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## RESEARCH ARTICLE

# MULTIFACTORIAL ANEMIA ASSOCIATED WITH LEECH TRANSMITTED TRYPANOSOMES IN WILD FISHES: A COMPARATIVE REVIEW

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**ABSTRACT**

Trypanosomes are flagellated protozoan, classified under the family *Trypanosomatidae* and known to cause serious disease conditions in all vertebrate animals under terrestrial and aquatic environments. The ubiquitous nature of the piscine parasite, warrants several vectors to accomplish their digenic lifecycle, involving arthropods and annelids. Sleeping sickness*, Nagana, Chagas disease, Surra, Dourine, Mal de Caderas,* Avian and Piscine trypanosomosis are associated with man, cattle, camels, horses, birds and fishes respectively, with tsetse flies, mosquitoes and leeches identified as respective vectors. Anemia, nervous disorders, abortion and loss of condition are principal pathologic manifestations. Hemolytic anemia is common to all species, due to accelerated erythrocyte destruction. However, in fish, other salient factors predispose and complicate the anemia observed. Anemia from heavy leech blood meal, and severe bleeding at the site of bite constituted multifactorial nature in fish. Leeches are equipped with specialized organs and biologically active substances for attachment, piercing and sucking blood from the host with mild pain sensation. These factors are considered and compared with trypanosomosis in other animals.



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## INTRODUCTION

Several possible etiological factors are associated with anemia in fish, notably accelerated Red blood cell (RBC) destruction, delayed erythropoeisis or severe loss of blood as seen from external and internal parasitic infestations (Witeska, 2015). Anemia remains an important clinical manifestation in most trypanosomal infections, though its severity differs from one host to another. While transmitting infectious agents, excessive blood loss is encountered either through voracious feeding by the vectors, accelerated hemolysis, excessive hemorrhage to exterior or combination of these factors, as observed with leech bite (Letch, 1977; Muhammad, 2014). Humans, domestic and wild animals, birds and aquatic mammals suffer from a devastating disease upon infection with the deadly protozoan parasite (trypanosome),that has worldwide distribution and is indeed of serious public health significance in Africa and Latin America.

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The disease is transmitted cyclically by tsetse flies, causing sleeping sickness in man and *Nagana* in domestic animals (Uilenburg, 1998; FAO, 2012). Avian and piscine trypanosomes are similarly transmitted through the bite of mosquitoes and leeches respectively (David and Nair, 1955; Simon and Woo, 1991; Neelima, 2006; Jan *et al.,* 2012; Polly *et al*., 2014). Mechanical transmission of the parasite in man (South America), is accomplished by triatomine bugs (*Trypanosoma cruzi)* and several hematophagus flies responsible for trypanosomosis among animal species in Africa, Middle East and Asia. Sexual transmission of the disease (*Dourine)* is known to occur in horses. Over the years concerted efforts in research, surveillance, and control of the disease in man and animals were made through combined efforts of several international organizations: World Health Organization (WHO), Office Internationale des Epizooties (OIE), Food and Agricultural Organization (FAO), International Atomic Energy Agency (IAEA), Inter-African Bureau for Animal Resources of the Africa Union (AU-IBAR), Programme Against Tsetse and Trypanosomosis (PAAT), Nigeria Institute for Tsetse and Trypanosomosis Research

(NITR), Non-governmental organizations (NGOs), regional governments and other research organizations within the affected areas (FAO, 2012), with overwhelming results. The disease was targeted for eradication by the year 2020 (WHO, 2012), as vividly seen from drastic reduction of reported cases of Human African Trypanosomosis (HAT) to 3,796, which is regarded as the lowest record in the last 75years (PAAT, 2015). Little or no effort is made towards addressing the condition in fish (Muhammad, 2014). Several factors are known to predispose fish to anemia under wild environment, notably internal and external parasites, infectious diseases, high concentration of heavy metals, agro-based chemicals, toxic and industrial byproducts in sewage and run off from urban areas (Witeska, 2015). Identifying a specific etiological agent may pose a challenge to fish pathologists, as observed from extremely low hematocrit values recorded in some wild fishes that were apparently healthy (Muhammad *et al.,* 2017). Voracious and repeated feeding by leeches could jeopardize the survival of many fish species, with subsequent hypovolemic shock, while the direct mechanical action of trypanosomes on the RBC could be responsible for hemolytic anemia (Brian, 1989). Another important factor is the loss of blood from the point of attachment of the leech parasite. Thus, anemia observed in trypanosome infected fish was suggested to have multidimensional and multifaceted causation (Esuruoso, 1993, personal communication), that requires epidemiological approach for better elucidation. This forms the basis of this review of anemia in trypanosome infected fish, in comparison with trypanosome- infected humans, cattle, horses and birds.

