Animal Nutrition Association of Canada

REDUCING THE IMPACT OF ERGOT IN LIVESTOCK FEED

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Contents

Introduction

Ergot is a term that describes the sclerotia (also known as ergot bodies) produced by fungi in the *Claviceps* genera, particularly *C. purpurea*. Several cereal grain species are vulnerable to ergot infection, producing ergot bodies that vary in size depending on the crop host, ranging from a few millimeters to greater than 4 cm in length (1). These sclerotia serve as intermediary bodies that form by replacing seeds in susceptible grass species after infection and facilitate reproduction upon germination the following growing season. Proliferation of the fungi is caused by 1) the wind-assisted transfer of spores from germinated sclerotia to flowering host plants, and 2) the spread of conidia produced during the honeydew stage by insects to surrounding susceptible plants (2). The prevalence of ergot infections can be influenced by climatic factors such as cool moist conditions, specifically during the flowering phase of host plants (2). Consequently, many regions in Canada have observed higher rates of ergot in recent years. While ergot bodies can be removed during seed screening and cleaning processes (i.e. colour sorting, gravitational separation), their presence in these grain screenings still presents a health risk for livestock that may consume these screenings. Toxic alkaloids produced by the fungi are stored in the ergot bodies. When fed in sufficient concentrations, these alkaloids can produce neurotoxic and/or vasoconstrictive symptoms in livestock. Since low ergot alkaloid concentrations (levels not exceeding current recommended maximum limits) may cause adverse health or performance effects in most livestock species (3, 4), exercising proper sampling technique prior to analysis of ergot alkaloid concentration is critical to minimize the risk of ergot toxicity. While much has yet to be learned regarding the effects of ergot alkaloids on animal performance, research is presently underway to examine current recommended maximum ergot concentrations for specific species as well as several preventative strategies to minimize their risk in feed.

History and Background of Ergot

Perhaps the earliest appearance of ergot toxicity on record was observed in humans after consuming bread that had been baked with infected rye grain. Although mistaken for witchcraft or insanity at the time, it is now believed that these cases could be attributed to hallucinations and convulsions induced by certain ergot alkaloids. In fact, the geographic location of the Salem Witch Trials during the late-17th century represents a region that may have favoured ergot growth and has led many to believe that their behaviour was a result of ergot poisoning (5).

In addition to the neurological manifestations, the vasoconstrictive action of ergot alkaloids has also been documented throughout history. Constriction of blood vessels resulting from ergot toxicity leads to a tingling or burning sensation in mild cases for humans – this has been referred to as St. Anthony's Fire (6). The vasoconstrictive properties of ergot alkaloids have also been utilized in pharmaceutical applications. Ergot was used during the Medieval Ages as a treatment to hasten labour and prevent post-partum bleeding (7). Several countries also continued using ergot until the mid-19th century as a treatment for "vascular headaches". This application, however, is currently limited due to the inherent qualitative variability that exists between ergot extract sources (8). Clinical cases of ergot toxicity have been observed in both humans and animals, with an increased incidence in livestock in recent years, presumably due to changing environmental conditions. One of the first well-documented outbreaks of ergot toxicity in cattle occurred during the early 1980's in Australia (9), although incidences of ergot in livestock feed certainly occurred before this case.

Life Cycle and Susceptible Plants

In addition to containing harmful alkaloids, ergot sclerotia also serve as a means to spread the fungi and to continue their life cycle season after season. Once the dark ergot sclerotia have formed to replace kernels of seed on an infected plant, they tend to dislodge and fall to the ground. These sclerotia can range from the same size as healthy kernels to several times larger, reaching up to 4 cm in some grass species (1). During the spring season, ergot sclerotia will germinate in the presence of adequate moisture and produce spores which target host plants. Their growth is augmented with the assistance of wind or transport by insects. Moist, cool and cloudy conditions, particularly when a plant is flowering, will favour the incidence of ergot infection by extending the duration of the flowering stage and thus enabling more opportunity for infection to occur. After a plant has become infected the affected seed ovary will begin to enlarge and harden, ultimately forming a mature ergot body (2). The complete life cycle of ergot fungi is illustrated in Figure 1.

