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Poisoning In Pigs

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Poisoning In Pigs

Mycotoxin poisoning of pigs:

Mild cases of mycotoxin poisoning in pig normally goes un-noticed in most pig farms.

Mycotoxins are poisons produced by moulds growing in feedstuffs. Moulds like ergot can grow on grains and produce mycotoxin before harvest. Other moulds infect grain before harvest but produce most mycotoxins during storage. The moulds that produce mycotoxins are not always visible, but feedstuffs that become visibly mouldy during storage are very likely to reduce productivity.

There are several key questions that should be asked when faced with options on either the purchase of mould-infected (often weather damanged) grain or the use of feed that has become mouldy in storage. These include:

- Whether mycotoxins are present in concentrations sufficient to affect pig health and performance;
- Whether the palatability and nutrient content has been altered for better or worse;
- Whether the lower price of the grain or other feed component compensates for these effects and the risk involved.

Many moulds cause only a slightly reduced growth rate or poor feed conversion, but some mycotoxins with more drastic effects are aflatoxins, ochratoxins, zearalenone, trichothecenes (deoxynivalenol, DON), fuminisins and ergot alkaloids. These mycotoxins do occur in particular regions and in particular situations, so that prior knowledge of these circumstances will greatly reduce the risk of poisoning.

Common symptoms of mycotoxin poisoning in pigs are:

1. **Aflatoxins:-** Pigs go off their feed and some may die, some are anaemic (pale) and jaundiced (yellowish in color). Characteristic damage is caused to the liver.

2. **Ochratoxins:-** Ochratoxin A causes kidney damage. Depressed appetite and reduced growth may result.

3. **Zearalenone:-** Has some properties of the female hormone, oestrogen. When fed to female grower pigs, zearalenone causes swelling and reddening of the valva similar to that seen at natural heat (oestrus). This can progress to straining and prolapse of the rectum and vagina. Also causes slight development of the teats of gilts and occasionally swelling of the prepuce of boars.

4. **Deoxynivalenol (DON):** - Pig initially reject feed and thereafter eat barely sufficient for survival. If the pigs are hungry when the feed is first offered, they may eat and then vomit, which is why DON is also called vomitoxin.

5. **Fumonisins:-** produced before harvest. Drought stress is a contributing factor in increased contamination. Have heen associated with pulmonary oedema (fluid in the lungs) in pigs.

6. **Ergot alkaloids:-** The most dramatic effect of these substances is cessation of milk by sows, leading to starvation of piglets.

Rodenticide Poisoning:

Many poisons have been used against rodent pests in grain and feed stores. Farm animals, pets and wildlife often gain access to these poisons via the baits or the poisoned rodents.

The most commonly is anticoagulant rodenticides like warfarin. Intoxications in pigs have resulted from contamination of feed with anticoagulant concentrate, malicious use of these chemicals and feed mixed in equipment used to prepare rodent bait.

Clinical signs of anticoagulant rodenticide toxicosis generally reflect some manifestation of hemorrhage, including anemia. Signs dependent on hemorrhage, such as weakness, ataxia, colic and polypnea, may be seen. Depression and anorexia occur before bleeding occurs.

Insecticide and acaricide toxicity:

Poisoning by organic insecticides and acaricides may be caused by direct application, by ingestion of contaminated feed or forage treated for controlling of plant parasites, or by accidental exposure.

Among the insecticides and acaricides, organophosphates (OP) are a major cause of animal poisoning. They vary greatly in toxicity, residue levels and excretion. Many OP have been developed for plant and animal protection, and in general, they offer a distinct advantage by producing little tissue and environmental residues.

In general, OP pesticides have a narrow margin of safety. Signs of OP poisoning include hypersalivation, frequent urination, diarrhea, vomiting, colic and dyspnea due to increased bronchial secretions, broncho-constriction, muscle fasciculations and weakness. Affected animals also show nervousness, seizures and even convulsions.

Severity and course of intoxication is influenced principally by the dosage and route of exposure. In acute poisoning, the primary clinical signs may be respiratory distress and collapse followed by death due to respiratory muscle paralysis.

Dioxins and their effect on human health:

Dioxins are environmental pollutants, mainly by products of industrial processes like smelting, chlorine bleaching of paper pulp and the manufacture of some herbicides and pesticides. In terms of dioxin released into the environment, waste incinerations (solid waste) are the worst culprits, due to incomplete burning.

Although formation of dioxins is local, environmental distribution is global. The highest levels of these compounds are found in soil sediments and food, especially dairy products, meat, fish and shellfish. Very low levels are also found in plants, water and air.

Extensive stores of PCB (polychlorinated biphenyls)-based waste industrial oil, many with high levels of PCDFs, exist around the world. Long term storage and improper disposal of

this material may result in dioxin pollution of the environment and the contamination of human and animal food supplies.

Incidents of dioxin contamination reported include:

1. Higher than permitted levels of dioxin in milk in 2004 in Netherlands, traced to a clay used in the production of the animal feeds.

2. Elevated dioxin levels were detected in animal feed in the Netherlands in 2006 and the source was identified as contaminated fat used in the production of the feed.

3. In 2007, the European Commission issued a health warning to its Members States after dioxins were detected in a food additive – guar gum – used as a thickener in small quantities in dairy, dessert or delicatessen products. The source was traced to guar gum from India that was contaminated with pesticide, pentachlorophenol which contained dioxin as contaminant.

4. In 2008, it was suspected that Irish farmers use of inappropriate type of fuel oil in a burner that dried out-of-date (expired) bread, dough and confectionary. Vapour from the burning oil worked their way into the food, resulting in dioxin levels 80-200 times above legal limits. This, when used in feed for finishing hogs, resulted in high residue levels of dioxin in the pork.

Being highly lipophilic, dioxins dissolve in fat. They need to be transformed in the liver to become water soluble before they can be excreted. However, dioxins are metabolized slowly and therefore tend to bio-accumulate, especially in fat and in the liver. In pigs, long term consumption of feed contaminated with dioxins, considerable quantities can be accumulated in the pork when slaughtered. The dioxins will find their way into the system of the people consuming the pork.

In human, in addition to cancer, exposure to dioxin can also cause severe reproductive and developmental problems. Dioxin is well known for its ability to damage the immune system and interfere with hormonal system.

Dioxin exposure has been linked to birth defects, inability to maintain pregnancy, decreased fertility, reduced sperm counts, endometriosis, diabetes, learning disabilities, immune system suppression, lung problems, skin disorders, lowered testosterone levels and much more.

Prevention and control of dioxin exposure in pigs:

- Feed only diets compounded from pesticide free grains and other feedstuffs.
- Do not use re-cycled cooking oil
- Do not use biscuit meal (out-of-date biscuits and bakery goods from food industry) which may be dried using non-feed grade burner oil.
- Do not use feed ingredients which may contains high level of pesticide residues