**Human Trypanosomosis**

The highly fatal sleeping sickness is confined to Africa, and South America. Tsetse flies of the genus *Glossina* serve as vectors of the disease, notably *G. submorsitans, G. longipalpis and G. tachynoides*. *Trypanosoma brucei gambiense* (*T. b. gambiense), Trypanosoma brucei rhodensiense (T. b. rhodensiense) and T. brucei* are responsible for the disease, with *T. gambiense* accounting for 95% of the reported cases annually in Africa. Less than 10,000 cases were reported from 2009 (PAAT, 2012). The disease progresses in 2 forms-hemolymphatic phase, which is characterized by fever, anemia, lymphadenophathy, infertility and neurologic stage (Prashant, 2013), in which severe nervous disorders (somnolence and insomnia) are seen, hence the name sleeping sickness (FAO, 2012). In South America, the disease is transmitted by bugs and referred to as *Chaga*s disease which occurs in twenty one Latin American States (FAO, 2012).

Though neurological signs are of major concern in human trypanosomosis, commonly seen in chronic stages of the hemolymphatic stage of sleeping sickness, anemia was observed among other clinical signs, possibly due to mechanical action on the blood cells prior to subsequent nervous invasion (Prashant, 2013). While conducting survey with hospital patients admitted with trypanosomosis, Chisi *et al*. (2004) observed jaundice, hepatosplenomegaly in some, and hypochromic anemia, in 85 % of the cases. According to Karle (1974), anemia could accompany febrile state of the patient due to exposure of RBC to high body temperatures, with resultant increase in osmotic fragility and permeability, or decrease in its plasticity and longitivity as reported (Woodruff *et al.*, 1972). Truc *et al.* (2013) reported incidental cases of animal trypanosomosis in man (*T. evansi, T. vivax, T. congolense* and *T. lewisi*), while Joshi *et al.(*2005) and Prashant (2013) reported an un usual human case, whose etiological agent was verified through clinical manifestation, parasite morphology and molecular studies, and confirmed to be *T. evansi.*.

**Trypanosomosis in Cattle**

The disease complex is referred to as *Nagana*, as coined by the Zulu tribe in Africa and *Samore* among Fulani pastoralists in Nigeria. *Glossina* species are responsible for cyclic transmission of *Nagana* in domestic animals within the tsetse fly belt of Africa, while mechanical transmission is also accomplished by *Tabanid, Muscid and Hippoboscid* flies, as well as by vampire bats (Clair, 2017). Farmers suffer serious economic losses due to decreased milk production, wasting condition, abortion and death in cattle, from 37 African countries. Important pathogenic species are *T. congolense,* and *T. vivax,* while less pathogenic *T. uniforme, T. simiae* and *T. brucei brucei* are also on record (OIE, 2013). Predominant clinical signs of the disease in most of these species include mild fever, anemia, emaciation, abortion and death, while neurological disorders are often recorded in horses (Uilenberg, 1998). The pale mucosa primarily observed in cattle and several animal species due to *T. congolense* or *T. vivax* infection are often considered among key indicators of anemia and important tool for differential diagnosis. Anemia due to *T. congolense* was considered more severe than the combined anemia seen in all other blood protozoan and gastrointestinal helminths affecting Zebu cattle in Nigeria (Jenkins and Facer, 1985).

**Trypanosomosis in Horses**

Trypanosomosis in horses is caused by *T. evansi* as the predominant etiological agent. It is mechanically transmitted by Tabanus, Stomoxys and vampire bats. *T. equiperdum* is another pathogen sexually transmitted by infected animals during coitus (Uilenberg, 1998; FAO, 2012). The disease originated in camels from North Africa, and later spread to Middle East, Asia and Latin America; hence it has the widest geographical distribution and host range among animal trypanosomoses. It has different vernacular names as per the regional occurrence, popularly known as *Surra* in Africa, Midlle East and Far East, *Murrina* in Central America, *El Debab* in Algeria, *Mal de Caderas* in Argentina, *Mbori* in Sudan, *Peste -Boba* in Venezuela, *Guifar* in Chad and *Mechaec* in Niger (Desquesnes *et al.,* 2013), with characteristic signs of anemia, abortion, wasting, hind limb paralysis and death. Pale mucous membranes and icterus with microcytic normochromic anemia were also described in horses (Roberto *et al*., 1995). According to Levine (1973), anemia, intermittent fever, alopecia, urticaria, oedema and progressive weight loss were common findings. On rare occasions, medicinal leech was incriminated in the transmission of *Mal de Caderas* in South America (Van-Bassewitz, 1929), where relapsing fever, emaciation and complete paralysis of hind limbs were observed.

