THE UNIVERSITY OF MANITOBA

THE EFFECT OF ERGOT ON THE PERFORMANCE OF GROWING CHICKS

BY

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ABSTRACT

Ergot produced by the fungus *Claviceps purpurea* has deleterious effects on chick performance. A series of experiments were conducted on growing chicks to (1) compare the toxic effects of ergot from different sources, (2) observe the effects of increasing ergot levels, (3) develop means of reducing ergot toxicity, (4) observe recovery patterns following short term exposure to ergot, and (5) attempt to isolate the toxic components in ergot. Changes in weight gain, and feed consumption relative to control birds and mortality were followed.

Leghorn chicks were fed ground ergot from rye, wheat and triticale from different Canadian sources, at equivalent total dietary alkaloid concentrations. The relative weight gains for birds fed diets containing 45 and 90 ppm total alkaloids varied considerably, ranging from 49.2±2.0 to 64.8±3.0% and 17.8±0.9 to 41.6±2.6%, respectively. Also, the degree of depression in relative performance was not uniform among the different ergot samples when their concentration in the diet was increased. Up to 1% dietary ground ergot (0.308% total alkaloids) was tolerated by both Leghorn and broiler chicks without significant ($P<0.05$) effects. Above the 1% level, there was a progressive decrease in the relative performance of chicks as the dietary concentration of ergot increased and also with increased time of exposure. Broiler chicks tended to be more sensitive to the effects of ergot, in terms of performance, than Leghorns, but Leghorn chicks appeared to be more sensitive in terms of mortality. After about three weeks however, in both strains of chicks, the differences in weight gain and feed consumption appeared to remain constant or increase slightly relative to control birds.

Leghorn chicks, previously fed up to 4% of a wheat ergot contaminated diet for four weeks, demonstrated a significant ($P<0.0001$) and continuing ability to recover from ergot ingestion when given an ergot-free diet. The above findings suggest the presence of an active alkaloid detoxification or extraction mechanism in chickens. Treatment of ground wheat ergot with chlorine gas or heat at 200°C for 30 minutes reduced the ergot effect about 50%, while addition of Antitox Vana (polyvinyl polypyrrolidone), a mycotoxin antitoxicant, had no effect. In a fractionation study, not all the toxic activity in ergot was isolated in the alkaloid fraction, suggesting the presence of additional non-alkaloid toxic components.

Chickens may be able to tolerate higher levels of dietary ergot than previously reported, but the total alkaloid content and composition should be considered when determining the maximum allowable level of inclusion in a feed for a given ergot sample. Even so, chemical analysis may give a low estimate of the alkaloid content of a sample and so may not provide a totally reliable prediction of a sample's true toxicity. These studies showed that there does not seem to be a clear and simple relationship between the alkaloid content of ergot and its effect on chick performance.

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It is the author's wish to dedicate this dissertation in honor of his parents, Rudi and Marianne Rotter, and his wife Barbara, and thank them for their love, faith, understanding and support.

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LIST OF ABBREVIATIONS

cAMP	cyclic Adenosine-3´, 5´-monophosphate
C	celcius
cm	centimeter(s)
CNS	central nervous system
feed to gain	feed to gain ratio
GI	gastrointestinal
HCl	hydrochloric acid
g	gram(s)
kcal	kilocalorie(s)
kg	kilogram(s)
$\leq$	less than or equal to
LD_{50}	median lethal dose
mg	milligram(s)
N	nitrogen
PEI	Prince Edward Island
%	percent
ppb	parts per billion
ppm	parts per million
PRL	prolactin
SE	standard error
SCWL	Single Comb White Leghorn
SNK	Student-Newman Keuls
UV	ultraviolet
WL	White Leghorn
w/w	weight for weight

INTRODUCTION

Ergot, produced by plant parasitic Ascomycetes fungi of the *Claviceps* species, is an important concern of the food industry, particularly the animal sector. The term ergot is used in two ways: (1) as a common name for the species of *Claviceps*; and (2) more precisely to refer to the sclerotium produced by *C. purpurea* (Kingsbury 1964).

While best known as a disease of rye, ergot also infects wheat, triticale, barley, oats, corn, rice, sorghum, millets, other small grains and numerous grasses. Lorenz (1979) has identified thirty-two *Claviceps* spp. capable of parasitizing over six hundred host plants. The sclerotia or ergot bodies produced by the fungus contain a mixture of chemicals, including alkaloids, which can cause toxic effects when ingested (Young 1981a).

An ergot infection is highly variable from year to year in intensity and incidence. Farming practices and environmental factors are involved in determining an outbreak of the fungus (Young 1979; Lorenz 1979). It can mean: reduced yield and downgrading to the cereal grower; unacceptable formulations for the feed manufacturer; reduced performance and quality of livestock and poultry; and for the consumer, contaminated food (Young 1981a).

Sporatic epidemics of ergot poisoning in humans have occurred throughout history, one of the most recent occurring in 1926 (Bové 1970). During the Middle Ages alone, up to 40,000 people suffered from ergotism. It became known as St. Anthony's Fire because of miraculous cures from the rotting flesh, falling off of limbs and death for those who travelled to the grave of St. Anthony (van Rensburg 1977).

Ergot has long been used in human medicine with references to it dating back to ancient Greece and earlier. Today, individual ergot alkaloids are important therapeutic drugs, particularly the amides of lysergic acid. The major uses of the alkaloids and their derivatives in medical science include applications in obstetrics, treatment of migraines and of hypertension (Lorenz 1979). They are widely used in scientific research, particularly in the area of endocrinology.

Ergot ingestion has also been shown to affect animal performance. The toxic effects of ergot are dependent on the animal species involved, ergot source and composition, amount ingested and the time of exposure to the contaminated feed (Young 1979). Griffith et al. (1978) showed that LD_{50} values of individual alkaloids, using laboratory animals, can differ by a factor of 40 and that the order of toxicity is species specific. Unfortunately, in most reported studies, the ergot ingested by animals was not chemically characterized (Young 1979; Lorenz 1979), thereby complicating comparisons between and also within animal species.

LITERATURE REVIEW

Life Cycle of Ergot

The life cycle of ergot has been described by several authors, among them Kingsbury (1964) and Seaman (1980). The cycle begins in a previous fall when mature ergot sclerotia fall to the ground from an infected plant. Unless covered by soil or debris, it germinates in spring when moisture and temperature conditions are favorable. Spore producing heads are extended above the soil covering so the spores can be carried away by air currents (Kingsbury 1964; Seaman 1980).

Some spores become deposited in the open florets of susceptible plants, and then the fungus grows, spreading throughout the ovary killing host tissue in the process. After five days, a sticky honey dew, containing thousands of condia or secondary spores, collects on the surface of infected florets. Condia are then carried away to healthy florets by contact, rain and numerous insects attracted by the honey dew (Kingsbury 1964; Seaman 1980). Plants at field margins and edges are particularly vulnerable to infection because of their proximity to infected grasses and insect activity (Seaman 1980).

Condia spread as long as flowering continues, the fungus growing as the crop matures, replacing the ovule and eventually forming the sclerotium. This allows the fungus to survive the winter and thus complete the cycle (Seaman 1980; Kingsbury 1964). Moisture is needed by the sclerotia to germinate. Frequent showers promote release of ascospores and germination of the condia. It is during wet springs followed by hot summers therefore that a problem of ergot infection and the subsequent contami-

nation of feed grains is of major concern.

Chemistry

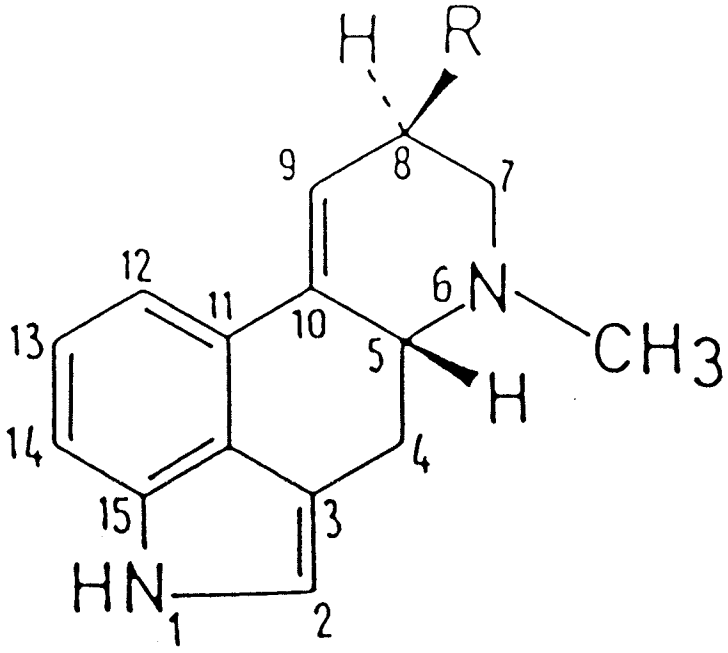
Ergot contains a number of physiologically active and inactive alkaloids, amines and other N-containing compounds. The most active of the alkaloids are ergotamine, ergocryptine, ergocornine, ergocristine, ergosine and ergometrine (Kingsbury 1964; Lorenz 1979). They have no universal pharmacological action, each possessing its own pharmacologic characteristics (Mercier 1982). Many demonstrate a wide range of actions, and if the doses required for a certain effect are considered, it is evident they exhibit a high degree of specificity (Berde and Sturmer 1978).

All alkaloids are derived from a tetracyclic ring called lysergic acid (Burfening 1973) (Fig. 1). They fall into two main groupings: (1) the clavine alkaloids, in which the carboxyl group of lysergic acid has been reduced to a methyl or hydroxymethyl group and (2) all lysergic acid derivatives. The latter is further divided into the lysergic acids, simple lysergic acid-amides and the peptide alkaloids (ergopeptines) (Berde and Sturmer 1978; Berde 1980). Only the amide and peptide alkaloids are considered to be of pharmacological significance.

The total alkaloid content of ergot may be highly variable among sclerotia from the same head, field or regions within a country. Young (1981a) reported alkaloid levels in Canadian ergot ranging from 0.011 to 0.42%, averaging 0.249%, while Atal and Schwarting (1957) in a similar study reported a range of 0.007 to 0.729%.

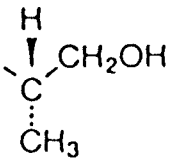
The proportion of individual alkaloids may be the same throughout a single sclerotium or in different sclerotia of the same head, but is highly

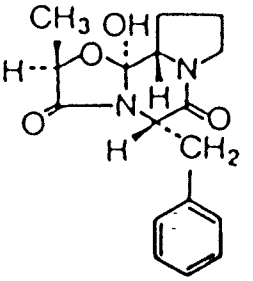
Figure 1. General formula of ergot alkaloids and examples of side chains from each of the four alkaloid types; (a) clavine, (b) lysergic acid group, (c) lysergic acid amide and (d) peptide. (Modified from Lorenz 1979 and Berde 1980).



a) R= CH_3 or CH_2OH

b) R= COOH

c) R= 

d) R= 

variable from head to head in a given field. However, the average alkaloid composition for different fields within a region tend to be uniform (Young 1981a). It may be postulated that the relative toxicity of a given ergot sample is dependent on its alkaloid content and the proportion of individual alkaloids.

Comparing the alkaloid contents of Canadian rye, wheat, triticale and barley ergots, Young and Chen (1982) concluded there were no significant differences between ergots from the prairies and that differences between fields in that region tended to be less than elsewhere in Canada. They stated that, except for a few samples from the Maritimes, Canadian ergots are fairly uniform in alkaloid composition. It must be noted however, that some *Claviceps* species produce large numbers of alkaloids, while others produce none. Species that do produce alkaloids only do so during the sclerotial stage of development (Sim 1965 in Watkins 1982). While most *Claviceps* species produce the lysergic acid-type alkaloids, many do not produce significant levels of the clavine alkaloids.

Some thirty different alkaloids have thus far been isolated from ergot (Robinson 1981). The ability to synthesize alkaloids is also variable from one strain of *Claviceps* to another. Bhat et al. (1976) found ergotamine was the dominant alkaloid produced by *C. purpurea* in wheat and rye, whereas agroclavine and elymolavine were the major alkaloids found in bajra (pearl millet) that was infested with *C. fusiformis*.

Absorption, Breakdown and Excretion

In mammals, alkaloids are poorly absorbed after oral administration. As a result, similar physiological effects are only produced when oral

doses are eight to ten times the intramuscular dose (Lorenz 1979). According to Eckert 1978 (in Franz et al. 1980), intestinal absorption of hydrogenated ergot alkaloids varies between 10-30%. Franz et al. (1980) studied the rate of disappearance of radiolabelled alkaloids from the GI tract of rats *in situ*. The absorption was found to be slow and incomplete. They suggested that the mucus layer is the rate limiting barrier for intestinal absorption due to interactions via hydrogen bonds. Generally, however, the higher the molecular weight of the alkaloid, the poorer its absorption from the GI tract.

Bailey et al. (1973) suggested that ergot alkaloids are likely poorly absorbed from the GI tract of pigs despite the fact that the acid conditions of the stomach would favor solution of the alkaloids. In contrast, Whittemore et al. (1976) found that about 70% of the total alkaloid ingested was removed in the stomach. Some may have reentered that tract in bile, but 90% was absorbed into the animal's body. The efficient extraction and absorption apparently was followed by an equally efficient breakdown of the alkaloids (Whittemore et al. 1976). Once absorbed, the alkaloids are rapidly degraded, apparently being detoxified by the liver (Lorenz 1979). Insignificant amounts are excreted in the urine and penetration of significant amounts into the brain and cerebrospinal fluid has not been demonstrated.

Degradative pathways may vary from species to species, but most animals have some degree of natural detoxification mechanisms. Alkaloids are thought to be metabolized by oxidation, one of the most common detoxification processes (Robinson 1981). It involves dehydrogenation and the introduction of oxygen into the molecule via hydroxylation. Another

process is that of conjugation, where foreign compounds, such as alkaloids, are converted to less toxic derivatives by combination with some conjugating agents. Further, in the case of some nitrogenous compounds in mammals, some are N-methylated, others acetylated and some N-methylated compounds are demethylated (Robinson 1981). These mechanisms may only be important at low level ergot contamination or when the contaminant has been removed from the feed. Several reports have noted recovery of ergotized animals when clean feed was substituted for contaminated feed.

Whittemore et al. (1976) found no evidence of intact alkaloids in tissue or urine samples from ergot-fed swine. It was postulated that they may be fragmented into unrecognizable structures and excreted in the bile or urine. In mice, subcutaneous injection of dihydroergosine, a related ergot alkaloid, resulted in 30% recovery in the feces. Presumably it was excreted in the bile (Mantle 1968).

No accumulation of ergotamine was found in the body tissues of broiler chicks fed dietary ergotamine tartrate (Young and Marquardt 1982). Only at the highest ergotamine tartrate levels tested, 270 and 810 ppm, were trace amounts of less than 10 ppb detected. In the feces they found that the amount of ergotamine detected was directly related to the amount fed. About 5% of the alkaloid fed remained unchanged and there were also small quantities of possible metabolites detected (Young and Marquardt 1982).

The route of excretion is thought to vary with the alkaloid, but not the method of administration (Kiechel 1979). Naturally occurring peptide alkaloids seem to be excreted via the bile, while semisynthetic clavines are removed in the urine. Intermediate in character are ly-

sergic acid and amine alkaloids. Alkaloids found in feces may be due to biliary excretion and/or incomplete absorption (Mantle 1968; Whittemore et al. 1976).

Physiological Actions

There are three major areas of physiological activity of ergot alkaloids. Peripheral effects are due to direct stimulation of smooth muscle, including vasoconstriction, uterine contraction and increased peristaltic activity (Lorenz 1979; Mercier 1982). Neurohumerol effects result from interference by the alkaloids with amines at receptor sites of myoneural junctions causing 5-hydroxytryptamine (HT) antagonism and adrenergic blockage. The third area of alkaloid activity is the central nervous system, affecting both the vasomotor and sympathetic systems (Müller-Schweinitzer and Weidemann 1978). The central actions are made up of stimulation activities, the dopaminergic properties and inhibitory effects, and the inhibition of vasomotor tone in the circulatory reflexes (Mercier 1982). There are many alkaloids which exert their effect through receptor-mediated interactions affecting c-AMP synthesis (Berde 1980).

In gangrenous ergotism, the majority of symptoms are due to direct stimulation of smooth muscle by ergot alkaloids. Contraction of arteriole musculature and smooth muscle of the GI tract are important in producing the characteristic symptoms and lesions due to vasoconstrictive alkaloids in *C. purpurea* (Kingsbury 1964; Sheridan 1980). They are responsible for the reduced blood supply to the extremities, areas where circulation is weakest. This results in loss of portions of ears, tails and limbs. Symptoms are exaggerated by cold weather when these extremities may become frozen and then slough off due to lack of circulation (Burfening 1973).

Clark (1979) noted that almost without exception, ergot alkaloids act to decrease heart rate. Most possess an affinity for more than one receptor type and so the effects are complex. For example, an alkaloid of particular importance, ergotamine, is a potent vasoconstrictor, but even within a single vascular bed, vasoconstriction is not uniform. In skeletal muscle, it has little effect on resistance vessels, but capacitance vessels are strongly constricted (Clark 1979). Ergotamine also increases peristaltic activity and the rate of passage of solid material along the GI tract (Lorenz 1979).

Long term, low level ingestion of ergot is thought to cause a generalized impairment of temperature regulation in ruminants. Watkins (1982) found that heat production in sheep was not affected by dietary ergot. He concluded that an increase in peripheral vasomotor tone was responsible for the observed lowering of the upper critical temperature by impairing heat flow to the environment.

Some effects on milk production in cows, ewes and swine are believed due to the action of ergot alkaloids on the release of PRL from the pituitary and to vasoconstriction in mammary tissue. The lack of appetite and increased body temperature in most species may be caused by effects of the alkaloids on the appetite and temperature control centres in the hypothalamus (Burfening 1973).

The effects due to ergot alkaloids are likely not due to a common mechanism at the cellular or molecular level. According to Berde (1980) and Mercier (1982), the actions are best explained if it is assumed that: (1) ergot compounds interact with more than one type of receptor site; (2) the chemical structure determines the affinity for receptors and the

intrinsic activity varies between compounds; and (3) access to and affinity for receptor sites varies from organ to organ. Unfortunately, it is still not clear whether the effects noted in animals which have ingested ergot alkaloids are due solely to unilateral actions of the alkaloids, or if other compounds enhance or synergize their apparent action (Mantle 1977).

Prevention and Detoxification of Ergot Contamination

The first step in preventing toxicity in animals due to ingestion of ergot or other mycotoxin contaminated feed is good farm management. Goldblatt and Dollear (1979) suggested crop rotation on fields that were infected the year before, planting crops which are not susceptible to the infection, using uncontaminated seeds and plowing the fields so as to bury ergot sclerotia at least 25-30 cm under the tillage. They further noted that insect control is effective in confining the spread of the disease.

