# THE EFFECTS OF FEEDING DIFFERENT LEVELS OF DEOXYNIVALENOL (DON OR VOMITOXIN) ON THE REPRODUCTIVE AND GROWTH PERFORMANCE OF SHEEP

A Thesis

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by

Chiwuike Ubawumadu Nwaerondu

In Partial Fulfilment of the

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of

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# THE EFFECTS OF FEEDING DIFFERENT LEVELS OF DEOXYNIVALENOL (DON OR VOMITOXIN) ON THE REPRODUCTIVE AND GROWTH PERFORMANCE OF SHEEP

BY

CHIWUIKE UBAWUMADU NWAERONDU

A Thesis submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements of the degree of

MASTER OF SCIENCE

## ABSTRACT:

Chiwuike U. Nwaerondu, M. Sc., The University of Manitoba, October, 1995. The Effects of Feeding Different Levels of Deoxynivalenol (DON or Vomitoxin) on The Reproductive and Growth Performance of Sheep. Major Professor; W. M. Palmer

An experiment consisting of four trials was conducted to determine the possible effects of deoxynivalenol (DON) on the reproductive and growth performance of two breeds of sheep (Suffolk and Outaouais).

Ewes and lambs received diets prepared with DON-contaminated barley containing either 0, 4, 10 or 20 ppm during their respective trials. Trial one measured the number of lambs born alive (NBA), number of lambs born dead (NBD) and average birth weight of lambs (ABW). From ewes fed the test ration during the last 47 days, treatment with 20 ppm in the concentrate diet resulted in a significant depression in ABW ($P < 0.05$) and a lower NBA. In Trial two, there was a significant difference ($p < 0.05$) in weaning weight of lambs, with the lambs nursed by ewes fed the highest level of DON having the lowest weaning weight when compared to the control and other treatment groups.

In Trial 3, the average daily feed consumption (ADFC), average daily gain (ADG) and average feed efficiency (AFE) of growing lambs fed DON-contaminated barley from an average weight of 17.3 kg to an average weight of 32 kg at levels 0, 4, 10 and 20 ppm were not different. Treatment did not affect the total weight gain and final weight of lambs. In Trial 4, ewes were fed DON-contaminated barley from a week before breeding and during the first and second trimester of pregnancy. There was no

observed difference between the control and the treated groups of ewes in the rebreeding interval, conception rate at first and second breeding as well as any other signs of possible toxicosis. The results of the lambing parameters signify that, there were no differences due to treatment. The only significant differences in ABW and NBA were due to breed.

These results show that, sheep experienced no major adverse effect when fed up to 20 ppm of DON in the concentrate portion of the diet.

## ACKNOWLEDGMENTS

It is with profound gratitude that I express my thanks to the Nwaerondus' family in Winnipegosis who paved my way to Canada, and the University, leading to this study through their financial help, advice,etc.

I wish to thank the Department of Animal Science, University of Manitoba for their financial support during this study. My special thanks goes to Dr W. M. Palmer for the opportunity of working under him as my advisor and Dr. R. J. Parker for giving me a job that ran throughout the duration of my program here. Other members of my research committee are not left out in this wave of thanks.

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# DEDICATION

DEDICATED TO

MY DAD

MR. PATRICK ISAIAH NWAERONDU UKASOANYA

AND

MY MOM

LATE MRS CHRISTINAH NMANWA UKASOANYA

[WHO PASSED AWAY DURING MY STUDIES HERE]

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# INTRODUCTION

Deoxynivalenol (DON) or vomitoxin, a trichothecene mycotoxin produced by *Fusarium graminearum*,occurs mainly on wheat, barley and other grains. Excessive rainfall and cool temperatures during silking and/or harvesting are the favorable conditions for the growth of this mold (DeHaan et al., 1983; Diekman and Green, 1992; Oltjen et al., 1984; Cote et al., 1985).

The weather conditions of the 1993 summer season in regions of the North American continent, caused an out-break of this mold on cereal grain crops harvested from many farms. There was great concern as regarding to the feeding value of these infected crops and the effects these contaminated feedstuffs would have on the performance of animals. Scarcity of feedstuff or lack of feedstuff, was also anticipated due to the high rate of contamination.

Several studies indicate that the mycotoxin, vomitoxin is the most predominant of all the trichothecenes, but the least in toxicity (Prelusky et al., 1990; Abramson et al., 1985; Richard et al., 1993). Vomitoxin is more toxic to monogastric animals, particularly swine than ruminants. Symptoms such as vomiting, dizziness, decreased body weight gain have been reported in pigs fed vomitoxin-contaminated feedstuffs (Forsyth et al., 1977; Friend et al., 1986; Coppock et al., 1985; Rotter et al., 1992; Young et al., 1983; Pestka et al., 1987). Ruminants are said to be less sensitive to vomitoxin because the effects of DON as seen in monogastrics were either reduced or totally eliminated in various studies involving ruminants (Trenholm et al., 1984; Prelusky et al., 1986; Swanson et al., 1987; Diekman and Green, 1992; Harvey et al., 1986). Zearalenone,

another compound produced by a *Fusarium* mold did reduce fertility/conception rate in heifers (Weaver et al., 1986a; Weaver et al., 1986b). However, DON has not been tested on pregnant ruminants to monitor the effects on pregnancy maintenance and other reproductive performance parameters. Therefore this study was, designed to investigate the possible effects of vomitoxin on the reproductive and growth performance of sheep, and to the insensitivity of ruminants to vomitoxin.

# LITERATURE REVIEW

## STUDIES WITH RUMINANTS:

### SHEEP:

#### Reproduction and Lactation:

The effects of vomitoxin on the reproductive performance of sheep have received little attention. However, considering the results from previous studies conducted with lactating ewes (Prelusky et al., 1987), lambs (Harvey et al., 1986; Oltjen et al., 1984; Prelusky et al., 1986), open ewes or sheep (Prelusky et al., 1985) and cattle (Trenholm et al., 1984; Charmley et al., 1993; Cote et al., 1985; Trenholm et al., 1985), it might be presumed that DON would have no adverse effect on the various parameters of reproductive performance such as estrus, conception and gestation.

Recently, Windels, (1994) fed DON at 2 levels (either 6 or 12 ppm) to pregnant Polypay ewes during the last 5 weeks of pregnancy and observed no apparent detrimental effect on number of lambs born and reared per ewe, ewe health, lamb vigor, birth weight and weaning weight. He also reported no problems with feed intake of ewes fed DON-contaminated diets (Windels, 1994). A study to confirm the effect of DON-contaminated diets on the conception rate and early pregnancy is also in progress in North Dakota as reported by Windels, (1994).

#### Growth and Development:

The result of a feedlot performance trial conducted by Oltjen et al., (1984) with 1.7, 3.5 and 5.2 ppm of DON did not show any difference in feed efficiency and weight

gains between lambs fed either of the three levels of DON or the control. However, reduced feed intake and average daily gain were observed for the first 28 days before lambs became adapted to the feed. There were no significant differences ($P > 0.05$) between the treated and control animals in all parameters measured at the end of the 70 day trial (Oltjen et al., 1984).

When fattening lambs were fed either dry rolled corn or scab-infested wheat screenings, the lambs receiving dry rolled corn tended to gain weight more rapidly and more efficiently than lambs fed the scab-infested wheat screenings (DeHaan et al., 1983). These results could have been biased because, the reduction in average daily gain could be due to feeding of wheat screenings to treated animals instead of the whole grains or kernels of normal size as was fed to the controls (DeHaan et al., 1983). Normal wheat tends to ferment faster than scab-infested wheat screenings, indicating that, there was lower energy content of the wheat screenings available to treated lambs than there was in dry rolled corn fed to control animals (DeHaan et al., 1983). The observed difference in weight gain could be due to the difference in energy concentration and not the presence of DON (DeHaan et al., 1983). The addition of alfalfa to the scabby-wheat diets resulted in no difference in lamb performance. However, it could have also diluted the toxic effects of DON on the scabby-wheat diets (DeHaan et al., 1983).

The feeding of DON ($< 10$ ppm) in diets to finishing cattle and lambs does not appear to affect feedlot performance. Harvey et al., (1986) administered 15.6 mg DON/kg to lambs for 28 days and observed no significant difference between that and the control groups. The parameters measured included gain/animal ($6.2 \pm 2.3$ vs 5.8

± 2.5 kg), feed consumption/animal (29.4 ± 5.6 vs 28.8 ± 5.9 kg) and feed:gain ratio (4.7 vs 5.0 kg/kg) for groups 1 and 2, respectively. This study indicated that, lambs may tolerate moderately high intakes of DON-contaminated diets without pathologic or clinical manifestation of toxicosis (Harvey et al., 1986).

**Metabolism and Excretion:**

The dependence of humans on animal products as a dietary source and the exposure of such livestock to DON-contaminated diets have been of immense concern to regulatory officials who fear that DON and its metabolites could be transmitted into animal products such as, eggs, milk and muscle tissue. This has prompted several research studies on the uptake, metabolism and elimination of DON in various species of livestock and poultry (Prelusky et al., 1985; Prelusky et al., 1986; Prelusky et al., 1990; El-Banna et al., 1983).

The conversion of DON to its chief metabolite 3$\alpha$,7$\alpha$,15 trihydroxy-trichothec-9,-12-dien-8-one (DOM-1), has been reported in various studies (Prelusky et al., 1985; King et al., 1984; Yoshizawa et al., 1983). DOM-1 is said to be less toxic than DON itself and about 15% of this conversion takes place in the rumen of ruminant animals, but the majority of the conversion takes place in the liver. Thus, this is referred to as an authentic hepatic metabolite of DON in sheep (Prelusky et al., 1985; Yoshizawa et al., 1983).

DON is rapidly distributed in the body following an intravenous (iv) administration. It also decreases rapidly and biexponentially with a half-life ($t^{1/2}$) of

about 12 to 23 minutes. A slower phase of elimination has a $t^{1/2}$ of 57 to 78 minutes and only trace levels could be detected in plasma 7 hrs after treatment (Prelusky et al., 1985). DON is conjugated to a glucuronide after iv administration and has a $t^{1/2}$ of about 77 min. Determination of DON conjugates accounted for about 15-23% of the total DON measured in plasma. The $t^{1/2}$ of DON conjugates which indicated the rate of metabolite formation averaged 35 min, with 177 min as the $t^{1/2}$ for the slow elimination terminal phase (Prelusky et al., 1985).

DON promptly appeared in plasma 30 minutes after oral dosing with 0.5 to 5 mg DON/kg/BW. The peak concentrations of DON were reached in about 4 to 5.3 hrs (Prelusky et al., 1985). It had a $t^{1/2}$ of 100-125 min when administered orally. The peak plasma concentration for DON levels (DON + conjugated DON) was 583 ng/ml (ppb). Complete clearance of DON from the system required a time range of 20-30 hrs after dosing, depending on the animal (Prelusky et al., 1985).

Similarly, an *in vitro* trial with DON (up to 10 ppm) incubated in rumen fluid by King et al., (1984), confirmed that rumen microorganisms were efficient in metabolizing DON and the results revealed a steady decrease in the quantity of DON. Studies suggest that, when diets containing DON at the commonly occurring levels (usually $< 3$ ppm) are to be fed to ruminants, oral ingestion is the best detoxification method for this particular toxin (Trenholm et al., 1985).

Only about 14-37% of total plasma DON is free; with 1.8-2.8% present as DOM-1, while the remaining 63-86% is present as a conjugate DON (Prelusky et al., 1985). The metabolic formation of the glucuronide conjugate after iv and oral administration of

DON appeared to occur quite efficiently and its elimination times were considerably longer , than that of the nonconjugated DON (Prelusky et al., 1985). The biexponential decay nature of conjugated DON showed that there may be compartmentalization of glucuronide conjugates. This may be responsible for the longer elimination $t^{1/2}$ compared to parent compounds.

Following the iv administration of DON to sheep only about 24% of the dose was recovered as the unchanged toxin in the blood (Prelusky et al., 1986). Data on the urinary and biliary elimination of DON suggested that renal excretion is the primary route of elimination for this compound (Prelusky et al., 1986).

Sheep were capable of rapidly converting the toxin to conjugated metabolites (42% of dose) which were readily excreted in the urine. DOM-1 has been identified as a minor metabolite (1.5%) in plasma after iv administration of DON; whereas, it was identified as a major metabolite in urine; revealing the efficiency of the glucuronide elimination pathway (Prelusky et al., 1986).

However, the recovery of DON and its metabolites after administration could not account completely for the administered dose. Approximately 66% of the total dose was recovered. Unconjugated DOM-1 accounted for only a small fraction (0.5%) of the recovery (Prelusky et al., 1986). Glucuronide acid conjugates of both DON and DOM-1 were excreted to a considerably greater extent in the urine. This showed that approximately 33% of the administered dose remained unaccounted for (Prelusky et al., 1986). This, therefore, suggests that there may be some unknown metabolites; the identification and toxicities of which are currently not known (Prelusky et al., 1986).

Orally administered DON appears to also undergo presystemic excretion via the feces, accounting for 55-75% of the dose recovered as unconjugated forms of DON and DOM-1 in feces. This value probably represents DON which was not absorbed from the gastrointestinal tract, because the extent of biliary elimination that occurs in sheep would only contribute a small fraction to the overall fecal recovery. This is supported by the overall low levels of DON and its metabolites (7.1% of dose) eliminated through the systemic routes (urine and bile) which is indicative of poor absorption of DON into the systemic circulation (Prelusky et al., 1986). Total recovery of DON in urine/bile by Prelusky et al., (1986), was 6.9-7.2% of the administered dose. This shows a good comparison with an earlier study which estimated systemic bioavailability of DON in sheep at approximately 6-10% (Prelusky et al., 1985).

