NUTRITIVE QUALITY OF LOW-GLUCOSINOLATE CANOLA MEAL FOR LAYING HENS

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INTRODUCTION

In Canada extensive plant selection programs to alter the glucosinolate content of rapeseed in an attempt to improve nutritional quality of the meal began in the mid 1950's (Bell, 1982). Low-glucosinolate rapeseed varieties now called canola were first made available for commercial use in the 1970's. Recently a new cultivar of canola with a very low level of glucosinolates has been made available on an experimental basis. The purpose of the current research was to evaluate the feeding value of meal produced from this new low-glucosinolate canola cultivar and to attempt to determine the "no-effect" level of glucosinolates in the laying hen.

MATERIALS AND METHODS

Low-glucosinolate canola (BC86-18, a *B. campestris* cultivar from Agriculture Canada, Saskatoon) and normal canola (Tobin, a standard *B. campestris* variety) were used in experiments conducted during two different years. In year 1 the canola was grown in Manitoba and was processed at a commercial processing plant (CSP, Altona) while in year 2 the canola was grown in Saskatchewan and processed at an experimental processing plant (POS, Saskatoon).

Two experiments were conducted with the canola meals to determine the feeding quality of the low-glucosinolate canola cultivar and to assess the effect of diets of varied glucosinolate content on antinutritive responses in laying hens. In Experiment 1, 1,728 Single Comb White Leghorn (SCWL) hens of two commercial strains (Shaver and Dekalb) were randomly allotted at 22 weeks-of-age to three dietary treatments. Diets were based on wheat with either canola meal as the sole dietary protein supplement (24% of the diet) or with soybean meal as the major protein supplement in a control diet. All diets contained similar levels of metabolizable energy, lysine and total sulfur amino acids. Each diet was fed to 20 replicate groups (12-16 hens/replicate) of each strain of hen for 336 days (12 28-day periods). Daily egg production, feed consumption determined on a replicate basis every period and final body weight data were collected. Mortality data was recorded throughout the experiment and cause of death was determined by a Poultry Pathologist. Egg weight was monitored throughout the experiment by collecting data from eight replicates for each strain. All eggs laid during three consecutive days at two week intervals during the first six periods of the experiment and at four week intervals during the last six periods were individually weighed. Percent distribution among standard Grade A egg-weight categories was calculated.

In Experiment 2, 1,216 SCWL hens (512 Shaver and 704 Dekalb) were randomly allotted at 23 weeks-of-age to eight dietary treatments. The diets (Table 1), similar to those used in Experiment 1, included a soybean control, BC86-18 and Tobin canola meals from year 1 and year 2, and a combination of BC86-18 and Tobin canola meals (year 1) to produce diets varying in glucosinolate content. Each diet was fed to 10 replicate groups (12-16 hens/replicate) for 196 days (7, 28-day periods). Egg production, feed consumption, body weight and mortality data were recorded as for Experiment 1. At the termination of the experiment 24 hens (12 from each strain) were chosen at random from each treatment and killed by cervical dislocation. Thyroids and livers were excised and weighed. Gross pathology was determined for all livers and liver samples were stored (-70°C) for glutathione analyses.

Diet nutrient analyses were conducted according to standard AOAC procedures and glucosinolates were determined by gas-liquid chromatography according to the method of Slominski and Campbell (1987). Liver glutathione was determined by the method described by Tietse (1969). Statistical analyses were according to Snedecor and Cochran (1980).

Table 1. Percent composition of diets used in Experiment II

Ingredients	Control Diet 1	BC86-18 (Year 1) Diet 2	BC86-18 (Year 2) Diet 3	Tobin (Year 1) Diet 7	Tobin (Year 2) Diet 8
Wheat	61.0	54.4	60.0	57.2	60.0
Barley	10.1	0.0	0.0	0.0	0.0
Canola meal[1]	0.0	25.0	25.0	25.0	25.0
Soybean meal	15.4	5.2	0.0	2.6	0.0
Meat meal	2.0	0.0	0.0	0.0	0.0
Tallow	0.7	4.4	4.0	4.2	4.0
Other[2]	10.8	11.0	11.0	11.0	11.0
Analyses (determined)					
Protein, %	19.2	18.4	18.0	18.9	18.4
Calcium, %	3.2	3.4	3.6	3.3	3.2
Phosphorus, %	0.6	0.7	0.7	0.7	0.7

[1]Canola meal additions were as specified in the diet description. Diets 4, 5 and 6 were produced by mixing diets 2 and 7 in the proportions; 75:25, 50:50 and 25:75, respectively.

[2]Included mineral and vitamin supplementation to meet NRC nutrient specifications. Lysine HCl and/or dl methionine were added to some diets to ensure that all diets contained at least 0.80% lysine and 0.40% methionine (calculated analyses).

