

LOW GLUCOSINOLATE RAPESEED MEAL AND EGG TAINT

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Introduction

It is estimated that global production of rapeseed now exceeds 13 million tonnes, equivalent to 4.5 million tonnes of refined oil. In the United Kingdom, production of this valuable crop has increased by a hundred fold since 1970. Approximately 220,000 hectares are presently under cultivation with a yield of 650,000 tonnes anticipated. This crop is entirely of the winter grown, low erucic acid, high glucosinolate (single 'zero') Brassica napus type.

To improve the palatability of the meal derived from rapeseed and also to reduce its thyrotoxicity, cultivars of both B. napus and B. campestris possessing much reduced levels of glucosinolates have been bred in Canada and Europe. Such double 'zero' varieties presently account for over 80% of the crop grown in Canada (where they are designated Canola) and may soon replace conventional varieties in Europe.

At present in the UK rapeseed meal is used almost exclusively in cattle rations, rather than as a replacement for soya in pig or poultry rations. In particular, rapeseed meal has not been included in diets for laying hens because of the problems of liver haemorrhage (1) and egg taint (2). The latter, characterised by a fishy or crabby odour (3), is due to the presence of trimethylamine (TMA) and is associated with a genetic defect which impairs the ability of the hen to metabolise this substance. It has only been reported in commercial brown egg strains although the tainting defect is not necessarily linked to shell colour (4). The recent increase in production of brown, rather than white, eggs in many parts of the world means that the use of rapeseed meal as a source of protein for poultry is being further restricted.

It has been found that (-)5-vinylloxazolidine-2-thione (goitrin) plays a central role in the production of fishy taint by depressing the oxidation (and hence excretion) of TMA (5). Goitrin is the most potent rapeseed goitrogen and may be formed in the gastrointestinal tract by the action of bacterial thioglucosidase (myrosinase) on 2-hydroxy-3-butenyl glucosinolate (6). Whilst 'zero' glucosinolate rapeseed varieties possess lower levels of

this glucosinolate (also called progoitrin) than conventional varieties it has not been entirely removed (7).

Furthermore there has been no general reduction in the levels of the other dietary components, sinapine (8,9) and tannins (10) now known to be involved in the production of egg taint, Fig. 1. Experiments to assess the tainting potential of meals derived from these new varieties were therefore undertaken.

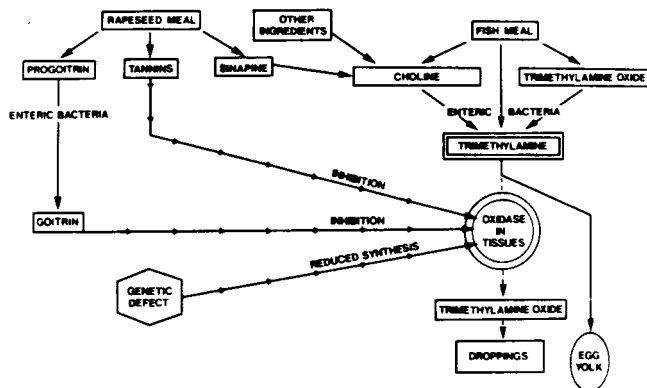


Figure 1
Production of egg taint by rapeseed meal and fishmeal

Materials and Methods

a) Animals: Experiments were conducted with Rhode Island Red x Light Sussex hens which had been bred at Houghton Poultry Research Station for low TMA oxidase activity and consequently for susceptibility to taint when fed rapeseed meal (11). They were housed in individual cages within unidirectional air flow isolators to protect them from infection.

b) Diets: The rapeseed meals investigated were prepared by commercial processes from 4 double 'zero' varieties of *B. napus* (Canadian cv Tower, French cvs Duo and Tandem and an unspecified French cv) and 2 varieties of *B. campestris* (Canadian cvs Yellow Sarson and Candle). Meal from a high glucosinolate variety grown in the UK was included for comparison. They were added to a basal ration containing maize and wheat as the main energy sources to give a concentration of 100g/kg. The control diet contained an equivalent amount of soybean meal.

To determine the contribution of the seed hulls to their tainting potential, dehulled flour and hull-enriched fractions obtained from Tower and French unspecified cv seeds were also fed (85g/kg and

15g/kg respectively). In addition the effects of a dehulled Yellow Sarson flour which had been extracted with hexane and acetone were also studied (100g/kg diet).

c) Design of experiments: Hens were maintained on the control ration prior to the start of the feeding trials. Eggs were collected for 4 days before to the introduction of the experimental diet (day 0) which was subsequently fed ad lib for 14 or 28 days. Eggs were also collected between days 10-14 or 24-28 as appropriate. The eggs from individual birds (typically 3-4 from each collection period) were combined and analysed by gas chromatography for TMA content (12). At days 0, 14 or 28 ^{14}C -TMA oxidation tests were conducted on the hens as described elsewhere (13). Sinapine, tannin and glucosinolate contents of the rapeseed meals and fractions were determined with published methods (14).

