Lohmann Information

Egg taint - A problem of practical importance

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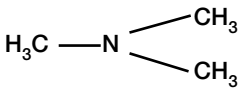
Introduction

Tainting eggs are a severe problem, because even faint malodours are noticed by consumers and lead to complaints. Egg taint is related to the transfer and excretion of smelling substances, resulting from increased intake or from endogenous metabolism, into the egg, mainly into the egg yolk. Trimethylamine (TMA, Figure 1), has been described as being associated with increased egg taint (HOBSON et al., 1973; HOBSON et al., 1975). Concentrations as low as 1 to 1.5 µg TMA g/egg contents are detectable by olfaction (HOBSON et al.,1973; HORIGUCHI et al., 1998).

Figure 1: Structure of trimethylamine (TMA), inducing fishy egg taint



Particularities of the metabolism of tainter hens

Brown layers with low endogenous trimethylamine oxidase activity can produce tainting eggs when exposed to adequate disposing factors (PEARSON et al., 1979). While the problem in hens is of considerable economic interest, it has to be noted that the formation of trimethylamine is found in diverse biosystems and has been demonstrated in different bacteria using TMA for the disposal of electrons and in humans with fishy odour syndrome, inducing badly smelling breath (BREWSTER and SCHEDEWIE, 1983; ZEISEL et al., 1985; BARRETT and KWAN, 1985; ZEISEL et al., 1989; CHAO and ZEISEL, 1990). Tainting hens are identified by breath tests as screening procedure. Eggs with unpleasant odour were found to be exclusively the product of hens with an unpleasant breath (MILLER et al., 1972). In White Leghorn hens, successively switched to 0.46, 0.68 and 0.90 % choline or to 5.0, 10.0 and 15.0 % rapeseed meal diets or to 4.0, 8.0 and 12.0 % fish meal diets no fish-tainted eggs in any dietary group were detected by the sensory test (HORIGUCHI et al., 1998), demonstrating that white hens normally have a high capability of dealing with problematic feedstuffs. The TMA levels were determined in the range of 0.46 to 1.01 µg/g egg yolk, remaining by that below the threshold for olfactory detection (1.1 µg/g). In hens, under identical housing and feeding conditions, we could demonstrate, that in a herd of brown and white layers only the brown hens were affected (ZENTEK and KAMPHUES, 2000; ZENTEK and KAMPHUES, 2002). Tainting was described with a high frequency in one white-shell strain in a study using diets with high levels of rapeseed. It was concluded, that tainting was conditional on the presence in the hen, in the heterozygous or homozygous state, of an autosomal semidominant mutant gene with variable expression, depending on environmental factors (BOLTON et al., 1976).

In crossbred hens selected for the tainting defect due to low trimethylamine oxidase activity it was confirmed, that dietary trimethylamine oxide from capelin meal was converted to TMA, presumably by bacterial reductase activity in the gut. It was concluded, that the fishy taint is caused by overloading hens that have inherently low oxidation capacities (FENWICK et al., 1981a). Additionally, higher levels of TMA could be found in eggs after dietary inhibition of TMA oxidase in hens with endogenously low activities of this enzyme. The authors concluded, that egg taint can even occur in hens given no TMA by dietary fish meal or TMA oxidase inhibitors from rapeseed meal.

