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Backyard Poultry Online 'Mini Series'

Session 1: Behaviour, Skin and Gastrointestinal Conditions

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These notes cover the conditions included in the session.The links below will direct you to poultry medicine texts for further information and also for conditions not included in the notes.

Prescribing in Chickens

The list in the link below is used to decide on the most suitable medicine. It is a hierarchy in that the first suitable medicine found as you work your way down the list should be used. A medicine from the lower tiers should not be used if a suitable medicine can be found higher up the list.

If there is a licenced veterinary medicine in the UK to treat the specific condition in the species being treated then use this and follow the stated withdrawal periods.

If there is a medicine licenced in the same species for different condition that would be appropriate then use this and follow the stated withdrawal periods.

If there is a medicine licenced in a different species intended for human consumption that would be appropriate then see below for information on withdrawal periods.

If there is a medicine licenced in veterinary medicine that would be appropriate to use then see below for information on withdrawal periods.

If there is a medicine licenced in human medicine that would be appropriate to use then see below for information on withdrawal periods.

If there is a medicine licenced in veterinary medicine in another EU member state,that would be appropriate to use then see below for information on withdrawal periods.

If there is a medicine listed in table 1 at http://ec.europa.eu/health/files/eudralex/vol-5/reg_2010_37/reg_2010_37_en.pdf that would be appropriate to use, see below for information on withdrawal periods.

Withdrawal periods for meat and eggs:

If the product is not listed in Table 1 then you cannot use it.

If licenced in another EU country for the same species then use the withdrawal period stated on the product literature.

If the product is listed in Table 1 and you able to recommend an appropriate drug withdrawal time for meat and, if appropriate, eggs for this drug then you can use the drug. The minimum withdrawal periods are 28 days for meat and 7 days for eggs (unless shorter periods are stated in the product literature).

If you are not able to recommend an appropriate withdrawal time then you cannot use this drug.

Below is the link to European Medicines Agency's Maximum Residue Limits reports. These documents summarise the available scientific information and from this information you may be able to set withdrawal times for eggs and meat. The withdrawal times will depend on the route and the dose given.

http://www.ema.europa.eu/ema/index.jsp?curl=pages/medicines/landing/vet_mrl_search.jsp&mid=WC0b01ac058008d7ad

Table 2 lists prohibited substances for which a maximum residue limit could not be established because residues of those substances, at whatever limit, constitute a hazard to human health. Important drugs on that prohibited list include Chloramphenicol and Metronidazole.

Enrichment
Environmental enrichment can make a big difference in the welfare of poultry.
Social interaction and foraging opportunities are the most important aspects of enrichment for chickens.

The ideal foraging material is soil that they have to scrape with their feet and will produce the occasional tasty worm or grub to eat.

Grassed areas are good but the safest plan is to keep the grass cut short. Also care with hay as long fibres of grass or hay are a common cause of crop impaction.
Chickens do not appear to play with toys but will if available to them spend most of their awake time foraging.

It has been shown that raising chicks in a barren environment even for as short a time as 10 days results in an increased incidence of feather picking when they are adult birds. So it is important that even chicks have foraging opportunities with grass, dirt, etc.

Regardless, chickens do well outside on mixed substrates that includes grass and packed earth. At a minimum, substrate can be added to cover a hard floor if outside foraging is not available. Also chicks need room to walk and run freely to ensure proper development of bone and muscle tissue. Chicks do not imprint as do waterfowl but they can become very tame.

Hens should be not kept alone. There are cases where a very tame bird appears happy with human interaction only but in most cases it will be a chronic stress situation leading to immunosuppression and increased susceptibility to opportunistic pathogens.

For the same reason avoid separating poorly birds from the flock unless essential, although if very ill they don't seem to care. Also it may upset the pecking order and they will often be attacked by others when they are returned to the flock. Better to separate in pairs if necessary. This will reduce the problem of bullying when they are re-introduced. In most cases of infectious disease all in-contact birds will have already been infected.

When introducing new birds it is easier to get them accepted into the group with minimum bullying if they are introduced in pairs rather than singularly. It is a common practice to introduce new birds at night to the flock. It appears to be associated with less problems.
If there is a Cockerel in the flock he will control the hens and there will be less inter-hen aggression.

If the social order (pecking order) has been lost then move them around in pairs.

Vent pecking is an abnormal behaviour where some hens cause damage by pecking the cloaca/vent and the surrounding skin and underlying tissue. Often these birds are just found dead and it can be extremely difficult to determine whether the damage around the vent occurred ante-or post-mortem. The finding of dead bird (s) with the vent portion of the body pecked out should raise suspicions of this behaviour. Beware that sometimes predators can do this, rats and stoats especially.

Sometimes birds are presented with wounds around the vent which should also raise suspicion of vent picking.

In general, where this is a chicken-to-chicken problem, birds will be affected one at a time. Predator attack often results in several bodies at a time.

Vent picking is a habitforming behaviour and will continue until the known Vent peckers are removed. If they are not removed other hens will copy.
Vent pecking may develop as an extension of pecking, or as an extension of egg eating as they figure out where the egg comes from, pecking at the vent during egg laying when there will be a partial prolapse creating a "bulls eye" target to pecking at a prolapse, or it may develop from pecking at a red perivent area from diarrhoea. Birds with fewer feathers around the vent appear more predisposed to be victims.

Crowing
This is normal behaviour but a problem in some neighbourhoods.
Rehoming is the best way to solve the problem (however cockerels do help maintain the pecking order among the hens reducing aggression and of course for those interested in breeding it means no more fertile eggs). Bantam cockerels are not as loud and are often tolerated. A good blackout hen house will reduce crowing until they are let out.