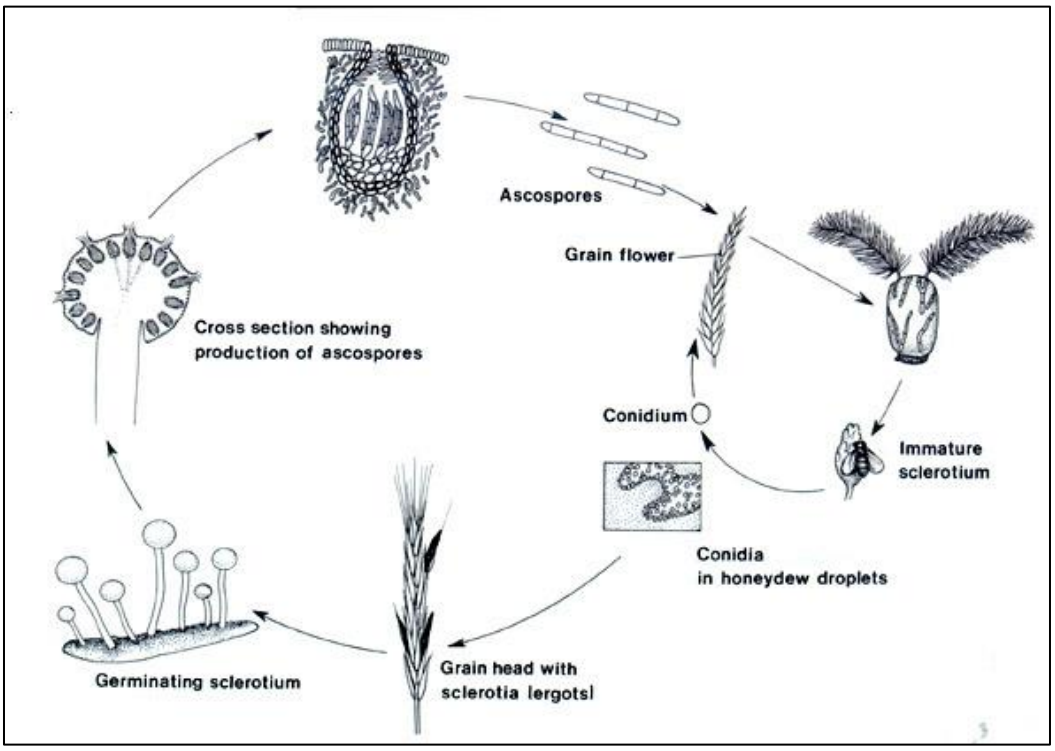

Figure 1. The life cycle of Claviceps purpurea. Source: Schumann (10).

Ergot has historically been associated with rye; however, several other grass species are also susceptible to ergot infections. Annual cereal crops including rye, wheat, triticale, barley and oats can become infected but only during the plant's flowering stage. For this reason, open-pollinated grasses such as rye are the most susceptible due to a comparatively lengthy flowering period.

Ergot Toxicity Symptoms in Livestock

Once ergot-contaminated feed has been consumed, the clinical symptoms may take effect in as little as a few hours or up to several months depending on the extent of feed contamination (9). The manifestations of ergot toxicity generally occur as either the convulsive form or the gangrenous form, although both may be exhibited concurrently (11). Convulsive symptoms can be characterized by seizures, staggering, confusion, hallucinations or partial paralysis. Historical records suggest that this manifestation of ergot toxicity is more common in horses and sheep and rarely observed in cattle (12).

The gangrenous effects of ergot alkaloids are a result of impaired circulation due to blood vessel vasoconstriction, resulting in a loss of blood supply to the extremities (i.e. limbs, ears or tail). Early symptoms may include elevated respiration rate, weight loss, reduced milk production and adverse effects on reproductive traits (i.e. abortion, low conception rates or dystocia) (13). While visual signs of gangrene may not be noticeable for several weeks, the consequences can be severe and may include the complete loss of tissues associated with extremities. Due to vasoconstriction, the gangrenous symptoms may be accentuated during periods of hot or cold temperatures outside of an animal's thermoneutral zone where thermoregulation is required (14). Severity of the infection is determined by the plant source of infection, ergot alkaloid concentration in feed, duration of exposure, ambient temperature and the mixture of ergot alkaloids (which will vary with geographic location).

