

Nasal Myiasis in animals due to Oestridae - A Mini Review

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Abstract— *Oestrus ovis*, the sheep 'nasal bot fly', somewhat looks like a honey bee, has a rudimentary mouth parts and do not feed. The larva of the sheep nasal fly is responsible for causing a condition in sheep and goat called nasal myiasis, oestriosis or 'false gid'. The disease occurs when adult fly deposit first larvae (L₁) into the nostrils of its host. The larvae develop into L₂ and L₃ in the nasal cavities and sinuses in due course. Sneezing and nasal discharges are the major clinical signs seen in infected animals. The pathogenic reaction occurs when the larvae irritate the mucosa with their spines and hooks during their development and also due to an allergic reaction induced by molecules excreted/secreted by the larvae. Sometimes damage of the skulls of the bones and injury to the brain occur to such an extent that signs of high-stepping gait and in-coordination suggesting the animal suffers from *Coenurus cerebralis* infection and hence the infection is also called false gid. Keeping in view the importance of the fly, the morphology, biology, pathogenesis, immunological reaction and various control measures of *Oestrus ovis* are discussed in this review.

Keywords— *Oestrus ovis*; morphology; biology; immunopathology; control measures.

I. INTRODUCTION

Oestrus ovis which is commonly called 'sheep nasal fly' or 'nasal bot fly', is responsible for severe economic losses in livestock across the globe (El-Tahawy *et al.* 2010). The larval phases (parasitic) of the fly irritate the nasal mucosa with their spines and hooks while feeding to support their growth and development (Cepeda-Palacios *et al.* 1999; Tabouret *et al.* 2003). The migratory larvae even penetrates and erode the dorsal turbinate bones,

frontal sinuses and occasionally the skull bones while entering into the cerebral cavity causing 'false gid' (Taylor *et al.* 2016).

Human in close contact with livestock, sheep particularly are at greater chance to become accidental hosts for the *O. ovis* larvae. So the disease is also have public health importance (Cepeda-Palacios 2001; Einer and Ellegard 2011; Hummelen *et al.* 2011; Hoyer *et al.* 2016). Clinical signs are manifested by mucopurulent nasal discharge, frequent sneezing and difficulty in grazing, restlessness, incoordination and dyspnoea (Dorchies *et al.* 1992; Dorchies and Alzieu 1997; Dorchies *et al.* 2000). The pathogenic effects of oestriosis are due to the damages in the nasal mucosa caused by the cuticular spines and oral hooks but also by immunological reactions of larval antigens (Jacquet *et al.* 2005). Important losses sue to *O. ovis* include upto 22% loss body weight, 16% in wool production and 10% in milk production (Shcherban 1973). The fly has cosmopolitan in distribution where sheep and goats rear. They also attack deer and occasionally horses, cattle, dogs and human. The current method of oestriosis is based on chemotherapy in sheep because no bait or traps are available to control the free living adult fly (Cepeda-Palacios and Scholl 2000b). Endectocides and fasciolicide drugs such as closantel and nitroxylnil are often used to treat nasal myiasis in sheep and goats (Dorchies *et al.* 1997).

The purpose of this review is to discuss the morphology, biology of *Oestrus ovis* together with immunopathology caused by the larvae of this fly vis-à-vis some of the controlling measures based on available literature.

II. MORPHOLOGY

The genus *Oestrus* contains four species *O. aureogentatus*, *O. caucasicus*, *O. ovis* and *O. variolosus*. *O. aureogentatus* and *O. variolosus* are found throughout Africa, south of the Sahara and *O. caucasicus* is present in domestic and wild animals of the Caucasus regions and central Asia. However, *O. ovis* is found in all sheep farming areas of the world. The adult fly is about 13-15 mm in length and grey in colour with black spots on the thorax and abdomen and somewhat resembles a bee (Fig. 1). The whole body is covered with light brown hair. The mouthparts are vestigial. The head is broad with small eyes. The segments of the antennae are small and the arista bare. They have a yellow-brown head, with small tubercles of equal size on the thorax and the yellow legs. They possess yellow-veined wings which have a strongly bent M vein joining the R₄₊₅ veins before the wing margin. It is an obligate parasite in sheep and goats in many parts of the world.