**Trypanosomosis in Birds**

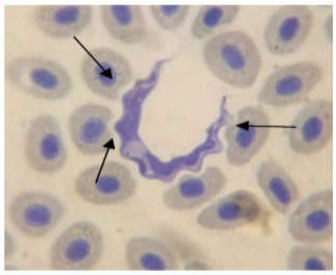
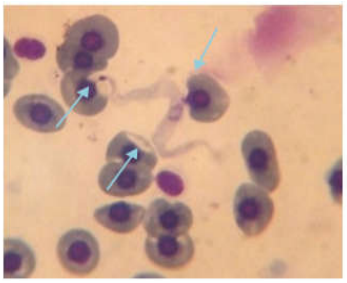
With the exception of the polar region, avian trypanosomes were reported worldwide (Baker, 1976: Zidkova *et al.,* 2012). Though over 100 species of the parasite were reported, the status of the disease and indeed specific vector in birds, is still poorly understood (Zidkova *et al.,* 2012). In most cases, the transmission of the disease was attributed principally to *Culex*

mosquitoes, Black flies and Hippoboscids (Votypka *et al.,* 2002; Peirce, 2003; Torello, 2005; Jan *et al*., 2012; Hartman, 2014).

hemorrhagic enteritis, resulting into internal ecchymosis, external hemorrhage and subsequently anemia. While conducting clinical trials with a canary that was infected with the parasite, splenomegaly, lymphoid hyperplasia and focal

**Table 1. Nature of trypanosomosis among vertebrates**

| | Man | Cattle | Horse | Birds | Fishes | References |
|---|---|---|---|---|---|---|
| Disease | Sleeping Sickness | Nagana | Mal de Caderas | Avian trypanosomosis | Piscine trypanosomosis | FAO (2012)<br>Merck (1986) |
| Vector | Tsetse fly, Bugs | Tsetse fly, Tabanus, Stomoxys. | Tsetse fly, Tabanus, Leeches. | Mosquitoes, Hippoboscids, Black flies. | Leeches | Balakrisnan & Zumla (2001)<br>Baker (1976)<br>Hartman (2014)<br>Egil *et al*. ( 2005) |
| Etiology (Trypanosomes) | *T. gambiense*<br>*T .rhodensiense*<br>*T. cruzi* | *T. vivax,*<br>*T. congolense,*<br>*T. brucei.* | *T. equinum,*<br>*T. equiperdum,*<br>*T. evansi.* | *> 100 species*<br>*T. culicavenu*<br>*T. avium* | *> 200 species*<br>*T. danilewhisky*<br>*T. aligaricus*<br>*T. mukasai* | Baker (1976)<br>Kolesnicov *et al*.(1995)<br>Neelima (2006)<br>OIE (2013)<br>Zidkova, *et al*.(2012) |
| Epidemiology | Africa, L/America | Africa, Asia | Africa, S/America Asia, Mid East | Worldwide | Worldwide | Zidkova *et al*. (2012<br>Nazrul Islam & Woo (1991)<br>OIE (2013)<br>Merck (1986) |
| Signs | Sleeping Sickness, Jaundice, Anemia. | Anemia, Wasting, Abortion. | Wasting, Hindlimb paralysis, Anemia. | Anemia | Lethagy, Anemia, Anorexia, Wasting, Death | Torello (2005), Neelima (2006), FAO (2012)<br>Desquesnes *et al*.(2013)<br>Merck (1986). |
| Pathology | Neuropathy, Anemia, Jaundice, Hepato-splenomegaly | Erythropenia, splenomegaly, Anemia. | Anemia, Neuropathy | Focal myocarditis, Anemia, Splenomegaly, Lymphoid-hyperplasia | Decreased PCV, RBC, Hb, blood proteins and Cholesterol, Anemia. | Osman *et al*. (2009)<br>Desquesnes *et al*. (2015)<br>Mbaya *et al*. (2012)<br>FAO (2012)<br>Nazrul Islam and Woo (1991)<br>Merck (1986) |
| Control | Vector control | Vector control | Quarantine<br>Vector control | Vector Control | Vector Control | Baba *et al*.(2017)<br>Dietmar (2008) |
| Treatment | Pentamidine, Suramin, Melarsoprol, Eflornithine. | Diminazine-aceturate, Suramin | Diminazin aceturate | Melarsomine | NA | Torello (2005)<br>WHO (2012)<br>Merck (1986) |