The effects of ergot toxicity vary among different livestock species with poultry seemingly able to handle greater dietary alkaloid concentrations than ruminants or swine. However, the fact remains that even minor dietary ergot concentrations (as low as 200 ppb) can have adverse health and performance impacts across all livestock groups, ultimately resulting in production losses. It has been estimated that losses due to ergot in just the ruminant and horse industries could exceed $1 billion annually (15).

Ruminants

Gangrene is a common response in cattle consuming ergot and usually affects the ear tips (Figure 2), tail and hooves. Clinical cases of ergot toxicity have resulted in the partial or entire loss of the tail and ears, or sloughing of tissues around the hooves. In severe cases, entire hooves may be lost due to a prolonged deprivation of blood supply. The loss of extremities is more pronounced in ruminants compared to swine or poultry due to the fact that they are generally housed outside rather than in a temperature-controlled environment. Therefore, these animals are more likely to experience hot and cold temperature conditions. Sheep tend to exhibit milder effects compared to cattle, although observed instances of convulsive symptoms are much more common.

Figure 2. Consumption of ergot-infected feed can lead to the loss of ear tips and other extremities in livestock. Source: NDSU (21).

In addition to reduced blood flow, less severe yet equally important symptoms exist for ruminants, including reduced growth performance (16, 17), shorter gestation lengths (18), lower conception rates (19), abortion (20, 21) and decreased sperm quality (22, 23). Ruminants also experience agalactia (loss of or failure to produce milk) after consuming ergot-infected feed (18, 24, 25). This can have significant effects on calf or lamb performance as it has been suggested that even after the infected feed has been removed, prolactin may need several months (or the entire lactation cycle) to return to normal.

Swine

The effects of ergot toxicity in swine are similar to those experienced by ruminants. Reduced growth performance and feed refusal have been associated with ergot-infected feed (26). Significant reproductive losses such as small litter sizes, premature farrowing, repeat estrus, metritis and mastitis have been observed in sows consuming ergot alkaloids (27). Additionally, milk production is reduced or halted entirely in the presence of ergot due to the inhibition of prolactin production (28, 29). The combination of agalactia and premature farrowing often results in the birth of smaller, weaker piglets who must be managed separately and generally do not meet expected performance measures.

In some cases, lameness of the hindquarters and necrosis of extremities have also been reported. Controlled temperatures in swine barns tend to reduce the impact of gangrene.

Poultry

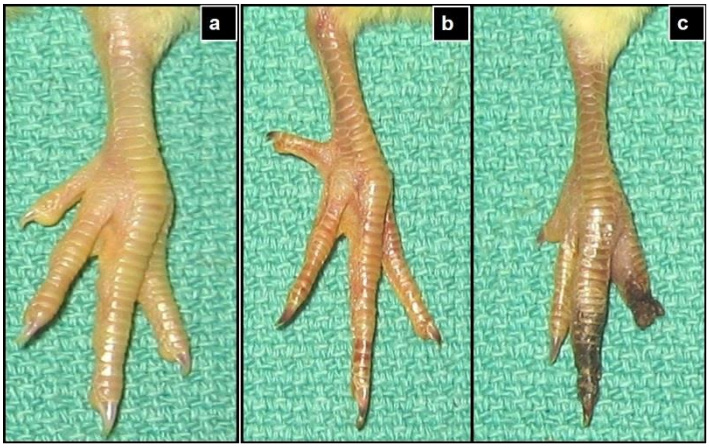

Figure 3. Comparison of feet from a healthy chick (a) to chicks consuming moderate (b) and high (c) concentrations of ergot. Source: Woinarowicz et al. (36).