Furthermore, the combined recoveries from feces, urine and bile did not total the administered doses, showing that, some unidentified metabolites of DON which must have been converted in either the rumen or the intestinal tract may be responsible for this unaccounted value (Prelusky et al., 1986). The major systemic excretion products were glucuronic acid conjugated metabolites which accounted for 70% and 63% of the total products recovered after oral and iv dosing respectively (Prelusky et al., 1986).

One reason why the proportion of conjugated DON was notably higher than free DON, after oral administration, as opposed to iv dosing (oral 51% vs iv 32%) was that, the DON absorbed from the gastrointestinal tract went straight to the liver where it possibly underwent extensive conjugation as a "hepatic first pass effect" before being distributed throughout the body. Whereas with iv dosing, a reasonable percentage of

systemic DON had been eliminated before it had the chance to be metabolized in the liver, thereby reducing the amount of DON that gets to the liver as well as its ability of being conjugated (Prelusky et al., 1986). The assumption that DON could be conjugated in the rumen or intestinal tract by microbial action prior to systemic absorption was not confirmed due to the absence of conjugated DON in the feces of treated animals (Prelusky et al., 1986).

The low recovery of biliary excreted DON could be attributed to the subsequent conversion of DON to unidentified metabolites in the liver before excretion in the bile. There was also the possibility of enterohepatic recirculation which may reduce excretion via the feces (Prelusky et al., 1986).

DON caused a notable reduction in the flow of bile 1 to 3 hr postdosing, decreasing from 20-25 ml/hr to 3-5 ml/hr which also corresponded to the decrease in the rate of biliary excretion of conjugated DOM-1. Following iv dosing, only a small reduction in bile flow was noted as the rate was from 20-25 ml/hr to 15-20 ml/hr over the initial 3 hrs, This change did not appear to impede overall biliary excretion of DON. An elimination rate of DON and its metabolites in urine showed that, the conjugated forms of DON and DOM-1 were excreted slower than the parent toxin DON and the corresponding elimination $t^{1/2}$ following oral administration were longer than iv dosing (Prelusky et al., 1986).

In summary, Prelusky et al., (1986) stated that, the predominant route for systemic elimination appeared to be through biotransformation prior to excretion. Excretion of DON and metabolites occurred through both urinary and biliary routes with

urinary excretion being the most important route. Since a significant proportion of the DON present in blood could not be accounted for it may be suggested that further metabolic pathways remain to be identified.

Prelusky et al., (1990) compared the distribution of DON in the cerebral spinal fluid (CSF) following iv administration of DON to swine and sheep. Results obtained for sheep showed that the toxin (DON) was detected at maximum concentrations in plasma at 2.5 min. after dosing and that, about 8-15% of the total dose administered was present in form of glucuronide conjugates with less than 1% as the DOM-1 metabolite (Prelusky et al., 1990). No metabolite was discovered in the CSF, but the parent compound was present. The amount of DON in the CSF constituted about 6% of the total DON residue (DON + conjugated DON) present in both plasma and CSF (Prelusky et al., 1990).

Peak concentration of DON in CSF following iv administration in sheep and swine, did not differ significantly (458 $\pm$ 171 vs 582 $\pm$ 175ng/ml) respectively. DON concentration declined in sheep with time but were maintained in swine for a longer period probably due to a delayed uptake of DON by the CSF. However, the low absorption, extensive metabolism by rumen microorganisms and the fast elimination of the toxin following oral ingestion of DON, all combine to greatly reduce the concentration of DON to low level in sheep (Prelusky et al., 1990). In sheep plasma, a conjugated metabolite of DON was present while the parent toxin was liberated upon hydrolysis (Prelusky et al., 1990). DON-glucuronide was the major metabolic product in plasma following iv administration of DON to sheep (Prelusky et al., 1986) and

current data suggests that DON-conjugates do not penetrate the blood-CSF-barrier, while the parent toxin DON does (Prelusky et al., 1990).

The distribution of DON in sheep appeared confined to the extracellular fluid. This results in a faster release from the compartments than in swine, thereby decreasing the time required for the terminal elimination phase (Prelusky et al., 1990).

In conclusion, the researchers explained that, differences exist in the kinetics of DON between the swine and sheep species. These have direct influence on the metabolism, absorption, distribution and elimination characteristics of the toxin (Prelusky et al., 1990). These differences were shown in the plasma and CSF profiles, suggesting that the pigs susceptibility to this toxin may be due to the concentration of (DON) in the CSF. Recent findings with rats (Fitzpatrick et al., 1988) and a review statement by Smith (1992) suggest that the feed refusal or loss of appetite is caused by elevated brain serotonin concentration.

## CATTLE:

### Reproduction and Lactation:

Little research has been published on the effects of vomitoxin on estrus, conception rate, pregnancy maintenance and calf birth weight in beef cattle. However, several studies have looked at the effects of zearalenone (ZEN) on the reproductive performance of cows and pigs (Weaver et al., 1986a; Weaver et al., 1986b; Trenholm et al., 1985; Vesonder et al., 1981).

The literature indicates that DON is not responsible for reproductive failure and

it is only associated with feed refusal and emetic effects in swine (Vesonder et al., 1981). Furthermore, Trenholm et al., (1985) reported that, one cow in the study which accidentally was 4 month pregnant and received the same treatment (1.5 mg DON for the first 3 wks, 6.4 mg DON for 6 wks and 1.5 mg for the last 3 wks of the trial), exhibited no unusual behavior or performance and gave rise to a healthy calf at term.

When diets containing up to 14.6 mg DON/kg in the concentrate mixture, was fed to lactating dairy cows, there were no significant effects on the feed intake, which however tended to be reduced during the third week of the trial in all treatment levels. This reduction was not attributed to DON, because the effects are not relative to the consumption as increased levels of DON did not cause greater reduction than the lower DON levels (Ingalls, 1994).

The study further looked at the milk composition, body weight change and rumen pH of the cows and concluded that, there was no treatment effects ($p > 0.05$) on all these parameters. Ingalls (1994) reported results which contradicted the earlier reports by Charmley et al., (1993), who indicated a change in milk fat of cows fed 6 ppm and not 12 ppm.

DON fed at 66 mg/kg of feed for 5 days caused no difference in milk yield between treated and nontreated groups. Similarly, concentrations of 0, 6 and 12 mg DON/kg of feed, fed to lactating dairy cows failed to influence feed intake and milk production (Charmley et al., 1993). Cows receiving the 6 mg DON/kg diet gained more weight than the others. Energy production was not influenced by treatments because, cows fed the 6 mg DON/kg diets tended to excrete less energy in milk (Charmley et al.,

1993; Cote et al., 1986). In contrast, Behlow et al., (1985), revealed a positive correlation between DON in the feed of dairy cows and a drop in milk production and reduced conception rate as cited in Cote et al. (1986).

**Growth and Development:**

*Fusarium* infected grains can be fed to ruminants without the serious deleterious effects that have been observed in swine (Vesonder et al., 1981; Noller et al., 1979; King et al., 1984; Trenholm et al., 1985; Charmley et al., 1993).

However, a slight reduction in feed intake was observed when nonlactating dairy cows were fed naturally-contaminated grains containing up to 6 mg DON/kg dry matter (DM), (Trenholm et al., 1985). This was corrected when the animals were returned to 1.3 mg DON/kg body weight (BW) following 6 wks of treatment. Nevertheless, the reduced feed intake, caused by DON consumption did not result in decreased BW (Trenholm et al., 1984; Noller et al., 1979). There was a decline in milk production with a slight increase from 0 to 0.8 mg DON/kg DM. Furthermore, an increase from 1.3 mg DON/kg BW in dairy cows resulted in a slight but statistically significant ($p < 0.05$) decrease in concentrate consumption (Whitlow et al., 1986; Whitlow et al., 1987)

Detrimental effects were more common when animals were changed from a low level of DON concentration to a higher level such as when Trenholm et al., (1985) changed from 1.5 mg DON/kg to 6.4 mg DON/kg or from 1.3 mg DON/kg to 5.8 mg DON/kg of BW and Whitlow et al., (1986; 1987), changed from 0 mg to 0.8 mg DON/kg DM.

**Metabolism and Excretion:**

Rumen fermentation and microorganisms act as the first line of defence against harmful or toxic substances in the diets of ruminants (Church, 1980; King et al., 1984). DON is readily metabolised in the rumen and ruminants appear to be relatively unaffected when fed DON-contaminated grain (King et al., 1984). An *in vitro* study with 10 ppm DON showed that DON was readily transformed by rumen microorganisms and results of ethyl acetate extracts taken from the *in vitro* rumen samples revealed a steady decrease in the level of DON concentration (King et al., 1984).

Metabolism of DON yielded the metabolite of DON called 3$\alpha$,7$\alpha$,15 trihydroxy-trichothec-9,-12-dien-8-one (DOM-1), which is relatively less toxic than DON and is easily detected in milk from a lactating cow or in urine and feces of a nonlactating cow, after feeding on DON-contaminated diets (King et al., 1984). Unconjugated DOM-1 was detected in milk of the cows, when 66 mg DON/kg of feed was fed (Cote et al., 1986); whereas Charmley et al., (1993) did not detect unconjugated DOM-1 in milk samples during their study. DOM-1 was still detected in the samples 16 hrs after treatment, but was totally cleared by 24 hrs after the treatment was terminated (Cote et al., 1986).

Previous studies reported that, only trace amounts of DON and DOM-1 were transferred to milk (Cote et al., 1986; Noller et al., 1979; Prelusky et al., 1984). However, the amount of unconjugated DON detected was very small when compared to the unconjugated DOM-1 (4% DON, 96% DOM-1 or 56.8 mg/ml DON and 1563 mg/ml unconjugated DOM-1) (Cote et al., 1986). Detectable concentrations of unconjugated DOM-1 was observed in both feces and urine from one cow for about 72

hrs, after the last treatment with DON, whereas the other cows had detectable concentrations for only about 4 hrs after the last oral dosing of DON (Cote et al., 1986). Cote et al., (1986) also detected marginally higher quantities of DOM-1 (4-26 ng/ml) from a single cow dosed with approximately 300 mg DON/day for 5 days. In contrast, Charmley et al., (1993) did not detect any amount of DON and DOM-1 in milk when diets containing 6 and 12 ppm were fed to lactating cows, but observed a quadratic response to milk fat concentration. In lactating dairy cows, diets containing 1.9 mg DON/kg BW as a single oral dose transmitted less than 1% of the total DON concentration into the system and extremely low concentrations ($<$ 4 ng/ml or $<$ 0.001%) were detected in the milk (Prelusky et al., 1985).

In general, it was estimated that, the total excretion of unconjugated DON and DOM-1 were (0 and 1.12 mg, respectively in milk ), (19.4 and 882.1 mg, respectively in urine) and (37.4 and 679.8 mg, respectively in feces). Cote et al. (1986) reported that about 20% of the total DON fed during the experimental period was excreted as unconjugated DON and DOM-1 through urine, milk and feces. In contrast, Charmley et al., (1993), failed to observe the same trend when 6 and 12 ppm of DON was fed to primiparous Holstein cows for about 10 weeks.

## STUDIES WITH NONRUMINANTS:

### POULTRY:

The effects of DON-contaminated grains on growing chicks and laying hens have been investigated, by various researchers (Kubena et al., 1984; El-Banna et al., 1983; Hamilton et al., 1985a; Hamilton et al., 1985b)

DON-contaminated wheat fed to growing chicks at concentrations of 0 and 9 mg/kg had no effect on body weight, body weight gain and feed efficiency. However, growing chicks fed a higher level of DON (18 mg/kg) in the same study exhibited a significantly ($p < 0.05$) heavier weight at 21 days of age than those fed control and lower DON concentrations (Moran et al., 1982; Kubena et al., 1984).

In other studies, it was reported that DON concentration of 4.6 mg/kg of feed, significantly influenced feed intake ($p < 0.01$) and body weight gains ($p < 0.05$) of chicks. Although the average daily gain of chicks fed DON-containing diets was higher than those of the controls, the feed/gain ratio and organ/body weight were similar (Hamilton et al., 1985a). Feed consumption was also higher in growing broiler chicks, fed a high DON level (4.6 mg/kg of feed) (Hamilton et al., 1985a; Moran et al., 1982). DON-contaminated diets decreased the feed/gain ratio in broilers between the age of 7 and 28 days when compared to the control, but not feed intake (Moran et al., 1982; Hamilton et al., 1985a).

Analysis for DON residues in liver, heart, kidney, breast, thigh and eggs indicated that DON and its residues were not transferred to any of the above tissues (El-Banna et al., 1983; Kubena et al., 1984; Trenholm et al., 1984).

A study by El-Banna et al., (1983), who fed 4 to 5 $\mu$g DON/kg body weight for a period between 28 and 190 days could not detect DON or its metabolites in eggs or muscle tissues of laying hens. However, slight and transitory anemia, described as hemolytic and hypochromic anemia was caused by DON (Kubena et al., 1984). Irritation due to the presence of DON-contaminated grains in diets or due to the difference in the density of the diets was found in the upper gastrointestinal tract of birds. Furthermore, DON did not influence egg production and/or yield, number of soft shelled eggs and number of cracked eggs (Hamilton et al., 1985b). It was stated that DON-fed hens utilized their feed more efficiently (1.4 to 4.4%) for egg production than birds given control diets. Hamilton et al. (1985b) also reported that dietary DON influenced embryonic mortality, which resulted in decreased hatchability of fertile eggs.