If found to be contaminated, a feed may still be decontaminated or detoxified before being fed to animals. Decontamination methods fall into three categories: (1) separation of contaminants from produce, a physical procedure; (2) removal of contaminants by extraction (Jemmali 1979); and (3) inactivation of toxic components by physical (heating), chemical or biological means (Jemmali 1979; Doyle et al. 1982). Ideally, the methods should be inexpensive and simple, destroy or inactivate all toxic components, yet not produce or leave any toxic residue itself, preserve or improve the nutritional and other parameters of the feed components and still use existing knowledge or technology (Jemmali 1979: Young et al. 1983).

Physical separation of ergot from grain is a practical method as whole ergot sclerotia of both wheat and rye are generally larger than the grain itself. Most sclerotia are removed by screening, but small or broken pieces of ergot are not (Seaman 1980; Goldblatt and Dollear 1979). An alternative is the use of a gravity separator. Using a brine solution, ergot, being of lower density than the grain kernels, is separated from them through flotation (Seaman 1980; Goldblatt and Dollear 1979), but this results in a damp, salty grain (Young et al. 1983).

Numerous chemical methods to detoxify ergot have been proposed (Young et al. 1983). They include treatment with gases, such as chlorine, hydrogen chloride, ammonia, ozone and sulphur dioxide, or various solutions including bleach, hydrogen peroxide and dilute acid or alkali. Physical methods suggested, besides separation, are heat and UV light treatment (Young et al. 1983).

Krzeminska (1968) heated rye mixed with ergot under pressure in an HCl solution for one hour at 126^{o}C. Based on a chemical test, they determined the product to be alkaloid-free, but no animal feeding trials were reported. Treatment of ground ergot with heat (150 and 200^{o}C) or chlorine gas by Young et al. (1983) greatly reduced the levels of alkaloids. Sulfur dioxide and hydrogen chloride had only moderate effects, while no significant improvement was seen with ammonia gas (24 hours) or ozone (16 hours) in reducing alkaloid content or composition (Young et al. 1983).

A reduction of 25% in the total alkaloid content was noted when ergot sclerotia were autoclaved at 121^{o}C for 30 minutes. This resulted in a reduced toxic effect on growing chicks and significant improvement

in the feed to gain ratio (Young et al. 1983). Heat generated in the feed pelleting process may reduce the total alkaloid level of ergot contaminated feed and so account for reduced toxicity.

Silber and Bischoff (1954) reported that alkaloids are concentrated in the outer layers of ergot sclerotia and decrease in concentration toward the centre, but light does not penetrate very far into dry sclerotal tissue. Young et al. (1983) irradiated ground ergot with UV light for 54 hours, but saw no change in total alkaloid levels nor in individual alkaloid composition. However, in solution, the alkaloids are light sensitive.

For animals suffering from ergotism, generally the only treatments applied have been to give the infected animals clean feed and clean surroundings, or to simply destroy them. Few other treatments have been examined. In humans suffering from ergot poisoning, treatment has included anticoagulants, low molecular weight dextran, various vasodilators, sympathetic blockage, surgical sympathectomy and periarterial stripping. Results have been variable, but success has been reported by several researchers using sodium nitroprusside, an extremely potent, fast acting vasodilator (Skowronski et al. 1979). Young and Marquardt (personal communication) fed male broiler chicks feed mixed with ergotamine tartrate or wheat ergot and sodium nitroprusside. There was no lessening of the effects of ergot alkaloids on chick performance when $\leq$90 ppm sodium nitroprusside was fed concurrently with ergotamine tartrate (300 or 800 ppm) or wheat ergot (4%). Fed alone, the sodium nitroprusside reduced chick performance at 240 and 500 ppm.

Effects on Domestic Animals

Effects on Livestock (General)

The responses of an animal to the ingestion of ergot are dependent on: (1) amount eaten; (2) toxicity of the sclerotia; (3) extent of absorption of toxin(s) from the GI tract; (4) animal species; (5) period of exposure; and (6) nutritional and health status (Young 1979). There are no general effects common to all animals except a reduction in feed intake and weight gain.

It is generally accepted that ergot sclerotia are unpalatable, especially when first offered. Possibly, some ergot symptoms are related to a reduced feed intake. Whittemore et al. (1976) suggested three phases of animal response: (1) initially feed is rejected due to an unpalatable contaminant; (2) the animal becomes accustomed to the contaminant and consumes the diet more readily, but not as readily as a contaminant free diet; (3) constituents of ergot induce toxic reactions which reduce appetite.

Although there are two distinct forms of ergotism, both are rarely seen simultaneously in the same group of infected animals (Burfening 1973). Convulsive or nervous ergotism is usually acute (Sheridan 1980) and is characterized by vertigo and muscle spasms of the hind limbs followed by temporary paralysis (Burfening 1973). Ergotism of horses, sheep and carnivora is almost always convulsive, but this form is rarely seen in cattle. The gangrenous form is chronic and is characterized by lameness, swelling of hind limbs, gangrene of feet, tail and ears and sloughing off of the hooves (Sheridan 1980). Cattle and sheep also show decreased rate of gain and increased body temperature and respiratory rate.

The most common sign in swine is agalactia, but they too display decreased weight gain and feed efficienty (Burfening 1973).

The apparent greater incidence and severity of ergotism in ruminants than non-ruminants may be due to the nature of metabolism that occurs in the rumen. The lower pH of the rumen may favor a more complete extraction of alkaloids from ingested ergot sclerotia when they are exposed for relatively long periods of time (Sheridan 1980). The chemical structure and therefore the effects of the alkaloids may also be altered by microbial fermentation. In addition, the alkaloids could interfere with rumen function, causing disorders that do not occur in non-ruminants (Mertens 1979).

Sheep tend to be less affected by ergotism than cattle which may be attributed to their practice of selective grazing. They avoid the coarser flowering heads of grasses and so may indirectly be avoiding ergot poisoning (Mantle 1978a; Young 1979).

Ergotism in Cattle

Convulsive ergotism with tremors, nervousness, muscle incoordination and even death has been observed in cattle ingesting *C. paspali*, but seldom in those consuming *C. purpurea* (Young 1979). Cattle more commonly suffer from the gangrenous form, but symptoms and effects are often contradictory. Typical signs are lameness and swelling of the hind limbs, gangrene and subsequent loss of extremities such as ears, tail and feet, depressed feed intake and weight gain (Lorenz 1979), as well as increased pulse and respiratory rates, nervousness and ataxia (Burfening 1973). There have been reports of decreased milk production (Burfening 1973),

but they are questionable (Young 1979). Other physiological symptoms reported include increased water uptake and salivation, diarrhea, formation of a rougher and heavier coat and increased heat stress. Occasionally internal hemorrhages occur (Young 1979). Differences in blood serum content of carotene, vitamin A, calcium and phosphorus have not been observed between affected and control animals. Earlier studies suggest adult animals are more susceptible to ergotism (Burfening 1973), while more recent reports say the young are (Young 1979).

Ingalls and Phillips (1971) fed early-weaned dairy calves diets containing triticale ergot at levels of 0, 0.07, 0.14 and 0.28%. Feed intake and weight gains on the ergotized diets were reduced, but feed efficiencies were not statistically different. These observations are supported by other studies (Young 1979). In contrast, Dinnusson et al. (1975) noted feed efficiencies as well as weight gains and feed intake were significantly higher from controls in heifers fed 0.4 and 0.8% ergot.

No adverse effects occurred when Greatorex and Mantle (in Mantle (1978a) fed pure ergotamine (as tartrate) at a level of 80 mg per day for ten days to a 70 kg calf. When given intramuscularly to cows shortly after calving, ergotamine caused the period of involution to be decreased by an average of 3.5 days and the time between calving to next service by 32 days (Mantle 1978a).

Cold predisposes cattle having ingested ergot to lameness. McKeon and Egan (1971) noted that thirty cows developed lameness leading to gangrene two weeks after starting to eat silage containing 0.1g dry ergot/ 100g wet weight of silage. The ergot contained only 0.13% total alkaloids,

mainly ergotamine, but the animals were exposed to mild winter conditions, the temperature ranging from -4 to +8°C (mean of 1.4°C) for two weeks (Mantle 1978a).

Ergot can affect pregnancy, but in cattle it more likely effects a peripheral gangrenous syndrome rather than cause abortion. Pregnant heifers dosed twice weekly as high as 40g of ergot for four months showed no effect on pregnancy (Rankin 1965). The largest dose was 90 mg of alkaloid, mainly ergotamine, but as the doses were given infrequently, the animals may have been able to recover before the next dosing (Mantle 1978a).

Few cases of agalactia in cattle have been reported. Drink (1955) (in Lorenz 1979) noted a decrease in milk production in a dairy herd six days after they were given access to a field heavily ergotized with *C. purpurea*. Sixteen of the 43 cows also became lame. Mainly the older, heavier animals were affected and it was confined to the hind feet (Lorenz 1979). A 28% fall in milk production was observed by Dring (in Mantle 1978a) in a herd of Friesian cows also during a six day period. Many animals developed lameness which progressed to gangrenous lesions, again due to *C. purpurea* ingestion. Dinnusson et al. (1971) also noted cases of complete agalactia.

Ergotism in Sheep

Carlevovo (1956) (in Mantle 1978b) stated that convulsive ergotism in sheep is extremely acute and may cause death without the appearance of other symptoms. Most studies however do not support such a dramatic effect. Symptoms of ergotism in sheep include difficulty in breathing,

excessive salivation, diarrhea, heat stress and inflammation of the GI tract, as well as ulcers and internal bleeding. Affected animals generally have reduced weight gain despite increased feed consumption and necrosis of the tongue (Burfening 1973; Young 1979; Lorenz 1979).

Ergotism was experimentally induced in sheep using ergot containing 0.29% total alkaloids, 80% of which was ergotamine (Greatorex and Mantle 1973). The animals were given varying doses of ergot based on ergotamine and four of six that received 1.0 mg/kg/day died within ten days. They exhibited anorexia, hyperventilation, cold extremities, salivation and some tongue necrosis. Extensive intestinal inflammation was found during post-mortem examination of seriously affected animals.

Forty lambs from a flock of 500 were reported by Spratling (1972) (in Lorenz 1979) to have developed signs of ergotism. They had dry gangrene of the ear tips, but not of the feet and tails. Although neither the amount of ergot nor total alkaloid content was determined, it was known that wheat in that region contained about 2% ergot. Cunningham (1949) fed ergot with a total alkaloid of 0.2 to 0.6 mg per kilogram body weight to sheep. Some were unaffected, but others developed alimentary and tongue lesions. In a few, the ergot was lethal, yet no peripheral gangrene occurred (Lorenz 1979).

Mantle (1978b) reported a study in which Czechoslovakian rye ergot, containing almost exclusively ergotamine, was fed to sheep. The animals tolerated the ergot even at levels which would have been lethal had they been given ergotamine alone. Lameness developed only in one animal kept on a cold, wet pasture.

Ewes in the latter half of pregnancy were given either an alkaloid free ergot or 1 mg ergotamine/kg/day (Greatorex and Mantle 1974). The alkaloid free ergot treated animals showed no clinical signs during two weeks of testing. At 3 1/2 to four months of gestation, those given ergotamine aborted after four days of dosing. Ewes at midterm did not appear to be affected in spite of general malaise, including tongue necrosis and lameness (Greatorex and Mantle 1974).

In a natural environment, the greater sensitivity to ergotism of ewes during pregnancy is of little concern since the time of high ergot levels does not naturally occur at the same time. However the danger occurs when ergotized grass seed is fed as a winter supplement to pregnant animals (Mantle 1978b).

Burfening (1975) observed that ewes given ergot during the prebreeding and/or gestation period in two separate trials had a lower lambing percentage, about 20% less than controls.

Ergotism in Horses

Ergotism in horses is rare and not well documented. The symptoms of the disease are not as stereotyped as in other species and are attributed to effects on the CNS (Lindley 1978). Mantle (1978c) noted a report of a nervous syndrome due to *Paspalum* ergot poisoning in Australia, but gave few details. Nicholson (in Lindley 1978) observed a number of rodeo horses which developed a trembling, drunken, ataxic condition, apparently due to ingestion of ergot contaminated hay. All these horses recovered within 24 hours after being given clean feed.

Lindley (1978) suggested that the shorter duration of symptoms in horses than cattle may be due to the greater amount of grass consumed by the ruminants. Another possibility may be the action of rumen protozoa and bacteria, but the reasons for the apparent differences have not been firmly established.

Ergotism in Swine

The classical symptoms of convulsive or gangrenous ergotism are not generally seen in swine (Young 1979). There are reports of lameness and gangrene of limbs, ears and tail, but more recent studies suggest they are not as sensitive to peripheral effects, rather only to centrally mediated effects (Mantle 1978d). The most serious problem is agalactia, seen in pregnant sows fed contaminated feed immediately prior to farrowing (Burfening 1973). Abortions do not occur, but while piglets are born healthy, high mortality results from malnutrition. There have been observations of gangrenous ear tips of newborns, but otherwise no developmental problems (Young 1979).

Ergot containing 0.29% total alkaloids fed to growing pigs caused severe reductions in feed intake and growth rate at the two highest levels tested, 1.0 and 2.0% ergot (Friend and MacIntyre 1970). At 0.05 and 0.1%, feed intake and weight gain were slight, but less than the controls. A subsequent experiment also indicated decreased feed intake and growth in growing-finishing pigs fed 0.05 and 0.10% ergot. This was further supported by a nitrogen-balance experiment in which control pigs retained 1.54 grams of N more than those fed 0.10% ergot (Friend and MacIntyre 1970).

Whittemore et al. (1976) induced pigs to eat a diet containing

10% ergot. The ergot contained 0.31% total alkaloids and although there was a reduction in feed intake and growth, there was no evidence of peripheral necrosis, internal or external hemorrhage or changes in hematological characteristics. Ingestion of ergot did not affect feed digestibility, although urinary excretion of N was increased and retention of N reduced, supporting the findings of Friend and MacIntyre (1970). In their study, Whittemore et al. (1976) observed increased water uptake in ergot fed animals. They suggested that the pigs may have wanted to reduce the distastefulness of the diet or that water in the intestines and increased urination may be important in detoxification. This is consistent with the centrally mediated pharmacological properties of some alkaloids (Whittemore et al. 1976).

Reduced reproductive performance was seen by Campbell and Burfening (1972) in gilts fed diets with 0.53% ergot. Although not significant, the pregnancy rates of ergot fed gilts tended to be lower than the controls, 72 versus 94% respectively. No effect was seen on the percent of corpora lutea in ergotized animals and there was no difference in birth weights of young born to control and ergotized gilts. This was in contrast to the observation of lighter young born to ergot fed gilts by Nordskog and Clark (1945).

Gilts given ergot during the 8 to 28 day stage of pregnancy showed no interference with the maintenance of pregnancy nor any adverse effects on blastocyst implantation or organ formation of the embryos (Bailey et al. 1973). While no direct effect during pregnancy itself has been noted, mammary function is affected by alkaloids, resulting in agalactia (Anderson and Werdin 1977; Mantle 1978d).

Affected sows also show no other signs of illness other than retarded growth of the mammary glands. In numerous studies where agalactia occurred, no cases of lameness, necrosis or gangrene of extremities were seen, even when up to 2.0% ergot was used (Lorenz 1979). Burfening (1973) reported sows fed up to 1.0% ergot during the latter third of gestation were in good condition at farrowing, but had no udder development and did not lactate. Gestation periods were shortened by five days, and all piglets died shortly after birth.

Shone et al. (1959) observed agalactia of sows leading to the death of newborn piglets. All were born live and healthy, but died from starvation within a few days of birth because of a failure of the mammary glands of the mother to develop normally during the last few weeks of pregnancy. Nordskog and Clark (1945) also reported agalactia in sows fed 0.1, 0.5 and 1.0% ergot.

Ergotism in Poultry

Before the advent of chemical assays for ergot, the sensitivity of the cock's comb to the vasopressor properties of ergot alkaloids was used. The contraction of the arterioles near the base of the comb causes a pallid coloration of the comb, and was the basis of the test (Mantle 1978e). Surprisingly little work has been done on poultry and water fowl as to the effects of ergot on them.

Perek (1958) fed boluses of 1g of ergot powder containing 1% total alkaloids twice daily for 24 days to 5 three month old WL cockerels and 5 ten month old WL hens. Between days 12 and 18, vesicular lesions of the comb and wattles were seen in all five cockerels and two hens, and foot lesions were noted in only two cockerels. The remaining three hens

demonstrated loss of appetite, purple combs and stopped laying. Only one mortality occurred, a cockerel on day 20, but no gross lesions of organs were seen (Perek 1958).

SCWL chicks fed ergotized diets up to 9.0% tolerated ergot only up to 0.3% without adverse effects (O'Neil and Rae 1965). Higher levels caused growth depression and death. The birds also showed signs of ergot poisoning which included the blackening of nails, toes, shanks, beak and comb. O'Neil and Rae (1965) reported that 0.2 to 0.4% ergot in the diet of laying hens caused reduced egg production, feed consumption and the maintenance of body weight. They did not see any effect on egg size, hatchability, shell texture and interior shell quality, even at the 9.0% ergot level. There was no mortality and the total alkaloid content of the ergot was not given.

Chickens avoid ergot sclerotia in their feed and are known to reject feed which was contaminated. Even after ergot is removed from grain, the smell or taste may still remain (Honkovaara in Mantle 1978e). This may account for the reduced feed intake observed when poultry are fed ergot contaminated feed.

In two studies by Dinnusson et al. (1973; 1974) and summarized by Johnson and Sell (1976), a total of 2000 SCWL laying hens were fed diets containing up to 2.4% ergot. No indication of the total alkaloid level was given. They noted that above 0.5 or 0.6% ergot, egg production was detrimentally affected. Hen day production was significantly reduced and feed consumption to produce one dozen eggs increased above 0.6% ergot. Body weights apparently were influenced at higher levels (Dinnusson et al. 1973). Above 0.5%, there was a significant increase in

the percentage of shelless and misshaped eggs, but egg quality, weight, shell thickness and mortality appeared to be unaffected. No alkaloids were detected in eggs, liver or fat tissue tested. In one collection period, feces contained intact alkaloids and their metabolites, but they were not detected in a second collection (Dinnusson et al. 1973). The overall conclusion of these experiments was that ergot levels of 0.5% and above, fed to laying hens, would cause economic losses to producers (Dinnusson et al. 1973; 1974; Johnson and Sell 1976).

The effect of feeding triticale ergot to broiler chicks for the first one to two months of life was examined by Bragg et al. (1970). They gave no indication of the source or chemical composition of the ergot, but noted that up to 0.8% ergot was tolerated in the diet. Ergotism in the chicks was characterized by depressed growth, poor feathering, nervousness, loss of coordination and then an inability to stand. The beaks, toenails and toes of severely affected birds gradually darkened until all tissue turned black at death. At 1.6% ergot, Bragg et al. (1970) observed a very severe growth depression and this depression continued as dietary ergot levels increased, up to 12.8%, where virtually no growth occurred. During the first four weeks of the trial, mortality at the 3.2% level and below was not significantly different, but at 6.4 and 12.8% ergot it was 60 and 90% respectively. By 48 days, mortality on the 3.2% ergot diet was 100%. It was concluded that the toxic components of ergot have a cumulative effect and can greatly affect survival of broiler chicks above 0.8% ergot in the diet.