Sometimes a low roof has been advocated as they have to hold their heads up for full crow volume.

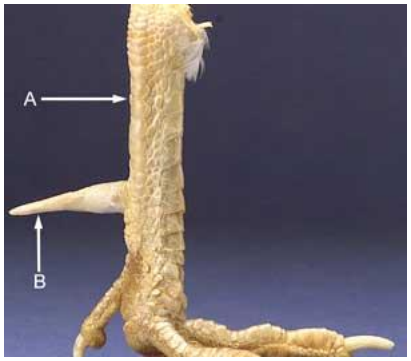
Deslorelin implants anecdotal suggested but no trials in cockerels (there are trials in hens which we will cover in the reproductive section in the 2nd webinar of this series.

Castration possible but it is very difficult to remove all of the testicles and regrowth common in 6 – 12 months. Many also continue to crow.

The bird's voice box, the syrinx is at the bottom of the trachea where surgical access is very difficult even if there was an effective and ethically acceptable surgical procedure.

Spurs
A spur is a bony, sharp conical projection from the tarsometatarsal bone of sexually mature roosters, and in many males of species in the Galliformes.The spur is used as a weapon by the bird, aggressive Roosters that are part of backyard flocks can be dangerous if they have spurs. Injuries to children, pets, and other chickens are likely if your rooster is. Deep wounds can be caused during mating to the hens. Not all breeds have spurs and older hens can sometimes grow spurs. It is surrounded by a cone of horny (keratinized) material. On the male chick, the spur appears as a projection, called a papilla. As the bird matures, the papilla grows larger, hardens, and starts to curve. Increasing the size of your backyard flock to include more hens and roosters may subdue the aggressive rooster because chickens are social birds that need to form a structured social order. Adding more birds to the flock will allow the rooster to establish a pecking order among its own species, instead of among other animal species, including humans.

The photograph shows the leg (A) and the horny layer of the spur (B).



Under this layer is the calcar bone.The sharp trim of the spur which is keratin can be filed down with a dremmel or other grinding instrument. Since the spur continuously grows, like toe nails, this procedure will have to be repeated as the spur tip grows out.

If these recommendations do not improve the situation, then surgical amputation of the spur may be indicated. Since this is an invasive procedure, the humane use of anaesthesia and postoperative pain relief should be included in the surgical protocol.

The spur papilla can be removed when the rooster is still a chick. Radiosurgical removal under a brief gaseous anaesthetic is the best way to remove the spur papilla. Some just snip off the papillae with a scissors but if the germinal cells are not all removed then some sort of spur may still grow but it is unlikely to be dangerous. Since the bony core is not developed at this age, the process is quick, non-invasive, and the bird recovers well. Just apply a local anaesthetic splash block to the surgical site before recovery.

Removing the spur from an adult rooster is a more invasive procedure, because it involves the amputation of bone. The rooster is anesthetized, and a bone saw is used to cut the spur at its base from the tarsometatarsal bone. If possible the skin should be sutured over the wound if this is not possible the wound is allowed to heal by secondary intention and the area kept clean. Postoperative infection of the bone is a possible complication. A peri-operative pain relief plan should be included in the surgical protocol.
Wound dressings may be indicated to prevent infection.

Broodiness is a natural behaviour. Many traditional methods are advocated (with poor success) to bring hens out of broodiness. Broodiness can often go on for longer than the normal incubation period.

A broody hen will sit on the nestbox in an attempt to incubate her eggs. Most birds will attempt to gather other birds' eggs as well. Many hens will lay their eggs in the nest box that the broody is in.

Sometimes the bird will remove herself to a hidden location to attempt to incubate a clutch. Simply removing eggs and bringing the hen back to the flock will rarely work. Most will continue to sit on the empty nest. Some owners will replace the eggs with dummy eggs which are removed at the end of "incubation" (approx 28 days), however many hens will just carry on being broody.

They can be usefully employed to incubate fertile eggs, even of different species.
Birds which are just broody will eat and drink when given this opportunity, I often find they may be stiff, especially the bigger ones, when 1st placed out but will loosen up. Usually they eat some food and have a drink of water and then race back to the nestbox/eggs.

I advise that the broody hen is lifted off the eggs/nestbox twice daily and placing her outside near the food and water. At least this way she will eat and drink more regularly.

In semi-commercial flocks, persistently broody hens may need to be replaced as they don't produce eggs when broody. Broodiness not common in exbats as they the selection process works against broodiness.

There are many traditional methods of bring hens out of broodiness which are often advocated however success is low and hens will eventually come out of broodiness in their own time.

Egg damaging and egg eating is another common behavioural problem that once developed is difficult to cure.
Try to check if it could be vermin damage (e.g. rats).
Are they normal eggs, shell less or thin shelled eggs, or could it be accidental breakage. Some hens will damage their own and others' eggs.
Try to distinguish if egg eating is the problem and not the occasional accidental breakages, or vermin damage (eg rats), Soft/ thin-shelled eggs often break on laying. It may be possible to piece together the broken egg to see what is missing. If soft shell eggs are being produced then try to identify the cause, is it nutritional or a disease process or sometimes the 1st egg laid in a young bird can be soft shelled but this should resolve on its own.