The ability of DON to inhibit protein and deoxyribonucleic acid (DNA) biosynthesis, causing inflammation of the skin, leukopenia and decreased feed intake and weight gain has been described as the mechanism of its action (Newberne and Rogers, 1981; Ueno et al., 1980; Hamilton et al., 1985b).

The relative insensitivity of poultry to DON has led to the conclusion that, DON-contaminated grains may be incorporated into poultry diets without affecting their performance (Kubena et al., 1984; Moran et al., 1982; El-Banna et al., 1983), whereas in swine, DON-consumption has been associated with emesis or vomiting, hence, the origin of the common name "vomitoxin" (Trenholm et al., 1984; Young et al., 1987; Friend et al., 1986).

Purified DON was tested with broiler chicks and the median lethal dose (LD50) was given as 140 mg/kg BW. There was no observed effect when purified DON was fed at concentrations of less than 49 mg/kg of diet or 140 mg/kg BW (Hamilton et al., 1985a).

**SWINE:**

Pigs respond promptly to DON-contaminated diets with signs, such as vomiting, reduced feed intake, reduced weight gain and decreased feed efficiency (Trenholm et al., 1994; Young et al., 1987; Forsyth et al., 1977; Coppock et al., 1985; Pollmann et al., 1984; Friend et al., 1986). These responses were more severe during the first 3 days of treatment and were attributed to the initial metabolism response, followed by a better subsequent utilization of the same diets after building up an adaptation to the toxin (Young et al., 1987). DON is rapidly absorbed from the gastrointestinal tract of swine as shown by the rapid appearance of the toxin in plasma and it is wide distribution throughout the body. The toxin does not appear to be stored in any tissue. However, its elimination is very slow suggesting that, there is a slow release from one of the compartments (such as the fat) (Prelusky et al., 1990).

A wide range of reduction in both feed intake and body weight gain have been reported by various researchers. These include reductions in feed intake and weight gain 18 and 23%, respectively, when 3.4 to 19.1 mg/kg of pure DON was fed to pigs (Trenholm et al., 1994), 20 and 90% reduction in feed intake with 3.6 and 40 ppm of DON, respectively (Forsyth et al., 1977), 23-29% and 30-72% for reduced feed intake

and weight gain respectively with 3.7 and 4.2 mg/kg fed to pigs (Friend et al., 1986). However, these differences could be due to the level of contamination, source of toxin (natural or pure), the physiology of the animal, type of feedstuff and the route of administration (Trenholm et al., 1994; Prelusky et al., 1991; Pestka et al., 1987).

The major routes of administration of DON described in the literature include oral (Trenholm et al., 1994; Young et al., 1987; Pollmann et al., 1984; Friend et al., 1986; Young et al., 1983), intravenous (iv) (Prelusky and Trenholm, 1991; Coppock et al., 1985) and intraperitoneal (ip) (Forsyth et al., 1977; Pestka et al., 1987). Intraperitoneal administration of DON was reported to be more effective than oral dosing as the animal responds within a shorter period of time after treatment (Forsyth et al., 1977). The minimum emetic doses of DON to swine of about 9-10 kg BW for the two routes were given at 0.05 mg/kg body weight and 0.1-0.2 mg/kg body weight for ip and oral administration respectively (Forsyth et al., 1977; Coppock et al., 1985). Emesis occurs at a shorter time from administration, if plasma DON concentration is high and after a prolonged period if the concentration is low (Coppock et al., 1985).

Naturally infected grains elicited more severe effects on feed refusal than pure compounds, suggesting that additional natural factors were involved in the induction of feed refusal (Pestka et al., 1987; Forsyth et al., 1977; Trenholm et al., 1994; Young et al., 1983). Feed refusal in pigs may be related to irritative action of DON on mucous membranes of the gastrointestinal tract (Friend et al., 1982; Young et al., 1987). DON is also implicated to cause thinning and erosion of the gastric mucosa with a higher degree of folding in the esophageal region (Trenholm et al., 1994; Pestka et al., 1987).

In contrast, thicker and heavier esophageal mucosa were found in pigs fed DON-contaminated diets (Rotter et al., 1992; Friend et al., 1984; Friend et al., 1983). It was therefore suggested that, the effects of DON on feed intake was systemically induced, as it was rapidly absorbed (about 67%) from the gastrointestinal tract. Excretion was also reported to be rapid (Friend et al., 1986).

The mechanism of trichothecene-induced emesis is believed to be via the chemoreceptor zone in the medulla oblangata (Ueno, 1983; Pestka et al., 1987), rather than involving direct effects on the gastrointestinal tract as was observed during acute toxicity (Forsyth et al., 1977; Pestka et al., 1987). Smith, (1992) stated that, behavioral responses including loss of appetite, may be initiated by inhibition of hepatic protein synthesis by trichothecenes and their metabolites absorbed from the gastrointestinal tracts. Feed refusal by animals consuming diets contaminated with trichothecene mycotoxins is caused by trichothecene-induced aminoacidemia; resulting in elevated brain serotonin concentrations which produce behavioral changes, such as loss of appetite and muscle incoordination (Smith, 1992).

**Metabolism and Excretion:**

Despite the possibility of human exposure to potential toxic residues, as mentioned earlier, severe financial losses may be incurred by the producers, due to DON contamination (Berry, 1988; Bullerman, 1986). DON is efficiently absorbed, but poorly metabolized and eliminated at a slower rate in pigs, compared to ruminants and poultry where DON is poorly absorbed and extensively metabolized, resulting in rapid clearance

from the biological fluids and tissues (Coppock et al., 1985; Prelusky et al., 1988; Prelusky and Trenholm, 1991). Other clinical effects of DON in swine includes depletion of hepatic glycogen, changes in blood glucose concentration, irritation of the gastrointestinal tract and erosion of the gastric mucosa (Pestka et al., 1987; Coppock et al., 1985; Trenholm et al., 1991).

Although tissue concentration of DON was said to be highest in the kidney and liver, its elimination was correspondingly high in urine and bile which was probably associated with their role in the elimination of the xenobiotics (Prelusky et al., 1991). The rate of urinary excretion of DON appears to be dependent upon the rate of urine formation. This means that, reduced water intake associated with field conditions, could result in decreased urine formation, thereby contributing to increased toxicity of DON to the animals (Coppock et al., 1985).

According to the observation of Prelusky et al., (1990) on the distribution of DON in the cerebral spinal fluid (CSF), after iv administration to swine and sheep, it was suggested that, the localization of DON could be compartmentalized into the central, deep and terminal compartments in swine. The terminal elimination phase was described as the elimination phase with a $t^{1/2}$ of 250 $\pm$ 52.3 min. There was a delay of about 30-60 min after iv administration before peak DON concentrations, reached the cerebral spinal fluid in swine. DON was detected in the CSF in about 6-10 hrs post dosing at a concentration of about 25.3 $\pm$ 7.7% of the total residue level measured in plasma (Prelusky et al., 1990).

Administration of DON through the intragastric route resulted in a quick systemic

absorption of the toxin which increased the concentrations in both the CSF and plasma, within 2.5 min (Prelusky et al. 1990). DON in the CSF has a long $t^{1/2}$ as it could be detected 20 hrs after administration.

A second route of DON clearance called the "extrarenal route" has been suggested since it was observed that a high percentage of iv-administered DON could not be accounted for in urine These DON may have been eliminated by extrarenal [hepatic, metabolite] routes (Coppock et al., 1985). The presence of DON in the bile, demonstrated that some of the DON was subjected to hepatic excretion (Prelusky and Trenholm, 1991). Furthermore, Coppock et al., (1985), observed some differences in the clearance rate in pigs that received DON administration either by iv infusion or by ip exposure, and stated that, ip administered DON was rapidly cleared from the portal blood. This was attributed to the "hepatic-first-pass" effect of DON administration by ip exposure, which rapidly clears DON from hepatic portal blood and supports the extrarenal clearance route of DON. The renal excretion of DON was altered by iv infusion of a saline solution which may indicate that, DON was both secreted and reabsorbed by the renal tubules (Coppock et al., 1985).

Hyperglycemia and pancreatic islet cell lesions were observed in pigs after iv administration of DON. Depletion of liver glycogen and pancreatic islet lesions have also been observed in pigs after iv administration of diacetoxyscirpenol (DAS) (Shimizu et al., 1979). These were accompanied by changes in blood glucose, showing that, DON-induced changes in intermediary metabolism may be an insidious aspect of DON intoxication in pigs (Coppock et al., 1985; Young et al., 1983).

The toxic effect of DON is reported to be more severe on males than females in terms of performance (Friend et al., 1986). DON was more toxic to male rodents than females (Iverson et al., 1985; Friend et al., 1986). The uterine weight of the pigs fed DON-contaminated wheat was significantly heavier than that ofpigs fed corn that was inoculated with DON, possibly due to the fact that the ZEN concentrations in the DON-contaminated wheat were twice that found in the DON-inoculated corn (0.40 mg/kg-1 and 0.25 mg/kg-1 respectively). This level was presumed to be too low to induce such symptoms, but was able to reach an unanticipated effective level to cause increased uterine weight (Friend et al., 1986). Increased uterine weight had previously been reported with increasing levels of DON (0.18, 3.79 and 6.24 mg/kg-1) plus very low levels of increasing ZEN (0.0, 0.15 and 0.24 mg/kg-1) respectively (Friend et al., 1986).

DON-contaminated diets could be subjected to detoxification treatments to reduce, if not eliminate, the toxic effects of DON on the animals. DON reacts readily with sodium bisulfite, resulting in a product called 10-sulfonate product (DON-S) which is stable in acid, but hydrolyzes to DON under alkaline conditions (Young et al., 1986b; Young et al., 1987). DON reduction was found to be dose (sodium bisulfite concentration) and time (reaction time) dependent (Young et al., 1986a). Increased moisture content and temperature also aided in the reduction of DON concentration (Young et al., 1987).

Treatment of DON-contaminated corn prior to diet formulation, made the resulting products more acceptable to pigs as opposed to the nontreated corn, which caused acute reduction in feed intake (Young et al., 1987). DON administered at an

effective dose (ED66) level caused severe emesis within 20 min in all the animals under that treatment but DON-S at ED66 and ED98 failed to elicit any observable response in the treated animals 3 hrs after dosing (Young et al., 1987).

Pigs are particularly susceptible to the adverse effects of DON in their diets and exhibited reduced feed intake and weight gain at concentrations equal to or lower than 2 mg/kg diet (Friend et al., 1982; Pollmann et al., 1985; Trenholm et al., 1994). However, subjecting contaminated corn to the detoxification processes by using sodium bisulfite effected a marked reduction in the DON concentration. Such treatment appeared to remove short term toxic effects of contaminated feeds and increased their acceptability by pigs (Trenholm et al., 1994; Young et al., 1986a).

## MATERIALS AND METHODS:

The research study consisted of four trials to determine the effects of vomitoxin on various aspects of sheep reproduction and growth.

The DON-contaminated barley used in all the trials was obtained from North Dakota and contained 29 ppm of DON. This was mixed with DON-free barley during formulations (Tables 1 and 2) to obtain the treatment levels used. Thus, the concentrates were formulated to contain the following DON-concentrations 0 ppm for the control, 4.0, 10.1 and 20.2 for treatments 1, 2 and 3, respectively in all trials.

The DON analysis was done in the Veterinary Diagnostic Laboratory, North Dakota State University, Fargo using a gas chromatographic (GC) technique as described by Bennett and Shotwell, (1990).

**Trial 1:**

Objective: To determine the effects of feeding barley containing vomitoxin (DON) to pregnant ewes.

### Animals and Routine:

One hundred and twenty ewes from two breeds [60 Suffolk (Su) and 60 Outaouais (Ou)], bred in October, 1993 were in their last trimester of pregnancy when the trial started on January 19, 1994. The Outaouais breed was developed by the Agricultural Research Center of Agriculture Canada at Ottawa. It was derived from the infusion of several breeds and contained about 50% Finnish Landrace breed when the flock was closed. It is now a recognized breed and is registered with the Canadian Livestock Record.

TABLE 1: Concentrate formulations used in Trials 1, 2 and 4 showing the amount of grain and supplement used for ration preparation/500 kg mix.

| Feedstuff (Kg) | Treatments | | | |
|---|---|---|---|---|
| | 0 | 1 | 2 | 3 |
| Clean barley | 405.0 | 324.0 | 202.5 | - |
| DON-contam. barley | - | 81.0 | 202.5 | 405.0 |
| Premix | 95.0 | 95.0 | 95.0 | 95.0 |
| Calculated DON | - | 4.0 | 10.1 | 20.2 |

DON- contam = Deoxynivalenol or vomitoxin contaminated barley
Calc. DON = Calculated deoxynivalenol (ppm)
Premix contained
Soy bean meal 35 kg; Low copper sheep mineral 50 kg containing (Ca 22.0%, P 14.0%, Fe 350 mg/kg, I 125 mg/kg, Cu 5 mg/kg, Zn 230 mg/kg, Co 50 mg/kg, Mn 550 mg/kg, Sel 8.2 mg/kg, F 2000 IU/kg, Vit A 80,000 IU/kg,
Vit D-3 45,000 IU/kg, Vit E 40 IU/kg); Molasses 10 kg.