In separate experiments of up to ten days duration, day old male SCWL and day old female broiler chicks were fed diets that contained

ergotamine tartrate (Young and Marquardt 1982). Dietary ergotamine tartrate levels of up to 27 ppm did not affect chick performance, but at or above 81 ppm, weight gains and feed consumption were greatly reduced. Feed to gain was only affected at 729 ppm ergotamine tartrate. No skin or toe necrosis was seen in birds fed up to 243 ppm. At 243 and 729 ppm, the incidence of toe necrosis was 60 and 100% respectively and at the higher level, 63% also had skin necrosis. No mortality was noted. Broilers fed greater than 30 ppm dietary ergotamine tartrate suffered from greatly reduced weight gains while feed consumption was decreased above 10 ppm. Skin necrosis was not seen at any level, but the incidence of toe necrosis was 63 and 78% at 270 and 810 ppm of dietary ergotamine tartrate respectively. Mortality at these two levels was 8%. Results obtained in a 51 day trial using male broiler chicks were similar to those seen in the 10 day studies. Only during the initial 2-3 weeks of the experiment were the reductions in weight gains noticeable. After this period, the relative weight differences between experimental birds and the controls remained constant (Young and Marquardt 1982).

Young and Marquardt (1982) also examined liver, brain and heart weights per unit body weight. The weights of all three tissues in broiler chicks were higher than the controls only when the dietary concentration of ergotamine tartrate was 810 ppm. Brain weights of leghorn chicks at 243 and 729 ppm ergotamine tartrate were also significantly higher. When sacrificed, the heart size for the two highest levels were considerably larger, but returned to normal shortly after exsanguination.

Muscovy ducks and ducklings were fed tail wheat cleanings con-

taminated with 1.17% ergot with an unknown alkaloid composition and with blackgrass seed (Swarbrick and Swarbrick 1968). Birds aged two to three months suffered heavy mortality after 48 hours, the older birds were seemingly unaffected. Lethargy and diarrhea preceeded death in seriously affected birds. Post-mortem examination showed no significant macroscopic lesions, but the livers were engorged with blood, kidneys congested and alimentary tracts empty. There was no evidence of parasitic or bacterial disease (Swarbrick and Swarbrick 1968).

It may be concluded from existing studies that birds in general, are able to tolerate higher levels of ergot in their feed than most other species of animals. Despite the problem of ergot contamination of animal feed however, very little research has been conducted in this area, particularly in poultry. The objectives of the experiments described herein therefore were designed to examine, over short time periods, a wide range of questions, namely to: (1) compare the effects of ergot from different sources and of different types; (2) attempt to isolate the toxic component(s) in ergot; (3) examine the effects of feeding increasing levels of dietary ergot; (4) compare the performances of SCWL and broiler chicks to increasing levels of ergot; (5) examine the ability of SCWL chicks to recover from short term exposure to ergot; and (6) examine methods aimed at reducing ergot toxicity.

MATERIALS AND METHODS

Formulation of Diets

All diets were formulated to meet the minimum NRC (1977) requirements for replacement pullets (Diet 1) and for broiler chicks up to four weeks of age (Diet 2) (Table 1). Diet 1 was mixed on two occasions, approximately six months apart, Diet 2 only once. Both formulations were stored in a walk-in-cooler (at $3^{o}C$) and required amounts removed when needed.

The total percentage of cornstarch and ergot added to diets varied between experiments. In Experiments I, V and VI, the cornstarch and ergot, and in Experiment VII also the Antitox Vana, accounted for 4.5% (w/w) of a given diet. For diets used in Experiments III and IV, cornstarch and ergot made up 4% of the total weight except in Experiment III at the 8% ergot level when only ergot was added. No cornstarch was added to any diet in Experiment II. In all cases, except Experiments III and IV, ergot and cornstarch were mixed into the basal diet by means of a Hobart 3-speed mixer. Because of the volumes involved, individual diets in Experiment III and IV were mixed using a Marion mixer (160 kg capacity). All diets were fed as mash.

Chicks and Management

One day-old male chicks were purchased from a commercial hatchery and housed in electrically heated, thermostatically controlled batteries with raised wire floors and provided with continuous lighting for seven days prior to the start of an experiment. All chicks were fed commercial chick starter crumbles containing a minimum of 21% protein. SCWL chicks

Table 1. Dietary formulations for basal diets

Ingredient		g/kg Diet 1[e]		Diet 2[f]
Wheat		585		577
Soybean meal	(44%)[a]	200	(47.5%)	210
Fishmeal (65%)		60		70
Tallow		60		60
Calcium phosphate		24		16
Calcium carbonate		10		11
Vitamin mix[b]		10		10
Mineral mix[c]		5		5
Methionine		1		1
Ergot + cornstarch		45[d]		40

[a]The percent protein of the soybean meal used.

[b]The vitamin mix per kilogram diet consisted of: retinyl palmitate, 7500 IU; cholecalciferol, 1000 IU; alpha-tocopherol acetate, 10 IU; menadione, 2.2 mg; thiamine, 2.2 mg; riboflavin, 4.4 mg; pantothenic acid, 14.3 mg; niacin, 33 mg; pyridoxine, 4.4 mg; biotin, 0.13 mg; folic acid, 1.3 mg; cholinechloride, 1320 mg; vitamin B_{12}, 0.11 mg; anti-oxidant (santoquin, 50% ethoxyquinone), 250 mg.

[c]The mineral mix supplied (mg/kg of diet): manganese, 330 as MnO; zinc, 110 as ZnO; iron, 30 as $FeSO_4 \cdot 7H_2O$; copper, 2.5 as $CuSO_4 \cdot 5H_2O$; iodized NaCl, 4930.

[d]Cornstarch was added to ground ergot sclerotia so that the total amount equalled 4.5% of the total diet, except for Experiments II, III and IV, when the total was 4.0%.

[e]ME = 3043 kcal/kg; protein = 21.4%.

[f]ME = 3055 kcal/kg; protein = 23.1%.

were used in all experiments, and in Experiment III, broiler chicks were also tested.

All experiments were set up as completely randomized designs. Where appropriate, a factorial arrangement of treatments was used. In addition, when correlated measurements were taken, a split plot analysis was performed. At seven days of age, immediately prior to the start of an experiment, the chicks were starved for a four hour period to decrease GI tract fill. Birds were then individually weighed and placed into appropriate small weight range groups. Those birds used in an experiment were then randomly taken from the median weight range groups in such a manner that initial pen weight variation was minimal and so that there would be no statistical difference between pens on the same experiment. In Experiment III, the initial weights of pens with Leghorns differed from those of broilers, but within each strain, the weights were similar. Initial pen weights in Experiment IV differed not only between treatments, but also within treatments. This was unavoidable however because of the nature of the study and will be discussed later in this section. The number of treatments, replicates per treatment and number of birds per replicate varied between experiments and will be discussed separately.

Birds on test were housed in Petersime battery brooders with continuous lighting and heat. Feed and water were provided ad libitum. During and at the end of an experiment, prior to pen weights being taken, the birds were starved for a four hour period.

In Experiment II however, as the birds were weighed more than once a week, only the initial and final weights are on a "starved" basis. All the intermediary pen weights were on a "full fed" basis as starving the

birds more than once a week may have deleteriously affected their performance.

All weight gain data given in this thesis is reported on a per bird basis and relative to the control data. This provides a good basis for comparison of results among and within experiments. The relative weight gain was taken as:

$$\text{Pen relative weight gain} = \frac{\left(\frac{\text{Final pen weight}}{\text{\# Birds alive in pen}} - \frac{\text{Initial pen weight}}{\text{\# Birds in pen}}\right)}{\text{Average weight gain per bird on control diet}}$$

Feed consumption is also expressed on a per bird basis, relative to the control data. It was taken as:

$$\text{Pen relative feed consumption} = \frac{\dfrac{\text{(Initial feed + feed added) - (Feed remaining + feed spilled)}}{\text{\# of birds alive during period in pen}}}{\text{Average feed consumption per bird on control diet}}$$

Feed to gain ratio per pen was calculated by dividing feed intake per bird by bird weight gain. The relative weight gain, relative feed consumption and feed to gain ratio for each treatment was calculated by averaging the pen values.

If a mortality occurred early in a given time period, the dead bird was assumed not to have consumed any feed. If it occurred however after the third day of a seven day period, the following correction method was applied to the above: (1) The number of days between feed weighings was determined and divided into the total feed consumed during that period in that pen. This would give an estimate of how much feed was consumed by the whole pen per day during that period. (2) The number of

days the dead bird was alive during that period was determined, and for that number of days, the feed per day for the pen was summed and divided by the number of birds alive prior to death. This gave a feed per bird value for the time before mortality. (3) The pen feed consumption data for the days after death was then divided by the number of remaining birds to give a feed per bird per day value for the days after the mortality. (4) The values of the feed per bird per day before and after death were then added to give an estimate of the feed consumed per bird during that time period. (5) This value in turn was then divided by the average feed consumption per bird on the control diet to give the relative feed consumption.

Sample calculation:

Feed consumed by pen over 7 day period: 271 g

Initially 6 birds in pen, one died on day 5

(1) Average feed consumption per day for pen = 271 ÷ 7 = 38.7 g/day.

(2) Dead bird consumed feed for 4 days, therefore 4 days x 38.7 g/day = 154.9 g consumed by 6 birds over four days

154.9 g ÷ 6 birds = 25.8 g/bird for the four days.

(3) Feed consumed by remaining 5 birds after death was 116.1 g

116.1/5 birds/3 days ÷ 5 birds = 23.2 g/bird/3 days.

(4) 25.8 g/bird/4 days + 23.2 g/bird/3 days = 49.0 g/bird/7 days.

(5) Average feed consumption of control birds was 108.4 g

Therefore, the relative feed consumption for this pen was:

$$\frac{49 \text{ g/bird/7 days}}{108.4 \text{ g/bird/7 days}} = 0.45$$

Ergot Preparation and Analysis

The ergot samples used in Experiments I, V and VI were obtained ground, treated (if applicable) and ready for use from Dr. J.C. Young, Agriculture Canada, Ottawa, Ontario. The ergot employed in all other studies was obtained as whole sclerotia from Dr. S.C. Stothers, Dept. of Animal Science, University of Manitoba, Winnipeg, Manitoba, and was originally purchased from Northern Sales Ltd., Winnipeg. The ergot sample 125A from Dr. J.C. Young is believed to have come from the same collection. All samples were ground to fit through a #20 mesh screen, and stored in a freezer until needed.

Analysis of all ergot samples was conducted by Dr. J.C. Young using the HPLC methods described in Young (1981a) and modified in Young (1981b). The percent total alkaloids, source and description of the ergots used are presented in Table 2. The alkaloid composition of all but sample ES02 are given in Appendix 1.

Table 2. Sources and descriptions of ergots used

Ergot source	Sample number	(%) Total alkaloids	Description
Rye	CY-XVI-126A	0.269	Volunteer fall rye collected from field of barley, Meadowbank, PEI August 8, 1978.
Rye	CY-XVI-126B	0.312	Fall rye (variety Kustro) collected from North River, PEI, August 23, 1978.
Rye	CY-XVI-126C	0.269	Fall rye (variety Tetra Petkus) collected from Central Experimental Farm, Ottawa, Ontario, July 27, 1979.
Triticale	CY-XVI-126D	0.259	Spring triticale collected from Central Experimental Farm, Ottawa, Ontario, 1977 crop.
Wheat	CY-X-690	0.336	From Canadian Grain Commission, Thunder Bay, Ontario, 1978 crop.
Wheat	CY-XVI-125A (Also ES02)	0.308	From Western Canada, 1979 crop. (Obtained from Northern Sales Ltd., Winnipeg, Manitoba).

Statistical Analysis

Analysis of variance was conducted for all data using the General Linear Models (GLM) procedure of the Statistical Analysis System (SAS Institute, Inc. 1982a). The GLM procedure utilizes the least squares principle to fit linear models. The statistical models used for each experiment are given in Appendix 2. Where appropriate, results were compared using the SNK test (SAS Institute, Inc. 1982b).

Experiment I

The Variability of Effects of Ergots from Different Sources on the Performance of Leghorn Chicks

In order to compare the effects of ergots of different types and sources, five ergot samples from rye, wheat and triticale from different areas of Canada and of different total alkaloid levels were mixed in diet 1 at levels of 45 and 90 ppm total alkaloids (Table 3). A series of three trials were conducted. Because of limited amounts of some of the ergots, in two of the trials, four birds were employed per replicate with six replicates per treatment per trial at each ergot level. For the rye ergot sample 126B, there was only enough ergot to use two birds per replicate per treatment. The second of these two trials involved a repeat of two ergot samples used in the first, 126C and 126D, but only at the 90 ppm level. The third trial was basically a repeat of the first, except it did not include sample 126B. In this trial, six birds were used per replicate and six replications per treatment as larger quantities of the ergots had been made available. Each of the three trials lasted a period of seven days and body and feed weights were taken at the start and end of each trial.

Table 3. Experiment I dietary mixtures using Diet 1 as the basal diet

Trial	Ergot sample	Level	% Ergot	Amount of ergot (g)	Amount of basal diet (g)	Amount of cornstarch (g)	Total diet (kg)
1	Control	-	0	0	3400	-	3.40
	125A (wheat)	Low*	1.46	36.5	2388	75.5	2.50
		High**	2.92	64.2	2101	34.8	2.20
	126A (rye)	Low	1.67	41.8	2388	70.2	2.50
		High	3.34	73.5	2101	25.5	2.20
	126B (rye)	Low	1.44	19.4	1289	41.6	1.35
		High	2.88	31.7	1050	18.3	1.10
	126C (rye)	Low	1.67	41.8	2388	70.2	2.50
		High	3.34	73.5	2101	25.5	2.20
	126D (triticale)	Low	1.74	43.5	2388	68.5	2.50
		High	3.47	76.3	2101	22.7	2.20

Continued .....

Table 3 (Continued)

Trial	Ergot sample	Level	% Ergot	Amount of ergot (g)	Amount of basal diet (g)	Amount of cornstarch (g)	Total diet (kg)
2	Control	-	0	0	3400	0	3.40
	126C (rye)	High	3.34	70.1	2005	24.9	2.10
	126D (rye)	High	3.47	72.9	2005	22.1	2.10
3	Control	-	0	0	5000	0	5.00
	125A (wheat)	Low	1.46	58.4	3820	121.6	4.00
		High	2.92	87.6	2865	47.4	3.00
	126C (rye)	Low	1.67	66.8	3820	113.2	4.00
		High	3.34	100.2	2865	34.8	3.00
	126D (triticale)	Low	1.74	69.6	3820	110.4	4.00
		High	3.47	104.1	2865	30.9	3.00

*45 ppm total alkaloids.

**90 ppm total alkaloids.

Experiment II

Effect of Increasing Levels of Ergot on Leghorn Chick Performance

The purpose of this experiment was to observe the effect of increasing levels of dietary ergot on Leghorn chick performance. Since wheat is a major component of chicken diets in many areas, especially western Canada, the wheat ergot used in Experiment I was used in this and subsequent studies.

Leghorn chicks were fed diet 1 mixed with Prairie wheat ergot (ES02) at levels of 0, 1, 2, 3, 4, 5 and 6% ergot (w/w) (Table 4). There were eight replicates per treatment and six birds per replicate. The experiment lasted a total of 28 days. Feed was weighed on days 0, 1, 2, 3, 4, 5, 6, 7, 10, 14, 17, 21, 24 and 28 and birds on days 0, 3, 7, 10, 14, 21 and 28 of the trial. All body weights were recorded on a "full fed" basis except on day 0 and on day 28 when both "full fed" and starved bird weights were taken.

Table 4. Experiment II dietary mixtures using Diet 1 as the basal diet

Ergot level (%)	Amount of* ergot (kg)	Amount of basal diet (kg)	Total diet (kg)
0	0	120.0	120.0
1	1.20	118.8	120.0
2	2.00	98.0	100.0
3	2.85	92.15	95.0
4	3.36	80.64	84.0
5	3.50	66.5	70.0
6	3.60	56.4	60.0

*No cornstarch added,

Experiment III (a and b)

Comparison of the Effect of Increasing Levels of Ergot on the Performance of SCWL and Broiler Chicks

This experiment was designed to compare the effect of increasing ergot levels on the performance of Leghorn and broiler chicks using wheat ergot (ES02). The ergot was mixed into each of diets 1 and 2 at levels of 0, 1, 2, 4 and 8% (w/w) (Tables 5 and 6). Leghorn chicks were fed both dietary formulations, whereas broilers only received diet 2. At each level for each of the three groups of birds, there were six birds per replicate and six replicates per treatment. The birds were on test for 19 days and body and feed weights were taken on days 0, 7, 14 and 19.

For the purposes of analysis, this experiment was considered as two experiments, Experiments IIIa and IIIb, although run concurrently. Experiment IIIa compared the Leghorn chicks fed the two dietary formulations (Diets 1 and 2) at the various ergot levels. Experiment IIIb compared the performance of Leghorn chicks to broilers on the same dietary formulation (Diet 2).

Table 5. Dietary mixtures fed to Leghorn chicks in Experiments III and IV using Diet 1 and Diet 2 as the basal diets

Ergot level (%)	Amount of ergot (g)	Amount of basal diet (g)	Amount of cornstarch (g)	Total diet (kg)
0	0	30240	0	30.24
1	212	20352	636	21.20
2	304	14592	304	15.20
4	424	10176	53	10.60
8	544	6256	0	6.80

Table 6. Dietary mixtures fed to broiler chicks in Experiment IIIb using Diet 2 as the basal diet

Ergot level (%)	Amount of ergot (g)	Amount of basal diet (g)	Amount of cornstarch (g)	Total diet (kg)
0	0	72000	0	72.0
1	720	69120	2160	72.0
2	1296	62208	1296	64.8
4	1440	34560	0	36.0
8	2016	23184	0	25.2

Experiment IV

Ability of Leghorn Chicks to Recover from Exposure to Ergot Contaminated Diets

Using the Leghorn chicks employed in Experiment IIIa, the ability of Leghorn chicks to recover from short term exposure to ergot contaminated diets when given an ergot free diet was examined. Birds (age 32 days) which had been on diets containing wheat ergot (ES02) levels of 0, 1, 2 and 4% (w/w) (Table 5) for a 25 day period were divided into two groups. Half were continued on the same ergot mixture and the rest placed on an ergot free diet. This procedure was followed for the birds on both dietary formulations, diets 1 and 2 (Table 1).

Birds from Experiment IIIa were kept on the ergot diets for an additional five days prior to the start of this study. The birds were then rerandomized by mixing cages of the same dietary formulation and ergot level. Then they were assigned to treatment groups so that initial pen weights were similar. Half of the birds received the ergot level previously fed and the other half the ergot free diets. Each treatment group contained three replicates with five birds per replicate. Exceptions to this were the control birds on diet 2 which had six replicates. Also, because of high mortality during Experiment IIIa for birds fed the 4% ergot level, only two cages could be assigned per treatment and three birds per cage on diet 1 and four birds per cage on diet 2. The birds were fed the experimental diets for 14 days and body and feed weights were recorded on days 0, 7 and 14.

Experiment V

Effect of Feeding Ergot Extracts on Chick Performance

Based on the results of Experiment I which suggested the possibility of toxic components other than alkaloids in ergot, this study attempted to isolate the toxic activity using ground wheat ergot (CY-X-690). While some of the ergot sample was set aside, the rest was subjected to the extraction scheme outlined in Figure 2. The fractions extracted and used in this study were obtained from Dr. J.C. Young, Agriculture Canada, Ottawa.