Factors which are regarded as predisposing towards egg eating:

- Excess light in the nesting box- this is easily checked and corrected
- Too little space or overcrowding in the nesting box – these should be of adequate size for the size of the birds and there should also be enough nesting boxes
- Some feel that having the nestboxes too low so eggs are seen can attract the egg-damaging bird

Lack of foraging opportunity:

If available they will spend most of their time foraging.
Some place the artificial eggs marketed to teach chickens where to lay in the nest box. The theory is that they find that they cannot break and eat these and so hopefully they will stop breaking and eating eggs. Artificial eggs are marketed to teach chickens where to lay.
As with vent picking it is usually a few hens that are the culprits. They need to be identified and removed.

Skin
Avian skin is very thin under the plumage but thicker on the feet and around the beak.It contains keratinocytes. Cells that secrete a thin lipid film that helps in the maintenance of the plumage.
The dermis is thinner than mammals, it contains the feather follicles, smooth muscle and a network of nerves and blood vessels.

The subcutaneous layer has loose connective tissue and some adipose tissue but is a much smaller space than in the mammals. Sub-cutaneous injections are more difficult to give and so it is not a useful space to give fluids.

Skin pigmentation of domestic fowl tends to be either yellow pink or white depending on the breed and the presence of carotenoids in the diet. Some breeds such as the silkie has blue- black skin.

Feather loss Caged hens are more likely to have light plumage with large bare patches. Once in a free range environment they will grow a new set of plumage quite quickly. The cause is unknown maybe it is less important in the climate controlled poultry house, they also tend to be heavier than free range birds.

Normally chickens moult once a year usually in the autumn, when moulting they stop laying. The severity of the moult can vary from year to year.
There are featherless areas of skin called aptera that are present in gallinaceous birds and in many other orders of birds. These are useful in the clinical examination and also provide easier access for collecting blood samples or for surgery in contrast with the situation in waterfowl for example where there are no aptera and access to the skin is much more difficult.

The brood patch is an area of skin on the mid-ventral chest between the caudal sternum and pubic bones. In broody hens oestrogen causes feather loss here and the skin becomes thickened and very vascular to provide extra warmth for egg incubation.
Two last general skin points.

Jaundice does not occur as they lack bilirubin. Yellow colour is due to dietary pigments.
Bruising instead of turning black and blue will be a bright green. Birds bruise green because they have biliverdin instead of bilirubin like mammals.

External Parasite Treatments
Diatomaceous earth is used as an insecticide. It has abrasive and absorptive properties. The fine powder absorbs lipids from the waxy outer layer of insects exoskeletons, causing them to dehydrate. Arthropods die as a result of the water pressure deficiency.

Ivermectin is often listed as a suitable insecticide. However there is no data on withdrawal times. It does penetrate into the eggs and has a long half-life. Some have recommended a half life of a minimum of 8 weeks but it would be prudent to advise testing the eggs are free of residues before allowing them to enter the human food chain again.

Pyrethrum extract and the synthetic derivatives e.g. Permethrins used to be available with information on the label for poultry. I used to get clients to purchase these products from agricultural merchants and follow the instructions on the label to get around the withdrawal information I would have to give. However these products appear to have disappeared and pyrethrum products are now only available for environmental treatment

This problem with the availability of suitable medication will make an accurate diagnosis with identification of the potentially pathogenic species even more important. Is this really a pathogen? Do we need to treat? What is the life cycle? Will treatment of the environment be enough?

Feather lice
These are large enough to be seen without magnification. There are many different species that tend to be found in different areas of the bird depending on the species. Common areas are the neck and around the vent. Eggs are laid and cemented at the base of the feather shafts at species specific sites They are not considered pathogenic as they mainly live on feather debris. Healthy birds keep numbers low by removal during preening.

Unhealthy birds sometimes have large numbers present and they are regarded as a marker of poor health also birds with damaged beaks may have larger numbers of these lice as they cannot use their beaks effectively. It is important do a thorough clinical exam to identify the underlying causes. Often, depending on the level of the infestation, it is enough to treat the predisposing cause. White feathers can be stained by the faeces of feather mites and other external parasites

Red mites are one of the most common ectoparasites in backyard poultry. They can cause extreme irritation in some birds but being blood-suckers the most common symptoms are anaemia and debility, especially in young birds where deaths may occur. The anaemia will be seen as a pale comb. They can survive for months between feeds so resting the hen house will not solve the problem.They will not be found on the hens during the day as they hide in cracks and crevices in the hen house by day.Treatment involves thoroughly washing the hen house. If they are in areas where you cannot reach (such as under roofing felt) it will be very difficult to eliminate the infestation.

Pyrethrum powders and permethrin sprays have been described. However, penetration of housing materials is poor and so only effective in limiting mite numbers. Permethrin/ pyriproxifen spray (Indorex, Virbac) has a persistent action so may be more effective overall.

Fipronil spray is very effective against feather mites and lice but is not licensed for use in food-producing species.

Go out at night with a torch to check for mites.This will reveal many small dark mites moving rapidly away from the light

Adults are approx 0.7mm by 0.4mm although when engorged females can measure over a 1mm

Get owners to collect mites as definitive diagnosis is by identification. They may be collected on acetate strips

They will feed on people and can cause localised skin reactions.

Northern Poultry Mite *Ornithonyssus sylviarum i*smore pathogenic than the red mite. Northern mite is not as common as the red mite but is becoming more common.

Similar to red mite this is a blood sucking parasite that does not cause skin lesions. However, unlike red mite these spend their entire life cycle on the bird. There is a more rapid onset of anaemia and debilitation. Affected birds can be very pruritic, continuous restlessness and itch a lot.

Diagnosis is by clinical signs and finding mites on the body of the bird. These should be distinguished from feather mite. Being an obligate on-bird parasite they are much more easily treated and controlled.They can only survive approximately 2 – 3 weeks off the bird.