**Plates 1 & 2. Photomicrograph of trypanosome in blood of fish, (indicating adhesion with RBC, & zone of lysis : Arrows show cyto adhesion) Plate 3; Common leech from Sokoto River, Nigeria**

Biting midges and mites were also incriminated as vectors of the disease (Molyneaux, 1977). Earlier on, Baker (1976) and Terry and Christine (2007) considered the parasite less harmful to birds. The division of procyclic epimastigotes in the mid and hind gut of mosquitoes suggested cyclic transmission of the parasite (David and Nair, 1955). An alternative oral transmission was also reported by Jan *et al.* (2012), to re affirm the direct life cycle and dynamism of transmission, similar to *Chagas* and *dourine* in man and equides respectively. Though the condition was considered relatively non pathogenic (Baker, 1976; Votypka *et al*., 2002; Pierce, 2003). Torello (2005) recorded success in treating a confirmed outbreak of avian trypanosomosis in a flock of falcons in Kuwait. Clinically, the birds were presented with signs of blood- tinged droppings, anorexia, inability to fly high, dyspnoea, and death. The falcons were treated with melarsomine at the dose rate of 0.25mg/kg x 4/7. The blood stained feces was an evidence of myocarditis were observed at postmortem (Molyneaux *et al.,* 1983). These lesions were considered similar to those recorded in mammalian infections.

## Trypanosomosis in Fish

Trypanosomes in fish are reported globally, with over 200 species described among freshwater and marine fishes (Koleneskov *et al.,* 1995; Neelima, 2006). Under aquatic conditions, freshwater and marine fishes were reported to be infected with trypanosomes globally, though most of them were considered relatively less harmful to fish (Nazrul Islam and Woo, 1991). With overwhelming evidence through dedicated studies, some piscine trypanosomes were however considered harmful to host, depending on the pathogenicity of the species and level of parasitemia. These records were documented under laboratory and natural conditions (Bhaskar

*et al.,* 1984; Nazrul Islam and Woo, 1991; Osman *et al.,* 2009; Neelima, 2012; Fujimoto *et al.,* 2013; Muhammad *et al*., 2017), with leeches incriminated as vectors for transmission in fish.

anesthetic, for minimizing pain (Mark, 2008). The head of the parasite is modified with specialized organs for piercing, cutting and sucking blood from the host (Govedich and Bonnie, 2005).

**Table 2. Assessment of blood loss in trypanosome infection among different animal species**

| | Species of Animal | | | | | |
|---|---|---|---|---|---|---|
| | Human | Bovine | Equine | Avian | Piscine | Reference |
| Hemolysis | ** | ** | ** | * | * | Nazrul Islam & Woo (1991); |
| Internal Hemorrhage | - | - | - | * | - | Torello (2005) |
| External Hemorrhage | - | - | - | -* | * | Torello (2005) Abdulkader*et al. (*2013) |
| Vector blood meal | * | * | * | * | *** | Comfort *et al,* (2002); Eco-Raider, (2015) |
| Quantification of blood meal size (mg /mls) | 7 – 55 mg; 19 – 78, mg | 7 –55 mg; 19 – 78 mg | 7 – 55 mg ; 19 – 78, mg | 0.001 - 0.01mls | 2 -5 mls | Gaston and Randolph, 1993: Taylor, (1997); Laden (1997) |

*Mild, ** Moderate, *** Severe

**Table 3. Comparative features of trypanosomosis (vector/ host relationship in mammals)**

| Trypanosomosis | Man | Domestic/wild animals | Birds | Fishes | References |
|---|---|---|---|---|---|
| Vector invasive features | Piercing/ sucking | Piecing, /sucking | Piercing/ sucking | Attachment /piecing/ cutting /sucking | Govedich and Bonnie (2005) |
| Vector effect | Chancre, irritation, pain | Local irritation | NR | Anticoagulant, local anaesthetic, Y-shaped mark, Hemorrhage, hypovolumic shock | Govedich and Bonnie (2005) Pietrangelo *et al* (2017) |
| Vector / Host wt .ratio | 22 : $60^7$ (22mg fly / man of 60 kg) | 22: $10^8$ (22mgfly / 300 kg animal) | 5 : $10^6$ mg (5mg fly / 1kg bird) | 11: 100g (1 g leech /100g fish) | Konishi (1989) Merck (1986) Colless & Challapah (1960) |