Dietary treatments used in this study consisted of the control diet containing no added ergot and two levels of each of the four fractions and of the unextracted ergot, equivalent to 1.5 and 3.0% of unextracted ergot (w/w) (Table 7). Six replicates of six Leghorn chicks each were fed each experimental diet for seven days.

Figure 2. Extraction scheme used to separate ergot fractions used in Experiment V (Young, pers. comm.).

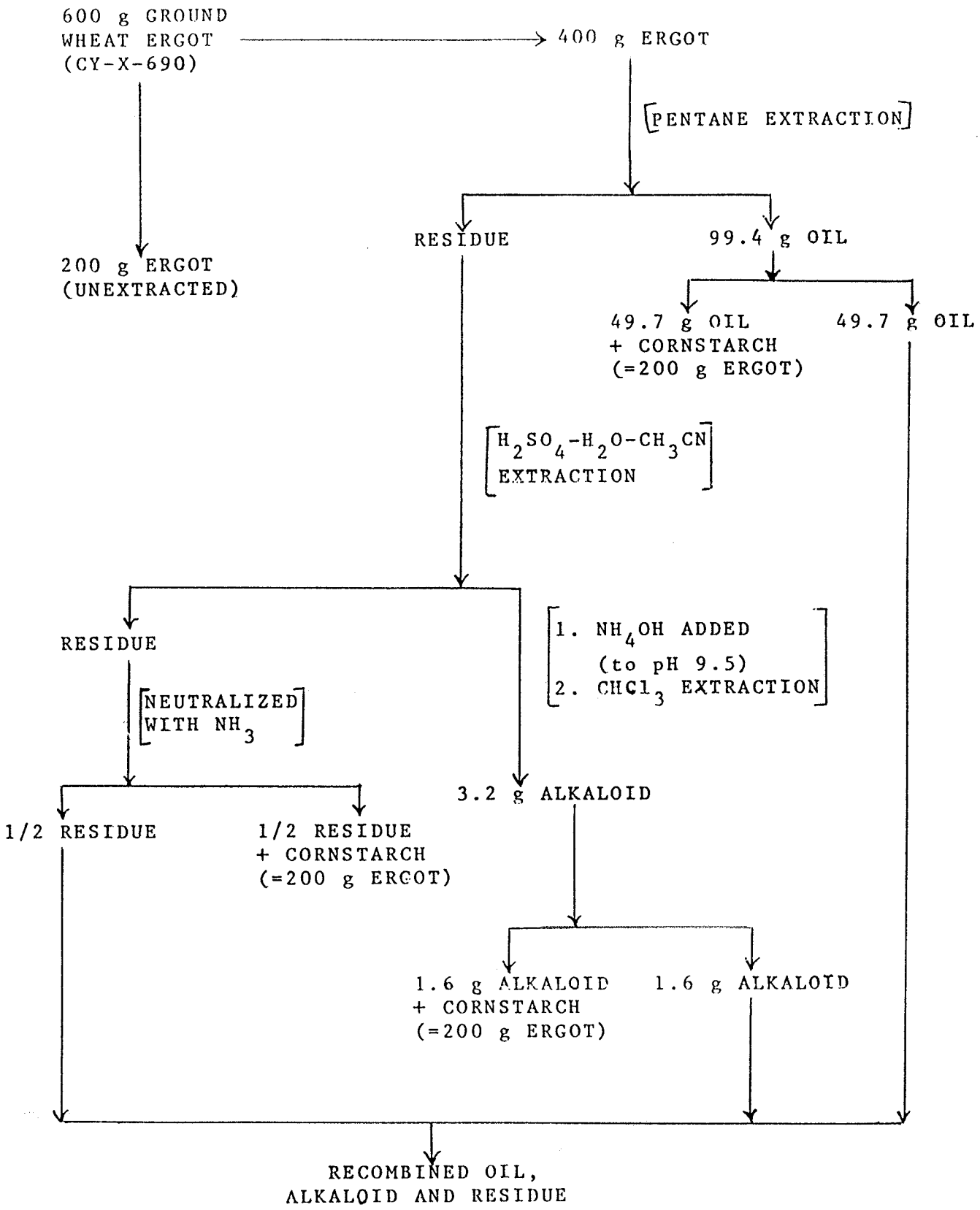

600 g GROUND WHEAT ERGOT (CY-X-690)
400 g ERGOT
[PENTANE EXTRACTION]
200 g ERGOT (UNEXTRACTED)
RESIDUE
99.4 g OIL
49.7 g OIL + CORNSTARCH (=200 g ERGOT)
49.7 g OIL
[H_2SO_4-H_2O-CH_3CN EXTRACTION]
RESIDUE
[1. NH_4OH ADDED (to pH 9.5) 2. $CHCl_3$ EXTRACTION]
[NEUTRALIZED WITH NH_3]
3.2 g ALKALOID
1/2 RESIDUE
1/2 RESIDUE + CORNSTARCH (=200 g ERGOT)
1.6 g ALKALOID + CORNSTARCH (=200 g ERGOT)
1.6 g ALKALOID
RECOMBINED OIL, ALKALOID AND RESIDUE

Table 7. Experiment V dietary mixtures using Diet 1 as the basal diet

Ergot fraction	(%) fraction	Amount of fraction (g)	Amount of basal diet (g)	Cornstarch (g)	Total diet (kg)
Control 1	0	0	4500	0	4.50
Control 2	0	0	4500	0	4.50
Unextracted	1.5	51.9	3304	104.1	3.46
	3.0	78.0	2483	39.0	2.60
Oil	1.88	78.0	3963	109.0	4.15
	3.75	155.6	3963	31.4	4.15
Alkaloid	1.56	64.7	3963	122.3	4.15
	3.11	123.5	3791	55.5	3.97
Residue	0.93	36.9	3791	142.1	3.97
	1.86	73.8	3791	105.2	3.97
Recombination	1.32	54.8	3963	132.2	4.15
	2.64	100.3	3629	70.7	3.80

Experiment VI

Effect of Feeding Heat- or Chlorine-Treated Ground Ergot on the Performance of Leghorn Chicks

To test methods of reducing the effect of ergot on chick performance, samples of Prairie wheat ergot (125A) were treated either with heat at 200°C or chlorine gas for varying lengths of time. Heat treated ground ergot was spread thinly over a metal tray and then placed in an oven preheated to 200°C for 10, 15, 20 or 30 minutes. Chlorine treated ground ergot was placed in a darkened Erlenmyer flask (covered with aluminum foil) and then subjected to chlorine gas at room temperature (24°C) and standard atmosphere for 15, 30 or 60 minutes. The treated ergot samples were prepared by Dr. J.C. Young, Agriculture Canada, Ottawa.

The seven treated ergots were each mixed with diet 1 at a level of 4% (w/w). In addition, four more experimental diets using untreated samples of the same wheat ergot were mixed at levels of 0, 1, 2 and 4% ergot (w/w), for a total of 11 treatments (Table 8). For each treatment there were six replicates and six male Leghorn chicks per replicate. The duration of this study was seven days with body and feed weights taken only at the start and termination of the experiment.

Table 8. Experiment VI dietary mixtures using Diet 1 as the basal diet

Treatment	Amount of ergot (g)	Basal diet (g)	Cornstarch (g)	Total diet (kg)
Control	0	5100	0	5.10
Untreated 1%	50.0	4775	175	5.00
Untreated 2%	90.0	4298	112	4.50
Untreated 4%	150.0	3581	19	3.75
Heated 10 min	180.0	4298	22	4.50
Heated 15 min	180.0	4298	22	4.50
Heated 20 min	180.0	4298	22	4.50
Heated 30 min	180.0	4298	22	4.50
Chlorine 15 min	180.0	4298	22	4.50
Chlorine 30 min	180.0	4298	22	4.50
Chlorine 60 min	180.0	4298	22	4.50

Experiment VII

Effect of Inclusion of Antitox Vana (Polyvinyl Polypyrrolidone) on the Reduced Performance of Leghorn Chicks due to Ergot Ingestion

This experiment was designed to test the effect of a commercially produced mycotoxin antitoxicant, Antitox Vana, on reducing the effect of ergot on Leghorn chicks.

Four levels of wheat ergot (ES02) (0, 1, 2 and 4% w/w) were combined with four levels of Antitox Vana, (0, 0.1, 0.2 and 0.8% w/w) (Table 9) in a 4x4 factorial arrangement of a completely randomized design. The dietary treatments were fed to Leghorn chicks for a period of two weeks and body and feed weights were taken on days 0, 7 and 14 of the test.

Table 9. Experiment VII dietary mixtures using Diet 1 as the basal diet

Ergot level (%)	Amount of ergot (g)	Antitox Vana level (%)	Amount of Antitox Vana (g)	Amount of basal diet (g)	Amount of cornstarch (g)	Total diet (kg)
0	0	0	0	10750	0	10.75
1	105	0	0	10027	368.0	10.50
2	168	0	0	8022	210.0	8.40
4	252	0	0	6165	31.5	6.30
0	0	0.1	10.5	10027	462.5	10.50
1	105	0.1	10.5	10027	357.5	10.50
2	210	0.1	10.5	10027	252.5	10.50
4	420	0.1	10.5	10027	42.5	10.50
0	0	0.2	21.0	10027	452.0	10.50
1	105	0.2	21.0	10027	347.0	10.50
2	210	0.2	21.0	10027	242.0	10.50
4	420	0.2	21.0	10027	32.0	10.50
0	0	0.8	84.0	10027	389.0	10.50
1	105	0.8	84.0	10027	284.0	10.50
2	210	0.8	84.0	10027	179.0	10.50
4	420	0.8	84.0	9996	0	10.50

RESULTS

The performance criteria in each table are presented on an average per chick basis, and the weight gain and feed consumption data as values relative to control birds (1.00).

Experiment I

This experiment was designed to compare the effects of ergot from different sources fed at the same concentrations of total alkaloids on chick performance. The data presented in Table 10 is a summary of the results from three trials. No significant differences were seen between trials 1 and 3 for relative weight gain, relative feed consumption and feed to gain ratio ($P>0.74$, 0.18 and 0.98, respectively). Also, when similar treatments from trials 1, 2 and 3 were compared, no significant differences were noted for relative weight gain and relative feed consumption ($P>0.17$ and 0.54, respectively), but there was a difference in terms of feed to gain ratio ($P<0.049$). The average feed to gain ratios for the two treatments common to trials 1, 2 and 3 were 4.72, 5.99 and 4.71±0.70, respectively.

Analysis of variance (Table 11) indicated significant ergot source x level interactions for relative weight gain ($P<0.0015$) and feed to gain ratio ($P<0.0009$). These interactions are evident when comparing the results due to the North River rye and triticale ergots (Table 10). At the 45 ppm total alkaloid level, the relative weight gains and feed to gain ratios due to the two samples were similar, but they were very different at 90 ppm. At this higher concentration, the triticale ergot was much more toxic than the rye ergot from North River, PEI. Addition

Table 10. The performance of Leghorn chicks fed diets containing ergots from different sources at equivalent concentrations of total alkaloids (Experiment I, Trials 1-3)

Ergot source	Total alkaloid content of diet (ppm)	Relative weight gain[a]	Relative feed consumption[b]	Feed to gain
Wheat (Prairies)	45	0.69	0.82	2.28
	90	0.32	0.55	3.46
Rye (Meadowbank, PEI)	45	0.51	0.68	2.64
	90	0.21	0.45	4.37
Rye (North River, PEI)	45	0.65	0.74	2.23
	90	0.42	0.55	2.60
Rye (Ottawa)	45	0.49	0.68	2.70
	90	0.18	0.46	5.43
Triticale (Ottawa)	45	0.60	0.75	2.43
	90	0.21	0.49	4.84
SE		0.12	0.11	0.48

[a,b]Actual control values were 56.1 and 108.4 g per bird respectively.

Table 11. Summary of analysis of variance (Experiment I, Trials 1-3) for ergot samples only

		Relative weight gain		Relative feed consumption		Feed to gain	
Source of variation	df	MS	PR>F	MS	PR>F	MS	PR>F
Ergot source	4	0.15	0.0001	0.050	0.0001	8.23	0.0001
Ergot level	1	2.35	0.0001	1.21	0.0001	63.8	0.0001
Source x level	4	0.018	0.0015	0.004	0.18	4.28	0.0009
Error	96	0.06		0.05		0.92	

of the ergot samples at a level of 45 ppm total alkaloid to the diet caused reductions in relative weight gains ranging from 31% for wheat ergot to 51% for the Ottawa rye ergot. At the higher level, the reductions ranged from 58 to 82% for the North River and Ottawa rye ergot samples respectively (Table 10). The feed to gain ratios varied between 2.23 and 2.70 for the ergots when fed at 45 ppm total alkaloids and 2.60 and 5.43 at the 90 ppm level. The control birds in contrast had a feed to gain ratio of 1.93.

Relative feed consumption was affected by both ergot level ($P<0.0001$) and source of ergot ($P<0.001$), but there was no interaction ($P>0.05$) between these factors as evidenced by the data in Table 10. The average relative feed consumptions were 0.74 and 0.50 on the 45 and 90 ppm total alkaloid diets. Comparing the averages for each of the ergot samples, the relative feed consumption reflects the same order of toxicity observed in the relative weight gain data at the 45 ppm total alkaloid concentration. The order of average toxicity, from most to least toxic in terms of relative feed consumption was Ottawa rye (0.55), Meadowbank rye (0.57), triticale (0.60), North River rye (0.64) and wheat ergot (0.68). According to the SNK test (SAS Institute Inc. 1982b), all averages were significantly different ($P<0.05$) from each other except that the Meadowbank rye sample was not different from the Ottawa rye and from the triticale ergot sample.

Although the same total alkaloid levels were added for each ergot sample, the proportions of the individual alkaloids were different (Appendix 1). For example, the Meadowbank rye ergot had almost twice as much ergotamine as the Ottawa rye ergot (40.1 vs 23.1%). The two

samples, however, had a somewhat similar effect on chick performance. In contrast, the two rye ergot samples from PEI had similar compositions (Appendix 1), yet significantly different effects ($P<0.05$) (Table 10). The reductions in relative weight gain and relative feed consumption due to the ergot samples tested were found to be significantly correlated ($P<0.01$) with the levels of ergosine, ergotamine and ergocristine present.

Only three birds died during this study, all during trial 2 on 90 ppm total alkaloid diets. One occurred early in the trial on the Ottawa rye ergot, the others on day 7 in separate pens fed triticale ergot. No classical symptoms of ergotism were noted in any of the birds.

Experiment II

This experiment was designed to see the effects of increasing levels of ergot on chick performance over a four week period (Tables 12-14). Statistical analysis (Table 13) indicated a significant time x ergot level interaction for relative weight gain (full fed basis) and relative feed consumption, as well as significant ergot level and time effects for both parameters ($P<0.0001$). The feed to gain ratio was not significant ($P>0.19$) for any factor.

Based on the observed daily results, the predicted daily relative feed consumptions depicted in figure 3, demonstrated a marked decrease with increasing levels of dietary ergot. As illustrated in the plots for the 2 to 4% ergot diets, the effect became more pronounced over time while the relative rate of decline decreased. These trends are given by the quadratic prediction equations. During the latter part of the

Table 12. The effect of increasing levels of wheat ergot on the relative weight gain (full fed basis) and relative feed consumption of Leghorn chicks over time (Experiment II)

	Relative weight gain				Relative feed consumption			
Week	1	2	3	4	1	2	3	4
Ergot level (%)								
0	1.00 (63.3 g)*	1.00 (83.0 g)	1.00 (107.0 g)	1.00 (116.2 g)	1.00 (115.6 g)	1.00 (159.9 g)	1.00 (215.8 g)	1.00 (258.4 g)
1	0.90	0.92	0.89	0.93	0.94	0.93	0.94	0.88
2	0.72	0.57	0.55	0.59	0.83	0.71	0.63	0.64
3	0.57	0.30	0.27	0.31	0.72	0.48	0.43	0.42
4	0.38	0.23	0.08	0.28	0.58	0.43	0.27	0.33
5	0.30	0.23	0.08	0.32	0.51	0.39	0.24	0.30
6	0.24	0.20	0.16	0.02	0.46	0.35	0.33	0.15
SE	0.025	0.025	0.025	0.025	0.01	0.01	0.01	0.01

*Numbers in brackets are actual values for controls (per bird).

Table 13. Summary of analysis of variance (Experiment II)

Source of variation	df	Relative weight gain		Relative feed consumption		Feed to gain	
		MS	PR>F	MS	PR>F	MS	PR>F
Time	3	0.22	0.0001	0.41	0.0001	119.88	0.19
Ergot level	6	3.78	0.0001	2.44	0.0001	91.60	0.30
Time x ergot level	18	0.04	0.0001	0.04	0.0001	83.69	0.35
Error	147	0.005		0.001		75.16	

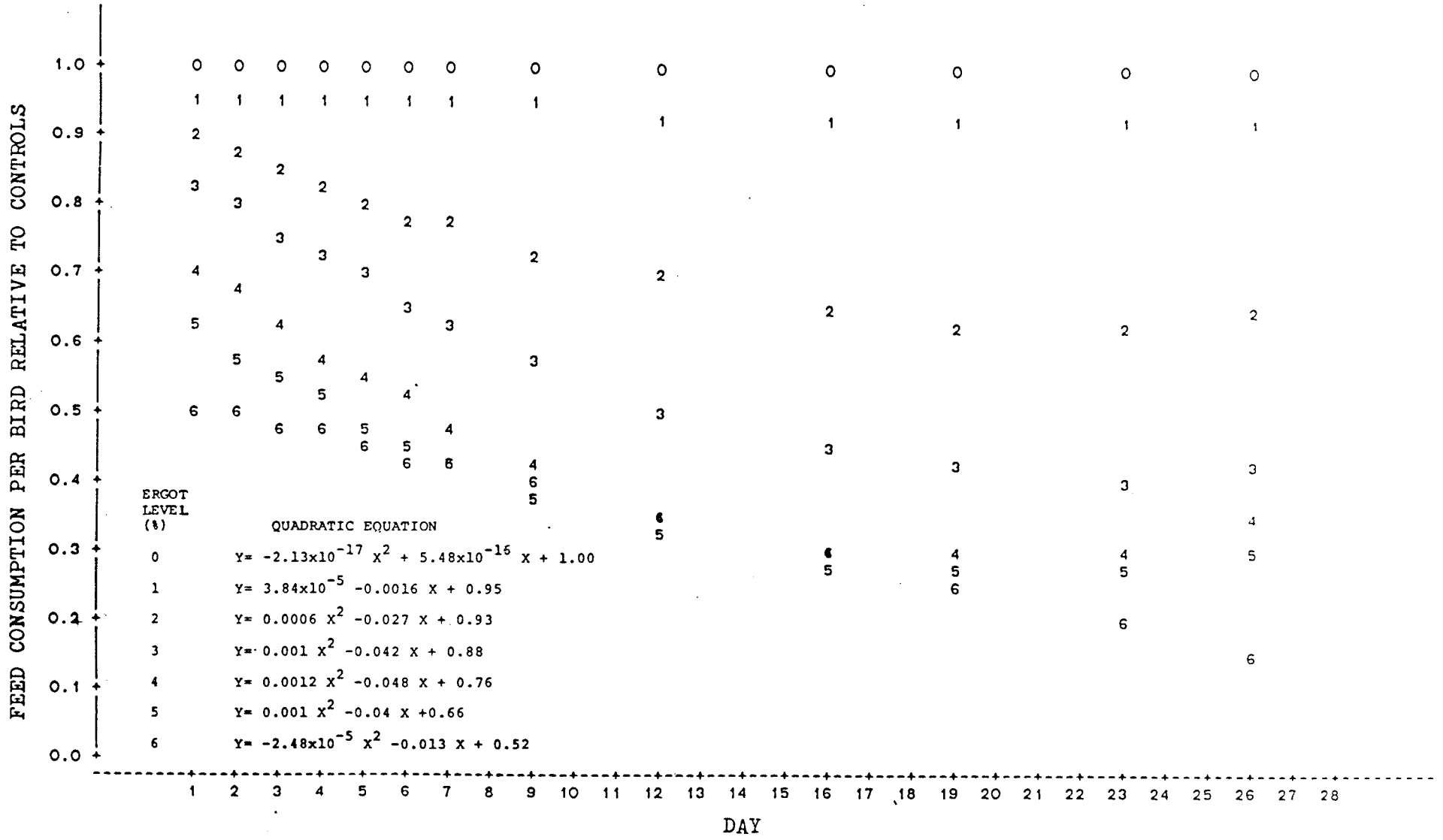
Table 14. Overall effect of increasing wheat ergot levels on the performance of Leghorn chicks (4 week totals - starved basis) (Experiment II)

Ergot level	Relative weight gain	Relative feed consumption	Feed to gain
0	1.00^{a*} (360.21 g)**	1.00^{a} (749.69 g)	2.08^{a}
1	0.90^{b}	0.94^{b}	2.16^{a}
2	0.59^{c}	0.68^{c}	2.40^{ab}
3	0.33^{d}	0.48^{d}	3.07^{bc}
4	0.22^{e}	0.37^{e}	3.64^{c}
5	0.21^{e}	0.34^{f}	3.43^{c}
6	0.12^{f}	0.29^{g}	5.28^{d}
SE	0.01	0.01	0.24

*Means in the same column with different superscripts are significantly different ($P<0.05$).