The tiny first-stage larvae are white or slightly yellow in colour and spindle in shape, 1-3 mm long with relatively large cephaloskeleton which may be seen during post-mortem by sawing the skull in half-longitudinally (Fig. 2). They are provided with strongly bent sclerites (gently curved mouth-hooks) and 22-25 terminal spines arranged in two groups. The third segment contains a row of denticles on the dorsal side. Ventrally the segments at their anterior margins show two to three rows of spine and hair like structures. Laterally, they have 22-25 hooks (Zumpt 1965).

The second instar larva is white in colour and 3.5-12 mm long with few weak denticles on the dorsal side of the second segment, the median part of the post-anal bulge is spinulose, ventrally the segments are provided with spines, the posterior peritremes are more or less circular, the channels are indicated by distinct suture (Zumpt 1965).

The mature larvae are about 2-3 cm long and brown with transverse, dorsal blackish bands. The anterior end is somewhat tapering but the posterior surface has a flat surface. The larvae bear large, black oral hooks, connected to an internal cephalopharyngeal skeleton (Fig. 3) The dorsal surface is devoid of spines while the ventral surface bears rows of small spines and the black stigmatic plates are circular, with a central ecdysial scar, and without a distinct suture (Zumpt 1965).

III. PREVALENCE AND DISTRIBUTION

The prevalence and incidence are greatly varied depending on climate and ecological factors. In temperate countries the flies occur in late spring and summer whereas in warm climate it can infest the sheep throughout the year. The prevalence and distribution of the fly is shown in Table no. 1.

Table.1: Prevalence rates of *Oestrus ovis* infestation in different countries

Country	Prevalence Rate (%)	Reference(s)
Saudi Arabia	5.5	Alahmed <i>et al.</i> , 2002
India	8.1	Pathak, 1992; Godara <i>et al.</i> , 2010
Zimbabwe	6 – 52	Pandey, 1989
Morocco	10 – 100	Paney and Ouhelli, 1984
Ethiopia	21.0	Gebremedhin, 2011; Bekele and Mukasa-Mugerova, 1994
Libya	22.6	Gabaj <i>et al.</i> , 1993
France	33.2 – 65	Dorchies <i>et al.</i> , 2000
Jordan	58.0	Abo-Shehada <i>et al.</i> , 2000
Algeria	67.4	Benakhla <i>et al.</i> , 2004
Spain	71.1	Alcaide <i>et al.</i> , 2003
Italy	91.0	Caracappa <i>et al.</i> , 2000
Spain	27.3	Barroso <i>et al.</i> , 2017

IV. BIOLOGY AND HABITS

The life cycle of the sheep nasal fly is shown in figure 4. The life cycle begins when the young larvae are deposited by the adult flies. Each female can produce up to 500 larvae. Bart and Minar (1992) reported that many L₁ are destroyed in the nasal cavities during the hypobiotic period. After being deposited the larvae crawl onto the mucous membrane of the nasal passage where they spend at least two weeks and are found attached to the mucous membrane by means of oral hooks. First stage larvae are deposited in packages directly into the nostrils with accurate precision. Thermo sensible cuticular sensilia and a quick mobility of the larvae allow them to overcome the first defence reactions such as sneezing and rubbing against close objects (Colwell and Scholl, 1995). It has been reported that the rate of larval establishment is 0-48% in sheep (Frugere *et al.* 2000) and 29-40% in goats (Angulo-Valadez *et al.* 2009). It is known that better water economy in goats leads to len humid noses than in sheep. This higher humidity is more conducive for larval survival in sheep than goats (Papodopoulos *et al.* 2010). Inside the host's nose, the larvae either continue to grow or hibernate in response to a combination of intrinsic rhythms and external environmental stimuli. Temperate countries, the first stage larvae hibernate but very high temperature slows the growth rate (Yilma and Dorchies 1991). Limited information is available about second stage larvae. They quickly develop into the sinuses of the

host and trigger a strong cellular reaction with infiltration of mast cells and eosinophils. This stage relies on a high protein diet which mainly they derive from mucus and seroproteins by way of trypsin like enzymes (Tabouret *et al.* 2003b). Finally the full grown larvae crawl out and expelled by the sneezing of the host onto the ground where they pupate. Under favourable environmental conditions, the duration of the first, second and third stages vary from 10-25 days, 7-15 days and 13-18 days, respectively (Cepheda-Palacios 2002). Intrapuparial metamorphosis occurs over 19-27 days before adult flies emerge.