TABLE 2: Formulations of pellet concentrates used in trial 3, showing the amount of grains and supplement used for ration preparation/500 kg mix

| Feedstuff (kg) | Treatments | | | |
|---|---|---|---|---|
| | 0 | 1 | 2 | 3 |
| Clean barley | 398.0 | 318.0 | 199.0 | - |
| DON-contam barley | - | 80.0 | 199.0 | 398.0 |
| Premix | 102.0 | 102.0 | 102.0 | 102.0 |
| Calculated DON | - | 4.0 | 10.0 | 20.2 |

DON- contam = Deoxynivalenol or vomitoxin contaminated barley
Calc. DON = Calculated deoxynivalenol (ppm)
Premix contained
Soy bean meal 80 kg; Low copper sheep mineral 5 kg containing (Ca 22.0%, P 14.0%, Fe 350 mg/kg, I 125 mg/kg, Cu 5 mg/kg, Zn 230 mg/kg, Co 50 mg/kg, Mn 550 mg/kg, Sel 8.2 mg/kg, F 2000 IU/kg, Vit A 80,000 IU/kg, Vit D-3 45,000 IU/kg, Vit E 40 IU/kg); Molasses 10 kg; Cobalt salt 2 kg and Limestone 5 kg.

Ewes were housed in a totally enclosed building with 10 animals to a pen measuring approximately 4 by 5.5 m. The animals received 8 hrs of light/day. Ewes were allocated as equally as possible according to breed and age to one of four treatments. The treatments were concentrate containing 0, 4, 10 or 20 ppm of vomitoxin. Each pen of ten ewes received 4.54 kg (0.45 kg/ewe) of concentrate and approximately 16 kg (1.6 kg/ewe/day) of alfalfa brome hay/day during the 47 day trial. Analysis of the diets are given in tables 3, 4 and 5. Water and salt lick were provided ad libitum.

Samples of concentrate were collected from 6 bags of feed in each treatment for analysis to confirm the level of vomitoxin after preparation of the feed. Samples of hay were also collected for proximate analysis of feed nutrients (Tables 3, 4 and 5). Number of lambs born alive, number of lambs born dead, average birth weight of lambs and number of ewes not lambing were recorded.

**Observations:**

Two ewes aborted their lambs, one of them died after abortion and five others were open at lambing. Total mortality rate of the lambs at lambing was 7.3% and ewes was 0.83%. However, autopsy examinations of the aborted lambs and dead ewe did not implicate the treatment as the cause.

**Trial 2:**

**Objective:** To determine the possible effects of feeding barley containing vomitoxin (DON) to lactating ewes, using lamb weaning weights as a measure of milk production.

## Animals and Routine:

Twenty-eight lactating ewes from Trial 1, were used in this trial. Ewes (16 Suffolk and 12 Outaouais) each with two lambs were selected to standardize the effects of number of lambs on milk yield. The ewes were allowed a period of 14 days from the time the first trial was terminated (7 days prior to lambing) to the time the second trial started (7 days after lambing) for acclimatization. The ewes were fed non-contaminated diets during this period. However, the ewes were maintained on the same treatments they received in Trial 1 during Trial 2.

The animals were housed in the Animal Science Research Unit (ASRU). They were housed in individual pens measuring about 1.2 by 2 m on plastic coated expanded metal floors. The ewes were blocked by breed with four Suffolk and three Outaouais ewes per treatment. These replicates with 2 lambs each gave a total of 14 lambs/treatment. The hay fed to the animals in the barn was chopped for ease of feeding and reduced wastage. Water and salt were provided ad libitum. The initial weight of lambs and ewes were considered to be the birth weight and ewe weight at lambing respectively, while the final weights were recorded at weaning which was 6 weeks after lambing, for both lambs and ewes.

Ewes were fed once a day with 0.68 kg of concentrate in the morning and 1.1 kg of alfalfa brome hay twice daily from March 22 to April 6, 1994 (14 days). The amount of grain and hay fed to the ewes was increased to 0.9 kg and kg to 3.0 kg for grain and hay, respectively, fed in equal amounts twice daily for the rest of the trial. The animals were fed each day between 08.00 to 09.30 hrs and 16.00 to 17.00 hrs for the morning and evening periods, respectively. A pelleted lamb creep feed ration was provided to the lambs ad libitum which contained non-contaminated barley. The lighting regime in the barn during this trial period provided for 16 hrs of light and the temperature was maintained at 19°C. The trial was terminated after 36 days or 43 day after lambing.

**Sampling/Data Collection:**

Lambs were weighed weekly from birth to the end of the trial at weaning (6 weeks of age). Ewes were weighed at the beginning (lambing period) and end of the trial (weaning) to determine the initial and final weight.

**General observations:**

Two lambs died during the trial giving a mortality rate for the lambs at 3.57% while none of the ewes died. The cause of the deaths were not determined and appeared not to be due to treatment.

**Trial 3:**

**Objective:** To determine the effects of feeding vomitoxin (DON) on the growth rate of lambs.

**Animals and Routine:**

One hundred and fourty one lambs born between March, 9 and 13, 1994 at the Glenlea Research Station from Trial 1 were used for this experiment. They were comprised of 69 Suffolk and 72 Outaouais lambs with approximately equal number of males and females from each breed.

Lambs averaged 17.3 kg in weight at the start of the trial and allocated 8-9/pen measuring approximately 4 by 5.5 m. They were self-fed a pelleted grain ration formulated to contain vomitoxin at levels of 0, 4, 10 and 20 ppm (Tables 2 and 4). They also received hay and water ad libitum. The trial was concluded when an average pen weight of approximately 32 kg per lamb was reached.

**Sampling/Data collection**

The lambs were weighed every two weeks and feed consumption was also recorded. Pellet samples were collected for analysis of DON and feed nutrient contents. The alfalfa brome hay was similar to that used in Trials 1 and 2 and fed long.

**General observations:**

Two lambs died during the trial. Autopsy revealed suggested a possible thiamine deficiency which did not occur in any one treatment.

**Trial 4:**

**Objective:** To determine the possible effects of feeding barley containing vomitoxin (DON) to ewes during breeding and early gestation.

## Animals and Routine:

Ninety mature ewes of the two breeds (45 Suffolk and 45 Outaouais), which were at the point of breeding were used in this trial. The trial took place in the sheep barn at the Glenlea Research Station and ran from September 26, 1994 to January 23, 1995. 11 or 12 ewes were placed in a pen measuring approximately 4 by 5.5 m. Feeding of the DON-contaminated diets started on September 26, 1994, with 0.2 kg of concentrate/ewe/day for the first week. This was increased to 0.45kg/ day from the time of sponging at the end of the first week and continued to the end of the trial. The concentrate contained either 0, 4, 10 or 20 ppm of vomitoxin (Tables 1 and 5) to provide the three same levels of vomitoxin intake as in previous trials. Ewes received an average of 1.4 kg of alfalfa brome hay/ewe/day.

Veramix sponges (Upjohn) were inserted on October 1, 1994 and removed on October 15, 1994 for synchronization of estrus. Rams were introduced into the pens on October 17, 1994, and remained with the ewes for two estrous cycles (30 days). One ram was placed in the pen containing 11 or 12 ewes. The rams brisket was painted with sheep branding paint and the color was changed after 15 days.

### Data Collection and observation

The parameters recorded included: dates of mating, pregnancy rate and lambing data. Samples of grain and hay were collected for proximate analysis and analysis for DON-concentration. Observations were made on the ewes to determine if there was feed refusal or loss of body weight associated with the consumption of DON-contaminated diets.

## Statistical Analysis

Data were analyzed as a randomized complete block design with four treatments in each breed group (Block) using the General Linear Model Procedure (Proc. GLM) (SAS Institute Inc. 1988)) for Trials 1 and 4. Treatment and breed effects on number of lambs born alive (NBA), number of lambs born dead (NBD), average birth weight (ABW) of lambs and their interaction were analyzed for Trials 1 and 4. The two breed groups of ewes in each trial were Suffolk and Outaouais and the ewes varied in age from yearlings to 6 years. Preliminary analysis of Trial 1 and 4 data showed that age of ewe was not an important effect, so it was not included in the analysis reported here.

Trials 2 and 3 were analyzed as a split plot repeated measures design with the main plot as a randomized complete block arrangement where block was breed and sex of lamb. For average daily feed consumption (ADFC) and average feed efficiency (AFE), pens were considered as main plots with weeks as subplot and for ADG, animals were the main plot with week as subplots. Repeated measurements were taken on the animals on a weekly basis, for the 6 weeks of Trial 2 and the 2-week interval in Trial 3 from 6 to 8 weeks of the trial.

A comparison of means test was done using the Bonferoni's T-test for multiple means in all trials.

## RESULTS

The results of the proximate analysis of the feeds used in the trials are shown in Tables 3, 4 and 5. The protein content of the feeds decreased slightly with increased level of toxin. Although these differences were not great, these differences could be due to the dilution of the clean barley which had about 12.8% protein with DON-contaminated barley which presumably had a lower protein content. However, the protein levels of the DON-contaminated grain was not determined. The protein level for diets used in Trials 1, 2 and 4 ranged from 13.6 to 16.7%, whereas those of Trial 3 were higher for the young growing lambs and ranged from 16.0 to 18.4%. These protin levels met the suggested NRC requirements (NRC, 1985). There were no major differences in the crude fat (CF), dry matter (DM), calcium ( Ca) and phosphorus (P) concentration within treatments as well as between Trials (Tables 3, 4 and 5).

The results of the analysis of feed samples for DON concentration, determined by the Veterinary Diagnostic Laboratory of North Dakota State University laboratories are listed in Table 6. There were some variations in analyzed concentrations but in general, they were close to concentrations that were calculated for the various treatments in the trials.

In Trials 1 and 2, the determined concentrations were 0.9, 4.5 10.8 and 18.6 ppm for treatments 0, 1, 2 and 3, respectively. The concentrations in Trial 3 were 0.4, 4.6 12.2 and 25.1 ppm for treatments 0, 1, 2 and 3, respectively. The determined concentrations in Trial 4 were 0.4 5.3, 12.4 and 19.0 ppm for treatments 0, 1, 2 and 3, respectively. Hay consumption in all trials ranged from 65 to 75% of total feed

TABLE 3: Percentage nutrient composition of concentrates and hay fed in Trials 1 and 2

| Rations[1] | Prot | CF | DM | ADF | NDF | CA | P |
|---|---|---|---|---|---|---|---|
| 0 | 16.7 | 1.4 | 90.8 | - | - | 1.0 | 1.0 |
| 1 | 15.5 | 1.5 | 91.0 | - | - | 0.7 | 0.8 |
| 2 | 13.6 | 1.5 | 91.1 | - | - | 1.1 | 1.0 |
| 3 | 14.2 | 1.5 | 91.1 | - | - | 0.8 | 0.9 |
| Clean barley | 12.8 | 1.8 | 89.8 | - | - | 0.1 | 0.4 |
| Hay 1 | 13.2 | 1.5 | 92.7 | 37.9 | 54.5 | 0.9 | 0.2 |

Rations[1] 0, 1, 2 and 3 contained 0, 4, 10.1 and 20.2 ppm of DON, respectively.
Prot = Protein
CF = Crude fat
DM = Dry matter
ADF= Acid detergent fibre
NDF= Neutral detergent fibre
Ca = Calcium
P = Phosphorus
Hay 1 = Hay used for Trials 1, 2 and 3

TABLE 4: Percentage nutrient composition of concentrates (pellet) and hay fed in Trial 3

| Rations[1] | Prot | CF | DM | ADF | NDF | CA | P |
|---|---|---|---|---|---|---|---|
| 0 | 18.1 | 2.2 | 91.3 | | | 0.9 | 0.6 |
| 1 | 18.4 | 2.0 | 90.5 | - | - | 0.8 | 0.6 |
| 2 | 17.1 | 1.6 | 90.6 | - | - | 0.8 | 0.6 |
| 3 | 16.0 | 1.3 | 90.8 | - | - | 0.6 | 0.6 |
| Clean barley | 12.8 | 1.8 | 89.8 | - | - | 0.1 | 0.4 |
| Hay 1 | 13.2 | 1.5 | 92.7 | 37.9 | 54.6 | 0.9 | 0.2 |

Rations[1] 0, 1, 2 and 3 contained 0, 4, 10.1 and 20.2 ppm of DON respectively.
Prot = Protein
CF = Crude fat
DM = Dry matter
ADF= Acid detergent fibre
NDF= Neutral detergent fibre
Ca = Calcium
P = Phosphorus
Hay 1 = Hay used for Trials 1, 2 and 3

TABLE 5: Percentage nutrient composition of concentrates and hay fed in Trial 4

| Rations[1] | Prot | CF | DM | ADF | NDF | CA | P |
|---|---|---|---|---|---|---|---|
| 0 | 15.5 | 1.4 | 89.9 | | | 0.9 | 0.9 |
| 1 | 14.4 | 1.9 | 90.1 | - | - | 0.8 | 0.8 |
| 2 | 13.8 | 1.8 | 90.5 | - | - | 0.5 | 0.7 |
| 3 | 13.2 | 1.2 | 90.0 | - | - | 0.8 | 0.9 |
| Clean barley | 12.8 | 1.8 | 89.8 | - | - | 0.1 | 0.4 |
| Hay 1 | 13.2 | 1.5 | 92.7 | 41.0 | 60.0 | 0.6 | 0.3 |

Rations[1] 0, 1, 2 and 3 contained 0, 4, 10.1 and 20.2 ppm of DON respectively.
Prot = Protein
CF = Crude fat
DM = Dry matter
ADF= Acid detergent fibre
NDF= Neutral detergent fibre
Ca = Calcium
P = Phosphorus
Hay 2 = Hay used for Trial 4

TABLE 6: Deoxynivalenol (DON) concentrations (ppm) in diets from the different Trials.

| Trials | Treatments | | | | |
|---|---|---|---|---|---|
| | Items | 0 | 1 | 2 | 3 |
| 1 | Concentrate | 0.9 | 4.5 | 10.8 | 18.6 |
| | Amount (total feed) | 0.2 | 1.0 | 2.3 | 4.0 |
| 2 | Concentrate | 0.4 | 4.6 | 10.8 | 18.6 |
| | Amount (total feed) | 0.1 | 1.2 | 2.8 | 4.9 |
| 3 | Concentrate | 0.4 | 4.6 | 12.2 | 25.1 |
| | Amount (total feed) | 0.1 | 1.2 | 3.4 | 7.3 |
| 4 | Concentrate | 0.4 | 5.3 | 12.4 | 19.0 |
| | Amount (total feed) | 0.1 | 1.3 | 3.0 | 4.6 |

consumption/day, resulting in reduced contamination levels in the total feed in all the trials. DON concentration in the total feed for treatments 0, 1, 2 and 3 of Trial 1with the hay dilution effect were calculated to be 0.2, 1.0, 2.3 and 4.0 ppm, respectively. Similarly, 0.1, 1.2, 2.8 and 4.9 ppm were the DON concentrations for treatments 0, 1, 2 and 3, respectively in Trial 2. Concentrations of 0.1, 1.2, 3.4 and 7.3 ppm were calculated for Trial 3 for treatments 0, 1, 2 and 3, respectively. Finally in Trial 4, the diets contained 0.1, 1.3, 3.0 and 4.6 ppm for treatments 0, 1, 2 and 3 respectively, (Table 6).