**Numbers in brackets are actual values for controls (per bird).

Figure 3. Predicted effect of consumption of various levels of ergot in the diet on the average daily feed consumption per bird relative to control birds over a 28-day period (Experiment II). Numeric symbols used correspond to the ergot level used in the experiment.


FEED CONSUMPTION PER BIRD RELATIVE TO CONTROLS
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28
DAY
ERGOT LEVEL (%)
QUADRATIC EQUATION
0 Y= -2.13x10^-17 X^2 + 5.48x10^-16 X + 1.00
1 Y= 3.84x10^-5 -0.0016 X + 0.95
2 Y= 0.0006 X^2 -0.027 X + 0.93
3 Y= 0.001 X^2 -0.042 X + 0.88
4 Y= 0.0012 X^2 -0.048 X + 0.76
5 Y= 0.001 X^2 -0.04 X +0.66
6 Y= -2.48x10^-5 X^2 -0.013 X + 0.52

study, the relative feed consumption at the intermediate ergot levels (2-5%) tended to stabilize or slightly increase as seen in the daily (Figure 3) and weekly (Table 12) results. There was a different pattern of change in relative feed consumptions at the high and low ergot levels compared to that obtained with the intermediate treatments. The relative feed consumption for the 1% ergot fed birds decreased only slightly over the course of the study whereas the birds fed the 6% ergot diet tended to show a linear decrease in relative feed consumption over time (Figure 3, Table 12).

Although there was also a significant ($P<0.0001$) ergot x time interaction for relative weight gain (full fed basis), its nature is not as evident as that for the relative feed consumption (Table 12). The results however do suggest a similar trend for both performance parameters.

The overall effects of ergot on chick performance (using starved basis relative weight gain) over the four week test period indicated significant ($P<0.0001$) differences for all parameters due to increasing ergot levels (Table 14). Increasing dietary ergot levels generally caused a marked and progressive drop in relative weight gain and relative feed consumption, and a corresponding increase in feed to gain ratio.

Analysis of the mortality data (Table 15) demonstrated a significant ergot level x time interaction and significant ergot level and time effects ($P<0.0001$). Mortalities occurred only at the 4% and higher ergot levels. Generally the incidence of mortality increased with higher ergot levels and increased time of exposure.

Experiment IIIa

The purpose of this experiment was to examine the performance of

Table 15. Average percent weekly pen mortalities of Leghorn chicks fed increasing ergot levels (Experiment II)*

Ergot level (%)	Week 1	2	3	4
0	0.00	0.00	0.00	0.00
1	0.00	0.00	0.00	0.00
2	0.00	0.00	0.00	0.00
3	0.00	0.00	0.00	0.00
4	0.00	0.00	12.63	6.75
5	2.13	4.25	24.25	27.25
6	6.38	6.75	4.25	72.25
SE	2.31	2.31	2.31	2.31

Experiment II

Summary of analysis of variance - mortality

Source of variation	df	Mortality MS	PR>F
Ergot level	6	2593.1	0.0001
Time	3	2366.4	0.0001
Ergot x time	18	1358.1	0.0001
Error	147	42.54	

*Each week is considered as a new period. Average cumulative mortalities for the 0.3, 4.5 and 6% ergot diets were 0.0, 18.3, 47.9 and 77.1%, respectively.

Leghorn chicks over a three week period when fed broiler and Leghorn type diets containing different levels of ergot (Tables 16-19). Analysis of variance (Table 18) demonstrated a significant ergot x time interaction ($P<0.001$) and the ergot level effects ($P<0.0001$) for relative weight gain and relative feed consumption. There was also an ergot x diet interaction for relative feed consumption ($P<0.015$) and feed to gain ratio ($P<0.02$) and a significant effect ($P<0.0001$) for ergot level for the feed to gain ratio data.

The interacting effects of increasing ergot levels on chick performance over time (Table 16) were similar to those seen in the previous study (Experiment II). In general, the higher the concentration of ergot in the feed, the greater the effect on relative weight gain and feed consumption. The effect of the ergot, especially at the higher concentrations, was also more pronounced over time.

The data for the ergot level x diet interaction (Table 17) shows the same trend as the weekly data; poorer performance with increasing ergot levels. The relative feed consumption for birds fed diet 2 was higher for all treatment groups except at the 1% ergot level. Diet 2 also had consistently lower feed to gain ratios than diet 1, with the differences between the two dietary formulations being more pronounced at the higher ergot levels.

A significant ergot x time interaction ($P<0.0001$) was obtained for chick mortality (Table 19). The greatest number of deaths occurred during the first week, most notably on the 8% ergot diets. The relatively high incidences of mortalities during week 1 for birds fed the 0, 1 and

Table 16. Average effect of increasing wheat ergot levels on the performance of Leghorn chicks over time (Experiment IIIa)

	Relative weight gain			Relative feed consumption			Feed to gain		
Week	1	2	3	1	2	3	1	2	3
Ergot level (%)									
0	1.00 (45.5 g)*	1.00 (69.8 g)	1.00 (66.8 g)	1.00 (86.2 g)	1.00 (140.5 g)	1.00 (131.3 g)	1.90	2.04	1.97
1	0.90	0.84	0.91	0.94	0.90	0.92	1.98	2.21	2.01
2	0.77	0.55	0.51	0.87	0.68	0.60	2.16	2.53	2.43
4	0.44	0.36	0.32	0.64	0.43	0.40	2.85	2.83	2.87
8	0.20	0.15	0.07	0.43	0.28	0.19	6.29	4.94	2.91
SE	0.04	0.04	0.04	0.03	0.03	0.03	0.98	0.98	0.98

*Numbers in brackets are actual values for controls (per bird).

Table 17. Comparison of Leghorn chick performance due to increasing ergot levels on two dietary formulations (Experiment IIIa)

	Relative weight gain		Relative feed consumption		Feed to gain	
Diet	1	2	1	2	1	2
Ergot level (%)						
0	1.00(181.2)*	1.00(182.8)	1.00(360.6)	1.00(355.2)	2.00	1.94
1	0.88	0.88	0.95	0.89	2.17	1.96
2	0.57	0.64	0.69	0.74	2.45	2.29
4	0.35	0.40	0.47	0.51	3.04	2.66
8	0.14	0.14	0.29	0.31	6.46	3.36
SE	0.05	0.05	0.03	0.03	0.98	0.98

*Numbers in brackets are actual values for controls (per bird).

Table 18. Summary of analysis of variance (Experiment IIIa)

Source of variation	df	Relative weight gain		Relative feed consumption		Feed to gain	
		MS	PR>F	MS	PR>F	MS	PR>F
Main plot comparisons							
Ergot level	4	4.54	0.0001	3.09	0.0001	43.70	0.0001
Diet	1	0.03	0.10	0.007	0.23	23.15	0.013
Ergot x diet	4	0.01	0.49	0.03	0.015	11.28	0.02
Main plot error (ergot x diet)	50	0.01		0.005		3.47	
Sub plot comparisons							
Time	2	0.18	0.0001	0.39	0.0001	5.00	0.43
Ergot x time	8	0.04	0.001	0.06	0.0001	10.80	0.44
Diet x time	2	0.003	0.78	0.002	0.63	2.66	0.63
Ergot x diet x time	8	0.12	0.46	0.004	0.50	2.49	0.90
Error	100	0.01		0.005		5.81	

Table 19. Average percent weekly pen mortalities of Leghorn chicks on diets 1 and 2 fed increasing ergot levels (Experiment IIIa)*

Ergot level (%)	Week 1	Week 2	Week 3
0	5.33	1.33	0.00
1	2.67	0.00	1.67
2	5.50	1.33	0.00
4	5.33	24.5	1.33
8	40.00	28.83	44.50
SE	6.12	6.12	6.12

Experiment IIIa

Summary of analysis of variance - mortality

Source of variation	df	Mortality MS	Mortality PR>F
Ergot level	4	8655.0	0.0001
Dietary formulation	1	32.09	0.71
Time	2	83.49	0.69
Ergot x diet	4	43.59	0.94
Ergot x time	8	687.6	0.003
Diet x time	2	61.76	0.76
Ergot x diet x time	8	73.09	0.96
Error	150	224.5	

*Each week is considered as a new period. Average cumulative percent mortalities for the 0, 1, 2, 4 and 8% ergot diets were 6.9, 4.2, 6.9, 30.6 and 83.3%, respectively.

2% ergot diets were not seen during weeks 2 and 3. In contrast, the mortality rate was high throughout the study for birds fed diets containing 4 and 8% ergot. Over the three weeks, 83% of the birds on the 8% ergot diet died compared to 31% on the 4% diet and 4% on the 1% diet (Table 19). At death, the majority of the birds displayed the following signs of ergot poisoning: black toes and nails, black feather shafts and a few with darkened beaks. Many also had poor feathering, particularly at the higher ergot levels. None of these symptoms were seen in birds alive at the end of the experiment, with the exception of some poor feathering.

Experiment IIIb

This experiment compared the effects of increasing ergot levels on the performance of Leghorn and broiler chicks (Tables 20-22). Analysis of variance (Table 22) indicated a number of significant interactions and main effects. There was an ergot x bird x time interaction ($P<0.001$) for the relative feed consumption. Ergot x time and ergot x bird interactions were obtained for relative weight gain ($P<0.006$ and $P<0.001$, respectively) and relative feed consumption ($P<0.0001$). All main effects were also significant ($P<0.0001$) for both parameters. The feed to gain ratio was only affected by ergot level ($P<0.001$).

The interaction of relative feed consumption within each time period and bird type due to increasing ergot levels (Table 20) was similar to that seen in earlier experiments. The general trend towards reduced feed consumption at the higher ergot levels was true for both the Leghorns and broiler chicks, although the broilers consistently consumed lower

Table 20. Effects of increasing wheat ergot levels on the relative feed consumption of Leghorn and broiler chicks over time (Experiment IIIb)

Week	1		2		3	
Bird type	Leghorn	Broiler	Leghorn	Broiler	Leghorn	Broiler
Ergot level (%)						
0	1.00 (87.2 g)*	1.00 (198.3 g)	1.00 (140.0 g)	1.00 (380.8 g)	1.00 (128.2 g)	1.00 (389.9 g)
1	0.93	0.95	0.88	0.93	0.87	0.70
2	0.86	0.69	0.72	0.54	0.64	0.55
4	0.66	0.45	0.44	0.29	0.44	0.27
8	0.43	0.26	0.30	0.14	0.20	0.11
SE	0.03	0.03	0.03	0.03	0.03	0.03

*Numbers in brackets are actual values for controls (per bird).

Table 21. Average effect of increasing wheat ergot levels on relative weight gain over time (combined Leghorn and broiler chick data) (Experiment IIIb)

	Relative weight gain		
Week	1	2	3
Ergot level (%)			
0	1.00 (88.4 g)*	1.00 (155.3 g)	1.00 (146.4 g)
1	0.91	0.87	0.91
2	0.70	0.52	0.54
4	0.38	0.27	0.31
8	0.16	0.13	0.04
SE	0.04	0.04	0.04

*Numbers in brackets are actual values for controls (per bird).

Table 22. Summary of analysis of variance (Experiment IIIb)

Source of variation	df	Relative weight gain		Relative feed consumption		Feed to gain	
		MS	PR>F	MS	PR>F	MS	PR>F
Main plot comparisons							
Ergot level	4	4.95	0.0001	3.42	0.0001	16.19	0.001
Bird type	1	0.18	0.0001	0.44	0.0001	3.87	0.26
Ergot x bird	4	0.05	0.001	0.055	0.0001	0.41	0.97
Main plot error (ergot x bird)	51	0.009		0.005		2.97	
Sub plot comparisons							
Time	2	0.094	0.0001	0.32	0.0001	1.78	0.65
Ergot x time	8	0.025	0.006	0.31	0.0001	1.56	0.93
Bird x time	2	0.008	0.39	0.001	0.79	2.22	0.58
Ergot x bird x time	8	0.008	0.47	0.015	0.001	1.68	0.91
Error	98	0.0085		0.004		4.05	

relative amounts of feed during the same period and at the same level, with the exception of the 1% level during weeks 1 and 2. There was also a general decrease in relative feed consumption over time. This decrease was more evident in the broiler chicks, suggesting that they were more susceptible to ergot toxicity than the Leghorns. There was however, little or no change in the relative feed consumption between weeks 2 and 3 for both bird types when fed the diets containing 4% ergot, and for the broilers also when fed 2% ergot. This trend was noted in Leghorn chicks in previous experiments, Experiments II and IIIa.

The nature of the ergot x time interaction for the relative weight gain is presented in Table 21, and it was similar to that described above for the relative feed consumption. Within each time period, there was a decrease in relative weight gain with increasing levels of ergot, but the effects became more pronounced over time, especially at the high ergot levels.

The overall relative weight gain values for the ergot x bird interaction for the 0, 1, 2, 4. and 8% ergot diets were 1.00 (182.8 g), 0.89, 0.64, 0.40 and 0.14, respectively for the Leghorn chicks and 1.00 (597.5 g), 0.91, 0.53, 0.24 and 0.08, respectively for the broilers. The broilers, with the exception of the 1% ergot diet, tended to have lower relative weight gains, particularly at the higher ergot levels, than Leghorn chicks. The general pattern of relative weight gain reduction due to increasing ergot levels again was similar to that seen in previous studies.

The overall average feed to gain ratios for birds fed 0, 1, 2, 4 and 8% ergot (w/w) were 1.77, 1.74, 2.06, 2.50 and 3.67, respectively.

Only the feed to gain ratio of the 8% ergot diet birds differed ($P>0.05$) from the other values.

A total of 150 Leghorn chicks (83%) died over the course of the study compared to 105 broilers (58%). Analysis of the mortality data (Table 23) revealed a significant ergot x bird x time interaction ($P<0.0001$) as well as bird x time ($P<0.007$) and ergot x time ($P<0.035$) interactions. Significant main effects were ergot level ($P<0.0001$), bird type ($P<0.04$) and time ($P<0.02$). The percent mortality seen in both bird types and generally in all time periods increased as the level of ergot increased (Table 23). The combined incidence of mortality during week 1 was unusually high for the Leghorn chicks on the low ergot (0 to 2%) diets, especially when compared to the broilers (6 vs 1% overall mortality, respectively). During weeks 2 and 3, the overall percent mortalities for both bird types did not exceed 1% for the combined lower ergot levels. The highest percentage of deaths occurred on the 8% ergot diet, and although the total percentage was similar between bird types (81% for Leghorns and 78% for broilers), the pattern over time was somewhat different. The Leghorn mortality on the 8% ergot diet was high throughout, but it dropped markedly in week 2, rising again in week 3 (Table 23). In contrast, broiler mortality was moderate and low in weeks 1 and 3, respectively jumping dramatically in week 2.

The majority of birds displayed the same symptoms in death, regardless of strain. Most had black toes, nails and feather shafts, and many had poor feathering. The most severely affected also had dark beaks. No other birds showed any signs of ergot poisoning at the end of the experiment, with the exception of poor feather development

Table 23. Average percent weekly pen mortality of Leghorn and broiler chicks fed increasing ergot levels (Experiment IIIb)*

	Week					
	1		2		3	
Bird type	Leghorn	Broiler	Leghorn	Broiler	Leghorn	Broiler
Ergot level (%)						
0	5.33	0.00	0.00	2.67	0.00	0.00
1	2.67	0.00	0.00	0.00	3.33	0.00
2	11.00	2.67	2.67	0.00	0.00	0.00
4	2.67	2.67	25.33	6.00	2.67	2.67
8	41.33	24.67	24.33	65.00	47.33	8.33
SE	4.80	4.80	4.80	4.80	4.80	4.80

Experiment IIIb

Summary of analysis of variance - mortality

		Mortality	
Source of variation	df	MS	PR>F
Ergot level	4	7649.1	0.0001
Bird type	1	583.2	0.04
Time	2	571.4	0.02
Ergot x bird	4	44.9	0.86
Ergot x time	8	296.3	0.035
Bird x time	2	709.3	0.007
Ergot x bird x time	8	1211.1	0.0001
Error	150	137.9	

*Each week is considered as a new period. Average cumulative percent mortalities for the 0, 1, 2, 4 and 8% ergot diets were 5.6, 5.6, 13.9, 30.6 and 80.6%, respectively, for the Leghorns and 2.8, 0.0, 2.8, 11.0 and 77.8%, respectively, for the broiler chicks.

in some individuals on the 8% ergot diet.

Experiment IV

The ability of Leghorn chicks to recover from short term (32 days) exposure to ergot contaminated feed was examined in this experiment (Tables 24-27). Analysis of the data (Table 27) indicated highly significant interactions between ergot level and time for relative weight gain ($P<0.0016$), relative feed consumption ($P<0.0001$) and feed to gain ratio ($P<0.0017$). Significant ergot x diet x time ($P<0.01$) and diet x time ($P<0.02$) interactions were also found for feed to gain ratio. In addition, significant effects were noted due to dietary formulation, ergot treatment and time for relative weight gain and relative feed consumption, and also due to time for feed to gain ratio (Table 27).