Mites congregate first on the vent, then on the tail, back, and legs of female birds; they are more scattered on male birds. As the mite population increases, feathers become soiled from mite eggs, cast skins, dried blood, and excrement. The soiling produces the characteristic blackened feathers in the vent area. Scabs also may form in the vent area.

In untreated flocks populations tend to build up to a peak at approximately point of lay and then slowly decrease. Older birds are often regarded as carriers. All birds in the flock will be infected and need treatment.

Again topical products should be effective at control. But will need repeating at 5-7 day interval. The life cycle is completed within 7 days

Epidermoptid mites
Epidermoptid mites are relatively common burrowing mites causing feather loss and irritation on the face and feathered skin of the face and neck("depluming itch") Figs 5 &6
Severe infestations may cause damage to the growing tissue of the beak.
Feather loss and skin irritation (and sometimes secondary bacterial infection) will result
Diagnosis is by typical clinical signs and skin scrape or biopsy.
A different species *Neocnemidocptes gallinae related to the scaly leg mite* causes a syndrome similar to that of the depluming mites.

Therapy of non-food species using ivermectin given percutaneously, orally or intra-muscular at 200µg/kg every 2 weeks on 3 occasions is very effective however a withdrawal time cannot be given for ivermectin. It may be possible to get round this by testing for drug residues after an 8 week period. All in-contact birds should also be treated even if showing no signs.

Scaly leg mite *Knemidocoptes mutans*
This is a very common skin ectoparasite. You get the formation of extensive crusts on the scaled skin of the feet and legs. There are varying degrees of irritation. Sometimes they don't appear irritated at all. Occasionally there is secondary bacterial infection and pododermititis can also be a sequalae.

It should be in the differential list if the typical clinical signs are present and confirmed by identifying the mites on skin scrape.

It is also important to note that birds may carry these mites without clinical signs and that clinical signs may represent immunosuppression or presence of an underlying stressor. In most cases only the occasional bird will have scaly leg but they will all be infected. The bird with clinical signs usually also has other problems.

In older hens you need to distinguish from the hyperkeratosis of the feet and legs which is seen as a normal aging process. In that case the scales will have their normal shape whereas on these feet you cannot make out individual scales as they are crumbly.

These mites look different to others and can easily be missed in a smear if you are unfamiliar with their appearance. They are a round shape and have very short limbs.

Traditionally petroleum jelly or liquid paraffin were smeared over the legs to block burrows and suffocate mites and this may be sufficient in mild infestations. This is repeated weekly until the crusting has gone.

Ivermectin is an effective treatment in non-food producing species. All in-contact birds are treated. Many cases resolve completely with treatment although some scarring may remain.

Bacterial skin infections are 2nd with **Erysipelothrix rhuiopathiae** an exception as it can cause skin lesions as a primary infection (actually systemic) There can be areas of dark thickened skin over any part of the body. It is a gram positive rod.

Erysipelothrix is very susceptible to penicillins. Amoxicillin is a good treatment choice. There is a vaccine available but only available for turkeys in this country.

Mycobacterial dermatitis is described but is very rare. Nonetheless this may be suspected in any case of granulomatous dermatitis and it is advisable to biopsy for this in such cases owing to the zoonotic nature of such infection.

Abscesses can occur after injury or skin penetration. Open and flush out and treat as an open wound, chlorhexidine or povidine iodine.

Flavus is a fungal skin infection seen in chickens.
White, powdery spots and wrinkled crusts along with scabs are seen on the comb and wattles and less commonly on the legs.

Feather loss can also be seen on the head. The skin becomes thick and crusty with a honeycomb appearance. This is caused by superficial invasion of the stratum corneum by the fungus resulting in the hyperplasia and hyperkeratosis that is seen as the thick crusty scaly skin. Deep infection may result in permanent damage to the eyelids and /or beak. Spread is by direct contact and also by fomites.

Diagnosis by microscopy as *Trichophyton megnini* doesn't grow well on conventional ringworm media.

Take skin scrapes for cytology or a skin biopsy for histology. It does not fluoresce under ultra-violet light.

Topical antifungal agents such as Povidone iodine can be used to treat this infection. Povidone iodine is fungicidal and sporicidal (as a 1% aqueous solution). Povidone acts by slowly releasing iodine to tissues. It has prolonged activity (4–6 hours). Povidone iodine also has mild degreasing and debriding activity and it can also be irritating to the skin so care. Enilconazole topically is an alternative treatment.The antifungal agents more commonly used in companion animals are not allowed in food producing animals.

Fowlpox
Poxvirus is an occasional finding in poultry.Transmission is by biting insects with the result that most of the lesions are seen on featherless areas, around the eyes nares on the comb, wattle and legs. These consist of either raised plaques or pale yellow scabs that may coalesce together.

An alternative means of spread is by inhalation which leads to the diphtheritic form of the disease which has a much higher mortality rate. The cutaneous form is self-limiting and the scabs will drop off after several weeks. Diagnosis is by finding of typical lesions and biopsy.

There is no direct therapy. However lesions are usually self-limiting and there is little scarring unless there is secondary infection. Topical antibiotics are useful especially if lesions are around the eyes. Attention must be paid to prevent spread to in-contact birds and strict barrier nursing is required. In severe cases with extensive lesions around eyes, nares and mouth supportive care may be required to help the bird feed. Vaccination is available but is generally not indicated as the infection is not very common nor particularly severe in most cases.

Marek's disease occasionally may present as swelling of individual feather follicles.
The virus replicates in the feather follicles and persists in the feather dander. Sometimes these swollen feather follicles can become roughened and ulcerated. These ulcers may develop secondary infections, exacerbated by the immunosuppressive nature of Marek’s disease.