RESULTS AND DISCUSSION

The total glucosinolate content of the canola meals used in this study was 6.05, 0.96, 17.70 and 15.50 μmoles g^{-1} for BC86-18 and Tobin meals in year 1 and year 2, respectively. It was apparent that the canola in year 1 was contaminated with weed seeds as the meals contained a relatively high content of 4-OH-benzyl glucosinolate. No contamination was evident for meals produced in year 2. With the contamination excluded the total glucosinolate content for meals produced in year 1 was: BC86-18, 2.98; Tobin, 13.30 μmoles g^{-1}.

The productive performance of laying hens fed canola meal and control diets for an entire production cycle in Experiment 1 is shown in Table 2. A high rate of egg production was maintained by all treatment groups although hens fed Tobin canola meal laid fewer eggs than those fed the control diet and the hens fed the low-glucosinolate canola (BC86-18) diet had an intermediate level of egg production. Feed consumption data showed the opposite response with a reduced intake for BC86-18 relative to control and an intermediate feed intake for Tobin-fed hens. No differences among treatment groups were apparent for final body weight. A marked treatment effect was evident for liver hemorrhage mortality rate with hens fed Tobin canola meal showing a high incidence of death due to liver hemorrhage (21/576) relative to hens fed low-glucosinolate canola meal (3/576) or control (0/576) diets which had similar mortality rates. A similar highly significant treatment effect was evident for egg weight distribution. Hens fed Tobin canola meal produced more small and medium eggs and fewer large and extra large eggs than those fed the control diet. The egg weight distribution for the hens fed the low-glucosinolate canola meal diet was similar to that of control hens for medium and large eggs although an effect similar to that for the Tobin treatment was evident for small and extra large eggs. In general, these data indicate a high rate of production performance among hens fed low-glucosinolate canola meal and a reduced performance among hens fed Tobin canola meal as the sole dietary protein supplement (24% of the diet).

The effects of dietary glucosinolate content on laying hen performance are shown in Table 3. All treatment groups had a high rate of

egg production with no consistent relationship between dietary glucosinolate content and egg production rate. A significant reduction in egg production rate relative to control, however, was evident for hens fed the diet containing the highest (3.84 μmoles g^{-1}) glucosinolate content. Although no consistent effects were evident for feed consumption and final body weight, hens fed canola meal diets tended to have lower feed intakes and final body weights than those fed the control diet. The responses however, did not appear to be related to diet glucosinolate content.

The influence of diet glucosinolate content on the antinutritive effects of canola meal in laying hen target organs is also shown in Table 3. As noted in Experiment 1, liver hemorrhage mortality was evident among hens fed canola meal diets in Experiment 2 and although not significant the effect tended to be related to diet glucosinolate content. Liver score, in contrast, which is a measure of a sublethal effect with regard to hemorrhages, showed no apparent differences among treatment groups. Although hens fed canola meal diets tended to have heavier livers than control hens the effect was not significant and was not related to diet glucosinolate content. Liver glutathione levels and thyroid size showed marked treatment responses and the effects were directly related to diet glucosinolate content with both parameters increasing with increasing diet glucosinolate content. The response in thyroid size has been well documented (Fenwick et al., 1989) as a goitrogenic effect of glucosinolates while the reason for the glutathione response is less clear and may be related to an induction of liver detoxification mechanisms in response to glucosinolate intake.

Liver hemorrhage has not been reported as a cause of mortality among hens fed canola meal in several different studies (Leeson et al., 1987; Clandinin, 1986; Thomke et al., 1983; Hulan and Proudfoot, 1980) while it has been listed as a cause of death in other reports (Campbell, 1987; Summers et al., 1985; Ibrahim and Hill, 1980). Liver hemorrhage mortality rates ranging from 1.9-3.3% were noted in recent studies in our laboratory (Campbell, unpublished results) in which commercial canola meal was fed as the sole dietary protein supplement with diet total glucosinolate content varying from 3-4.2 μmoles g^{-1}. These data are corroborated by the results of the current experiments where the Tobin diet (Experiment 1) with a glucosinolate content of 3.19 μmoles g^{-1} resulted in an elevated liver hemorrhage mortality rate and in Experiment 2 liver hemorrhage mortality rate tended to be related to diet glucosinolate content. It is difficult, however, to ascertain at which level of dietary glucosinolates the incidence of liver hemorrhage is discernible from that of controls. This might explain why liver hemorrhage incidence has been reported in some studies and not in others. In this regard a "no-effect" level of glucosinolates of from 0.71-1.43 μmoles g^{-1} is indicated if liver glutathione and thyroid weight data are considered (Table 3). Whether or not this "no-effect" level can be applied to the liver hemorrhage response requires further study. While liver hemorrhage mortality was evident at these dietary glucosinolate levels (Table 3) the responses were not significantly different from the control response. It is of interest to note that a glucosinolate level of 1.43 μmoles g^{-1} approximates the level that would be present in a diet containing 10% commercial canola meal which is the upper limit for canola meal inclusion in laying hen diets recommended by the Canola Council of Canada (Clandinin, 1986).