Anemia, anorexia, lethargy and deaths were observed in Goldfish infected with *T. danilewhskyi* (Nazrul Islam and Woo, 1991). Reduction in hemoglobin level by 30% was recorded in *Heteropneustes fossilis infected with T. danilewhsky* (Bhaskar *et al.,* 1984). Ahmed *et al*. (2011), however, observed decrease in PCV, erythrocyte and hemoglobin levels with increased mortality in juvenile carp infected with *T. danilewhskyi.* Neelima and Gupta (2012), recorded 30 - 33% reduction in erythrocyte counts from infected *Clarias batrachus*, *Wallu atta*, and *Channa punctata*, that were challenged with *T. batrachi*, *T. aligaricus* and *T. atti* respectively. Evidences of anemia due to natural infections were also documented by Shahi *et al.* (2013), in Kashmir, Fujimoto *et al*.(2013), in Brazil and Muhammad *et al.*(2017) in Nigeria. Evidence of cytoadhesion with subsequent lysis of RBC was observed among wild species of fish infected with trypanosomes in Nigeria (Plates 1 and 2). Similar phenomenon was earlier reported by Brian (1989). These parasites are known to be transmitted from one fish to another through the vector leech (Simon and Woo, 1991; Neelima, 2006; Polly *et al.,* 2014; Moara *et al.,* 2015; Muhammad *et al.,* 2016). The common leech parasitizing wild fish of northern Nigeria is presented in plate 3.

## Leeches

With over 500 species worldwide, very few leeches are parasitic to humans, amphibians, reptiles and fishes (Govedich and Bonnie, 2005). The parasites could occasionally engorge with large blood meal from their hosts (Gingerich *et al.,* 1989; Odoya *et al.,* 2015). Leeches are hermaphrodites whose saliva contain hirudin (Knobloch *et al.,* 2007), a potent anticoagulant that prevents blood clotting at the site of bite, in addition to other enzymes that prevent platelet aggregation, notably: calin, suratin and lefaxan, as factor –X inhibitors; buriden and hirudin, as anti thrombin agents (Abdulkader *et al.,* 2013). Though not yet proven, leech saliva might contain a local

These features were fully exploited in human medicine as originated from ancient Egyptian and Greek physicians, and still applicable in many hospitals worldwide. Hence *Hirudin medicinalis* (a leech) became popular in the history of medicine, for the past 2500 years. The application of leech in modern medicine (leeching) for relieving venous congestion in reconstructive surgery is gaining momentum worldwide (Munro *et al.,* 1989; Knobloch *et al*., 2007; Abdulkader *et al.,* 2013). Notwithstanding their beneficial come back in medicine, leeches are associated with cyclic transmission of trypanosomes in fish and several aquatic mammals globally (Simon and Woo, 1991; Egil *et al.,* 2005; Neelima, 2006; Polly *et al.,* 2014). Important leeches associated with piscine trypanosomes are mostly members of *Rhynchobdellae* under *Glossiphonidae* or *Piscicolidae* families (Paperna, 1996). From Canada, developmental stages of *Trypanosoma. phaleri* were observed in a leech vector *–Desserenobdella phalera* (Simon and Woo, 1991). Likewise, procyclic forms- amastigotes and epimastigotes of trypanosome from marine fish were observed from adult and juvenile leeches (*Zeylanicobdella arugamensis*) in South Africa (Polly *et al.,* 2014).

From the continent of South America, developmental stages of *Trypanosoma abeli* were recovered from different parts of the gastrointestinal tract (GIT) of the leech *Haementteria brasilensis*, recovered from Brazilian armored catfishes *Hypostomus affinis* and *H. luetkeriand* (Moura *et al.,* 2015). The preponderance of leeches and high prevalence of piscine trypanosomes were earlier reported in ten out of eleven water bodies of north- western Nigeria (Muhammad *et al.,* 2016), which conform with Baker (1961) for occurrence of leeches in all waterways of Africa (Plate 3). Similarly, the teeming number of piscine trypanosomes documented from India (Neelima, 2006) and Brazil (Fujimoto *et al.,* 2013) were attributed to several species of leeches as potential vectors. Hence, recognition of leeches as vectors of fish trypanosomes is fully appreciated globally (Muhammad *et al*., 2017). Though

reliable data on quantification of fish blood meal of leeches under natural conditions are lacking, previous records documented for *Hirudin medicinalis* in hospitals from Europe were considered as replica of what is expected with fishes in the wild. Several experimental studies and records suggested an unprecedented withdrawal of blood from human body of up to 2 mls (Comfort *et al*., 2002). Records also showed single blood meal of some leech species in the range of 1 – 15mls of blood from one patient (Chris, 2017).