Chickens fed diets containing ergot alkaloids have exhibited reduced growth and feed intake, depressed feed conversion, respiratory difficulty, diarrhea and high mortality (30, 31, 32). In birds, prolactin concentrations are also reduced which affects both incubation and broodiness behaviour. This is a result of reduced prolactin causing lower circulating concentrations of gonadotropins and thus ovarian regression (33). As with other livestock species, poultry are also susceptible to gangrene in their extremities following consumption of ergot, particularly at their feet (Figure 3), beaks and comb (34).

Horses

The gangrenous effects of ergot toxicity are typically not observed in horses, although they may be present in extreme cases of chronic or high exposure. Rather, horses tend to exhibit signs of convulsive ergot toxicity as well as other secondary symptoms described below.

Ergot alkaloids reportedly affect mare reproductive performance as well. Mares seem to be quite sensitive to ergot and have been shown to experience abortions (35), extended gestation periods (36), retained placentas (35) and higher incidence of dystocia (36, 37). These reproductive losses can mostly be attributed to the depression of hormones such as progesterone, estradiol and prolactin (38). If ergot is consumed during pregnancy, mammary development will be affected. Furthermore, a lack of prolactin following foaling means that milk production will be poor (39), further jeopardizing the survival of the foal. Milk production is also depressed when ergot toxicity occurs after foaling and during lactation.

Recommended Tolerance Levels of Ergot and Ergot Alkaloids

Several countries have established legislative maximum limits for concentrations of ergot sclerotia in grain for humans and animal feed (Table 1). Each of the countries listed in Table 1 have ergot concentrations between 0 - 0.05% total ergot by weight in cereal grains. Canada's tolerance limit for ergot concentrations in cereal grains intended for livestock feed range from 0.10 to 0.33% while the United States limits ergot to 0.30%. The European Union and the United Kingdom have the lowest limits for ergot in animal feed at 0.1% and 0.001%, respectively. For reference, 0.1% would visually represent approximately 10 ergot bodies present in one litre of grain. Note that these limits are based on the presence of ergot bodies in a sample rather than the concentration of ergot alkaloids. Due to the variation in ergot body potency between crop species along with the uneven distribution of ergot bodies in grain, ergot alkaloid concentrations (rather than the proportion of ergot bodies in a sample) would be a better indicator of potential toxicity in livestock.

Table 1. Allowable ergot concentrations in grain and feed from several countries (adapted from Scott (40))

Region	Ergot limit in cereal grains for humans (% net wt)	Ergot limit in cereal grains for livestock feed (% net wt)	Other comments
Canada	**0-0.05**	**0.10-0.33**	**Varies with grade of wheat for each type of grain**
Australia and NZ	0.05	N/A	0-0.10% (triticale)
European Union	0.05	0.10	-
Japan	0.04	N/A	-
Switzerland	0.02	N/A	0.05 limit on cereals destined for milling
United Kingdom	Zero tolerance	0.001	-
United States	0.30 (wheat, rye)	0.30 (wheat, rye)	0.10% (barley, oats, triticale)

In Canada, maximum ergot alkaloid concentrations are recommended for specific livestock species (41). Due to the high potency of ergot alkaloids, maximum concentration limits are typically only reported by livestock species rather than by age or stage of production. Dairy, beef cattle, calves and horses have recommended tolerance levels of 2 – 3 ppm while swine and poultry have maximum values of 4 – 6 ppm and 6 – 9 ppm, respectively (Table 2). Recommended practical alkaloid tolerance limits are also listed in Table 2. While it can be agreed that relatively low concentrations of ergot alkaloids in feed can elicit an adverse response, discrepancies exist between maximum levels recommended by regulatory bodies and those suggested in the literature. The suggested maximum ergot concentrations have generally not been substantiated through animal toxicology studies (40, 41). In fact, dietary ergot alkaloid concentrations (ergovaline) as low as 0.1 – 0.2 ppm have been shown to adversely affect animal growth performance, especially for animals that are also suffering from heat stress (4). There is a common belief that current guidelines in many countries may exceed the actual no effect concentrations for livestock. It is evident that additional information is needed to more accurately determine maximum tolerance levels in specific livestock species.