Thomke et al. (1983) reported a negative relationship between feed intake of laying hens and diet glucosinolate concentration but this relationship was not evident in the current experiments. In this regard it is possible that any effect of dietary glucosinolates on feed intake was masked by an effect of BC86-18 meal that was unrelated to glucosinolates. In Experiment 1, a significant reduction in feed intake was apparent for hens fed the BC86-18 diet in relation to those fed the control diet (Table 2) and this trend was also apparent in Experiment 2 (diet 3 vs. diet 1, Table 3). The lack of a feed intake depression among hens fed diet 2 (BC86-18, year 2) in Experiment 2 might indicate that the contamination of BC86-18 with 4-OH-benzyl glucosinolate in year 1 could have caused the feed intake response. However, Tobin meal produced in year 1 also had a high content of 4-OH-benzyl glucosinolate and consistent effects on feed intake for treatments containing Tobin meal (year 1) in Experiments 1 and 2 were not evident. Further work is needed to establish if diet glucosinolate content has a direct effect on feed intake in laying hens. The results

from the current experiments indicate that if there is an effect it is not likely to be a marked effect.

CONCLUSIONS

Low-glucosinolate canola meal (BC86-16) supported a high rate of egg production when fed to laying hens as the sole dietary supplement (24-25% of the diet). Diet glucosinolate content influenced egg size with Tobin canola meal resulting in an increase in the number of small and medium eggs and a decrease in the number of large and extra large eggs relative to controls. Tobin canola meal fed as the sole dietary protein supplement resulted in an increased incidence of liver hemorrhage in laying hens. The liver hemorrhage effect appeared to be related to diet glucosinolate content although the diet glucosinolate level at which the rate of mortality was discernible from that for controls was not clear. A "no-effect" dietary level for glucosinolates of 0.7-1.4 μmoles g^{-1} was evident for the target organ responses, liver glutathione level and thyroid weight.

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Table 2. Productive performance of SCWL laying hens[1] fed canola meals varying in glucosinolate content[2] as the sole dietary protein supplement (24% of the diet) for a complete laying cycle (336 days)

Treatment	Egg production % Hen Day	Feed consumption g/hen/day	Final body weight kg	Liver hemorrhage mortality, %	Egg weight distribution (% of total eggs)			
					Small	Medium	Large	X-large
Control	84.6a[3]	108.8a	1.73	0.00a	2.5b	26.0b	50.9a	20.7a
BC86-18 (year 1)	83.0ab	106.4b	1.73	0.52a	3.2a	27.3b	52.6a	16.8b
Tobin (year 1)	81.9b	107.3ab	1.73	3.65b	4.2a	30.0a	47.4b	18.2b
P-value	.001	.005	.973	.0012	.0001	.0001	.0001	.0003

[1]Number of hens per treatment group was: Shaver, 288; Dekalb, 288.

[2]Glucosinolate content (total glucosinolates less 4-OH-benzyl glucosinolate) of canola meals was: BC86-18, 2.98; Tobin, 13.30 μmoles g^{-1}.

[3]Means within a column followed by different letters are significantly different.

Table 3. Influence of diet glucosinolate content on egg production and antinutritive parameters in SCWL hens[1] during a 196-day feeding period

Diet[2]	Glucosinolates[3] μM/g diet	Egg production % Hen Day	Feed consumption g/hen/day	Body weight kg	Liver hemorrhage mortality, %	Liver weight[4] g/kg BW	Liver score[4] (1-6)	Liver glutathione[4] mg/g liver	Thyroid weight[4] mg/100 g BW
1	0.00	87.7a[5]	108.3	1.70	0.0	23.8	1.0	1.14d	9.5a
2	0.19	87.0ab	108.7	1.68	0.0	24.9	1.0	1.39cd	9.6a
3	0.71	85.9ab	106.1	1.69	0.7	24.6	1.2	1.33cd	9.5a
4	1.43	87.5ab	107.4	1.70	1.3	24.7	1.0	1.48bc	11.7ab
5	2.08	88.7a	107.9	1.67	2.0	23.8	1.0	1.54abc	13.2bc
6	2.84	87.1ab	107.3	1.64	2.6	26.1	1.0	1.72ab	15.8cd
7	3.46	87.2ab	107.0	1.65	2.6	24.7	1.2	1.68ab	15.7cd
8	3.84	84.0b	107.4	1.67	2.6	24.8	1.2	1.81a	19.2d
P-value		.013	.434	.045	.202	.170	.660	.0001	.0001

[1]Number of hens per treatment was: Shaver, 64; Dekalb, 88.

[2]See Table 1 for description of diets.

[3]Total glucosinolates less 4-OH-benzyl glucosinolate.

[4]Data from 24 hens per treatment (12 of each strain) sampled at the termination of the experiment. Liver score was determined as follows 1 = no visible hemorrhage; 2 = few petechial hemorrhages; 3 = extensive petechial hemorrhages; 4 = small hematomas (<2 cm); 5 = large hematomas (>2 cm); 6 = massive hematomas covering a major portion of the liver.

[5]Means within columns followed by different letters are significantly different.