



**Table 4: Diagnostic parasitology sample throughput and positive results for the third quarter 2008**

	Number of Samples Tested	Number Positive	Number of Contributing Laboratories
<b><u>Endoparasites</u></b>			
Ascarids	669	26	7
Cyathostomes	800	195	9
Dictyocaulus	121	68	6
Strongyles	767	165	8
Tapeworms (ELISA based testing)*			
Tapeworms (Faecal exam)	641	2	5
Trichostrongylus	28	8	1
Strongyloides	341	14	8
Oxyuris equi	16	1	1
<b><u>Ectoparasites</u></b>			
Mites	228	4	7
Lice	142	0	5
Ringworm	236	34	8
Dermatophilus	104	16	6

## **FOCUS ARTICLE**

### **Anthelmintic resistance in equines – a new perspective**

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In this article it is hoped to bring the reader up to date with the current situation and give information on how to do something about it.

Anthelmintic resistance in horses has been reported since the 1970's, mainly to the benzimidazole group of drugs. The widespread use of ivermectin based products means it is only a matter of time before we see a marked increase in resistance levels to that drug. Resistance has in the past been a very significant problem in ruminants most especially sheep and it is from these species that we can learn how resistance develops and how to avoid the situation becoming so significant in the equine population. Ivermectin resistance has also been widely reported in humans in Africa where River Blindness caused by the nematode *Onchocerca volvulus* is a significant problem. If we get to the stage where no horse wormers are efficacious then equine welfare will be severely compromised.

Steps to prevent or reduce resistance to anthelmintics include:

- 1) Use of existing products sensibly – minimise excessive frequency of dosing, estimate weight of animal accurately and do not over or under dose, rotate the class of drug (not brand) annually, monitor the treatment with regular faecal worm egg counts.
- 2) Try to minimize pasture contamination (pick up droppings) and make pastures low risk (de-horse them; graze with other species for a year or two).



- 3) Most of the worms live within a few susceptible animals; these animals need identifying and treating accordingly.
- 4) Treat brought in animals on arrival and keep indoors until clear of infestation.

Understanding the life cycle of the parasite is important in devising successful control measures. The worms are not in the host for very long as for most of the cycle they are as free living infective larvae on the pasture. The phenomenon of “refugia” has been known of since the early 1980’s and it may well be a major way forward in reducing/preventing anthelmintic resistance. “Refugia” refers to the population of worms that are not exposed to drug treatments and therefore have not developed resistance.

Three factors that influence the numbers of worms in “refugia” are:

- 1) Number of larvae on the pasture,
- 2) Percentage of animals treated with anthelmintics,
- 3) The ability to kill all developmental stages within the host (if inhibited worms in the gut mucosa are not treated then the young emergent worms are in “refugia” and this is how the worm can survive drought).

In order to make the numbers of worms in “refugia” greater, reliance should be placed on removal of faeces and limiting treatment of animals with wormers to only those that require it, possibly only young stock and those with positive worm egg counts). The population of worms in “refugia” then dilute the population of resistant worms, with a pool of sensitive genes.

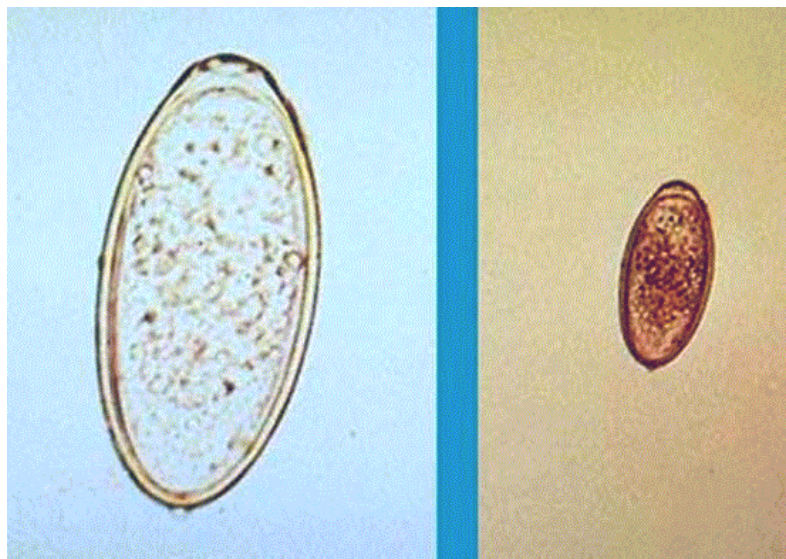


Figure 1: Strongyle eggs as seen under the microscope.

The molecular method of drug resistance within the parasites is poorly understood, the only gene associated with resistance that has been isolated so far is the beta tubulin gene.

It is thought that genes for pyrantel/ivermectin resistance are naturally rare in nematodes of equines and therefore resistance should be slow to develop. However, the advent of drugs that do kill the encysted stages (such as moxidectin) is decreasing the worms in “refugia” and thus increasing both the likelihood of resistance and the speed at which it will



arrive. As no new anthelmintic products are likely to be developed in the foreseeable future we have to look to alternative methods of worm control.

As well as increasing the numbers of worms in “refugia” other areas that should be researched as methods of worm control, include possible vaccine development, forms of biological control and host selection. All of which require a greater understanding of the genetics and molecular methods of survival employed by the nematodes both within the host and in the environment.

### **Report on *Post Mortem* Examinations for Third Quarter 2008**

#### **East Anglia**

*Thirty one cases were examined including fourteen fetuses, one of which was from a miniature donkey.*

Of the fourteen fetuses examined this quarter six were from thoroughbred mares. One thoroughbred fetus of 186 days gestation was positive for EHV-1 which was diagnosed by virus isolation on mixed fetal tissues. This diagnosis was confirmed by PCR testing on mixed fetal tissues. The mare was vaccinated against herpes virus and none of the in contact mares have shown any clinical signs of disease. One thoroughbred fetus of 200 days gestation had placental malperfusion, one thoroughbred fetus of 160 days gestation had acute umbilical cord torsion with eleven twists of the cord identified and one thoroughbred fetus of 210 days gestation was found to have umbilical herniation with eventration of small intestine and liver, however it was unclear if this was the cause of the abortion. One thoroughbred mare had an abortion at 180 days gestation no cause could be found. This was not the first occurrence of this problem in this mare and it was recommended that she had a thorough pre-breeding examination. One thoroughbred mare aborted at 150 days gestation; no cause could be identified. Seven other fetuses were examined but no definite cause of the abortions could be identified.

A multiparous miniature donkey jenny gave birth to a still born full term foal. The foal was still in the fetal membranes (apart from the nose where the jenny had tried to release it from the membranes). The foal was tested negative for EHV however *Areomonas hydrophilia* was isolated from swabs of fetal lung, the chorion and the jenny's uterus. There were unconfirmed reports that the jenny had been ill during the pregnancy and post delivery bloods revealed lipaemia, the significance of which is unknown. A maiden jenny from the same stud had also had a red bag delivery, which the foal did not survive, a few weeks earlier.

A neonatal foal was examined and found to have a congenital heart defect. It was euthanased and mitral valve dysplasia was identified at *post mortem* examination.

A two month old foal died of haemothorax which was thought to be caused by breakdown of an adhesion between the right lung and a healing rib fracture. The rib fracture had no acute pathology associated with it and was thought to be an injury from parturition.

A six month old foal with a history of previous strangles infection was found dead in the field. It had been being treated for suspected *Rhodococcus equi* infection at the time. *Post*