Little or no improvements were seen in birds which remained on the ergot contaminated diets (Table 24). In contrast, those birds which had an ergot-free feed substituted for the ergot-containing diets showed dramatic improvements in performance. The greatest degree of recovery was observed in those birds previously fed diets which contained the higher concentrations of ergot. For example, the birds maintained on the 4% ergot diets had a relative weight gain of only 0.14 during week 1, whereas those switched to the ergot free diet had a value of 0.50; an improvement of 357%. The difference was even more pronounced during week 2, 0.82 versus 0.17, respectively; a 482% improvement. These results demonstrate that chicks given the ergot free diets displayed an increased recovery rate over time, while little or no change occurred in those remaining on ergot contaminated feed. Although not

Table 24. Comparison of the performance of Leghorn chicks, previously fed ergot contaminated diets, when given ergot free or ergot containing diets (Experiment IV)

		Relative weight gain		Relative feed consumption		Feed to gain	
Week		1	2	1	2	1	2
Ergot level fed (%)	Previous ergot level fed (%)						
0	0	1.00 (126.3 g)*	1.00 (134.5 g)	1.00 (288.8 g)	1.00 (354.2 g)	2.32	2.65
0	1	0.96	1.01	0.99	0.99	2.60	2.40
0	2	0.84	0.99	0.75	0.86	2.05	2.30
0	4	0.50	0.82	0.48	0.63	2.23	2.01
1	1	0.94	0.92	0.98	0.97	2.40	2.78
2	2	0.64	0.73	0.68	0.75	2.44	2.73
4	4	0.14	0.17	0.34	0.33	5.55**	5.11**
SE		0.04	0.04	0.005	0.005	2.90	2.90

*Numbers in brackets are actual values for controls (per bird).

**Values based on total feed consumption and total weight gain of treatment pens rather than the the averages of pen feed to gain ratios because of an extreme bias due to one pen.

Table 25. Comparison of the feed to gain ratio of Leghorn chicks on two dietary formulations previously fed ergot contaminated diets when given ergot free feed to those kept on ergot diets (Experiment IV)

		Diet 1		Diet 2	
Week		1	2	1	2
Ergot level fed (%)	Ergot level previously fed (%)				
0	0	2.26	2.61	2.34	2.67
0	1	2.57	2.61	2.22	2.59
0	2	2.46	2.72	2.34	2.83
0	4	2.12	2.36	1.98	2.23
1	1	2.42	2.86	2.45	2.61
2	2	2.13	1.91	2.32	2.12
4	4	4.61	5.32	7.84*	4.92*
SE		2.90	2.90	2.90	2.90

*Values based on total feed consumption and total weight gain of treatment pens rather than averages of pen feed to gain ratios because of an extreme bias due to one pen.

Table 26. Effect of diet on weight gain and feed consumption of Leghorn chicks (Experiment IV)

Diet formulation	Relative weight gain	Relative feed consumption
1	0.76	0.78
2	0.86	0.84
SE	0.03	0.01

Table 27. Summary of analysis of variance (Experiment IV)

Source of variation	df	Relative weight gain		Relative feed consumption		Feed to gain	
		MS	PR>F	MS	PR>F	MS	PR>F
Main plot comparisons							
Ergot treatment	6	0.85	0.0001	0.63	0.0001	53.31	0.80
Dietary formulation	1	0.09	0.0026	0.024	0.032	117.71	0.30
Ergot x diet	6	0.014	0.16	0.005	0.37	100.41	0.47
Main plot error (ergot x diet)	27	0.008		0.0047		105.05	
Sub plot comparisons							
Time	1	0.156	0.0001	0.037	0.0001	295.16	0.013
Ergot x time	6	0.033	0.0016	0.01	0.0001	204.80	0.0017
Time x diet	1	0.002	0.59	0.00001	0.91	257.57	0.02
Ergot x diet x time	6	0.005	0.32	0.0007	0.55	197.11	0.01
Error	27	0.0067		0.0001		42.01	

as dramatic, the same trend was also evident in the relative feed consumption results. The pattern for the feed to gain ratios was less clear (Table 24). Generally there was a decrease in the feed to gain ratio over time for birds that were fed the ergot-free formulations with the values, in most cases, being lower than control values. In contrast, birds that continued to consume ergot in their diets, with the exception of those fed 4% ergot, had feed to gain ratios similar to the control groups.

The ergot x time x diet interaction for feed to gain ratio (Table 25), though significant ($P<0.02$), did not demonstrate any clear trends. The high negative and positive values with the diets containing 4% ergot, particularly on diet 2, may have biased the data, thereby suggesting the interaction. An interpretation of this data will therefore not be attempted.

The data summarized in Table 26 demonstrated that birds fed diet 1 ate ($P<0.032$) and gained ($P<0.0026$) less relative to the controls than those on diet 2. There was however no difference ($P>0.30$) in the average feed to gain ratios.

A total of two birds died during this study, one each on the 4% ergot containing and ergot-free diets. Signs of ergotism included dark toes and nails and black feather shafts.

Experiment V

The purpose of this experiment as summarized in Table 28 was to isolate the toxic component(s) in ergot. Statistical analysis of the data indicated highly significant fraction type x level interactions

Table 28. Effect of feeding different wheat ergot fractions on Leghorn chick performance (Experiment V)

Fraction type	Ergot equivalent level in diet (%)	Relative weight gain	Relative feed consumption	Feed to gain
Control	0	1.00 (54.7 g)*	1.00 (110.0 g)	2.01
Ground ergot	1.5	0.49	0.68	2.80
	3.0	0.19	0.44	4.94
Oil extracted	1.5	0.99	0.95	1.95
	3.0	0.95	0.94	2.00
Alkaloid extracted	1.5	0.88	0.89	2.05
	3.0	0.83	0.84	2.05
Residue	1.5	0.95	0.93	1.98
	3.0	0.97	0.96	1.99
Recombination	1.5	0.88	0.91	2.09
	3.0	0.85	0.87	2.06
SE		0.018	0.013	0.08

*Numbers in brackets are actual values for controls (per bird).

and fraction type and level effects for all parameters ($P<0.0001$) (Table 29). The majority of these effects are attributable to the difference between birds fed the control diet and those fed the unextracted ergot-containing diet. Chick performance was not greatly affected by any of the ergot fractions, including the recombination fraction. The recombination and alkaloid fractions, however, tended to be more toxic than the oil extracted and residue fractions.

Only one death occurred during the study, and it was on the 1.5% level of the unextracted ergot. At the end of the experiment, only one bird was seen to have black toes, nails and feather shafts, and it was on the 3% unextracted ergot.

Experiment VI

This experiment examined the effect of chlorine gas or heat treatment at 200°C on reducing ergot toxicity. The treated ergot samples were fed at a level of 4% while untreated ergot was fed at various levels in an attempt to determine the effectiveness of the treatments. The results are presented in Table 30.

Chlorine gas treatment of ground ergot for 30 or 60 minutes reduced the effect of ergot on chick performance to a level equivalent ($P<0.05$) to a diet containing 2% ergot. Treatment of the ergot with chlorine gas for only 15 minutes was less effective ($P<0.05$) causing a response somewhat intermediate to those obtained with the 2 and 4% untreated ergot diets.

The results due to the heat treated ergot suggest a time dependent

Table 29. Summary of analysis of variance (Experiment V)

Source of variation	df	Relative weight gain		Relative feed consumption		Feed to gain	
		MS	PR>F	MS	PR>F	MS	PR>F
Fraction type	4	0.81	0.0001	0.31	0.0001	8.24	0.0001
Fraction level	1	0.10	0.0001	0.06	0.0001	2.85	0.0001
Type x level	4	0.05	0.0001	0.03	0.0001	2.74	0.0001
Error	61	0.002		0.001		0.14	

Table 30. Comparison of the effect of untreated wheat ergot at various levels to treated ergot samples fed at 4% (w/w) on Leghorn chick performance (Experiment VI)

Treatment		Relative weight gain	Relative feed consumed	Feed to gain
Untreated ergot (%)	0.0	1.00^{a*} (61.4 g)**	1.00^{a} (121.5 g)	1.98^{a}
	1.0	0.96^{a}	1.00^{a}	2.05^{a}
	2.0	0.61^{b}	0.82^{bc}	2.64^{b}
	4.0	0.28^{ef}	0.59^{e}	4.20^{e}
Treated ergot (4%)	Cl_2 15 minutes	0.46^{d}	0.72^{d}	3.10^{c}
	Cl_2 30 minutes	0.61^{b}	0.81^{bc}	2.66^{b}
	Cl_2 60 minutes	0.62^{b}	0.83^{b}	2.66^{b}
	Heat 10 minutes	0.33^{ef}	0.62^{e}	3.75^{d}
	Heat 15 minutes	0.37^{e}	0.65^{e}	3.48^{d}
	Heat 20 minutes	0.52^{cd}	0.75^{d}	2.87^{bc}
	Heat 30 minutes	0.55^{bc}	0.76^{cd}	2.78^{bc}
SE		0.02	0.02	0.12

*Means in the same column with different superscripts are significantly different ($P<0.05$).

**Numbers in brackets are actual values for controls (per bird).

inactivation of the toxic components in ergot; the longer the time of treatment, the greater the effect. The 30 minute heat treatment reduced ergot toxicity by approximately 50%. Only slight improvements ($P>0.05$) in performance were obtained when ergot was heated for 10 or 15 minutes (Table 30).

No mortalities occurred during this study, and other than some reduced growth and general sluggishness at the high ergot levels, no physical evidence of ergot ingestion was noted.

Experiment VII

The purpose of this experiment was to see if the toxic effects of ergot could be mitigated by the addition of Antitox Vana to the diets (Tables 31 and 32). Statistical analysis of the data showed that ergot level, time and an ergot level x time interaction significantly ($P<0.0001$) affected relative weight gain and relative feed consumption. The effect of the increasing ergot levels over time (Table 31) was similar to that seen in earlier experiments, with the toxic effect being greatest in birds given the highest ergot concentration in the feed. Over time, the effect of the ergot also became more pronounced, particularly at the 2 and 4% ergot levels. The average percent reduction in relative weight gain and relative feed consumption, comparing week 2 to week 1, was approximately 30% for both parameters at both levels.

The feed to gain ratio was only affected by ergot level ($P<0.0001$) and time ($P<0.06$). There was an increase in feed to gain ratio with increasing levels of dietary ergot with the overall values for the 0, 1, 2 and 4% ergot diets being 1.94, 2.06, 2.43 and 3.39 respectively. The

Table 31. Average effect of feeding different levels of wheat ergot in combination with different levels of Antitox Vana on Leghorn chick performance (Experiment VII)*

	Relative weight gain		Relative feed consumption		Feed to gain	
Week	1	2	1	2	1	2
Ergot level (%)						
0	1.01 (57.35 g)**	0.99 (78.42 g)	0.99 (109.63 g)	0.99 (154.82 g)	1.91	1.98
1	0.92	0.89	0.95	0.94	2.03	2.08
2	0.66	0.46	0.79	0.57	2.35	2.52
4	0.36	0.25	0.60	0.41	3.31	3.46
SE	0.02	0.02	0.016	0.016	0.016	0.16

*Antitox Vana was added to each ergot level at levels of 0.0, 0.1, 0.2 and 0.8% (w/w).

**Numbers in brackets are actual values for controls (per bird).

Table 32. Summary of analysis of variance (Experiment VII)

Source of variation	df	Relative weight gain		Relative feed consumption		Feed to gain	
		MS	PR>F	MS	PR>F	MS	PR>F
Main plot comparisons							
Antitox Vana level	3	0.002	0.58	0.001	0.58	0.12	0.39
Ergot level	3	4.95	0.0001	2.50	0.0001	20.67	0.0001
Ergot x Antitox Vana	9	0.003	0.54	0.003	0.13	0.09	0.65
Main plot error	80	0.0029		0.0017		0.12	
Sub plot comparisons							
Time	1	0.38	0.0001	0.53	0.0001	0.56	0.06
Antitox Vana x time	3	0.0001	0.98	0.0003	0.91	2.27	0.91
Ergot x time	3	0.09	0.0001	0.16	0.0001	0.04	0.86
Ergot x Antitox Vana x time	9	0.002	0.79	0.0003	0.99	0.05	0.98
Error	80	0.003		0.0015		0.16	

average feed to gain ratios during the first and second week of the experiment were 2.40 and 2.51, respectively.

Antitox Vana had no effect ($P>0.10$) on reducing ergot toxicity, even at four times the manufacturer's recommended level, 0.8%. Differences between relative weight gain and relative feed consumption did not exceed 1%.

A total of ten birds died during the experiment, all at the 4% ergot level. All but one died during the second week of the test, and they tended to be evenly distributed over the four levels of Antitox Vana (0 to 0.8%). At death, all the birds showed signs of ergotism including darkened nails and toes, and in some, black feather shafts and poor feather development. None of the birds alive at the end of the study displayed these symptoms.

DISCUSSION

The presence of ergot in a feedstuff can substantially affect the performance of domestic livestock. As a general rule, a limit of up to 0.1% ergot is considered allowable in the feed of most species (Young 1979). While this appears to hold true for cattle, sheep, horses and swine, poultry can tolerate higher levels. Earlier studies proposed limits of 0.3 (O'Neil and Rae 1965) and 0.8% (Bragg et al. 1970) ergot in poultry feed for Leghorn and broiler chicks, respectively. Unfortunately, the majority of reports on ergot poisoning in animals have not taken the alkaloid composition or the level of the total alkaloids into account when suggesting limits. The composition and levels of total alkaloids may be highly variable between sclerotia even from the same head, field and region (Young 1981a), so differences between studies in terms of ergot toxicity are not surprising. In the current studies, the alkaloid composition and levels of all ergot samples utilized were chemically determined (Appendix 1) and so could be related to the toxicity of the ergots. This will be discussed later.

The effects of increasing levels of dietary ergot on chick performance over short periods of time were examined in a series of experiments (Experiments II, IIIa and IIIb) using prairie wheat ergot containing 0.308% total alkaloids. The general trends were similar to those obtained by other authors (O'Neil and Rae 1965; Bragg et al. 1970). In addition, ergot level x time of exposure interactions were noted for relative weight gain and relative feed consumption in the current studies. The same trends were seen in all three experiments. In these studies,

birds fed the 1% ergot diets showed a very slight but uniform decrease in relative feed consumption and relative weight gain over time, while the relative performance of the 6 and 8% ergot-fed birds decreased progressively and uniformly during the time course of the studies. At the intermediate ergot levels (2-5%) however, the relative weight gain and relative feed consumption initially tended to decrease at a steady rate, and then after about two weeks, stabilize or even increase slightly (Fig. 3; Tables 12, 16 and 20). The results suggest that not only do the effects of ergot increase as its concentration increases, but they also changed over time.

The nature of the diet however did not appear to modify the toxicity of ergot. Similar performances at all ergot levels were obtained for birds fed the diets formulated to meet the requirements of Leghorn (diet 1) and broiler (diet 2) chicks (Experiment IIIa). The lack of differences in response is likely due to the fact that the two diets differed only in protein content and that this difference was only 1.5%. Possibly a greater difference in composition between the diets would have resulted in different conclusions. No effect of dietary composition on ergot toxicity has been reported by other researchers.

Broiler chicks appeared to be much more sensitive to the effects of ergot, particularly at higher levels, than Leghorn chicks (Experiment IIIb). Some of this difference may be attributed to the faster rate of gain and therefore the greater feed consumption by broiler chicks. Even at the 1% ergot level, the broilers ate progressively lesser relative amounts of feed over time than Leghorns (Table 20). As they ingested

greater amounts of feed in absolute terms, the broiler chicks would also have ingested greater amounts of ergot and so displayed a greater response, especially at the high dietary ergot levels. Young and Marquardt (1982) in contrast, found that broiler and Leghorn chicks fed ergotamine tartrate were affected in a similar manner. The number of birds used in their studies may not have been large enough to demonstrate a difference or the response of the two strains of poultry to ergot and ergotamine tartrate may be different.

The reason for the observed lower weight gains and feed consumption of ergot fed animals compared to controls has not been established for any species. For poultry, part of the effect may be due to a direct appetite depressing effect of the ergot, or merely to the presence of ergot in the feed. Poultry are known to avoid whole ergot sclerotia in their feed and to reject previously contaminated feed as the smell or taste of the ergot may still be present (Mantle 1978c). In the current experiments, the ergot was finely ground and thoroughly mixed in the diet. The birds had no other source of nutrition and so were forced by hunger to consume the contaminated feed. Therefore, the amount of feed consumed, at least initially, may have been dependent on the relative balance between hunger and the repulsive nature of the ergot contaminated feed.

Having ingested the contaminated feed, it is likely that some constituent alkaloids acted on the appetite centre in the hypothalamus, causing a reduction in appetite (Burfening 1973), leading to a further reduction in relative feed consumption and in turn, lowered relative

weight gains and increased feed to gain ratios. A reduction in efficiency of feed utilization can readily occur when feed intake is reduced, due to the greater percentage of the absorbed nutrients being utilized for maintenance purposes. Another possible factor which may be involved in reducing chick weight gain and increasing the feed to gain ratio, particularly at the higher ergot levels, is that the ingested ergotamine could have acted to increase peristalic activity and the rate of passage of solid material along the GI tract (Lorenz 1979). This would tend to reduce the amounts of nutrients which the chicks could extract from their feed. Also possible is that some components of ergot interfere with nutrient digestion and absorption. In contrast, however, ergotamine and possibly other alkaloids may act indirectly to improve nutrient utilization through vasoconstriction, reducing heat loss from the extremities, and thereby reducing some metabolic costs. This is supported by the results of Young and Marquardt (1982) who reported that when ergotamine tartrate was fed at low levels, feed intake and weight gain tended to be reduced, but feed efficiency was improved. In the current studies, at all levels of dietary ergot tested and the higher levels of ergotamine tartrate fed by Young and Marquardt (1982), there was a dramatic increase in the feed to gain ratio. More extensive studies are needed to examine these possibilities.

An important question which has not yet received much attention is the degree to which ergot alkaloids are absorbed and broken down. While no such work has been reported in poultry, Franz et al. (1980) found

that only 10-30% of ingested hydrogenated alkaloids were absorbed from the GI tract of rats. Bailey et al. (1973) suggested that alkaloids are poorly absorbed in swine, while Whittemore et al. (1976) reported that up to 90% were absorbed. It is conceivable that the degree of alkaloid absorption in poultry is very low as they are able to consume much higher concentrations of dietary ergot than mammals without showing any effects. In addition, the alkaloids could be more efficiently modified by the intestinal flora of the chicken before absorption, or may be more efficiently detoxified by avian tissues.

