HINDGUT FERMENTATION EFFECTS ON THE ANTINUTRITIVE RESPONSES OF LAYING HENS TO DIETARY GLUCOSINOLATES

L.D. Campbell and B.A. Slominski
Department of Animal Science, University of Manitoba
Winnipeg, Manitoba Canada, R3T 2N2

INTRODUCTION

Glucosinolates have been implicated in the antinutritive effects of rapeseed meal in poultry (Papas et al., 1979a, 1979b; Ibrahim and Hill, 1980; Martland et al., 1984; Campbell, 1987a, 1987b). Recent evidence has indicated that substantial hydrolysis of intact glucosinolates can occur in the GI tract of poultry (Freig et al., 1987; Slominski et al., 1988 and Campbell and Slominski, 1989) but the role that this hydrolysis plays in the antinutritive effects of glucosinolates in the laying hen is not clear. It has been reported that the microbial status of the GI tract of chicks and rats is a significant factor in specific antinutritive responses of these animals to glucosinolates (Nugon-Baudon et al., 1988). Since the ceca of poultry contribute significantly to the microbial load in the GI tract of poultry, studies were conducted with laying hens to determine the antinutritive responses of glucosinolates when fed to hens with and without ceca.

MATERIALS AND METHODS

Two experiments were conducted with Single Comb White Leghorn (SCWL) hens to determine the influence of hindgut fermentation in the laying hen on the antinutritive effects of high-glucosinolate diets (Table 1). Hindgut fermentation was varied by using cecectomized and intact (normal) laying hens and the cecectomy operation was performed on hens (20-22 weeks of age) using the method of Payne (1971).

Table 1. Composition and glucosinolate content of experimental diets

Item	Experiment 1	Experiment 2
Composition (%)		
Wheat	54.5	54.5
Canola meal	24.0	30.0
Rapeseed meal	6.0	-
Sunflower oil	5.0	5.0
Limestone	8.4	8.4
Calcium phosphate	0.6	0.6
Vitamin premix[1]	1.0	1.0
Mineral premix[1]	0.5	0.5
Glucosinolate analyses (μmoles g^{-1})		
3-butenyl	1.49	0.64
4-pentenyl	0.32	0.03
2-OH-3-butenyl	4.72	1.34
2-OH-4-pentenyl	0.18	0.02
4-OH-benzyl	0.81	0.43
3-indolylmethyl	0.09	0.09
4-OH-3-indolylmethyl	0.67	0.50
Total glucosinolate	8.28	3.05

[1]Provided vitamin and mineral nutrients as specified by NRC (1984).

Cecectomized and intact hens (50 each in Experiment 1 and 32 each in Experiment 2) were housed in individual cages and fed the high-glucosinolate diet ad libitum for 140 days (Experiment 1) or 168 days (Experiment 2). Feed consumption, mortality and daily egg production data were recorded and at the termination of the experiments all birds were killed by cervical dislocation to obtain target organ data. Livers and thyroids were removed from the birds and were examined and weighed. During the experiments excreta was collected according to the procedure of Slominski et al. (1988) to determine glucosinolate balance. Glucosinolate analyses of diets and excreta were conducted as described by Slominski and Campbell (1987).

RESULTS AND DISCUSSION

The influence of cecectomy on feed consumption, egg production and antinutritive responses is shown in Table 2. In Experiment 1 feed consumption and egg production were similar for both intact and cecectomized hens while in Experiment 2 egg production was lower for cecectomized as compared to intact hens although feed intake levels were similar for both groups. In Experiment 2 the apparent reduction in egg production in the cecectomized hens may have been as a consequence of incomplete recovery from the cecectomy operation rather than an effect of glucosinolates per se. In this regard the glucosinolate content of the diet fed to the hens was 2.7 fold higher in Experiment 1 than in Experiment 2 and no effect on egg production was evident.

Table 2. Influence of cecectomy on the antinutritive effects of glucosinolates in laying hens

	Experiment 1[1]		Experiment 2[2]	
Item	Intact hens	Cecectomized hens	Intact hens	Cecectomized hens
Number of hens	50	50	32	32
Feed intake g/hen/day	104.3a[3]	105.5a	95.7a	92.8a
Egg production % Hen Day	80.5a	81.8a	84.5a	69.6b
Thyroid weight mg/100 g BW	22.4a	23.3a	12.7a	12.7a
Liver weight g/kg BW	21.1a	21.2a	23.6a	22.9a
Liver score[4]	1.9a	1.9a	1.6a	1.5a
Liver hemorrhage mortality, %	2.0	2.0	0.0	0.0

[1]Hens were fed a diet containing 8.28 μmoles g^{-1} total glucosinolates for 140 days.