**Comparative Study**

An overview of trypanosomosis among different classes of animals is presented in table 1, while blood loss due to trypanosome infection, as predisposing factor to anemia in different species, is summarized in table 2. The parasite-host relationship compares the mode of transmission, local effect on the host and relative weight of the vector to its host, assessing quantity of blood withdrawn that could jeopardize the survival of the host. Vectors involved include tsetse flies, belonging to *Glossina* species in man, and domestic animals, *Culex* mosquitoes for birds and leeches in fish respectively. Trypanosomosis in livestock is reported from several regions, depending on the species of the animal and trypanosome involved. Some are cyclically transmitted by tsetse flies (*T. vivax, T. congolense*) and others are transmitted mechanically (tabanid flies). Yet other trypanosomes are sexually transmitted (*T. equiperdum*). The effect of vector could be seen at the site of the bite by inflammatory swelling (Chancre), or Y-shaped mark, from the site of leech bite (Govedich and Bonnie, 2005; Kara, 2017). Similar lesions associated with tsetse in animals and mosquitoes in birds are less pronounced.

The size of blood meal for tsetse flies is in the range of 19 -78 mg for *G. palpalis,* and 7- 55 mg for *G morsitans* (Gaston and Randolph, 1993; Taylor, 1997). Though values for different species of mosquitoes differ, Konishi (1989), experimenting with a dog, reported volumes in the range of 1 nl - 6µl. However, earlier records of 5mg by weight of blood was documented by Colless and Chellapah (1960), for *Aedes aegypti* mosquito. In recent time, Eco Raider (2015) projected blood meal of mosquitoes in the range of 0.001 to 0.01 millilitre per bite. The voracious blood meal of medicinal leech *Hirudin medicinalis* were assessed from regular blood letting in venous decongestion within the range of 2 mls (Comfort *et al.,* 2002) to 15 mls (Chris, 2017) at a time, by a single leech, depending on the species, in comparison to the negligible quantity from tsetse and mosquito bites (Table 2). The parasite host weight relationship compares the size of the vector (mg / g), as 5 mg for mosquitoes: 20- 30 mg for tsetse fly and 1- 2 g for a leech.

The ratio is wider for mosquitoes and tsetse flies, in relation to size of their respective hosts, than a closer ratio of 1: 100, for leeches in relation to the size of 100g fish. Considering the total blood volume for most fishes, which is about 3% of body weight (Shukla, 2009) or in the range of 3- 5 mls /100g body wt., as projected (Gingerich *et al*., 1989), withdrawal of a minimal quantity of 2 mls from a 100g fish could subject the host to severe anemia, or hypovolemic shock and death. The extreme low PCV recorded in some smaller fishes (Muhammad, 2014) revealed the adverse effect of high volume withdrawn probably from repeated leech meals. For engorgement, a leech could take about 5 times its normal size / GIT capacity. An average size leech therefore, could consume up to 1 -5 mls of blood from the host. High infestation with more than one leech per fish could result into severe hypovolemic shock as earlier observed (Letch, 1977). Natural infection and histopathological records of clinical trials of avian trypanosomosis as reported by Torello (2005) and Molyneaux *et al.* (1983) respectively, suggested pathogenecity of avian trypanosomes, contrary to some reports (Baker, 1976; Votypka *et al.,* 2002; Peirce, 2003; Hartman, 2014). The biologically active substances in leech saliva: hirudin (Knobloch *et al*., 2003), calin, lefaxan, buriden, and suratin (Abdulkader *et al.,* 2013), facilitate free flow of blood at the site of bite after detachment, contrary to tsetse and mosquito bites. These factors account for another probable cause of anemia in fish.

**Conclusion**

Anemia is an important clinical manifestation of trypanosome infection in man, aquatic and terrestrial animals, the severity of which differs with the species of host and pathogenecity of the parasite, possibly due to accelerated RBC destruction. However, in leech transmitted piscine trypanosome infection, anemia has multidimensional and multifaceted causation. Accelerated RBC destruction, bleeding at the site of bite and additional loss of blood to heavy leech meal, constituted multifactorial nature in fish.

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