Poultry appear to have the same general mechanism of detoxification as has been proposed for other animal species (Mantle 1968; Whittemore et al. 1976; Robinson 1981) and man (Lorenz 1979), and the liver appears to be the site of degradation of absorbed alkaloids. Studies on poultry and other avian species have demonstrated the presence of liver microsomal drug metabolizing enzymes which seem to be responsible for the metabolism of a variety of endogenous (steroids, fatty acids and bile acids) and exogenous (drugs, carcinogens and pesticides) compounds. The rate at which these compounds are metabolized varies greatly and is dependent on the species and strain as well as age, tissue nutritional status and pretreatment of the animal (Lu 1976). O'Brien et al. (1983) noted that of a range of animal species tested, quail showed the most rapid metabolism of aflatoxin B by liver microsomal enzymes. This detoxification mechanism may also be involved in the metabolism of ergot alkaloids, but it may be slow to develop and then only be effective at low levels of the toxin(s). Chicks fed diets containing 2-5% ergot in Experiment II and those given the 2 and 4% diets in Experiments IIIa and

IIIb tended to stabilize or slightly increase their weight gains and feed consumption relative to the control birds, but only after about two and one half weeks of exposure (Tables 12, 16 and 20; Fig. 3). Young and Marquardt (1982) had also previously noted that differences between control and ergotamine tartrate fed birds remained constant after about three weeks on test. The increase in relative weight gain and relative feed consumption evidenced during week 4 of Experiment II for the 2-5% ergot diets was also seen in those Leghorn chicks from Experiment IIIa which were carried over on the 2 and 4% ergot diets during Experiment IV (Table 24). These results appear to lend support to the idea of an active detoxification mechanism in poultry, but whether it is only poorly developed in young birds or needs to be stimulated is unclear.

Very little information has been published concerning the ability of animals to recover from ingestion of ergot contaminated feed. The incident cited by Lindley (1978) resulted in apparently full recovery of affected horses, but they were only exposed to the ergot containing feed for about 24 hours. Bragg et al. (1970) suggested that the toxic components of ergot had a cumulative effect on chicks, but this is probably not due to an accumulation of toxicants in the body. Only trace amounts of ergotamine were detected in the body tissues of chicks fed up to 810 ppm of ergotamine tartrate in their diets (Young and Marquardt 1982). It is conceivable though that there may have been an accumulation of ergotamine metabolites.

In the current study (Experiment IV), it was shown that Leghorn chicks which were fed up to 4% of wheat ergot in their diets for a 32

day period demonstrated an ability to recover to a considerable degree when given ergot free feed. The birds showed a dramatic improvement in performance after only one week on an ergot free diet, and this recovery continued through week 2 (Table 24), particularly for those previously fed the higher ergot levels. The feed to gain ratios were also greatly improved. The data suggests that birds given ergot free feed appear to recover more in a compensatory fashion than merely as delayed growth. There is no evidence to suggest that these birds would ever have fully gained the weight they should have put on during the period they were consuming ergot. The results do show however that a considerable recovery potential exists for Leghorn chicks even after more than four weeks of exposure to diets containing as high as 4% wheat ergot (0.308% total alkaloids). A more complete and rapid rate of recovery may occur in birds exposed for shorter periods.

The highest incidences of mortality were seen in Experiments II, IIIa and IIIb. The number of deaths increased as the ergot level rose above 4% in the diet. Although the birds in Experiments IIIa and IIIb appeared to be healthy prior to the start of the experiment, an unusually high number died early in the studies on the control and low ergot level diets. Shortly after the start of these studies, the birds were subjected to stress due to internal building construction directly adjacent to their location. This stress may have contributed to the deaths of some Leghorn chicks as no signs of ergot poisoning were visible in birds fed low ergot levels. Stressed animals may also have been more susceptible to the effects of ergot (Young 1979). The broiler chicks

did not appear to have been stressed in a similar manner.

The incidence of mortality tended to increase with increasing ergot levels and time. This was most clearly seen in Experiment II, as mortalities occurred only on the 4% and higher ergot level diets (Table 15). Similar mortalities were also obtained by Bragg et al. (1970). In the current studies, the percentage of birds dying each week was low for weeks 1 and 2, being under 7%, even on the highest ergot level. In weeks 3 and 4, the percentage jumped dramatically, most notably to 72% on the 6% ergot diet during week 4. Overall, the total percent mortalities after four weeks were 18.3, 47.9 and 77.1% for the 4, 5 and 6% ergot fed birds. The results for the Leghorn chicks during week 1 of Experiments IIIa and IIIb are somewhat misleading due to the high mortalities especially for those on the low ergot level diets (0 to 2%) (Tables 19 and 23). The reason for this effect was discussed previously.

A higher incidence of mortality was anticipated for the broiler chicks, especially considering the dramatic effects of ergot on reducing their performance (Experiment IIIb). Overall however, only 58% of the broiler chicks on test died compared to 83% of the Leghorns. Possibly, the broilers still consumed enough feed to maintain themselves or were of sufficiently larger size that the toxic effects of ergot was less severe compared to the Leghorns. Also, as the broilers appeared initially to be healthier than the Leghorns, they may have been better able to resist the effects of ergot. The overall trend of mortalities for the broilers over time at the various ergot levels however was similar to that noted for the Leghorn chicks, only the values were somewhat lower for the latter.

Bragg et al. (1970) observed poor feathering, loss of coordination and an inability to stand in addition to the darkening of beaks, toenails and toes in many of the ergot affected birds. Similar observations were noted for the most severely affected birds in all the current studies. While Bragg et al. (1970) stated that these signs appeared gradually until the tissue turned black at death, they generally appeared only a day or so before death in the majority of cases observed in the current study. Also, there was no incidence of skin necrosis as evidenced by Young and Marquardt (1982) when they fed chicks pure ergotamine tartrate. Part of the differences in results noted by different researchers may be attributed to the nature of the ergot samples and their alkaloid composition. The observed darkening of toenails, toes and beaks and poor feather development in the more severely affected animals was likely caused by ergotamine and possibly other vasoconstricting alkaloids in the ingested ergot, as they cause reduced blood flow to the extremities. Had any of these birds been sacrificed prior to their "natural" death, it is likely that an enlarged heart would have been initially evident (Young and Marquardt 1982), and possibly the livers engorged with blood and congested kidneys as seen in infected ducks (Swarbrick and Swarbrick 1968). These factors could have directly contributed to an affected animal's death, or acted in combination with any number of factors, including malnutrition. The relative concentration of the vasoconstricting alkaloids may have been higher in the ergot ingested by these animals than was used in the current studies.

A number of treatments to reduce ergot toxicity were tested in

Experiments VI and VII. The best results were obtained when ground ergot was treated with chlorine gas for 30 or 60 minutes and with heat for 30 minutes (Table 30). All three treatments reduced the toxic effects of a 4% ergot contaminated diet to the equivalent of a 2% contamination. Lesser reductions were obtained with the shorter exposure times. There was little or no additional improvement in animal performance when the time of exposure of ergot to the chlorine gas was increased above 30 minutes. In contrast, the longer the ergot was kept at 200°C, the less toxic its effect. Young et al. (1983) conducted chemical analysis on whole ergot sclerotia, obtained from the same source as used in the current studies, following various treatments including chlorine gas and heat at 200°C for up to 96 hours. They found that the heat treatment reduced the percentage of total alkaloids remaining to 10% after only about one hour of exposure. In the current study it was assumed that a shorter exposure time would have been required to achieve a similar reduction in the total alkaloid level since ground ergot rather than whole ergot was subjected to chemical treatment. Presumably, had the ground ergot been exposed to 200°C for a longer time period, a further reduction in toxicity would have occurred. The chlorine gas treatment of whole ergot sclerotia by Young et al. (1983) also caused a reduction of the total alkaloids to 10%, but after a period of four hours. In contrast, pure alkaloids on a glass surface decomposed completely after only a few minutes. The difference between the results may be attributed to a delay in the diffusion of the gas into the sclerotia. Again, a much longer time of exposure of the ground ergot to chlorine gas may

have resulted in further improvements in chick performance, but to what extent is unknown.

All the chlorine and heat treatments used in Experiment VI were carried out on ground ergot sclerotia prior to its being mixed in the feed. How effective or economical these procedures would be on whole ergot mixed with grain is unknown and beyond the scope of this study. However, it is known that heating does affect proteins and carbohydrates, especially in the presence of moisture. The effect of proper heating is to denature protein and make its digestion more efficient, particularly in young monogastric animals (Sunde 1972). Heat treatment of contaminated grain may therefore not only affect its nutritive value, but also tend to destroy some of the toxic components in the ergot.

Addition of Antitox Vana to ergot contaminated diets (Experiment VII) had no effect on lessening the toxicity of ground wheat ergot containing 0.308% total alkaloids. The manufacturer, Vana, suggested inclusion of Antitox Vana at a level of 0.15 to 0.2% of a contaminated diet, but even 0.8% inclusion had no effect. The antitoxicant is claimed to be effective against mycotoxins, including ergot, by binding ingested mycotoxins and so preventing membrane irritation, absorption and damage to intestinal flora (Vana, promotional literature), but no evidence of this was seen. More detailed studies of this product, particularly with whole ergot sclerotia, may be necessary to demonstrate its effectiveness against ergot. The current results however, suggest that it is ineffective against ergot toxicity.

In a series of trials (Experiment I), it was shown that ergot from

different sources can have considerably different effects in terms of toxicity even when included in Leghorn chick diets at equivalent levels of total alkaloids. For instance, when fed at the 45 ppm level of total alkaloids, the triticale ergot depressed relative weight gain by 40% while the Ottawa rye ergot sample depressed relative weight gain by 51%. If they had been included in the diets on a w/w basis rather than equivalent proportions, the difference in toxic effect would have been greater as the total alkaloid contents of the triticale ergot was lower than that of the Ottawa rye ergot (Appendix 1). The toxic effects due to increased dietary concentrations of ergot alkaloids also did not produce a uniform response. When the total alkaloid content of the feed was doubled from 45 to 90 ppm, the relative weight gain of birds fed the North River rye ergot decreased by 35% for the 90 ppm level compared to the 45 ppm fed birds (0.65 vs 0.42, respectively). In contrast, the triticale ergot sample caused a corresponding decrease of 65% (0.60 and 0.21, respectively) in relative weight gain. The results from Experiment I also indicate the relationship between the toxicity of a sample and the individual alkaloid composition is not clear. The correlation coefficient between the percentage of the three predominant alkaloids common to all ergot samples tested, ergosine, ergotamine and ergocristine, and the effects on relative weight gain, relative feed consumption and feed to gain ratio for the 45 ppm total alkaloid diets were 0.54, 0.47 and -0.50, respectively and 0.21, 0.39 and -0.19, respectively on the 90 ppm diets. The questionable relationship between ergot composition and toxicity was further illustrated by the

effects on chick performance of the two rye ergot samples from PEI. While these samples had similar alkaloid compositions, their toxic effects differed when fed at equivalent total alkaloid concentrations (Table 10).

The results from this current study suggest that ergot toxicity for young chicks can not reliably be estimated from total ergot body counts, total alkaloid levels or even from the estimation of certain individual alkaloids in the ergot. There is considerable variation in the total alkaloid level and composition between ergot samples (Young 1981a), and this series of studies showed they can have very different effects on chick performance even when fed at equivalent total alkaloid levels. The failure to obtain a clear relationship between the toxicity of a given ergot sample and its total alkaloid content may be due to the different physiological and metabolic effects of the twenty plus ergot alkaloids (Berde and Sturmer 1978), each with its own particular dose response effects. Many of the effects of ergot can be attributed to the differential synergistic, antagonistic and additive responses among its toxic components. Other non-alkaloid or related compounds in the ergot may also affect chick performance.

In an attempt to isolate the toxic components of ergot and examine the possibility of the presence of non-alkaloid toxic components, wheat ergot was separated into different fractions (Experiment V). The majority of the toxicity was found in the alkaloid fraction, but the oil extracted and residue fractions also contained some toxic activity (Table 28). Analysis of the latter two fractions demonstrated they

contained little or no alkaloids (Young, pers. commun.), further suggesting the presence of another antinutritional factor, or possibly simply a palatability problem. Unfortunately, most of the ergot toxicity was destroyed during the isolation procedure and so no conclusive evidence for or against the presence of antinutritional factor(s) other than the alkaloids was established. Rather than attempting further isolation procedures, an alternative method to demonstrate the presence or absence of such a factor would be to formulate synthetic mixtures of ergot alkaloids so that the composition of the mixture would be the same as a given ergot sample. The mixture and ergot could then be compared in pair feeding studies. Young and Marquardt (1982) reported that a diet containing crude ergot was four times more toxic to chicks than an equivalent amount of pure ergotamine tartrate. It must be noted that in this study the effects of only a single alkaloid were considered.

The current study nevertheless did show that ergot alkaloids are fairly labile since a large percentage of the alkaloid toxicity was lost during a standard isolation procedure. It is also conceivable that some alkaloids are affected to a greater degree than others and that at least some destruction may occur during the extraction of an ergot sample for routine chemical analysis. Possibly too, the alkaloid extraction may be incomplete. If so, chemical analysis may give a low and somewhat variable estimation of the total alkaloid content of a sample. This too could explain some of the differences between observed and predicted results in animal feeding trials. Although chemical analysis is superior to merely estimating the percentage of ergot in a feed sample for

predicting ergot toxicity, it does not yet seem to provide a highly reliable measure of its toxicity to a given species. For this, feeding trials are indispensible. Development of a reliable model or method for estimating the toxicity of ergot contaminated grain from chemical analysis requires much more investigation. The toxicity of individual alkaloids, either singly or in combination, must be known before accurate predictions of the toxic effects of an ergot sample given to any animal species at any stage of life can be made.

SUMMARY AND CONCLUSIONS

1) Ergots from different sources and geographic locations have variable effects on chick performance, even when fed at equivalent concentrations of total alkaloids. Also, chick performance was not depressed uniformly among the different ergots when the dietary concentration was increased. These findings suggest that there is not a simple relationship between the total alkaloid content of an ergot sample and its effect on chick performance.

2) Growing chicks may be able to tolerate higher levels of dietary ergot than previously reported, but the total alkaloid level and composition must be considered when defining the exact permissible level of a given ergot sample.

3) When different fractions of extracted wheat ergot were fed to Leghorn chicks, not all the toxic activity was contained in the alkaloid fraction. This would suggest that there may be toxic components other than alkaloids in ergot.

4) Ergot alkaloids appear to be very labile when subjected to standard extraction procedures. During such procedures, there may be a loss of some alkaloids, resulting in low estimates of the actual alkaloid content of an ergot sample. Modified or new procedures should be developed to ensure that intact biologically active fractions are obtained from ergot.

5) Broiler chicks were found to be more sensitive to the effects of ergot in terms of decreased performance, than Leghorn chicks. The corresponding incidence of mortality, however, appeared to be

greater for the Leghorns.

6) Chick performance was affected by both the dietary concentration of ergot and the length of time the birds were exposed to it. There was a progressive decrease in relative performance as the dietary concentration of ergot increased and this decrease was enhanced as the time of exposure increased. However, the differences in weight gain and feed consumption appeared to remain constant or increase slightly relative to control birds after about three weeks of exposure.

7) Leghorn chicks, previously fed up to 4% of a wheat ergot contaminated diet for four weeks, demonstrated a significant ($P<0.0001$) and continuing ability to recover from the effects of ergot when placed on an ergot-free diet.

8) Treatment of ground wheat ergot with chlorine gas or heat at 200^{o}C for 30 minutes reduced its effect on Leghorn chicks by about 50%. Smaller reductions were obtained with shorter exposure times. It is not known if these treatments would be equally effective on whole sclerotia or ergot already in a grain or feed mixture.

9) Antitox Vana (polyvinyl pyrrolidone), a mycotoxin antitoxicant, when added to a diet at four times the manufacturer's recommended level did not reduce the toxic or antinutritive effect of ergot.

10) More detailed studies are required on the mechanism of how ergot affects poultry, particularly on the mode by which the toxic components of ergot are absorbed and their sites of action. Continued emphasis must also be placed on finding methods to reduce

or eliminate the toxic effects of ergot. Much more research must also be carried out to more clearly define the relationship between the individual alkaloid composition, their sum total concentrations in ergot and their biological effects on poultry.

LITERATURE CITED

ANDERSON, J.F. and R.E. WERDIN. 1977 Ergotism manifested as agalactia and gangrene in sows. J. Amer. Vet. Med. Assoc. 170:1089-1091.

ATAL, C.K. and A.E. SCHWARTING. 1957. The variability of alkaloid content of domestic ergot. J. Amer. Pharm. Assoc. 46:451.

BAILEY, J., A.E. WRATHALL and P.G. MANTLE. 1973. The effect of feeding ergot to gilts during early pregnancy. Brit. Vet. J. 129:127-133.

BERDE, B. 1980. Ergot compounds: a synopsis. In: Ergot Compounds and Brain Function: Neuroendocrine and Nueropsychiatric Aspects. M. Goldstein, A. Lieberman, D.B. Calne and M.O. Thorner (ed.). Raven Press, New York. pp. 3-23.

BERDE, B. and E. STÜRMER. 1978. Introduction to the pharmacology of ergot alkaloids and related compounds as a basis of their therapeutic application. In: Ergot Alkaloids and Related Compounds, Handbook of Experimental Pharmacology, Vol. 49. B. Berde and H.O. Schild (ed.). Springer-Verlag, Berlin, Heidelberg, New York. pp. 1-28.

BHAT, R.V., D.N. ROY and P.G. TULPULE. 1976. The nature of alkaloids of ergoty pearl millet or bajra and its comparison with alkaloids of ergoty rye and ergoty wheat. Toxicol. Appl. Pharmacol. 36:11-17.

BOVÉ, F.J. 1970. The Story of Ergot. S. Karger Publ. Basel, New York. 207p.

BRAGG, D.B., H.A. SALEM and T.J. DEVLIN. 1970. Effect of dietary Triticale ergot on the performance and survival of broiler chicks. Can. J. Anim. Sci. 50:259-264.

BURFENING, P.J. 1973. Ergotism. J. Amer. Vet. Med. Assoc. 163:1288-1290.

BURFENING, P.J. 1975. Feeding ergot to pregnant ewes. Theriogenology 3:193-198.

CAMPBELL, C.W. and P.J. BURFENING. 1972. Effects of ergot on reproductive performance in mice and gilts. Can. J. Anim. Sci. 52:567-569.

CLARK, B.J. 1979. Cardiovascular effects of ergot alkaloids. J. Pharmacol. 10:439-453.

CUNNINGHAM, I.J. 1949. A note on the cause of tall fescue lameness in cattle. Aust. Vet. J. 25:27-28.

DINNUSSON, W.E., C.N. HAUGSE and R.D. KNUTSON. 1971. Ergot in rations for fattening cattle. N.D. Farm Res. 29:20.

DINNUSSON, W.E., J.L. SELL, R.L. HARROLD, D.O. ERICKSON, R. JOHNSON, C.N. HAUGSE and M.L. BUCHANAN. 1973. Ergot in livestock and poultry rations. 1973 Ann. Rep. Dept. Anim. Sci., North Dakota State University. pp. 130-138.

DINNUSSON, W.E., J.L. SELL, R.J. HARROLD, D.O. ERICKSON, R.L. JOHNSON, C.N. HAUGSE and M.L. BUCHANAN. 1974. Ergot in livestock and poultry rations. 1974 Ann. Rep. Dept. Anim. Sci., North Dakota State University. pp. 89-92.

DINNUSSON, W.E., J.L. SELL, R.J. HARROLD, D.O. ERICKSON, R.L. JOHNSON, C.N. HAUGSE and B. MOORE. 1975. Ergot in rations for heifers. N.D. Farm Res. 33:144-146.