Table 2. Recommended practical and maximum ergot alkaloid levels for various livestock species (adapted from Coufal-Majewski (13)).

Species	Recommended ergot alkaloid practical limit (ppm) (3)			Maximum tolerance level of ergot alkaloid (ppm) (41)
	Low	Moderate	High	
Swine (piglets/sows/gilts)	0.50	1.0	2.0	4-6
Poultry (broiler/layer)	0.75	1.5	3.0	6-9
Dairy/Beef Cattle	0.50	1.0	2.0	2-3
Calf	0.25	0.5	1.0	2-3
Horse	0.25	0.5	1.0	2-3

Sampling and Screening Methodologies

Visual Detection

Visually inspecting feedstuffs for ergot infection has limitations, but is currently the most rapid and common screening method for ergot in the feed industry. However, due to variation in size, weight and alkaloid content (currently only verified by analytical methods), visual detection is viewed as highly unreliable for determining the degree of ergot infections. Ergot sclerotia can be several times greater in size than healthy kernels, but may also only grow to the same size of regular kernels. Furthermore, sclerotia may break apart during handling making it even more challenging to identify. A general rule of thumb would be to test all cereal grains for alkaloid concentration where ergot sclerotia are observed or may be an issue.

Figure 4. Visual detection is often used for detecting ergot in grain samples. However, this method is highly unreliable, especially for ground ingredients.

In general, mycotoxins are difficult to sample and screen in a reliable fashion. The inherent variability in which mycotoxins are distributed throughout crops presents the first challenge. This variability can translate into uneven distribution of mycotoxins within a single bin or lot of grain produced from the same field. For example, ergot infections in cereal crops tend to originate from surrounding grass sources (i.e. ditch grass) and may only penetrate a few hundred feet into the field. Therefore, the first grains harvested will likely be from the headland regions and could represent a very high ergot concentration compared to the remainder of the field. Once this grain has been deposited in a bin, there would presumably be a high concentration of ergot bodies near the bottom (first grain harvested) with lower concentrations progressing upwards through the bin. In addition to potential infection variability within the field, the high potency of ergot alkaloids also creates a challenge. For example, if a beef cow continuously consumes more than just 120 ergot bodies per day, adverse performance and health effects related to ergot toxicity may be experienced (42). As well, the density of ergot sclerotia is different compared to that of healthy seed kernels which may result in “layering” during grain transport. This reinforces the importance of retrieving a representative sample before analyzing ingredients for ergot alkaloids.

Bulk Ingredients

Some feed companies require their suppliers to provide pre-delivery samples for incoming grains. Regardless, it is essential that each facility carries out individual load sampling upon arrival of ingredients since sampling is an integral part of good manufacturing practices. Supplier sampling procedures should also be verified to ensure that any pre-delivery samples are an appropriate representation of the ingredient lot. If pre-delivery samples are not available, feed mills will need to rely on samples obtained at receiving. Subsamples should be collected and pooled from transport trucks prior to unloading. The number of samples will vary depending on the size of the truck. In general, a minimum of six subsamples (i.e. one sample from each corner and two from mid-trailer) probing the entire depth of the trailer should be taken for every load (i.e. super B trailer) of product. This can be accomplished using automatic or manual sampling probes.

Packaged Ingredients

The Canadian Grain Commission has recommendations for sampling packaged feed ingredients (43). Feedstuffs that are susceptible to ergot infection may be shipped in bags and should also be sampled using approved sample probes. Probes should be an appropriate length where they reach to the bottom or the full length of individual bags. If a single lot is comprised of less than 20 bags (i.e. 25 kg bag units) then a sample from each bag should be taken and pooled for a representative sample. In lots that range from 21 to 1000 units, samples should be randomly taken from a minimum of 6% of the bags (but not less than 20 bags), while lots containing more than 1000 units should have samples collected from a minimum of 3% of the total stock. If tote bags are used for ingredients, each unit should have a minimum of two subsamples collected from within.