[2]Hens were fed a diet containing 3.05 μmoles g^{-1} total glucosinolates for 168 days.

[3]Means within a row for each experiment followed by different letters are different ($P<0.05$).

[4]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.

Enlargement of the thyroid gland is a well recognized antinutritive response to glucosinolates in animals (Bell 1984). Normal thyroid size in SCWL hens has been reported to range from 8-10 mg/100 g BW (Papas et al., 1979a; Martland et al., 1984; Campbell, 1987b) and in this regard the results (Table 2) noted in the current experiments are indicative of an

antinutritive response. The data, however, showed no difference between intact and cecectomized hens. The liver hemorrhage mortality evident in Experiment 1 (Table 2) is also indicative of an antinutritive response to glucosinolates that has been well documented for hens fed rapeseed meal (Hill, 1979) and, as with thyroid size, no differences were indicated between intact and cecectomized hens. In addition, liver weight and liver score data were similar for intact and cecectomized hens in both experiments. The fact that the responses in target organs were similar in intact and cecetomized hens indicates that hindgut fermentation has little or no influence on the antinutritive effects of glucosinolates.

In contrast to the target organ data, glucosinolate recovery showed a marked difference between intact and cecectomized hens. As indicated in Table 3 glucosinolate recovery in cecectomized hens was greater than 2-fold that noted for intact hens. These data indicate that hindgut fermentation contributes significantly to the hydrolysis of glucosinolates in the GI tract of poultry. Similar data have been reported previously (Slominski et al., 1987, 1988). The hydrolysis, however, of the glucosinolates in the hindgut does not appear to contribute to the antinutritive effects of glucosinolates in the laying hen.

Table 3. Influence of cecectomy on the percent recovery of intact glucosinolates in the excreta of laying hens

Sampling time	Experiment 1[1]		Experiment 2[2]	
	Intact	Cecectomized	Intact	Cecectomized
Day 20	40.3 ± 7.9[3]	72.7 ± 17.4	-	-
Day 87	34.3 ± 8.5	75.6 ± 13.4	-	-
Day 128	-	-	27.9 ± 6.6	63.8 ± 4.9
Day 140	33.0 ± 6.2	68.6 ± 11.8	-	-
Day 158	-	-	28.2 ± 7.9	68.3 ± 13.1

[1]Hens were fed a diet containing 8.28 μmoles g^{-1} total glucosinolates for 140 days.

[2]Hens were fed a diet containing 3.05 μmoles g^{-1} total glucosinolates for 168 days.

[3]Mean ± standard deviation of 4 pooled observations per treatment.

The lack of effect of microbial hydrolysis of intact glucosinolates on the severity of the antithyroid response in the laying hen is similar to the response noted by Macholz and co-workers (Diedrich and Kujawa, 1987). They showed with in vitro incubation studies that the intestinal contents from rats resulted in the degradation of progoitrin but without the production of the antithyroid compound, goitrin. It was demonstrated, however, by Nugon-Baudon et al. (1988), using germ-free and conventional rats and chickens,that the intestinal flora was the sole factor responsible for rapeseed meal toxicity. In their studies rat intestinal microflora were shown to be responsible for only a slight hypertrophy of glucosinolate target organs while a strong goitrogenic effect was evident for chicken microflora. In a subsequent study Nugon-Baudon et al.(1990) showed that a lactobacillus strain of bacteria isolated from the crop of chickens was responsible for the dramatic goitrogenicity noted in animals fed rapeseed meal. In their studies the effects of the bacterium were confined to the upper parts of the GI tract. Consequently, it would appear that the antinutritive effects of glucosinolates in poultry are manifest in the upper part of the GI tract and relate to a small portion of the total glucosinolate intake. In this regard changes in the microbial status of birds could have a marked effect on the antinutritive effects of glucosinolates and could result in variable responses among birds and also mask potential differences between high and low glucosinolate diets.

CONCLUSIONS

Reduced hindgut fermentation as indicated by removal of ceca in laying hens results in a marked decrease in the extent of hydrolysis of

intact glucosinolates in the GI tract. This influence on breakdown of intact glucosinolate to aglucone products does not affect the antinutritive responses in target organs. These data indicate that the antinutritive effects of glucosinolates are as a consequence of events in the upper part of the GI tract and probably related to the direct absorption of intact glucosinolates or the absorption of hydrolysis products produced by microflora present in the upper part of the tract.

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