Adults do not bear functional mouth parts and therefore minimize the energy loss by localizing their potential hosts and suitable mates with their large eyes. They are swift fliers and since they do not feed, the lifetime of adult is short and females emerge from the puparium bear fully developed eggs which are ready to fertilize (Taylor *et al.* 2016).

V. IMMUNOPATHOLOGICAL REACTIONS

The intensity of local changes inoculated by *O. ovis* larvae in the mucosa of the upper respiratory tract of the host is not related to larvae density but Biggs *et al.* (1998) suggested that any larval number above 10 is potentially dangerous. The most significant lesions are seen in the sinus and ethmoidal mucosa. In natural or artificial infection, there is hyperplasia and abrasion of mucociliary film (Dorchies *et al.* 2006). A strong cellular response is elicited as many cells are positively marked as Ki67 epitopes (Nguyen Van Khanh *et al.* 1998). Many ultra-structural changes in the nasal sinus are due to a combination of mechanical damage associated with effects of secreted proteases from the larvae. These changes increase the permeability of the mucosa allowing the diffusion of antigenic/ excretory products through the mucosa to come in close contact with the locally recruited immune cells (Dorchies *et al.* 2006). Histopathological data indicates pathogenesis of ovine oestrosis is due to Type-I immediate hypersensitivity phenomenon. Many B and T lymphocytes, phagocytic mononuclear cells (PMC), eosinophils, mast cells and globule leucocytes have been observed where larval moult occurs (Tabouret *et al.* 2003b). Nasal myiasis also induces IgM and IgG production in both sheep and goats (Suarez *et al.* 2005; Angulo-Valadiz *et al.* 2008, 2009).

Clinical signs can be described into three categories: fly strike, sinusitis and other consequences.

5.1. Fly strike

Fly activity causes great annoyance when attack sheep to deposit larvae into the nostrils. The animals get nervous and congregate together, keeping their noses deep inside the fleece of the other sheep or close to the ground (Fig. 5, 6). Animals become restless and stop feeding. Goats

are less reactive because of their browsing habits (Hoste *et al.* 2001).

5.2. Sinusitis

The irritation of the nasal mucosa caused by the oral hooks and spines of the fly larvae manifests by nasal discharge and sneezing. Sheep are agitated and the nasal discharge occasionally become purulent tinged with blood (snotty nose, Fig. 7). Sometimes erosion of the bone and eventual injury to the brain with neurological signs including ataxia, nystagmus, high stepping gaits and in-coordination of movements may be misdiagnosed as *C. cerebralis* infection. For this reason, the infection is also called false gid.

5.3. Other consequences

In some breeds of sheep, neoplastic growth might be found (Bergeaud *et al.* 1994). In some cases, interstitial pneumonia with interstitial emphysema and pleural adhesion has also been observed (Dorchies *et al.* 1993).

VI. TREATMENT AND CONTROL

The objective of the treatment is to eliminate or at least suppression of clinical signs and to limit the extension of the endemic zone of the parasite. Sheep already infected with nasal bot flies can be successfully treated with several parasiticides. Ivermectin either as injectable form or oral drenches have excellent curative effect and gives protection against re-infestation after several weeks of treatment (Bowman 2014). Roncalli (1984) reported that larvae of *O. ovis* are highly susceptible to ivermectin at a dose rate of 0.2 mg/Kg body weight. The efficacy of eprinomectin at with 0.5 mg/Kg and 1 mg/Kg body weight ranges from 83.5 – 100% (Hoste *et al.* 2004; Habela *et al.* 2006).

Anthelmintics which are commonly used against nasal bots include closantel nitroxynil and rafoxanide. Closantel have a persistent effect on larvae and can give protection to animals from reinfestation during fly season (Dorchies *et al.* 1997).