**TRIAL 1**

The data collected from Trial 1 were analyzed for number of lambs born alive, number of lambs born dead (NBD) average birth weight of lambs (ABW) and number of ewes not lambing (NNL). Although insignificant, there tended to be a treatment difference on the number of lambs born alive as the animals that received the highest concentration of DON had a greater number of lambs born alive than the control and low level of DON (Fig. 1; Table 7). However, the only significant difference was observed when treatment 1 was compared to treatment 3, with treatment 3 exhibiting a significantly ($p < 0.05$) greater number of lambs born alive than treatment 1 (Fig.1; Table 7). The breed differences show that, the Ou breed had a significantly ($p < 0.05$) greater number of lambs born alive than the Su breed (Fig 2; Table 7). The number of lambs born dead were not different due to treatments but, was different due to breed. Su breed ewes had lower number of lambs born dead than the Ou ewes. The total number

breed ewes had lower number of lambs born dead than the Ou ewes. The total number of lambs born/ewe was higher in the Ou ewes (2.8) than the Su ewes (1.8).

The data on the average birth weight (ABW) of lambs showed that, as the NBA increased the ABW of the lambs decreased.

TABLE 7: The effects of treatments and breeds on the gestation length (GL; days), number of lambs born alive (NBA), number of lambs born dead (NBD), average birth weight (ABW; kg) and number of ewes not lambing (NNL) (Trial 1)[1]

| Treatment | n | NBA | NBD | ABW | NNL |
|---|---|---|---|---|---|
| 0 | 29 | $2.1 \pm 0.17^{ab}$ | $0.2 \pm 0.09$ | $4.2 \pm 0.14^{ab}$ | 1 |
| 1 | 27 | $1.8 \pm 0.17^{a}$ | $0.2 \pm 0.09$ | $4.3 \pm 0.15^{a}$ | 3 |
| 2 | 29 | $2.1 \pm 0.17^{ab}$ | $0.1 \pm 0.08$ | $3.8 \pm 0.14^{ab}$ | 1 |
| 3 | 28 | $2.5 \pm 0.17^{bc}$ | $0.1 \pm 0.09$ | $3.6 \pm 0.15^{bc}$ | 2 |
| **Breed** | | | | | |
| Ou | 55 | $2.5 \pm 0.12$** | $0.2 \pm 0.06$ | $3.8 \pm 0.10$ | 5 |
| Su | 58 | $1.7 \pm 0.12$ | $0.1 \pm 0.06$ | $4.8 \pm 0.10$** | 2 |

[1] Least square means ± SEM
n = Number of animals in a treatment
** = Significant ($p < 0.0001$) difference between breeds
Ou = Outaouais
Su = Suffolk
Means within a column with different superscripts are significantly ($p < 0.05$) different.

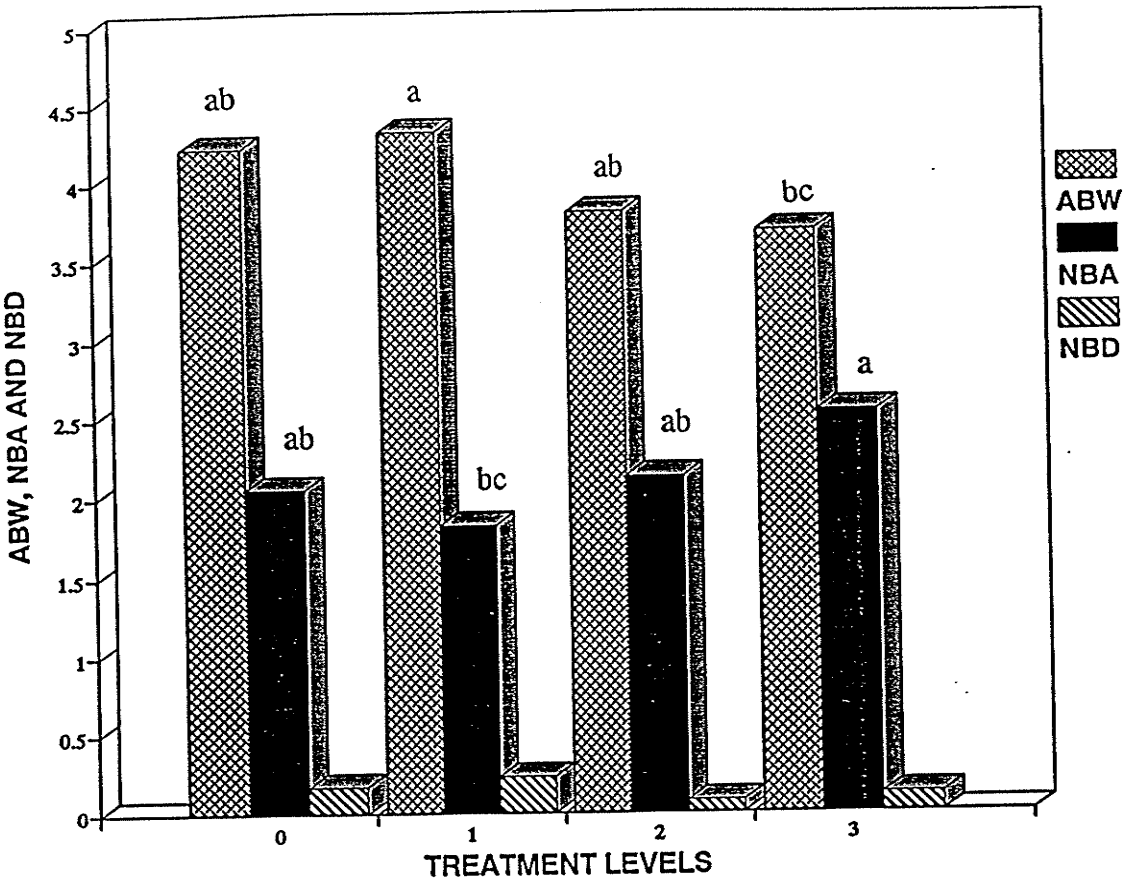


**Figure 1:** Comparison of treatment effects on average birth weight of lambs (ABW), number of lambs born alive (NBA) and number of lambs born dead (NBD). Similar bars with different superscripts are significantly different

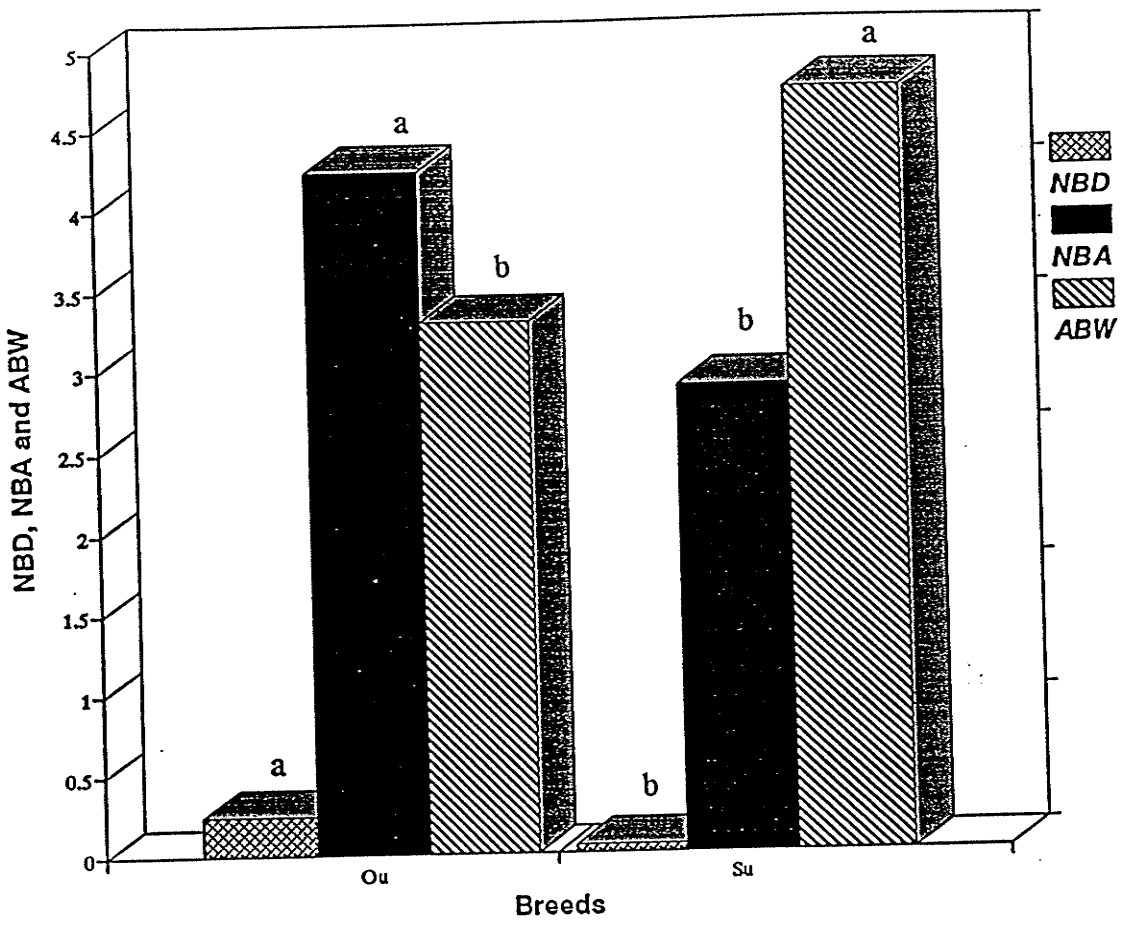


**Figure 2:** Breed differences on number of lambs born dead (NBD), number of lambs born alive (NBA) and average birth weight of lambs (ABW).
Similar bars with different superscripts are significantly different

The lambs from the ewes fed the highest level of DON had the lowest ABW which were significantly ($p < 0.005$) different when compared to treatment 1 but not the other treatment groups (Table 7). The breed effects showed that Su lambs had greater ABW than the Ou lambs. The ABW reflected those of the NBA and NBD with the lowest level of DON having the highest ABW and the highest level of DON having the lowest ABW (Fig 1). These results suggest that, as the number of fetuses carried by an ewe increased, there was increased number of lambs born dead as well as a decrease in ABW which is most likely due to neonatal competition for nutrients in the uterus.

**TRIAL 2**

a) LAMBS:

Trial 2 dealt with the effect of DON-contaminated diets on lactation performance of ewes as judged by growth of their suckling lambs. The weight data collected at weekly intervals were analyzed to determine if there was a time by treatment interaction to ascertain if there was any build up effect of the toxin as the trial progressed over time However, this interaction of treatment by week was not different, indicating no cumulative effect of the toxin.

The analysis of the weaning weight showed that, there were differences due to treatments as lambs whose dams were fed the highest DON-diet had a significantly lower ($p < 0.05$) weaning weight than the other treatments (Fig 3; Table 8). There tended ($p < 0.07$) to be a significant three way interaction of treatment by breed by sex, with Ou male lambs

TABLE 8: Effects of treatment, breed and sex on the average total gain and weaning weight of lambs during lactation[1]

| Treatments | n | Average total gain (kg) | Weaning weight (kg) |
|---|---|---|---|
| 0 | 14 | 11.4 ± 0.44 | 15.5 ± 0.51[ab] |
| 1 | 14 | 12.1 ± 0.47 | 16.5 ± 0.64[a] |
| 2 | 14 | 12.0 ± 0.56 | 16.2 ± 0.64[a] |
| 3 | 14 | 10.7 ± 0.54 | 14.0 ± 0.63[b] |
| **Breed** | | | |
| Ou | 24 | 11.7 ± 0.38 | 15.1 ± 0.44 |
| Su | 32 | 11.5 ± 0.34 | 16.0 0.39 |
| **Sex** | | | |
| Male | 30 | 12.1 ± 0.35 | 16.1 ± 0.40 |
| Female | 26 | 11.0 ± 0.37* | 15.0 ± 0.42* |

[1] Least square means ± SEM
n = number of animals
* = Significant ($p < 0.05$) difference between sexes
Ou = Outaouais
Su = Suffolk
Means within a column with different superscripts are significantly ($p < 0.05$) different.

