complex-that manipulation relies on the successful establishment and development of the parasite within the vector and this requires many intrinsic barriers to be overcome. Second, it shows evidence of ‘‘purposive design’’-the behavior is exhibited when the parasite is ready for transmission. Third, the production of secretions that appears to have arisen independently in other parasite vector combinations. Fourth and most important of manipulation are directly increased the fitness of the parasite through enhanced transmission. Parasites may select for collaborative behavior in their hosts by imposing extra fitness costs in the absence of compliance [9].

Pathogen Arthropod Interaction: Vector-borne parasites can be very selective in the host species in which they complete development (Figure 1). For example, human malaria can be transmitted only by mosquitoes of the genus *Anopheles*. But there are anopheline mosquito species that serve very poorly or not at all in the *Plasmodium* life cycle [10]. This may be due to many biologic reasons varying from the time the mosquito peritrophic matrix is formed to the mosquito’s ability to mount a melanotic reaction to the developing oocysts and to the sporozoite’s ability to invade the mosquito salivary gland and survive the mosquito’s hemolymph defense system. Other mosquitoes can develop heavy sporozoite infections of the salivary glands, even when feeding on lightly parasitemic hosts [5].

Parasite Development and Blood Feeding: Blood feeding is vital for the vector as well as for the parasite transmission [11]. This is because, in most cases, hematophagous female insects require the amino acids acquired from erythrocyte and plasma protein digestion to synthesize yolk proteins for egg production. Hematophagous arthropods saliva contains many pharmacologically active components such as anticoagulants, anti-platelet aggregation, vasodilators and immunomodulators that assist them to obtain a blood meal by neutralizing the vertebrate’s haemostatic and inflammatory system to their advantage [12]. Several studies suggest that parasites alter the feeding behavior of vectors in way that enhance completion life cycle. The host vector contact decrease at the early stage of the parasite development to reduce the risk of mortality because of host defensive behavior [13]. When the parasites reached the infective stage (invade the salivary glands of the vectors), the rate of feeding on host increases and hence, increases chance of the parasite transmission [14].

Parasite Development and Vector Longevity: Vector age is a critical determinant of the ability of most arthropod vectors to transmit a range of human pathogens. This is due to the fact that most pathogens require a period of extrinsic incubation in the arthropod host before pathogen transmission can occur. This developmental period for the pathogen often comprises a significant proportion of the expected lifespan of the vector. As such, only a small proportion of the population that is oldest contributes to pathogen transmission, which a vector is less successful in feeding during the parasite development time, which in turn affects the vectors fitness [6]. However, strategies may include increasing the amount of contact between vector and host (during sporozoite stage) [15], reducing vector reproductive output and consequently altering vector resource management to increase available nutrient reserves and increasing vector longevity. However, the frequent blood feeding behavior might increase host related mortality [16].

Parasite Development and Vector Reproductive Capacity: Blood feeding is vital for the vector as well as the parasite. Egg production is influenced by a variety of factors in hematophagous insects, many of them being interlinked, but with blood meal quantity and quality playing major roles [7]. In many insects the normal process of oogenesis is disrupted by parasites of various taxa; resulting in loss of reproductive fitness [17].

Implication of Parasite Manipulation for Disease Transmission: The majority of vector-borne diseases undergo a period of growth, development and sometimes reproduction within their vector. As a consequence the majority of infected vectors die before they take a blood meal that transmits infective parasites [6]. Parasite fitness is thus intrinsically linked to vector fitness. Growing number of studies demonstrate that vector-borne parasites manipulate phenotypic traits of their vectors and hosts in ways that increase contacts between them and hence favor the parasites' transmission [18]. For instance, the feeding behavior of sand flies infected with *Leishmania* was modified in way that parasite transmission appeared to be enhanced [19]. Gel-like plug composed of filamentous proteophosphoglycan (fPPG), a very high molecular mass glycoprotein unique to Leishmania [20], which causes physical blockage of the gut that ensures regurgitation of infective forms [21, 22, 23]; subsequent exacerbation of infection in the mammalian host through the action of fPPG and vector saliva [21 24]; and now the manipulation of feeding behavior according to the presence of infective forms available for transmission. fPPG is a potent virulence factor for Leishmania that benefits both the transmission of parasite from the sand fly and infection of the mammalian host [21].

CONCLUSION AND RECOMMENDATION

Points of contact between vector and parasites begin with ingestion of the infective blood meal. Once infected, the immune system may be manipulated, circulating metabolite titers changed, vector reproductive success and longevity altered and the process of further blood feeding affected. Strategies may include increasing the amount of contact between vector and host (at the infective stage), reducing vector reproductive output and consequently altering vector resource management to increase available nutrient reserves and increasing vector longevity. However, the mechanisms by which the parasite manipulate the vector is very complex and need further molecular and field studies as most studies come from the laboratories.

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