Subsamples should be subsequently pooled and mixed prior to inspection or analysis. While the size of each subsample will vary based on collection method, a minimum of 500 to 1000 g total sample is required when determining ergot sclerotia as a percentage of the net weight for most cereal grains (44). For ergot alkaloid determination, a minimum sample size of 1000 g is usually requested by analytical laboratories. Sampling methods may differ based on an organization's standard operating procedures or the equipment available. Regardless, it is critical that techniques to ensure representative sampling are applied as limited samples or inadequate sampling techniques may not accurately reflect the true ergot concentration in a grain source.

Higher Risk Ingredients

There are several feed ingredients that pose a higher risk for ergot contamination and should be monitored accordingly. Firstly, raw cereal grains that are susceptible to ergot infection should be inspected before entering a feed mill or farm operation. Incoming wheat, barley, rye, triticale and oat feedstuffs should be screened for ergot. Grain screenings are a common feed ingredient, particularly in beef diets. However, grain screenings pose a high risk for ergot contamination as they are the byproduct of the grain cleaning process. Ergot sclerotia larger than healthy kernels, broken and cracked sclerotia fragments and grain fines can be easily introduced into the screenings stream. In particular, dust or fines are a significant source of ergot contamination. Caution should be exercised when utilizing screenings at significant inclusion levels in

livestock diets. Finally, byproducts of different milling and biofuel industries can also present a risk of high ergot alkaloid levels. Unlike the case for raw grain or screenings where ergot bodies can be easily identified, visual identification of ergot sclerotia in these byproduct ingredients is difficult if not impossible as the ergot bodies are often broken up during processing. Compounding the issue is the fact that mycotoxins can be concentrated up to three times their original level through various processes during the manufacture of these byproducts. A list of feed ingredients susceptible to ergot contamination can be found in Table 3.

Table 3. Commonly used feed ingredients susceptible to ergot contamination.

Cereal grains	
	Rye
	Triticale
	Wheat
	Barley
	Oats
Grain terminal wastes	
	Cereal grain screenings
Milling byproducts	
	Wheat bran
	Wheat shorts
	Wheat flour
	Wheat or barley mill run
Distillers'/biofuel byproducts	
	Dried distillers' grains from wheat or barley

Forages should not be overlooked when considering possible ergot infection. If grasses have passed the flowering stage before they are harvested for hay, the possibility exists for ergot alkaloids to be present. Likewise, annual cereals planted for fall or winter grazing could reach the flowering stage prior to swathing. The popularity of crops such as fall rye and triticale as forages for winter grazing emphasizes the need to test forages prior to feeding.

Analytical Methods for Analyzing Ergot Alkaloids

More than 50 alkaloids have been discovered from grains infected with fungi in the *Claviceps* spp. These alkaloids are the cause of adverse health effects and performance responses exhibited by livestock and thus it is important to know the total alkaloid concentrations of infected feedstuffs. Although the relative importance of each may vary from region to region, the predominant alkaloids of concern in North America that can be detected by current analytical procedures are: ergometrine, ergosine, ergotamine ergocornine, ergocryptine and ergocristine. The total concentration and type of ergot alkaloids present is dependent upon plant species, environmental conditions and the species of fungus involved (14). Recent advancements have been made towards analytical methods for detecting and quantifying ergot alkaloids. While significant progress in enzyme-linked immunosorbent assays (ELISA) and near infrared reflectance (NIR) analyses have been achieved, accuracy is still an ongoing issue for these tests. An accurate on-site ergot alkaloid screening tool (rapid test) for the feed industry does not currently exist.

High-Performance Liquid Chromatography

The high-performance liquid chromatography (HPLC) method requires that a prepared sample solution be pumped at high pressure into an analytical column containing chromatographic material. Determination of alkaloids is carried out using fluorescence detection that measures light absorbance at excitation wavelengths between 235 and 250 nm (45, 49). This method is commonly used to detect the six predominant ergot alkaloids at detection limits as low as 0.01 – 0.5 ppb (50).