Highly Pathogenic Avian Influenza
It is a highly lethal, systemic disease which is listed by the OIE (The World Organisation for Animal Health) as a most serious disease and therefore in most countries is a notifiable animal disease.
It is mentioned here in the skin section because along with the high mortality and respiratory signs, oedema of the head and face can be seen with subcutaneous haemorrhage with cyanosis of the skin, especially the head and wattles. Although the haemorrhagic lesions typically target the non-feathered skin, here severe cutaneous hemorrhages are observed on the ventral abdomen. The eyelids of birds with HPAI are swollen and unable to fully open die to the diffuse oedema. The wattles and comb are often swollen and discolored red to purple with congestion and cyanosis.

Gastro-intestinal disease
To facilitate weight reduction for flight, birds have lost teeth. As a result the digestive system is adapted to process unmasticated food. Food is softened and stored in the crop (*ingluvies*) which is just above the thoracic inlet and then passed down to the proventriculus and into the ventriculus (gizzard) where it is mechanically ground down. Herbivorous birds like the chicken also have a caeca for microbial breakdown of cellulose.

Paratyphoid,Fowl Typhoid, Rotavirus and Pullorum Disease are common differentials in birds ages from hatch to 2 weeks old with diarrhoea.

Coccidiosis, Dysbacteriosis, Paratyphoid, Fowl Typhoid, Rotavirus, *Capillaria,* Blackhead, Ulcerative Enteritis, Pseudotuberculosis, Gumboro Disease and Infectious Bronchitis are common differentials for diarrhoea in birds from 2 to 16 weeks of age.
Capillaria, Dysbacteriosis, Fowl Typhoid, Blackhead, *Chlamydophila,* Pseudotuberculosis, Tuberculosis, *Brachyspira,* Infectious Bronchitis and Lymphoid Leukosis should be on the differential list for adult birds with diarrhoea.

Capillaria also sometimes called Hairworm or Threadworm in the hobbyist literature.
Some Capillaria spp may use earthworms as intermediate hosts, though the parasite's life cycle may be completed directly. The worm is approximately 2cm in length (1.5cm-8 cm) and burrows into the mucosa which is how it causes damage.
Affected birds are often inappetant and lose weight. In young birds severe burdens can lead to death.

The most common findings on post mortem are inflammation and thickening of the small intestine in chickens. In severe cases the result is the formation of a diphtheritic membrane. Upon close inspection (often aided by a hand held lens) the adult worms may be seen but mucosal scrapings should be taken for examination using a microscope.
If treated in the early stages of infection the prognosis is excellent.

Diagnosis in the living bird is based on faecal nematode egg counts where *Capillaria* eggs can be clearly identified by their elongated shape and bipolar plugs.
Due to their pathogenicity, the presence of any *Capillaria* eggs should constitute grounds for treatment. (See below for the treatment of nematodes). Flubendazole is the only licenced treatment.

I advise yearly faecals unless there is a history of internal parasites. Also if there are new introductions it's a good plan to do a faecal exam.
Routine treatment should not be done. Many intestinal nematodes are non- pathogenic and there are potential side effects of the treatment.
Traditionally it has been recommended that only when the worm egg count exceeds 400 eggs/gram of faeces that worming should take place.

There is an argument that the presence of any *Capillaria* eggs are grounds for treatment because of their pathogenicity compared with other worms.
Rotating the pasture will reduce the egg burden however some eggs are long lived and they can be carried by intermediate hosts such as earthworms. UV light is good at destroying eggs and as such keeping the pasture short will allow UV light to reach the soil helping to destroy worm eggs. Applying agricultural lime will also help control pasture burdens.

Coccidiosis
Different species of coccidiosis will vary in both their predilection site and pathogenicity.
There are no maternal antibodies against coccidiosis found in the young chick. As such young birds are vulnerable to clinical disease if the level of challenge is great enough until the birds have developed sufficient immunity.

Usually this takes up to three months of age. Ex-battery hens are also particularly vulnerable to infection since they have spent the preceding year separated from their droppings and also need to develop resistance.

Coccidiosis tends to be a flock problem and often the first birds in a flock infected do not show clinical signs but instead act as amplifiers as large numbers of oocysts can be passed before natural

immunity develops.The large numbers of oocysts then build up in the environment which go on to infect the rest of the flock leading to clinical signs.
Infection occurs through the ingestion of sporulated oocysts. Oocysts passed in droppings can sporulate in as little as 48 hours in warm (25-30°C) humid conditions. The chicks will need the warmth but keeping the substrate dry especially around water containers will reduce the challenge.

As the parasite undergoes several rounds of asexual reproduction followed by a round of sexual reproduction (to produce many more oocysts) the intestinal wall is damaged. This damage can lead to blood loss, bacterial overgrowth, necrotic enteritis, malabsorption and even septicaemia.

Affected birds are often dull, inappetant and are seen hunched up with ruffled feathers due to abdominal pain. They will have diarrhoea. They don't always have enough intestinal pathology to produce a bloody diarrhoea. But if there is bloody diarrhoea this will lead to anaemia and a pale comb and wattles will be seen.
Diagnosis is based upon clinical signs and history, together with high faecal oocyst counts (over 50,000 oocysts/gram of faeces).

Treatment of coccidiosis involves two components:
One to control the coccidia with an anticoccidial agent such as toltrazuril or amprolium (follow manufacturers recommendations).
It is also important to control secondary bacterial growth concurrently using antimicrobials such as tylosin or amoxicillin. When coccidiosis damages the intestinal wall leading to secondary bacterial overgrowth manifesting itself as a necrotic enteritis.
It is very important to keep the bird hydrated and comfortable. Always ensure the bedding is kept dry to slow down oocyst sporulation.