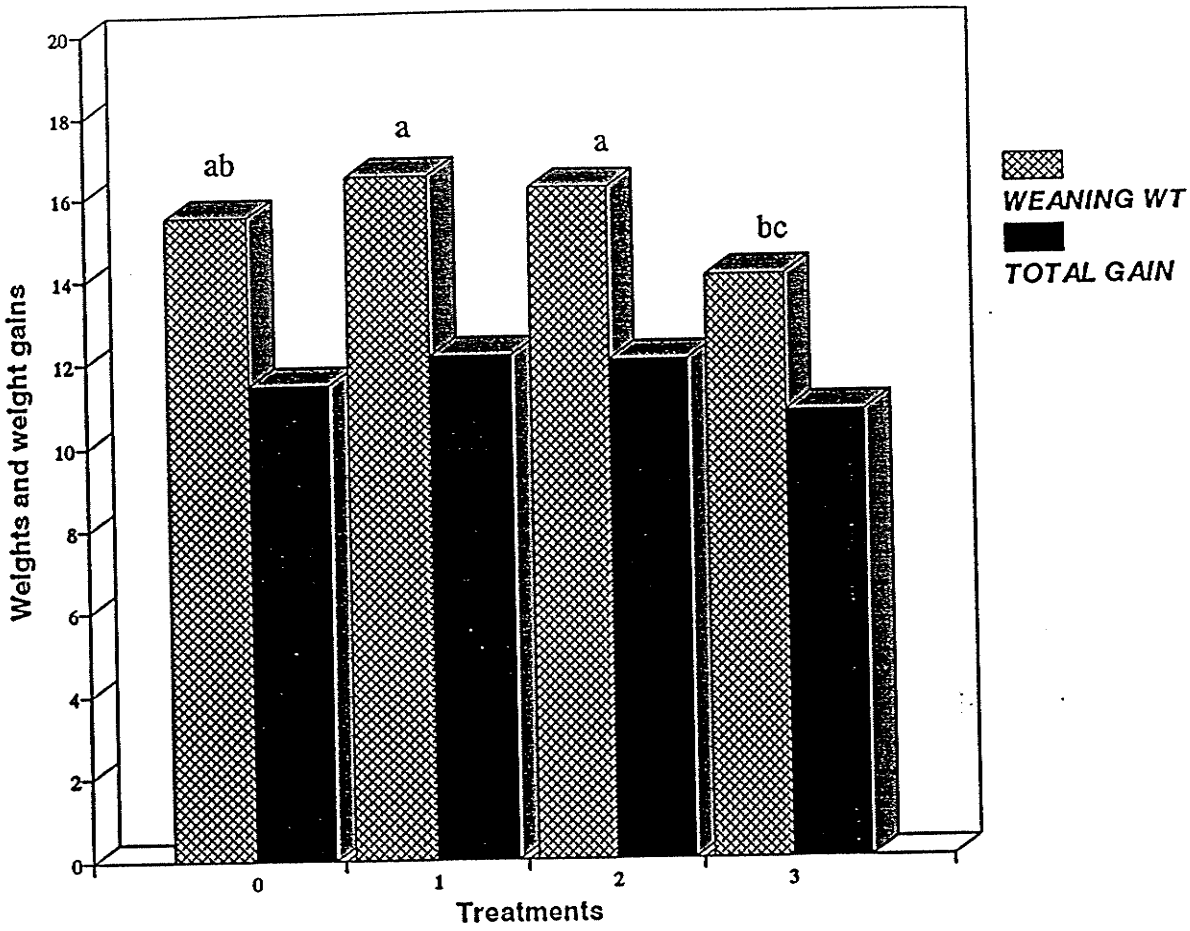


**Figure 3:** Treatment effects on average total gain and weaning weight of lambs.

Similar bars with different superscripts are significantly different

having greater weight decrease in response to DON than Ou females, Su males and Su females (Fig 4). There was also a significant sex difference ($p < 0.05$), but no treatment and breed interaction in weaning weight (Table.8). Weaning weight was significantly ($p < 0.05$) lower in treatment 3 as compared to the other treatments.

There was no breed difference in the overall weaning weight of lambs from both breeds. There was also no treatment differences in average total gain of lambs. There were significant differences in average total gain and weaning weight of lambs due to sex (Table 8).

b) EWES

The performance characteristics of ewes were also followed throughout the lactation trial. The data collected from the ewes includes the initial weight which was the lambing weight of the ewes, ewe weight loss during lactation, and weight gain by the ewe's 2 lambs during nursing period. The data showed that breed was significantly different ($p < 0.05$) for initial weight as the Su ewes were heavier than the Ou ewes (Table 9).

Although the Su ewes had a significantly higher initial weight, their weight loss during lactation was significantly ($p < 0.05$) higher than that of the Ou ewes (Fig 5; Table 9). However, the associated lamb gain to these losses was not different between breeds. Treatment was significantly ($P < 0.05$) different for initial weight with treatment 3 showing the lowest weight, when compared to the other treatment groups, but ewes in this treatment had a significantly less weight loss during lactation ($p < 0.05$) (Fig 6).

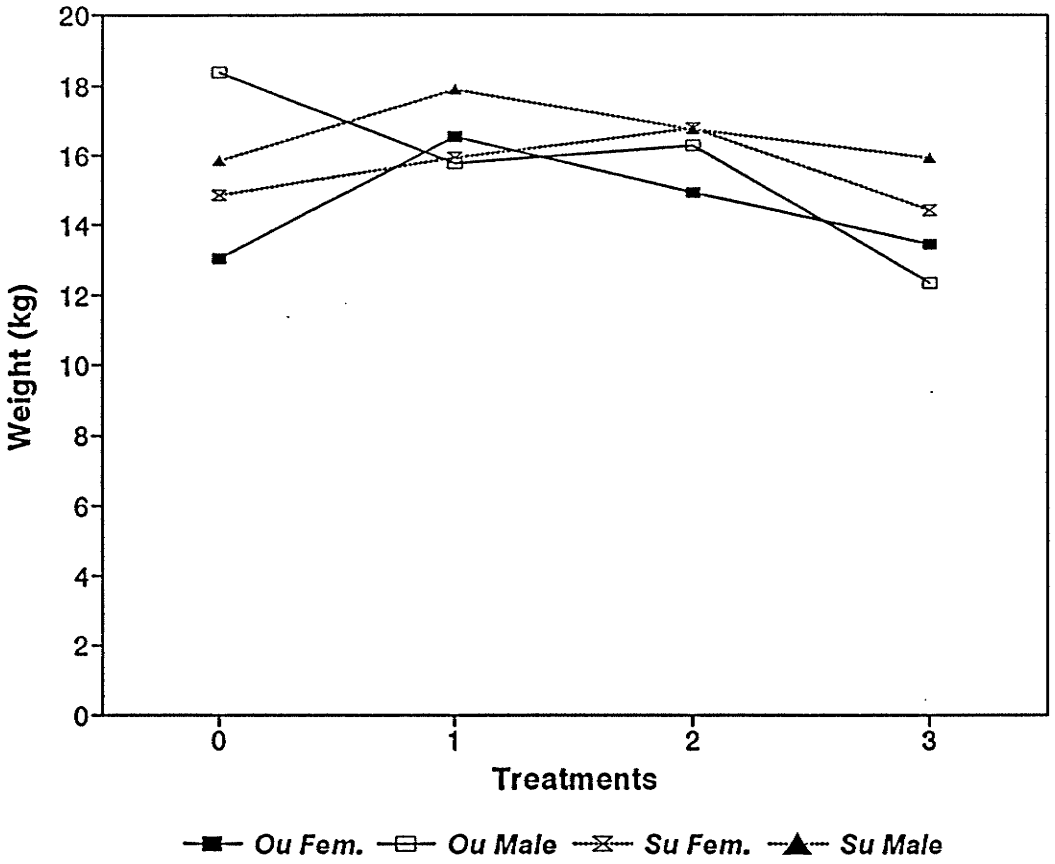


**Fig. 4 The interact of treatment, breed and sex on weaning weight**

TABLE 9: The effects of treatments and breeds on the initial weight, ewe weight loss and lamb weight gain during the lactation period[1]

| Treatment | n | Initial weight (kg) | Ewe loss (kg) | Lamb gain (kg) |
|---|---|---|---|---|
| 0 | 7 | 80.9 ± 3.63[b] | 12.4 ± 1.68[a] | 22.9 ± 1.20 |
| 1 | 7 | 90.8 ± 3.63[a] | 12.2 ± 1.68 [a] | 24.3 ± 1.20 |
| 2 | 7 | 81.1 ± 3.63[b] | 13.3 ± 1.68[a] | 24.0 ± 1.20 |
| 3 | 7 | 74.3 ± 3.63b[c] | 9.6 ± 1.68[b] | 21.5 ± 1.20 |
| **Breeds** | | | | |
| Ou | 12 | 73.0 ± 2.74 | 9.0 ± 1.27 | 23.1 ± 0.91 |
| Su | 16 | 90.6 ± 2.38* | 14.7 ± 1.10* | 22.9 ± 0.79 |

[1] Least square means ± SEM
n = Number of animals
* = Significantly ($p < 0.05$) greater than Ou
Ou =Outaouais
Su Suffolk
Means within a column with different superscripts are significantly ($p < 0.05$) different

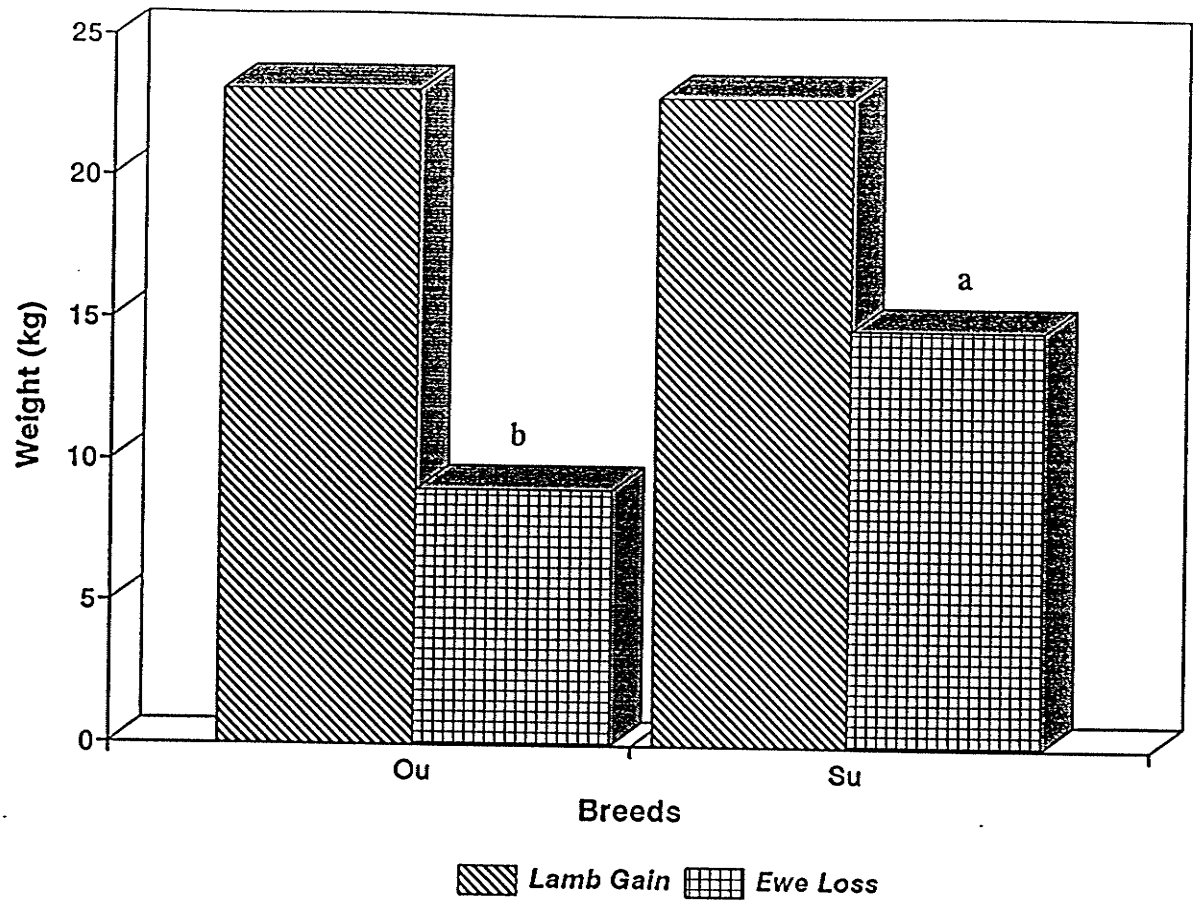


**Figure 5:** Effects of breed on lamb weight gain and ewe weight loss.
Similar bars with different superscripts are significantly different