Liquid Chromatography and Mass Spectrometry

The use of liquid chromatography in combination with mass spectroscopy (LCMS) is a common method for the quantification and identification of ergot alkaloids (46). Briefly, reverse phase-based chromatography using a liquid solvent is used for the preliminary separation of ergot alkaloids. Thereafter, individual alkaloids are ionized and subjected to gas molecules to produce charged ions that can be separated and identified during the final detection stage of MS (47). Currently, six ergot alkaloids (ergometrine, ergotamine, ergosine, ergocristine, ergocryptine and ergocornine) are routinely measured and just ten alkaloids in total can be accurately calculated using the LCMS method. Very sensitive limits of detection for these six ergot alkaloids and their epimers in wheat using LCMS have been determined, ranging from 0.0017 ppb for ergometrinine to 0.021 ppb for ergocristinine (48). Generally, LCMS is viewed as the most sensitive and reliable method available to measure ergot alkaloid concentrations.

Strategies to Reduce the Risk of Ergot Toxicity

It is important that feed mills discuss the issue of ergot contamination with their ingredient suppliers as prevention is the most effective means of reducing the risk. The simplest method to prevent ergot-infected feedstuffs from entering a feed mill is to pre-screen the ingredient prior to receiving. This begins with collecting a true representative sample. As well, adapting adequate sampling and receiving standard operating procedures that reflect thorough sampling and screening would provide another layer of control. Furthermore, it would be necessary to ensure employees and ingredient suppliers are properly trained to conduct these procedures in order to maintain consistent receipt of uncontaminated ingredients. Ideally, ingredient shipments containing any visible ergot bodies should be rejected. However, other mitigation strategies should be applied if this is not possible.

In addition to visual inspection of unground ingredients, ingredient quality assurance programs could be expanded to address the risk of ergot. A potential preventative strategy could be to request that suppliers submit ingredient samples to an analytical laboratory (Table 4) capable of performing ergot alkaloid analysis. In this case, ingredients are only accepted by a feed mill if the ergot alkaloid content falls below a maximum allowable concentration. While this positive release system approach requires additional coordination and associated analytical expenses prior to receiving, there would be additional assurances that ingredients do not contain harmful ergot alkaloids.

Table 4. List of diagnostic laboratories offering ergot alkaloid analysis of feedstuffs.

Lab	Location	Method	Contact Information
Prairie Diagnostic Services	Saskatoon, Saskatchewan, Canada	LCMS	Ph: (306)-966-7316 E: pds@info.usask.ca Web: www.pdsinc.ca
Actlabs	Ancaster, Ontario, Canada	LCMS	Ph: (905)-648-9611 ext 224 E: info@actlabsag.com Web: http://www.actlabsag.com/home
Endophyte Service Laboratory – Oregon State University	Corvallis, Oregon, United States	HPLC	Ph: (541)-737-2872 E: a.morrie.craig@oregonstate.edu Web: http://oregonstate.edu/endophyte-lab/
Iowa State University Veterinary Diagnostic Laboratory	Ames, Iowa, United States	LCMS	Ph: (515)-294-1950 E: isuvdl@iastate.edu Web: https://vetmed.iastate.edu/vdl
University of Missouri Veterinary Medical Diagnostic Laboratory	Colombia, Missouri, United States	LCMS	Ph: (573)-882-6811 E: evanst@missouri.edu Web: http://vmdl.missouri.edu/index.html

Once ingredients (particularly raw cereal grains) have entered a facility, it becomes difficult to manage the ergot risk potential. Conventional grain cleaning methods (i.e. scalpers, shaker decks) that remove impurities, dust and broken or shriveled kernels can reduce, but not eliminate, the risk of ergot toxicity (9, 43). Specific grain cleaning equipment such as gravitational separators and colour sorters are also effective in removing sclerotia from infected grain sources. At present, however, this type of cleaning equipment is not found in a typical feed mill. Soaking, dehulling, roasting or high velocity air cleaning of kernels can also be used to remove surface contamination that may contain ergot alkaloids (43).