Prognosis:
The prognosis for early cases and the flock is good but for severely affected individuals treatment may be too late.

Prevention:
Prevention of clinical coccidiosis but not exposure is key since the birds need to develop immunity.
Good hygiene is the first step as oocysts can persist for up to several years between batches of birds. After removing the bedding always use a detergent to remove dirt.

Next after letting the pen dry use a disinfectant with anticoccidial activity-
If a disinfectant doesn't state that it destroys oocysts then it should be assumed that it will not.
While the birds are being reared anticoccidial agents such as toltrazuril are used by some preventatively or a coccidiostat is often added to the feed.

However for the back yard flock the small numbers and small size of the brooding area should facilitate proper cleaning of the area between batches and greater ease in keeping the substrate dry.

A live attenuated vaccine is available for chickens which can be given in the first week of life.
However for the vaccination to be successful the environmental conditions must be conducive for the sporulation and cycling of the vaccine strains of *Eimeria*.

Paratyphoid
These are the salmonellas that we associate primarily with food poisoning outbreaks.
S. typhimurium is a common isolate but there are other Salmonellae which are collectively covered by the term paratyphoid.

Most salmonellae are spread by fomites, latently infected carriers, vertical transmission, rodents, wild birds and contaminated feed/water.

Clinical signs are mostly restricted to young birds. Vertical transmission can lead to poor hatchability. Young chicks that do become infected are often dull and huddled up and with faecal pasting around the vent due to the diarrhoea. Blindness can occur due to occular lesions.

When birds older than one month of age are infected, the majority of these birds will clear this infection within sixty days. However a small number of animals remain carriers and act as reservoirs within a flock.

Post mortem findings include yolk sac infection, septicaemic signs (a reddened congested carcase, inflamed liver, enlarged spleen and inflamed kidneys), widespread necrotic foci, pericarditis, peritonitis, haemorrhagic enteritis, caseous typhlitis and occular lesions.
Diagnosis is based upon bacteriological isolation and subsequent serotyping.
Treatment with antimicrobials may be attempted although all of the survivors should be regarded as carriers.

Prevention is based upon purchasing new birds from disease free flocks, controlling vermin, wild birds and flies along with restricting the movement of fomites. Always ensure clean feed is sourced (commercial mash and pellets are unlikely sources of the infection as they are routinely tested).

Competitive exclusion using probiotics may also be considered but must be given before infection (before the gut becomes populated with bacteria) to day-old chicks.
Vaccines are available in both live and inactivated forms. Vaccination should be initiated before the birds come into lay and boosted annually.

In the UK keepers with more than 350 laying hens or those wishing to sell their eggs through local shops must register with the National Salmonella Scheme which will involve routine monitoring.

Fowl Typhoid
S. gallinarum is a rarely isolated but pathogenic *Salmonella* of poultry, with the ability to infect all poultry species of all ages. The bacteria are shed in the droppings from latently infected birds. Infected hens can transmit the pathogen vertically. Vermin and fomites have been implicated in the epidemiology of fowl typhoid.

Clinical signs
Vertical transmission results in poor hatchability. Those birds that do hatch are often weak showing signs of dyspnoea and a yellow/white diarrhoea. Adult birds may appear depressed (hunched up with ruffled feathers), have respiratory signs, diarrhoea, pale comb and wattles, they are pyrexic and show weight loss.

Post mortem findings:
Findings include an enlarged, friable, congested inflamed liver (often with a bronze hue), an enlarged spleen and inflamed kidneys, catarrhal enteritis (often with bilious staining), milliary necrosis of the liver, intestine, heart and pancreas and pericarditis.

Diagnosis requires isolation and identification. A tentative diagnosis can be made based on history, clinical signs, mortality and lesions, along with serologic findings. Clinical signs and post mortem lesions are not diagnostic.

Treatment is based upon using antimicrobials and supportive therapy.
The prognosis is moderate with between 10% and 50% of birds dying, however most survivors will remain carriers.

Prevention is based upon good hygiene and biosecurity along with controlling pests and sourcing new stock from disease free flocks. Serology and bacteriology can be used to identify carriers. There are currently no licensed vaccines in the UK.

Salmonella Pullorum also called Bacillary White Diarrhoea or Pullorum Disease.
The bacteria are predominantly transmitted vertically from infected hen through the egg
Vertical transmission causes poor hatchability and the chicks that hatch are weak and depressed, huddling together with drooped wings.They will have a white diarrhoea and respiratory distress. Infected chicks can then go on to infect their flock mates.

The Post mortem findings are non-specific and the diagnosis is based on bacteriology.
Findings may include retained infected yolk sacs, inflammation of the liver (occasionally with haemorrhages), enlarged spleen and inflamed kidneys, peritonitis, caseous caecal cores, pericarditis and widely distributed necrotic foci.

Antimicrobial therapy can be attempted but the prognosis is poor and treatment will not eliminate the pathogen, only suppresses it.
Mortality can be variable from zero in birds over one month of age up to 100% in younger birds.The condition is prevented by sourcing new birds from disease free flocks. Bacteriology and serology can be used to detect carriers.

Rotavirus
Rotaviruses can infect all ages of chickens.
Spread is via the faeco-oral route including the surface contamination of eggs leading to early infection of chicks.