Enhanced immune responses may have a detrimental effect on *O. ovis* larvae led to immunological trials against *O. ovis* in sheep using excreted/secreted products and digestive tract protein extracts of third instars by previous works (Frugere *et al.* 2000; Angulo-Valadiz *et al.* 2007). It was predicted that immunized animals would develop humoral responses against such antigens. The authors concluded that a reduction of 40% mature larvae weight would reduce 38% adult population. Further experiment is required to ascertain whether such immunization can affect the adult population significantly.

The more general control measure includes feeding in narrow troughs, the edges of which are smeared with tar. This automatically tars the animals and thus acts as a repellent (Bowmann 2014).

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Figures:

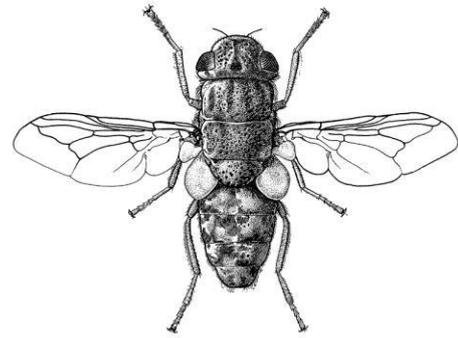


Fig.1: Adult *O. ovis* fly (Anon, 2018)

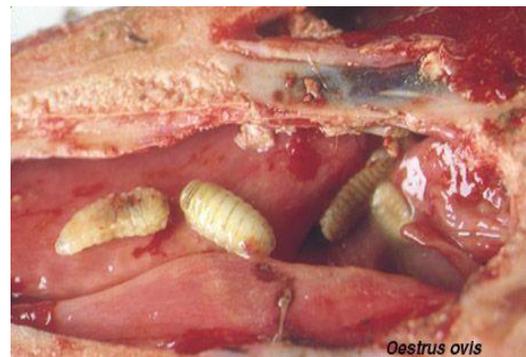


Fig.2: Third stage larvae of bot fly in nasal sinus (Anon, 2018)

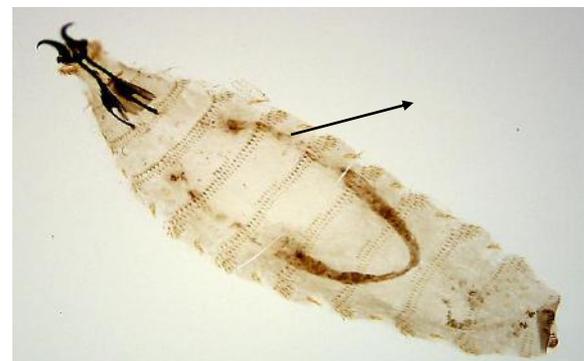


Fig.3: Cephalopharyngeal skeleton of *O. ovis* larvae (Anon, 2018)

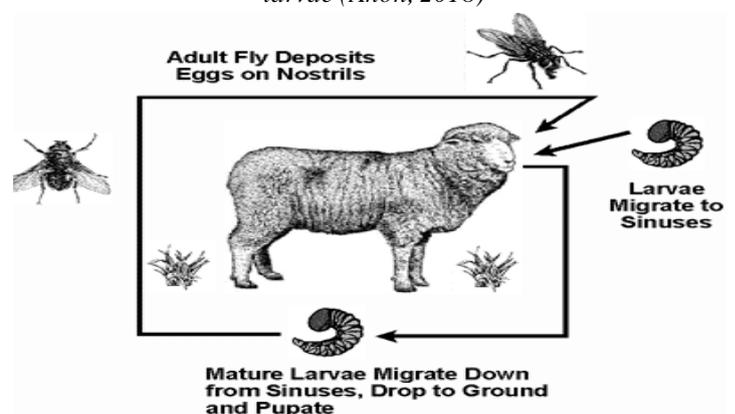


Fig.4: Schematic diagram of *O. ovis* life cycle (Anon, 2018)



Fig.5: *O. ovis* infested sheep (Anon, 2018)



Fig.6: Depression in a sheep due to false gid (Mozaffari et al., 2013)



Fig.7: snotty nose (Anon, 2018)