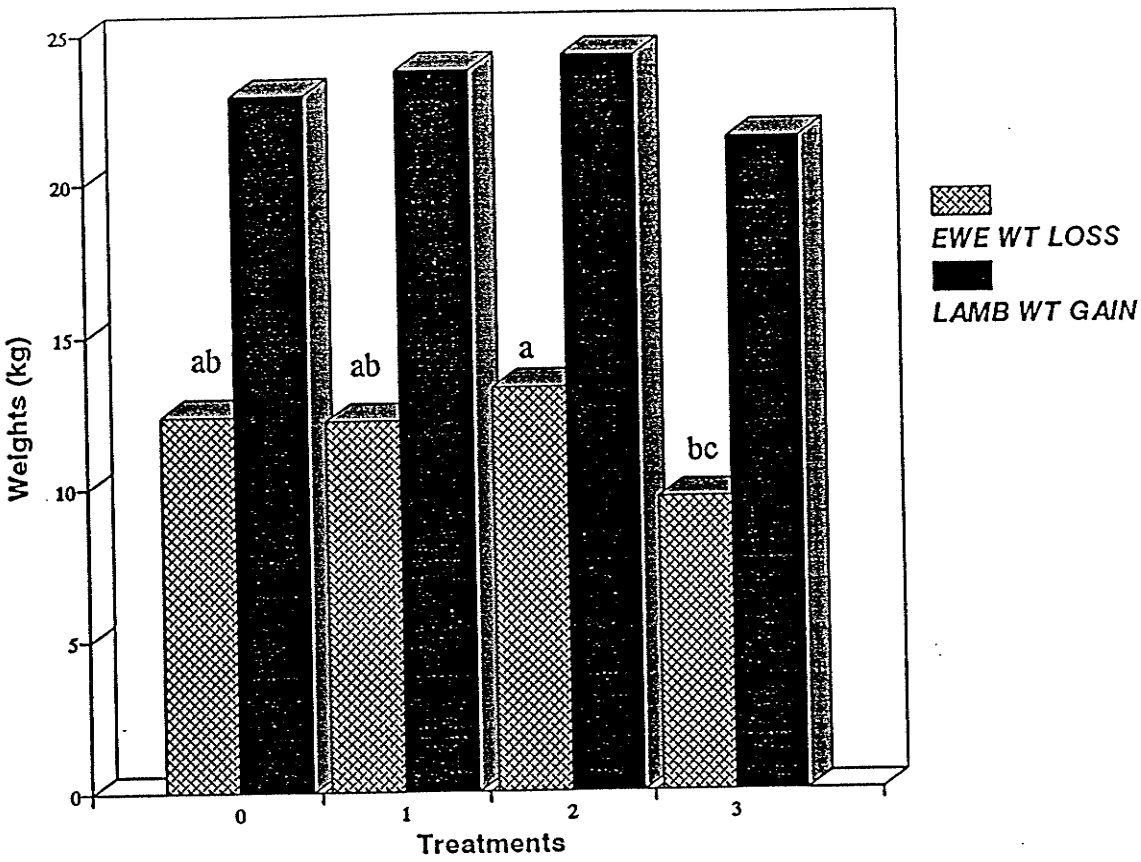


**Figure 6:** Treatment effects on ewe weight loss and lamb weight gain.
Similar bars with different superscripts are significantly different

**TRIAL 3**

The initial analysis which used period as a part of the split plot repeated measure analysis to test if there was period by treatment interaction, indicated that, there was weight difference ($p < 0.05$), due to period and period by sex interaction but not treatment by period and the 3-way interaction of treatment by breed by period was significant ($p < 0.05$). A closer look at the data revealed that, Ou lambs of treatment 3 out-performed the Su lambs in both period 1-2 and 2-3, resulting in a significant treatment by breed by period interaction, whereas, the differences in the other treatment levels and periods were very small and negligible.

This study was then analyzed for final weight and total weight gain of lambs measured for the 6 to 8 weeks of the trial as a split plot repeated measures analysis with randomized complete block arrangement and the results did not show any significant difference for treatments in both final weight and weight gain. Breed was significant for weight gain ($p < 0.05$) with Ou lambs having greater weight gain than Su lambs, but there were no such results found for final weight. Body weight and total gain were significantly ($p < 0.05$) different by sex with the male lambs exhibiting heavier weights and weight gains than the females (Table 10). There was also a significant ($p < 0.05$) interaction of treatment by breed (Fig 7), but the interactions of treatment by sex, treatments by breeds by sex, breeds by sex, and the overall interactions of treatments by breeds by sex by periods in the final weight and weight gain of lambs were not significantly ($p < 0.05$) different.

TABLE 10: Effects of treatment, breed and sex on the total gain and final weight of lambs[1]

| Treatments | n | Total gain (kg) | Final weight (kg) |
|---|---|---|---|
| 0 | 35 | 14.76 ± 0.48 | 32.1 ± 0.77 |
| 1 | 36 | 14.84 ± 0.47 | 32.1 ± 0.75 |
| 2 | 35 | 14.65 ± 0.47 | 32.1 ± 0.76 |
| 3 | 34 | 14.16 ± 0.49 | 32.3 ± 0.78 |
| **Breed** | | | |
| Ou | 70 | 15.35 ± 0.34** | 32.7 ± 0.54 |
| Su | 68 | 14.36 ± 0.34 | 31.6 ± 0.54 |
| **Sex** | | | |
| Male | 68 | 15.98 ± 0.34* | 34.2 ± 0.54* |
| Female | 70 | 13.73 ± 0.34 | 30.1 ± 0.54 |

[1] Least square means ± SEM
n = number of animals in a treatment
** = Significantly ($p < 0.0001$) greater than the Su
* = Significantly ($p < 0.05$) greater than the female
Ou = Outaouais
Su = Suffolk

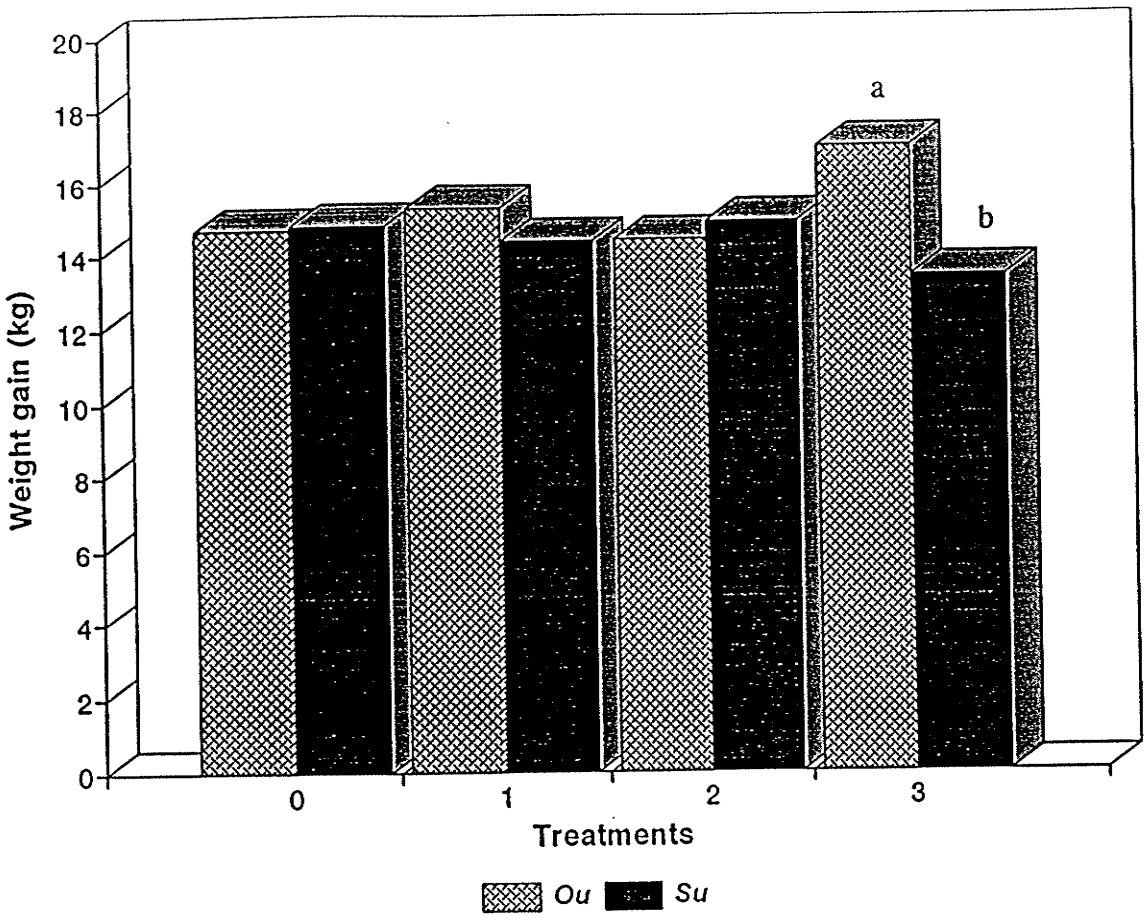


**Fig. 7 Effects of Treatment by breed on total gain of lambs**
bars with different superscripts are significantly different

The analysis which dealt with the average daily feed consumption (ADFC), average daily gain (ADG) and average feed efficiency (AFE), showed no significant difference between the treatments, breeds, and sexes. There was however, a trend towards some differences between sexes (Table 11) with the males gaining faster than the females (0.4 vs 0.3 kg/day). Although there was no significant difference in ADFC, ADG and AFE due to treatment, breed, sex, and their interactions (Fig 8), the males exhibited better feed efficiency and ADG, than the females in all parameters measured (Table 11).

**TRIAL 4**

The ewes were observed for changes in mating behavior, return to estrus, feed refusal and body weight changes. The observations showed absolutely no changes in mating behavior. There was also no observed loss of body weight, abortion or feed refusal. However, 16 out of 68 treated ewes returned to estrus, giving a total of 76.5% conception rate at first breeding. Five out of 22 ewes allocated to the control treatment also returned to estrus, giving a conception rate of 77.3% for the control groups. All the ewes that returned to estrus after the first breeding were rebred, but lambing results have shown that 6 of the Suffolk ewes, 2 each from treatments 0, 1 and 2 did not conceive. All ewes in treatment 3 had lambs showing that treatment had no effect on the performance of the Suffolk ewes. Only three of the Outaouais ewes did not conceive and no effect of DON treatment could be detected. The means for the ABW of lambs however does not include the data from Outaouais ewe lambs as all the data from the

TABLE 11: The effects of treatment, breed and sex on the average daily feed consumption (ADFC), average daily gain (ADG) and average feed efficiency (AFE) of lambs fed DON-contaminated diets[1].

| Treatments | n | ADFC (kg) | ADG (kg) | AFE (kg) |
|---|---|---|---|---|
| 0 | 35 | 1.3 ± 0.08 | 0.4 ± 0.01 | 3.7 ± 0.15 |
| 1 | 36 | 1.3 ± 0.08 | 0.4 ± 0.01 | 3.5 ± 0.15 |
| 2 | 35 | 1.2 ± 0.08 | 0.4 ± 0.01 | 3.5 ± 0.15 |
| 3 | 34 | 1.3 ± 0.08 | 0.4 ± 0.01 | 3.6 ± 0.15 |
| **Breeds** | | | | |
| Ou | 70 | 1.3 ± 0.55 | 0.4 ± 0.01 | 3.6 ± 0.11 |
| Su | 68 | 1.2 ± 0.55 | 0.3 ± 0.01 | 3.5 ± 0.11 |
| **Sex** | | | | |
| Male | 68 | 1.3 ± 0.06 | 0.4 ± 0.01 | 3.5 ± 0.11 |
| Female | 70 | 1.2 ± 0.06 | 0.3 ± 0.01 | 3.7 ± 0.11 |

[1] Least square means ± SEM
n = Number of animals
Ou = Outaouais
Su = Suffolk

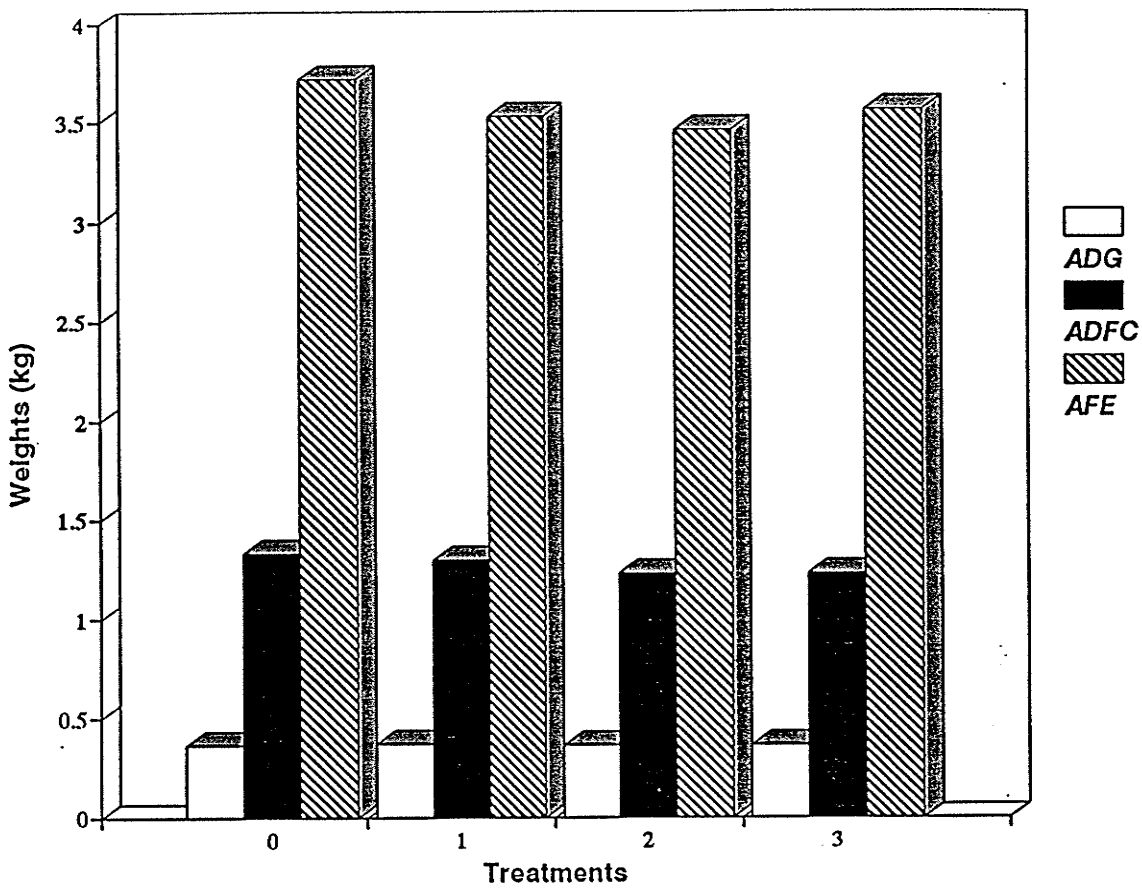


**Figure 8:** The effects of treatments on average daily gain (ADG), average daily feed consumption (ADFC) and average feed efficiency (AFE).

animals was not recorded. The total number of the ewes not lambing was 9 out of 90 ewes that were bred. These were distributed as follows among the various treatments; treatment 0 = 3, treatment 1 = 3, treatment 2 = 2 and treatment 3 = 1. Three of the 9 ewes were Ou ewes while the remaining 6 were Su ewes.