Infection can cause dullness, birds to be hunched up with ruffled feathers, inappetance, frothy yellow diarrhoea, dehydration and in some cases death.There are no pathognomonic post mortem findings. The caecae will be distended with yellow frothy contents.

The diagnosis is made using either ELISA kits, PolyAcrylamide Gel Electrophoresis (PAGE) or by PCR.

There is no specific treatment but maintaining hydration is crucial. Antimicrobials such as amoxicillin can be used to prevent secondary bacterial infection. The prognosis is fair but surviving birds can be left stunted. Prevention is based upon good hygiene especially in relation to hatching eggs. There is no licensed rotavirus vaccine.

Dysbacteriosis is a widely used term to describe a non-specific diarrhoea involving a disruption/imbalance to the normal intestinal flora It is more common in free ranging birds.
This disruption is usually caused by eating something inappropriate (or something gone off with mycotoxins) or an underlying pathogenic agent such as coccidiosis.
These birds are presented with diarrhoea but are otherwise bright and alert with no other clinical signs. Severe cases may progress to a necrotic enteritis.

On Post mortem:
Affected birds often have watery intestinal and caecal contents with a pale flaccid distended intestine.

Diagnosis is frequently based upon apparently healthy birds being presented with diarrhoea. Faecal cultures are useful to rule out other differentials but will not confirm the diagnosis of dysbacteriosis.

It is important to rule out other causes of chronic diarrhoea in the individual bird as in many cases of chronic diarrhoea there is an underlying problem. Most uncomplicated cases will often spontaneously recover.

Clostridium Colinum Ulcerative Enteritis (Quail Disease)
Ulcerative enteritis is a rare condition in a wide range of avian species. Ulcerative enteritis was first reported in quails. In chickens birds between 3 – 10 weeks of age are most commonly affected.

The condition is caused by *Clostridium colinum*. Infection is through the faeco-oral route though it needs a co-pathogen such as coccidiosis or other disease to enable the *Clostridium colinum* to cause disease. Infection usually leads to sudden death of birds in good condition but in some cases dullness and diarrhoea may be the main presenting signs.

Post mortem findings include ulceration of the intestine occasionally with the formation of a pseudomembrane. Some will also have splenomegaly and large areas of focal hepato-necrosis. Whilst post mortem lesions are not pathogonomic they are suggestive. Bacterial culture is needed for a definitive diagnosis.

Treatment involves treating/removing any stressors, maintaining the bird's hydration status and using antimicrobials. Amoxicillin and tylosin are common choices and are given for five days.

If detected early enough the prognosis is good for the flock, but is guarded for individuals.
Prevention involves controlling underlying factors such as coccidiosis and immunosuppressive agents together with good hygiene and biosecurity.

Gumboro Disease (Infectious Bursal Disease)
Gumboro disease is a birnavirus which attacks the bursae of fabricius. Infection tends to occur from 3-6 weeks of age when the levels of maternal derived antibody have fallen. The infection will damage B-cells causing immunosuppression. The virus is shed via the faeces and can survive in the environment for several months.

In acute cases birds will be dull, hunched up (with ruffled feathers) along with having a white watery diarrhoea. Many of these birds will rapidly die. In cases where the maternal derived antibody levels are high the birds will show poor growth and an increased predisposition to other diseases.

Post mortem examination will often reveal bursae filled with pus or blood which are regarded as pathognomonic changes for this condition.

Whilst post mortem findings are diagnostic in acute cases, for birds with subacute infection serology, bursal histopathology or PCR can be used to confirm clinical suspicion.Treatment involves supportive care and potentially using antimicrobials to control secondary bacterial infection.

The prognosis is variable depending on the levels of maternal derived antibody present.
Prevention is based upon vaccinating the parent flocks with a live vaccine at approximately twenty eight days followed by a killed vaccine at sixteen weeks to provide adequate levels of maternal derived antibody. This should be followed by vaccination of the chicks at approximately twenty eight days of age.

Chlamydia
There is some confusion about the correct name for this organism. About 8 years ago the name was changed from *Chlamydia to Chlamydophila ,* but 3 years ago after dna sequencing evidence, it was renamed Chlamydia i.e. the original name.
It is more commonly regarded as a respiratory disease but we mention it here because it can present as a diarrhoea.It is also a potential zoonosis.

C. psittaci is not a common pathogen of backyard chickens and if infected they are often asymptomatic while other poultry species incontact may have serious disease (esp duck, turkeys and pigeons).

Wild birds are the most common source of infection with the bacteria being shed in the faeces and respiratory secretions of carriers. Once infected they should be regarded as lifelong carriers.

Symptoms seen can include pyrexia, nasal discharge,conjunctivitis, respiratory distress, dullness, inappetance, biliverdinurea, weight loss, Gelatinous diarrhoea +/- blood and a drop in egg production.
Post mortem findings include pericarditis, pneumonia, air sacculitis, serositis, hepatomegaly and splenomegaly and both may have areas of necrotic foci/focal necrosis.
Whilst clinical signs can be suggestive, PCR testing (from cloacal swabs), histopathology (of the heart, spleen, liver and kidney) to stain for elementary bodies or retrospectively paired serology samples are necessary for a definitive diagnosis. Detecting asymptomatic carriers is difficult.

This is a potential zoonosis and in some countries it is a legal requirement to report all cases to the authorities. Treatment involves a 45 days course of doxycycline.
Most recover however there is no guarantee of elimination of the organism.

Pseudotuberculosis (Yersiniosis)
Yersinia pseudotuberculosis is a rare pathogen of domestic poultry. Turkeys are much more susceptible than chickens. The infection is spread primarily through the faeco-oral route and

occasionally through cutaneous abrasions. Wild birds and rodents are usually responsible for spreading this bacteria.This organism has zoonotic potential.