DOYLE, M.P., R.S. APPLEBAUM, R.E. BRACKETT and E.H. MARTH. 1982. Physical, chemical and biological degradation of mycotoxins in foods and agricultural commodities. J. Food Protect. 45:964-971.

FRANZ, J.M., J.P. VONDERSCHER and R. VOGES. 1980. Contribution to the intestinal absorption of ergot peptide alkaloids. Int. J. Pharmaceut. 7:19-28.

FRIEND, D.W. and T.M. MACINTYRE. 1970. Effect of rye ergot on growth and N-retention in growing pigs. Can. J. Comp. Med. 34:198-202.

GOLDBLATT, L.A. and F.G. DOLLEAR. 1979. Modifying mycotoxin contamination in feeds - use of mold inhibitors, ammoniation, roasting. In: Interactions of Mycotoxins in Animal Production. Proceedings of a Symposium, July 13, 1978. Nat. Acad. Sci. pp. 167-184.

GREATOREX, J.C. and P.G. MANTLE. 1973. Experimental ergotism in sheep. Res. Vet. Sci. 15:337-346.

GREATOREX, J.C. and P.G. MANTLE. 1974. Effect of rye ergot on the pregnant sheep. J. Reprod. Fert. 37:33-41.

GRIFFITH, R.W., J. GRAUWILER, CH. HODEL, K.H. LEIST and B. MATTER. 1978. Toxicological considerations. In: Ergot Alkaloids and Related Compounds, Handbook of Experimental Pharmacology, Vol. 49, B. Berde and H.O. Schild (ed.). Springer-Verlag, Berlin, Heidelberg, New York. pp. 805-851.

INGALLS, J.R. and G.D. PHILLIPS. 1971. Effect of triticale ergot on calf growth. Can. J. Anim. Sci. 51:819 (abstr.).

JEMMALI, M. 1979. Decontamination and detoxification of mycotoxins. Pure Appl. Chem. 52:175-181.

JOHNSON, R.L. and J.L. SELL. 1976. Effects of ergot on the productive performance of laying hens. Poult. Sci. 55:2049-2050 (abstr.).

KIECHEL, J.R. 1979. Pharmacoinétique et metabolisme des dérivés de l'ergot. J. Pharmacol. (Paris) 10:533-565.

KINGSBURY, J.M. 1964. Poisonous Plants of the United States and Canada. Prentice-Hall Inc., Englewood Cliffs, New Jersey. 626p.

KRZEMINSKA, K. 1968. Suitability of the fermentation broth obtained on utilization of ergot infected rye. Chem. Abst. 69:34803 (abstr.).

LINDLEY, W.H. 1978. Ergot toxicosis. Mod. Vet. Pract. 59:463-464.

LORENZ, K. 1979. Ergot on cereal grains. Crit. Rev. Food Sci. Nutr. 11:311-354.

LU, A.Y.H. 1976. Liver microsomal drug-metabolizing enzyme system: functional components and their properties. Fed. Proc. 35:2460-2463.

MANTLE, P.G. 1968. Studies on *Sphacelia sorghi* McRae, an ergot of *Sorghum vulgare*. Pers. Ann. appl. Biol. 62:443-449.

MANTLE, P.G. 1977. Chemistry of *Claviceps* mycotoxins. In: Mycotoxic Fungi, Mycotoxins, Mycotoxicoses. An Encyclopedic Handbook. Vol. I. T.D. Wyllie and L.G. Morehouse (ed.). Marcel Dekker Inc., New York, Basel. pp. 421-426.

MANTLE, P.G. 1978a. Ergotism in cattle. In: Mycotoxic Fungi, Mycotoxins, Mycotoxicoses. An Encyclopedic Handbook. Vol. 2. T.D. Wyllie and L.G. Morehouse (ed.). Marcel Dekker Inc. New York, Basel. pp. 145-151.

MANTLE, P.G. 1978b. Ergotism in sheep. In: Mycotoxic Fungi, Mycotoxins, Mycotoxicoses. An Encyclopedic Handbook. Vol. 2. T.D. Wyllie and L.G. Morehouse (ed.). Marcel Dekker Inc. New York, Basel. pp. 207-213.

MANTLE, P.G. 1978c. Ergotism in horses. In: Mycotoxic Fungi, Mycotoxins, Mycotoxicoses. An Encyclopedic Handbook. Vol. 2. T.D. Wyllie and L.G. Morehouse (ed.). Marcel Dekker Inc. New York, Basel. pp. 185.

MANTLE, P.G. 1978d. Ergotism in swine. In: Mycotoxic Fungi, Mycotoxins, Mycotoxicoses. An Encyclopedic Handbook. Vol. 2. T.D. Wyllie and L.G. Morehouse (ed.). Marcel Dekker Inc. New York, Basel. pp. 273-275.

MANTLE, P.G. 1978e. Ergotism in poultry. In: Mycotoxic Fungi, Mycotoxins, Mycotoxicoses. An Encyclopedic Handbook. Vol. 2. T.D. Wyllie and L.G. Morehouse (ed.). Marcel Dekker Inc. New York, Basel. pp. 307-308.

MCKEON, F.W. and D.A. EGAN. 1971. Lameness in cattle fed ergotised silage. Irish Vet. J. 25:67-69.

MERCIER, C. 1982. Peptide alkaloids of ergot and their semi-synthetic derivatives. I. Chemistry of the alkaloids (brief survey) II. Pharmacology of the ergot. J. Pharm. Belg. 37:219-227.

MERTENS, D.R. 1979. Biological effects of mycotoxins upon rumen funtion and lactating dairy cows. In: Interactions of Mycotoxins in Animal Production. Proceedings of a Symposium. July 13, 1978. Nat. Acad. Sci. pp. 118-136.

MÜLLER-SCHWEINITZER, E. and H. WEIDEMANN. 1978. Basis pharmacological properties. In: Ergot Alkaloids and Related Compounds, Handbook of Experimental Pharmacology. B. Berde and H.O. Schild (ed.). Springer-Verlag, Berlin. pp. 87-232.

NATIONAL RESEARCH COUNCIL. 1977. Nutrient Requirements of Domestic Animals. Number 1. Nutrient Requirements of Poultry. 7th Edition. National Academy of Science, Washington, D.C. 62p.

NORDSKOG, A.W. and R.T. CLARK. 1945. Ergotism in pregnant sows, female rats and guinea pigs. Amer. J. Vet. Res. 6:107-116.

O'BRIEN, K., E. MOSS, D. JUDAH and G. NEAL. 1983. Metabolic basis of the species difference to aflatoxin B_1 induced hepatotoxicity. Biochem. Biophys. Res. Comm. 114:813-821.

O'NEIL, J.B. and W.J. RAE. 1965. Ergot tolerance in chicks and hens. Poult. Sci. 44:1404 (abstr.).

PEREK, M. 1958. Ergot and ergot-like fungi as the cause of vesicular dermatitis (sod disease) in chickens. J. Amer. Vet. Med. Assoc. 132:529-533.

RANKIN, J.E.F. 1965. Abortions associated with ergotised pastures. Vet. Rec. 77:911-912.

ROBINSON, T. 1981. The Biochemistry of Alkaloids. 2nd Edition. Springer-Verlag, Berlin, Heidelberg, New York. 225p.

SAS INSTITUTE INC. 1982a. SAS User's Guide: Basic, 1982 Edition. Cary, NC: SAS Institute Inc. 923p.

SAS INSTITUTE INC. 1982b. SAS User's Guide: Statistics, 1982 Edition. Cary, NC: SAS Institute Inc. 584p.

SEAMAN, W.L. 1980. Ergot of grain and grasses. Agriculture Canada Publ. 1438. 14p.

SHERIDAN, J.J. 1980. Some observations on selected mycoses and mycotoxicoses affecting animals in Ireland. Irish Vet. J. 34:148-154.

SHONE, D.K., J.R. PHILIP and G.J. CHRISTIE. 1959. Agalactia of sows caused by feeding the ergot of the bulrush millet, *Pennisetum typhoides*. Vet. Rec. 71:129-132.

SILBER, V.A. and W. BISCHOFF. 1954. Die Konstanz des Alkaloid gehaltes bei verschiedenen Rassen von Mutterkorn. Die Pharmazie 9:46-61

SKOWRONSKI, G.A., M.D. TRONSON and W.G. PARKIN. 1979. Successful treatment of ergotamine poisoning with sodium nitroprusside. Med. J. Aust. 2:8-9.

SUNDE, M.L. 1972. Chemical and nutritional changes in feeds brought about about by heat treatment. In: Effect of Processing on the Nutritional Value of Feeds. Proceedings of a Symposium, Gainsville, Florida. January 11-13, 1972. Nat. Acad. Sci. pp. 23-33.

SWARBRICK, O. and J.T. SWARBRICK. 1968. Suspected ergotism in ducks. Vet. Rec. 82:76-77.

VAN RENSBURG, S.J. 1977. Role of epidemiology in the elucidation of mycotoxin health risks. In: Mycotoxins in Human and Animal Health. J.V. Rodricks, C.W. Hesseltine and M.A. Mehlman (ed.). Pathotox Publ. Inc. Park Forest South, Il. pp. 700-711.

WATKINS, W.G. 1982. The effect of ergot ingestion on thermoregulation in sheep. M.Sc. Thesis, University of Manitoba. 123p.

WHITTEMORE, C.T., R.C.F. MACER, J.K. MILLER and P.G. MANTLE. 1976. Some consequences of the ingestion by young and growing pigs of feed contaminated with ergot. Res. Vet. Sci. 20:61-69.

YOUNG, J.C. 1979. Ergot contamination of feedstuffs. Feedstuffs 51:23-33.

YOUNG, J.C. 1981a. Variability in the content and composition of alkaloids found in Canadian ergot. I. Rye. J. Envir. Sci. Health Part B16:83-111.

YOUNG, J.C. 1981b. Variability in the content and composition of alkaloids found in Canadian ergot: II. Wheat. J. Envir. Sci. Health, Part B16:381-393.

YOUNG, J.C. and Z-J. CHEN. 1982. Variability in the content and composition of alkaloids found in Canadian ergot. III. Triticale and barley. J. Envir. Sci. Health, Part B17:93-107.

YOUNG, J.C., Z-J. CHEN and R.R. MARQUARDT. 1983. Reduction in alkaloid content of ergot sclerotia by chemical and physical treatment. J. Agric. Food Chem. 31:413-415.

YOUNG, J.C. and R.R. MARQUARDT. 1982. Effects of ergotamine tartrate on growing chicks. Can. J. Anim. Sci. 62:1181-1191.

Appendix 1. Alkaloid compositions of ergot samples used, expressed as a percentage of the totals

Alkaloid	Rye Meadowbank P.E.I.	Rye North River P.E.I.	Rye Ottawa Ont.	Triticale Ottawa Ont.	Wheat Prairies	All-grain[a] Canadian average
Ergometrine	6.56	10.13	9.92	5.37	7.70	5.00
Ergometrinine	1.23	1.52	3.28	2.93	3.00	2.17
Ergosine	15.47	16.30	12.33	9.16	6.28	4.21
Ergosinine	3.78	3.90	2.95	2.53	1.30	1.99
Ergotamine	40.10	36.94	23.09	22.79	14.77	17.30
Ergotaminine	5.73	6.58	6.06	5.18	3.13	7.63
Ergocornine	1.09	1.26	5.75	5.77	10.35	4.01
Ergocorninine	0	0	2.33	2.04	2.67	2.97
Ergocryptine	0.93	2.24	3.52	3.67	10.05	5.33
Ergocryptinine	0	0	1.26	1.19	1.70	2.58
Ergocristine	22.04	16.95	22.15	32.76	33.09	31.12
Ergocristinine	1.62	1.33	3.97	4.50	3.15	13.23
Unknowns	1.45	2.85	3.39	2.11	2.81	3.36
% Total alkaloid	0.269	0.312	0.269	0.259	0.308	0.236

[a]Taken from Young and Chen (1982).

Appendix 2. Statistical Models Used

Experiment I

The model used for Experiment I was:

$$Y_{ijk} = \mu + \alpha_i + \beta_j + (\alpha\beta)_{ij} + e_{ijk}$$

where Y_{ijk} is an observation on the k^{th} pen at the j^{th} ergot level on the i^{th} ergot

μ is the mean parameter of all treatments

α_i is the fixed effect of the i^{th} ergot ($i = 1, 5$)

β is the fixed effect of the j^{th} ergot level ($j = 1, 2$)

$(\alpha\beta)_{ij}$ is the fixed interaction of the j^{th} level and the i^{th} ergot

e_{ijk} is the random effect of the observation from the k^{th} pen at the j^{th} level of the i^{th} ergot ($k = 1, 6$).

Experiment II

There were three models used for the analysis of data in Experiment II. The first model involved the analysis of the weekly totals (relative weight gain based on "full fed" birds, relative feed consumption and feed to gain). The model used was as follows:

Model 1

$$Y_{ijk} = \mu + \alpha_i + \delta_{ik} + \beta_j + (\alpha\beta)_{ij} + e_{ijk}$$

where Y_{ijk} is the observation on the k^{th} pen on the j^{th} week on the i^{th} ergot level

μ is the overall mean of all treatments

α_i is the fixed effect of the i^{th} ergot level ($i = 1, 7$)

δ_{ik} is the main plot error ($k = 1, 8$)

β_j is the fixed effect of the j^{th} time period ($j = 1, 4$)

$(\alpha\beta)_{ij}$ is the random effect of the interaction of the j^{th} time period and the i^{th} ergot level

e_{ijk} is the random effect of the observation on the k^{th} pen during the j^{th} time period and the i^{th} ergot level.

The second model (Model 2) used analyzed the four week totals (relative weight gain in terms of "starved" basis). The model used was the same as Model 1 of Experiment 1 with the following exceptions:

$i = 1, 7$

The third model was used to analyze the least square means and plot the predicted average daily relative feed consumption of the chicks over time of the different ergot levels. The analytical model was as follows:

Model 3

$$Y_{ij} = \mu + \alpha_i + \alpha_i^2 + e_{ij}$$

where Y_{ij} is the predicted relative feed consumption of the j^{th} pen on the i^{th} ergot level

μ is the mean parameter for each treatment

α_i is the i^{th} day of the study ($i = 1, 28$)

e_{ij} is the random effect of the observation from the j^{th} pen on the i^{th} ergot treatment.

Experiment IIIa

The model used for Experiment IIIa was as follows:

$$Y_{ijk\ell} = \mu + a_i + ab_{ij} + P_{k(ij)} + t_\ell + at_{i\ell} + bt_{j\ell} + abt_{ij\ell} + e_{ijk\ell}$$

where Y_{ijk} is an observation from the ℓ^{th} time period on the k^{th} pen which was fed the j^{th} dietary formulation on the i^{th} ergot treatment

μ is the mean parameter of all treatments

a_i is the fixed effect of the i^{th} ergot treatment ($i = 1, 5$)

b_j is the fixed effect of the j^{th} dietary formulation ($j = 1, 2$)

ab_{ij} is the fixed interaction of the j^{th} dietary formulation and the i^{th} ergot treatment

$P_{k(ij)}$ is the random effect of pen within an ergot-diet treatment combination ($k = 1, 6$)

t_ℓ is the fixed effect of the ℓ^{th} time period ($= 1, 4$)

$at_{i\ell}$ is the fixed interaction of the ℓ^{th} time period and the i^{th} ergot treatment

$bt_{j\ell}$ is the fixed interaction of the ℓ^{th} time period and the j^{th} dietary formulation

$abt_{ij\ell}$ is the fixed interaction of the ℓ^{th} time period, the j^{th} dietary formulation and the i^{th} ergot treatment

$e_{ijk\ell}$ is the random effect of the observation from the ℓ^{th} time period on the k^{th} pen which was fed the j^{th} dietary formulation and the i^{th} ergot level.

Experiment IIIb

The model used for Experiment IIIb was identical to that used for Experiment IIIa with the following exceptions:

$Y_{ijk\ell}$ is an observation from the ℓ^{th} time period on the k^{th} pen containing the j^{th} bird type and the i^{th} ergot treatment

b_j is the fixed effect of the j^{th} bird type ($j = 1, 2$)

ab_{ij} is the fixed interaction of the i^{th} ergot treatment and the j^{th} bird type

$bt_{j\ell}$ is the fixed interaction of the j^{th} bird type and the ℓ^{th} time period

$abt_{ij\ell}$ is the fixed interaction of the i^{th} ergot treatment, the j^{th} bird type and the ℓ^{th} time period

$e_{ijk\ell}$ is the random effect of the observation from the ℓ^{th} time period on the k^{th} pen which contained the j^{th} bird type fed the i^{th} ergot treatment.

Experiment IV

The model used for Experiment IV was:

$$Y_{ijk\ell} = \mu + a_i + b_j + ab_{ij} + P_{k(ij)} + t_\ell + at_{i\ell} + bt_{j\ell} + abt_{ij\ell} + e_{ijk\ell}$$

where $Y_{ijk\ell}$ is an observation from the ℓ^{th} time period on the k^{th} pen which was fed the j^{th} dietary formulation and i^{th} ergot treatment sequence

μ is the mean parameter of all treatments

a_i is the fixed effect of the i^{th} ergot treatment sequence ($i = 1, 8$)

b_j is the fixed effect of the j^{th} dietary formulation ($j = 1, 2$)

ab_{ij} is the fixed interaction of the i^{th} ergot treatment sequence (the j^{th} dietary formulation)

$P_{k(ij)}$ is the random effect of pens within an ergot-diet treatment combination

($k = 1, 2$ for $i = 4, 8$, $j = 1, 2$)
($k = 1, 2, 3$ for $i = 1, 3$ and $5, 7$, $j = 1, 2$)

t_ℓ is the fixed effect of the ℓ^{th} time period (= 1, 2)

$at_{i\ell}$ is the fixed interaction of the ℓ^{th} time period and the i^{th} ergot treatment sequence

$bt_{j\ell}$ is the fixed interaction of the ℓ^{th} time period and the j^{th} dietary formulation

$abt_{ij\ell}$ is the fixed interaction of the ℓ^{th} time period, the j^{th} dietary formulation and the i^{th} ergot treatment sequence

$e_{ijk\ell}$ is the random effect of the observation from the ℓ^{th} time period on the k^{th} pen which was fed the j^{th} dietary formulation and the i^{th} ergot treatment sequence.

Experiment V

The model used for Experiment V was the same as Model 2 used in Experiment I except that i = 1, 6.

Experiment VI

The model used for Experiment VI was the same as Model 1 used in Experiment I.

Experiment VII

The model used for Experiment VII was:

$$Y_{ijk} = \mu + \alpha_i + \beta_j + (\alpha\beta)_{ij} + e_{ijk}$$

where Y_{ijk} is the observation on the k^{th} pen on the j^{th} level of Antitox Vana and the i^{th} ergot level

μ is the overall mean parameter of all treatments

α_i is the fixed effect of the i^{th} ergot level (i = 1, 4)

β_j is the fixed effect of the j^{th} Antitox Vana level (j = 1, 4)

$(\alpha\beta)_{ij}$ is the fixed interaction of the j^{th} Antitox Vana level and the i^{th} ergot level

e_{ijk} is the random effect of the observation of the k^{th} pen on the j^{th} level of Antitox Vana and the i^{th} ergot level