Data on the rebreeding interval was analyzed and no significant difference in the rebreeding interval between the control and treated groups was noted. There also was no breed differences. The rebreeding interval averaged 16 days over all treatments and breeds which is the average length in days, of the estrous cycle of the sheep.

The lambing data was analyzed for GL, NBA, NBD and ABW. Bonferoni's multiple comparison T-test showed that, there was no significant difference ($p > 0.05$) in all the parameters due to treatment. The analysis however revealed a significant difference ($p < 0.0001$) due to breed in NBA, with the Ou ewes having an average NBA of 2.8 lambs/ewe while the Su ewes had 1.5 lambs/ewe. Furthermore, there was a significant breed difference ($p < 0.0001$) in ABW of lambs, with the Su lambs exhibiting greater ABW than the Ou lambs (Table 12).

The overall number of lambs born/ewe did not differ from the NBA average because, only a minimal number of stillbirths were recorded for both the treatments and breeds. There was no interaction effect of treatment by breed.

These results show that there was no observed toxicosis due to DON which affected pregnancy rate.

TABLE 12: The effects of vomitoxin-contaminated barley and breed on the gestation length (GL;days), number of lambs born alive (NBA), number of lambs born dead (NBD), average birth weight (ABW; kg) of lambs and number of ewes not lambing (NNL) (Trial 4)[1]

| Treat. | n | GL (days) | NBA | NBD | ABW (kg) | NNL |
|---|---|---|---|---|---|---|
| 0 | 19 | 144.5±0.48 | 2.0±0.19 | 0.17±0.07 | 4.5±0.18 | 3 |
| 1 | 19 | 144.3±0.48 | 2.2±0.19 | 0.11±0.07 | 4.3±0.18 | 3 |
| 2 | 21 | 143.5±0.46 | 2.1±0.18 | 0.0±0.06 | 4.4±0.17 | 2 |
| 3 | 21 | 144.5±0.47 | 2.3±0.18 | 0.15±0.07 | 4.3±0.17 | 1 |
| **Breed** | | | | | | |
| Ou | 41 | 144.5±0.33 | 2.8±0.13* | 0.07±0.05 | 3.6±0.13 | 3 |
| Su | 39 | 143.8±0.34 | 1.5±0.13 | 0.14±0.05 | 5.2±0.12* | 6 |

[1] Least square means ± SEM
* Significant ($p < 0.0001$)

# DISCUSSION

The concentration of DON in the feed samples collected from the last preparation (one year after the first preparation) was slightly higher than those of the first preparation (4.5 vs 5.3, 10.8 vs 12.4 and 18.6 vs 19.0) for treatments 1, 2 and 3, and for first and second preparations respectively, indicating that, the toxin level had not decreased during storage. Although El-Banna et al., (1983) noticed similar stability of DON in contaminated diets, they reported a slight increase in the concentration of DON IN broiler finisher diets stored at two different temperatures which is in contrast to the increased DON concentration observed in this study.

The high DON concentration in the treatment 3 pellets after preparation cannot be explained except perharps, it may have been due to sampling error. It was anticipated that, since the grain was subjected to heating during the process of pellet preparation, it would have decreased the DON concentration in the diets. This was not so, showing that, DON is resistant to heat. This result is in agreement with Kamimura, (1989), who stated that trichothecenes are heat stable at 120°C, moderately stable at 180°C and decompose within 30 to 40 min at 210°C (Hulan and Proudfoot, 1982).

## TRIAL 1:

The results from this study showed that, treatment was significantly different ($p < 0.05$) in ABW. The ABW of the lambs decreased as the NBA increased. Although, the NBA was significantly ($P < 0.05$) higher than in treatment 1, it is hard to explain this difference. It may have occurred by chance. This was assumed to be the first trial

to look at this line of DON effects, but coincidentally, Windels, (1994) conducted a similar study in North Dakota and observed that, the ABW of lambs was reduced with increased toxin level (from 11.1 lbs or 5.04 kg to 9.30 lbs or 4.20 kg), but the difference was not significant when compared to the other treatment groups. The difference could also be due to higher number of lambs born.

The number of lambs born per ewe in this study was highest in treatment 3 with 2.5 $\pm$ 0.17 (mean $\pm$ SD). Although there was no evidence of a significant difference between treatment 3 and the control, there was a significant ($p < 0.05$) difference between treatment 3 and treatment 1 (1.81 $\pm$ 0.17). Our results were very similar to those of Windels, (1994) who reported 2.5/ewe in the group fed the highest concentration of DON treatment (12 ppm) and 1.83 lambs/ewe for the other treatment groups. The overall number of lambs born per treatment was higher (15) in the highest DON level than in both the control or the 6 ppm treatments which had 11 lambs each. The number of lambs born alive in treatment 1 was significantly lower ($p < 0.05$) than in treatment 3 but not the control. There was a similar trend in the results for NBD, which was not statistically different with treatment 1 having higher NBD than treatment 3. The lambing rate for all the treatments was high with low mortality rates. However, treatment 1 had a higher mortality rate than the other treatment groups which is another interpretation of the high NBD result obtained.

Breed differences were noticed in almost all the parameters measured. The Ou breed had a greater number of lambs born alive as well as greater number of lambs born dead, and thus a higher overall number of lambs born per ewe (2.8 vs 1.8 respectively).

The Su lambs had a greater ABW (4.7 $\pm$ 0.1 kg) than the Ou (3.3 $\pm$ 0.1 kg) lambs, which could partially be interpreted to mean that, the lower the number of lambs, the greater the nutrients available from the ewe and the greater the ABW of lambs.

**TRIAL 2**

a) LAMBS

This study showed that, there were differences in weaning weight, due to treatments. Treatment 3 lambs were significantly lower in weaning weight ($p < 0.05$) or (14.0 $\pm$ 0.63 vs 15.52 $\pm$ 0.51 kg) than the control treatment. Treatments 1 and 2 exhibited the greatest weaning weight. There was also a trend towards significance in the three-way interaction of treatment by breed by sex ($p < 0.07$) with Ou males having greater weight decrease in response to DON than Ou females, Su males and Su females. The effects of DON is more severe on males than in females in rodents (Friend et al., 1986; Iverson et al., 1985), and this is in agreement with the results of the 3-way interaction obtained in our study . In contrast to the result of this study, there was no overall treatment effects on weight of lambs fed DON-contaminated diets (Oltjen et al., 1984; DeHaan et al., 1983).

Furthermore, there has never been any published study as far as the author knows which used the lambs weaning weight and weight gain to study the toxic effects of DON fed to the lactating ewes. Several studies also revealed that, the bioavailability of DON after administration is 7.5% of the total DON administered (Prelusky et al., 1985; King et al., 1984). This was due to the increased rate of absorption and efficient metabolism

by the rumen microorganisms. This may mean therefore that, the levels fed to the ewes in this trial may be too low to elicit a similar response on the lambs. However, considering the fact that the rumen of the lambs at this stage of life have not yet been fully developed with the microorganisms, it may be suggested that, any level of DON could affect the lambs because they do not have the functional rumen protection against the toxin.

The result of the statistical analysis of the total weight gain showed that there was no difference due to treatment. This is similar to the results reported by other researchers (Oltjen et al., 1984; DeHaan et al., 1983; Harvey et al., 1986). The sex difference in which the male lambs gained more weight than the females was normal but has not been reported for lambs fed DON containing diets. Other researchers in contrast have reported that males are more affected by DON than females.

**TRIAL 2**

b) EWES

Weight loss of treatment 1 ewes tended to be greater ($p < 0.06$) than those of treatment 3, indicating that treatment 3 did not affect the performance of ewes during lactation. Initial weight of Su ewes were significantly ($p < 0.0001$) greater than those of Ou ewes (Table 9).

It is clear that nursing caused weight losses but, the Su ewes which had a significantly higher ($p < 0.0001$) initial weight than the Ou ewes also showed a significantly higher final weight. However, there was a significantly ($p < 0.05$) greater weight loss of the Su ewes when compared to the Ou ewes. A look at the lambs weight gains achieved as a result of these losses showed that, there was no statistical difference ($p > 0.05$) between the two breeds. The breed differences may indicate that, Su ewes are more susceptible to the toxin (vomitoxin) than the Ou ewes or that they milk more from reserves or consumed less feed.

**TRIAL 3:**

The results of this trial did not show any significant ($p > 0.05$) difference in ADFC, ADG, AFE or total weight gain of the lambs due to treatments or breeds except for breed. The final weight of the lambs was approximately 32.4 kg between 12 to 14 weeks of age. Harvey et al. (1986) also reported that weight gain in Rambouillet lambs during a 28 day trial was not affected by the concentration of DON in the diet. Similarly, results were also reported by DeHaan et al. (1983). However, Oltjen et al., (1984) observed reduced feed intake and average daily gain in lambs fed contaminated diets during the first 28 days of the trial. This was followed by an adaptation of the lambs to the diet which eliminated the differences that initially occurred due to the consumption of contaminated diets. Our data taken once every two weeks on both feed intake and weight gain did not show similar trend.

There was no treatment effect on the AFE in this study as the results for the various treatment levels were 3.7 ± 0.15, 3.5 ± 0.15, 3.5 ± 0.15 and 3.6 ± 0.15 for treatments 0, 1, 2 and 3 respectively. There was also no breed difference in the AFE between the two breeds used ( 3.6 ± 0.11 and 3.5 ± 0.11 for the Ou and Su lambs, respectively). Harvey et al., (1986) reported a total feed:gain ratio of 4.73 and 4.98 for the control and treated lambs, respectively. These results show no significant difference between the treated and control groups, and as such no treatment effects. The AFE in males was slightly better than that observed in the females (3.47 ± 0.11 vs 3.67 ± 0.11, respectively) indicating that males require less feed to attain gain than females. The fact that the interaction of treatment, breed and sex was not different for final weight and weight gain of lambs, indicates that there were no overall treatment effects on ADFC, ADG and AFE which suggests that treatments (DON) did not in any way pose a problem to the lambs used in this trial. This result is similar to the previous studies (Harvey et al., 1986; DeHaan et al., 1983; Oltjen et al., 1984).

Weight gain was significantly ($p < 0.05$) affected by sex as the male lambs gained weight more rapidly than the females. No study has compared the sex of these two breeds before, but it is a common phenomenon that, males show faster weight gain than females. In this study, the males reached the final weight (31-34 kg BW) earlier (12 weeks) than the females (14 weeks).

**TRIAL 4:**

This study which was designed to answer further questions based on Trial 1, has shown that treatment had no effect on the reproductive performance of ewes. There was no treatment effect on all the parameters measured which included; gestation length (GL), number of lambs born alive (NBA), number of lambs born dead (NBD) and average birth weight of lambs (ABW), which agrees with the report of Windels, 1994.

However, breed differences were noted in NBA and ABW, showing that, as the Ou ewes had a significantly ($p < 0.0001$) greater number of lambs born, this significantly ($p < 0.0001$) reduced the ABW of the lambs. Similar observations were made in Trial 1 showing the repeatability of these breed differences.

However, Trial 4 provided a greater insight into the effects of the toxin on the reproductive performance of the ewes because, the feeding of the toxin started before breeding when it was anticipated that the toxin may have had an adverse effect on the conception rate and the developing fetuses. The outcome of the trial would indicate no clear cut deleterious effect of feeding DON-contaminated barley (up to 20 ppm) to sheep during any stage of production

## CONCLUSIONS:

Feeding of DON-contaminated diets during the last trimester of pregnancy had no major effect on the ABW and NBA of lambs. Similarly the lambs nursed by ewes that consumed up to 20.2 ppm of DON in their concentrate diets were not affected in any major way.

There was no treatment effect on either the total weight gain of lambs or the overall final weight attainment, showing that the sheep which is a ruminant is less sensitive to the effects of this toxin which has also been reported by other researchers.

The results of feeding DON-contaminated diets during breeding /early pregnancy proved to have no adverse effects on the reproductive performance. The high lambing rate seen in the reproductive studies (especially in Outaouais) could have been due to the high energy intake provided by the barley to provide a "flushing" effect which is known to maximize ovulation rate and thus lambing rate. There were breed and sex differences which were similar to those obtained previously.

Our studies suggest that DON-contaminated grains can be fed to ruminants since they have been found to be less sensitive to this toxin than any other species. Care should be taken when reporting the concentration of vomitoxin in ruminant diets. Since they animals consume a large amount of hay, this will dilute the level of contamination in the overall diet. It is therefore advisable that the level of contamination be calculated based on the total feed consumption which includes both the concentrates and hay.

# RECOMMENDATION

Based on the results of this study, farmers can be advised to feed contaminated grains containing up to 20 ppm of DON to ruminants (in particular to sheep) irrespective of their physiological status.

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