Once the organism gains entry into the body it enters the blood stream to cause a bacteraemia and subsequently septicaemia. Many birds die peracutely but in some birds the disease can have a chronic course with diarrhoea, weight loss and lameness being common clinical signs.

On post mortem examination hepatomegaly, splenomegaly and a congested bright red carcase are the most common findings. Inflammation of the digestive tract and caseous TB-like nodules may be found in the bird's visceral organs.
No post mortem lesions are diagnostic and bacteriology is needed for confirmation.
There is a poor prognosis when clinical signs are present

Avian Tuberculosis
Avian TB is caused by *Mycobacterium avium*. It is a relatively rare condition of non-aquatic fowl but is much more common in ornamental waterfowl. It is more common in older birds. The primary source of infection is through the faeco-oral route.

Wild birds are often carriers and faecal contamination of the soil and water (especially at a land water interface) is an important risk factor for infection Its very resistant in the environment and can survive in the soil for years.

Once infected the course of the infection is slow. It will take up to several months for clinical signs to develop. Avian TB leads to the formation of tubercle-like granulomas throughout the body. The clinical signs can be variable depending upon the location of the lesions but the most common finding is chronic weight loss and other non-specific signs. Diarrhoea can also be a symptom.

Post mortem examination findings include the finding of a thin carcase with white/yellow/grey granulomas throughout the internal organs and bones. Clinical signs and gross post mortem changes are suggestive of the diagnosis but the presence of large numbers of acid fast tubercle bacilli in the lesions help confirm the diagnosis. PCR tests are also available to confirm the presence of the acid fast organism. The tuberculin test may be used to confirm diagnosis in the live bird. This involves injecting 0.05ml of Avian Tuberculin intradermally into one wattle and checking the reaction site 48 hours later. The injected wattle should be compared to the un-injected wattle for comparison with any swelling in the injected wattle indicating a positive result. On post mortem examination suspected lesions can be stained to look for acid fast bacteria.

Treatment is very difficult and due to the risk posed to other birds in the flock, euthanasia should be considered. It should also be remembered that *M. avium* is a potential zoonosis.

Brachyspira (Intestinal Spirochaetosis)
Brachyspira Spp (*B. pilosicoli* and *B. intermedia*) are a genus of anaerobic bacteria
The bacteria are transmitted by the faeco-oral route from infected birds. It mainly affects laying hens and most common signs are chronic diarrhoea and a drop in egg production.
The diarrhoea is often a mucoid, frothy yellowish-brown diarrhoea in about 5-20% of effected birds together with mild weight loss. Most infected birds do not appear unwell.
Diagnosis is made on clinical signs confirmed by faecal cultures or PCR (which can be used for both confirmation and speciation).

Appropriate antimicrobials such as oxytetracycline are usually effective treatments. Attempting to prevent contact with the faeces of wild birds and mammals will reduce the risk of infection.

Lymphoid leukosis
Like Mareks disease, lymphoid leukosis causes both immunosupression and neoplasia. However unlike with Mareks disease the nervous system is spared from neoplastic invasion.
Lymphoid leukosis is caused by a retrovirus and is transmitted via biting insects, needles and vertically. Not all exposed birds succumb to the infection.

The tumours often form in the visceral organs, liver, spleen, kidneys, intestines and bursa and in some cases will form in the bones of the legs.

Affected birds show weight loss, poor growth and an increased susceptibility to other infections, including gastrointestinal infections. If the tumours invade the bones the legs may become bowed.

Post mortem lesions include neoplastic invasion of the visceral organs but histopathology will be required to differentiate lymphoid leukosis related tumours from those caused by Mareks.

Diagnosis is usually based upon histopathology.There is no treatment and the tumours are eventually fatal. Prevention is based upon sourcing disease free stock and controlling biting insects.

Clinical approach to Diarrhoea
As in any other species supportive care is very important. One of the prime aims is to keep the bird hydrated. Fluids should if possible be given orally. Parenteral fluids will be essential in some cases. Nutritional support can be tube fed to anorexic birds. Give small volumes frequently. Use either one of the commercially available products or powder up their pellets to make a liquid food that will flow through the crop feeding tube.

Thermal support may be needed in the early stage of treatment to prevent hyopthermia. If a critical care cage is not available then this can be improvised.
Pasty vent is a traditional poultry term for faecal material accumulation around the vent stuck to the skin and the feathers. If can dry in a hard concretion which can block the vent especially in young chicks and also will act as a magnet for flies to lay their eggs with the risk of fly strike. Keeping this area clean is also important.

Check what the bird is being fed, is it free ranging, and also check in regard to treats. Ingesting inappropriate items can lead to a disruption to the normal gut flora.

Many free range birds have loose droppings that spontaneously resolve. In cases where there are no other clinical signs it is worth monitoring the bird/s over a few days first

Take a fresh faecal sample and using the McMaster method Carry out a worm egg/ coccidial oocyst count.

>400 worm eggs per gram or the presence of any *Capillaria* eggs indicates treatment is necessary.

>50,000 coccidial oocysts per gram is likely to be pathogenic faecal culture/virus testing. Gumboro (AGIDT) Agar Gel Immunodiffusion Test Rotavirus (PAGE) Polyacrylamide Gel Electrophoresis.

If it is an individual bird the problem might not be an infectious agent and if a thorough clinical examination does not reveal the cause then continue to work up the individual animal blood samples for haematology/biochemistry +/- diagnostic imaging.

If antibacterials are needed tylosin and amoxicillin are common choices.