Results and Discussion

Table 1 shows clearly that the feeding of double 'zero' rapeseed meal to laying hens may seriously impair TMA oxidation and hence lead to an increase in the TMA content of the eggs. Some of the birds fed these meals laid eggs which were unacceptable when assessed organoleptically and which contained more than 0.8 g TMA/g egg. Lower levels (average 0.56 g/g) were found in eggs laid by hens maintained on the Candle ration. This is consistent with its much smaller effect on the impairment of TMA oxidation in vivo (primarily due to its low progoitrin content, 0.13g/100g meal). In addition it is likely that the low sinapine content of this meal, less than one quarter of that normally found in rapeseed meals (8,9), also contributed to the relatively low incidence of taint. It should be noted that there was little difference in the tannin contents of the samples examined.

Table 1 also shows the effect of dehulling on the tainting potential of the meal. It should be noted that the Tower fractions were obtained from the same batch of seed but the meal was prepared from a different batch.

It can clearly be seen that the dehulling of rapeseed meal does not lessen the taint. The hull fraction of both Tower and the French variety produced a slight depression of TMA oxidation and a corresponding increase in the TMA content of the egg, but this was insufficient to reach tainting levels. The dehulled flour which retained almost all of the sinapine and progoitrin and most of the tannins, however, strongly inhibited TMA oxidation and produced eggs which were strongly tainted. The meal derived from B. campestris cv. Yellow Sarson behaved entirely as would be expected.

However the dehulled product which had also been extracted had little effect on TMA oxidation or on the TMA contents of the eggs. As can be seen from Table 1, the unexpected result of these treatments had been to selectively remove the progoitrin. This loss is also evident in the small effect that including the fraction in layer rations has on the levels of TMA found in the egg. The experiment, therefore confirmed the importance of goitrin in the production of this taint.

It is evident from the results reported here that the introduction of the newer 'zero' glucosinolate rapeseed cultivars will not provide meal, even after processing, which can be fed to poultry without the possibility of fishy taint resulting. The tainting potential of rapeseed meal may be reduced by treatment with calcium hydroxide (15) or ammonia (16) but this is not

TABLE 1 Partial composition of rapeseed meals and fractions and their effect on trimethylamine (TMA) oxidation in hens.

Meal or fraction	Sinapine	Progoitrin (g/100g)	Tannins	Time fed (days)	Depression of TMA oxidation (%)
Duo meal	0.22	0.91	3.02	28	81
Tandem meal	0.26	1.32	3.45	28	94
French meal	0.41	0.32	2.64	28	70
French hulls	0.05	0.08	0.82	28	22
French dehulled flour	0.87	0.35	2.41	28	63
Tower meal	0.87	0.29	2.71	14	85
Tower hulls	0.03	0.15	1.60	14	14
Tower dehulled flour	1.80	0.35	3.91	14	91
Sarson meal	0.50	0.34	3.00	14	81
Sarson dehulled extracted flour	0.55	<0.01	3.38	14	12
Candle meal	0.20	0.13	2.85	14	39
UK single 'zero' meal	0.84	3.12	2.98	14	97

completely effective and we have found some tainting of eggs when such treated meal is fed to susceptible hens. Furthermore the cost of any additional processing of rapeseed meal such as to reduce its tainting potential, in the absence of any additional nutritional benefit, would seem likely to exceed the saving achieved by using it as a source of protein. The complex cause of the tainting makes it unlikely that plant breeding will offer a solution, since reductions in sinapine, progoitrin and tannins would be necessary without introducing undesirable agronomic or processing characteristics.

Recent work has shown that fishy taint in eggs may originate from the inclusion of dietary ingredients other than rapeseed (17) (18). In addition choline or betaine fed as a vitamin supplement for laying hens may cause taint (19) (20). Thus the only effective preventive measure would seem to be the removal of the metabolic defect from commercial flocks by selective breeding. Small scale experiments (21) have indicated that it may be possible to eliminate carriers of the defect within three generations.

It should, however, be emphasised again that the use of rapeseed meal in layer rations in the UK will also depend upon the solution of the liver haemorrhage problem. In the absence of information concerning the involvement of any particular component(s) of rapeseed meal in the initiation and development of this condition, rational methods for its prevention cannot yet be suggested.

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