If feed ingredients are found to contain ergot after being received by a feed mill, they should either be used in a manner that will reduce the risk of ergot toxicity to poultry and livestock or be discarded. Note that any mixing which may occur naturally for bulk ingredients when new material is unloaded on top of existing will not be sufficient to dilute contaminated feedstuffs. In some instances, limiting the dietary inclusion levels of ingredients that are at a higher risk of containing ergot can also reduce the risk of ergot toxicity. Several commercial products are currently marketed for mycotoxin control, some of which may prove effective in managing ergot toxicity to poultry and livestock.

Suggested Areas of Future Research

The recent increase in ergot infestation worldwide has led to renewed interest for a more complete understanding of the fungal disease, particularly in the livestock industry. Research involving ergot toxicity in livestock can be categorized into three general groups: 1) feeding studies investigating actual dietary tolerance levels; 2) new or improved methods for detecting ergot alkaloids; and 3) additional strategies aimed at reducing the impact of ergot toxicity.

Feeding studies to further evaluate toxicology effects of ergot in livestock would be useful to determine whether current suggested tolerance levels are adequate. Measurements of various performance (i.e. growth, milk production) and health parameters (i.e. ability to thermoregulate, effects on reproduction) in livestock fed different levels of ergot and data generated would help to either validate or disprove current tolerance limits. As well, the recovery of animals once infected feed is removed from their diet should be focused on, particularly with regards to lactation. Due to recent advancements in analytical technology for determining alkaloid concentrations, additional studies focusing on ergot toxicity in all livestock species would provide valuable information to researchers, nutritionists and feed manufacturers when addressing ergot concerns.

As previously discussed, alkaloid detection methods have significantly improved over the last several years in terms of accuracy, time required and cost effectiveness. However, further improvements to current methods and new methods of detection would benefit the livestock industry. Presently, ELISA analytical techniques are being improved and could soon offer a rapid and inexpensive method for on-site ergot alkaloid detection. As well, more information involving NIR analysis of ergot-infected feeds is required in order to build a reliable reference database before industry adoption can take place. Detection of ergot alkaloids in animal tissues also has utility in the livestock industry, especially for rapidly screening animals suspected of having consumed ergot-infected feed. The ability to test liver tissue, blood or milk for ergot alkaloid content would be a valuable tool for diagnosing toxicity, particularly in cases of abortion outbreaks.

Alternative strategies to reduce the impacts of ergot have been investigated. A possible vaccine to combat the toxic effects of ergot alkaloids would also be beneficial and has already been successfully studied in rabbits consuming tall fescue containing ergot alkaloids (51). Feed additives that are intended to mitigate the risk of ergot alkaloids are also becoming more common in Europe. Adding a supplement to feed that could bind harmful toxins and make them unavailable to livestock would be a practical method for preventing ergot toxicity. This approach has already shown promise for reducing the availability of other mycotoxins in feed (52, 53) with binders such as clays and charcoal proving effective. However, further evaluation of the efficacy for currently available mycotoxin binders to specifically reduce the impact of ergot alkaloids on poultry and livestock is necessary.

Finally, hydrothermal processing has been shown to reduce the alkaloid concentration of ergot-infected grain. Batches of rye grain containing up to 25% ergot were steam conditioned for approximately 2 minutes at 95°C (17% moisture) before being expanded over a 5 second period at a temperature of 120°C (18% moisture). Using samples taken before and after the entire hydrothermal process, the researchers determined that alkaloid levels had been reduced by approximately 10% (54). This indicates a potentially promising treatment strategy that could be adopted in feed mills, although further research is needed to support the concept.

Conclusion

The effects of ergot have been documented for several hundred years. Due to an increased awareness and advances in grain cleaning technologies, the risk to human health is low. Nevertheless, the potential still exists for ergot to enter livestock feed, especially in years when there is more contaminated grain. Ergot toxicity has significant performance and health effects on all livestock species and therefore the industry needs to consider possible strategies to reduce the overall impact of ergot. While accurate analytical detection methods are now available for screening feedstuffs, they are not always rapid enough to be practical. A combination of preventative strategies and management practices to address ergot in animal feed are required to minimize the potential adverse effects of ergot. Future research associated with ergot will help broaden our understanding of this dangerous fungal disease, particularly in areas addressing tolerance levels and innovative approaches to minimize its toxicity.

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