ANTINUTRITIONAL EFFECTS OF RAPESEED GLUCOSINOLATES IN POULTRY

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Glucosinolates (Thioglucosides) in rapeseed are hydrolysed by myrosinase enzyme (thioglucoside glucohydrolase, EC 3.2.3.1) to a variety of aglucone products. Hydrolysis may occur to a variable extent during extraction of the oil from rapeseed and following processing rapeseed meal (RSM) generally contains predominantly intact glucosinolate (IG) and thiocynate (SCN), variable levels of 1-cyano-2-hydroxy-3-butene (CHB) and traces of 5-vinyloxazolidine-2-thione (OZT) and other aglucones such as butenyl- and pentenylisothiocyanates (NCS). Glucosinolates, in general, have been implicated in the antinutritional effects of rapeseed meal in poultry (Jackson, 1969; Olomu et al. 1975; Smith and Campbell, 1976).

A series of experiments were conducted with broiler chickens and laying hens to determine the potential antinutritional effects of specific RSM-glucosinolate hydrolysis products. Diets varying in IG, CHB, OZT, NCS and SCN were formulated by 1) use of RSM's produced from rapeseed varying in glucosinolate content, 2) use of freeze-dried water extracts of rapeseed (RE), 3) the addition of a freeze-dried water extract of yellow mustard to RSM and RE diets as a source of myrosinase enzyme, 4) use of synthetic components, CHB and SCN. The parameters measured in the various experiments to assess the antinutritional effects of the diets included: liver damage and incidence of liver hemorrhage (HL) liver weight, liver microsomal mixed function oxidase activity, prothrombin time and plasma levels of coagulation factors V and X, thyroid weight, plasma thyroid hormone (T4 and T3) levels and egg iodine content. In addition, the influence of dietary menadione in counteracting the liver hemorrhage effect of RSM was studied.

Rapeseed meal and RE were analysed for intact glucosinolates and aglucones using procedures adapted from methods developed by Van Etten and Daxenbichler (1977) and Daxenbichler and Van Etten (1977). Thiocyanate ion content was determined by the method of Josefsson (1968). The extent of liver damage was assessed by gross observation for presence of lesions, weight of liver and histological observation while the activity of the microsomal mixed function oxidase system was determined by assay for cytocrome P 450 content (Omura and Sato, 1964). Blood coagulation-factor analyses and plasma thyroid hormone determinations were conducted using standard methodology employed in human medicine. Iodine was determined on freeze-dried hexane-extracted homogenized egg samples using the Hyoel procedure.

The results of the laying hen experiments indicated that RSM feeding caused a marked reduction (2-4 fold) in egg iodine content, thyroid enlargement (3-5 fold) and a variable incidence of liver hemorrhage. The egg iodine effect was associated with diet thiocyanate content whereas thyroid enlargement was produced primarily by diets in which IG was hydrolysed to OZT and NCS although a high dietary content of IG also resulted in thyroid enlargement. This latter effect may have been due to hydrolysis of IG to aglucones in the intestinal tract of the hens. Analyses of circulating thyroid hormone levels indicated that in the short term experiments, at least, thyroid enlargement was adequate to maintain a constant production of hormones. It is not known, however, if the hens could have maintain normal hormone production throughout on entire production cycle. Some evidence of thyroid damage was apparent through histological observation.

A clear cause and effect relationship between diet glucosinolate content and liver hemorrhage was not evident from the results of the laying hen experiments. The data indicate, however, the presence of a consistent association of high diet content of IG and CHB with HL. A high dietary level of IG alone did not cause a marked increase in the incidence of HL nor did a high content of CHB. It appeared necessary for these two compounds to be present in a diet as components of RSM to produce a marked effect in terms of an increased incidence of HL. This might suggest a synergism among these glucosinolate products in the development of HL or also could implicate other factor(s) in RSM. It would appear from the data obtained that these other factors, if involved, are present in RSM but not in RE.

Liver enlargement was evident among laying hens and broiler chickens showing an increased incidence of liver hemorrhage as a consequence of RSM feeding. In addition to the increase in liver weight a marked induction (1.5-2 fold) of the liver microsomal mixed function oxidase system was evident in broiler chickens. This enzyme system is involved in the biotransformation of xenobiotics or lipophylic foreign compounds such as glucosinolate aglucones from lipid-soluble to water-soluble substances for excretion. It could be hypothesized that this biotransformation might lead to the production of highly reactive intermediates that could be implicated in cell necrosis and possibly hemorrhage. It has been demonstrated that glucosinolate-like thionosulfur containing compounds can cause liver necrosis in rats and the necrosis was demonstrated to be more severe if the rats were first fed a known inducer of the microsomal enzyme system (Hunter and Neal, 1975; Dalvi et al. 1974). These relationchips of the liver microsomal enzyme system in the etiology of HL warrant further study.

TABLE 1

EFFECT OF RAPESEED MEAL AND SUPPLEMENTAL MENADIONE ON PROTHROMBIN TIME AND BLOOD COAGULATION PROTEINS IN BROILER CHICKENS AND ASSOCIATION WITH HEMORRHAGIC LIVER

Treatment		Prothrombin time[1]	Factor V[1]	Factor X[1]	HL incidence
Control	(14)[2]	79[b3]	75[b]	37[c]	0/240
Control + K	(16)	106[a]	145[a]	93[b]	0/240
RSM	(15)	97[a]	167[a]	98[b]	25/480
RSM + K	(23)	108[a]	186[a]	123[a]	7/480

1) Expressed as % of a "normal pool". Low values for prothrombin time represent a prolonged coagulation time
2) Values in brackets refer to the number of birds sampled for blood analyses
3) Means within a column not followed by the same superscript are significantly different ($P<0.05$).

Supplemental menadione was effective in reducing the incidence of RSM-induced HL (Table 1). This effect of vitamin K was apparently not mediated through the blood clotting mechanism as the data indicate normal, or above normal levels of plasma clotting factors (V and X) in RSM-fed birds. Furthermore prothrombin times were not altered markedly by diets. Similar data were obtained in studies with laying hens.

The predominant antinutritional effects associated with RSM feeding to poultry in these studies were liver damage ultimately leading to fatal

hemorrhaging, increased thyroid size and reduced transfer of iodine into eggs. Glucosinolate hydrolysis products were clearly implicated in the development of the latter two antinutritional effects whereas a cause and effect relationship, although evident, was less definitive in the case of liver hemorrhage.

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