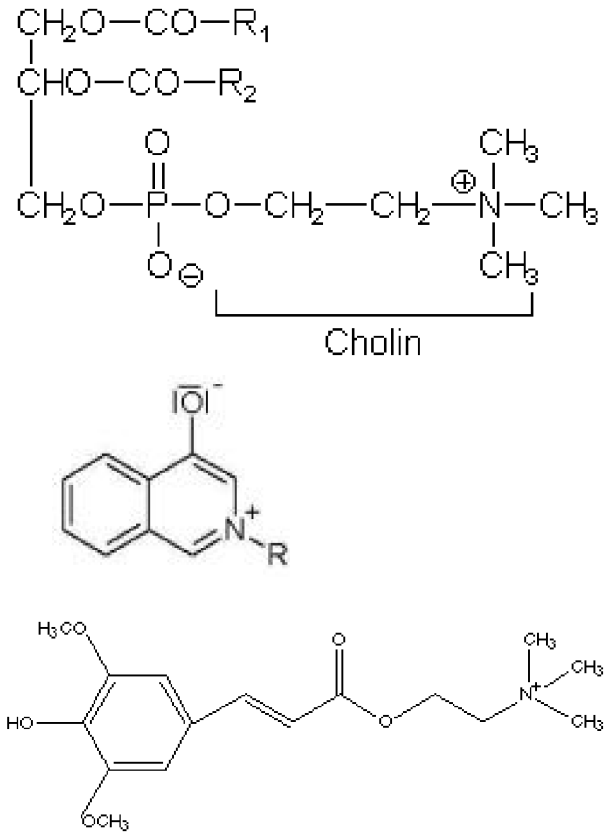
TMA metabolism was studied in intact and caecectomized tainter hens fed on a diet containing 10 % rapeseed meal and given a single intravenous load of [14C]TMA to determine pool size, half times and plasma levels of trimethylamine (EMMANUEL et al., 1984). Eggs from intact layers contained 5.2 µg TMA /g and had a fishy odour. Eggs from caecectomized hens were odour-free and contained TMA levels of only 0.06 µg/g. When both groups were fed on a wheat-soya bean meal diet, all eggs were odour-free and contained only trace amounts of TMA (less than 0.1 µg/g). In those experiments, it could be demonstrated, that both groups differed in terms of mean entry rate, pool size, half-life and plasma concentrations of TMA. Comparing to intact hens, the caecectomized hens had a lower mean TMA entry rate of 7 compared to 13 mg/day/kg bodyweight, the pool size was 921 compared to 2797 µg, the half-life was reduced to 63 compared to 97 min, and plasma levels were only 212 compared to 443 ng/ml. From these observations and from concomitant feeding trials the authors concluded, that the caecum as major location for bacterial fermentations has an important role in TMA production in hens. This does not exclude however, that a certain fraction of the excreted TMA is derived from the bacterial metabolism in the small intestine of tainter hens (MARCH and MacMILLAN, 1979).

Dietary factors influencing egg taint

Fishy taint has been demonstrated to be related to increased levels of TMA mainly in the egg yolk. Diet can affect egg taint either by increased dietary TMA levels, by providing precursors for the TMA formation to the intestinal microflora or by provision of inhibitors of the endogenous TMA oxidase. Fish meal has been investigated in several studies, for it may contain considerable TMA concentrations. Fish meal contains varying levels of TMA, but also TMA oxide. When 500 mg TMA oxide were added per kg of a fish meal free diet, egg TMA concentrations increased to 1.10 µg/g egg and egg taint (FENWICK et al., 1981a). Other precursors of TMA formation are betaine (WILLEKE, 1980) and choline (WILLEKE, 1980; BUTLER et al., 1982; PEARSON et al., 1983a; BUTLER and FENWICK, 1984) (Figure 2), that may induce egg taint after metabolisation and splitting of a TMA moiety by the intestinal microflora. Sinapine, one of the problematic constituents of rape seed is also a potential source for microbial TMA formation (HOBSON et al., 1977; FENWICK et al., 1979; GOH et al., 1979).

Beside those substances that can be considered as direct precursors of trimethylamine, other feedstuffs can have detrimental effects by inhibition of the activity of TMA oxidase. Rapeseed meal (200 g/kg) fed to laying hens depressed egg production when compared with a similar diet containing soyabean meal. Pathological findings included liver haemorrhage, enlarged thyroid glands and

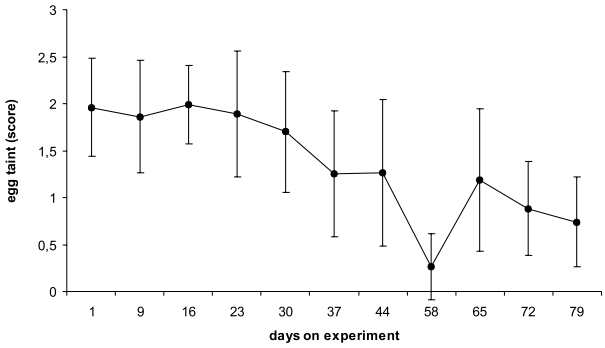
Figure 2: Choline, betaine and sinapine can be precursors for bacterial TMA formation in the intestinal tract and can cause egg taint



egg taint (IBRAHIM and HILL, 1980). When a different cultivar of rapeseed was used, egg production was equivalent to the performance with a soyabean diet but frequence of tainting eggs was not reduced despite of distinct differences in the glucosinolate contents, that were lower in the new rapeseed variety. Glucosinolates can be considered as important antinutritional factors but are obviously not directly involved in the generation of egg taint (GOH et al., 1983). The polyphenolic tannins have been described as inhibitors of TMA oxidase in tainter hens with a reduction of the activity of TMO oxidase in the liver by 52 to 68 % (FENWICK et al., 1981b; BUTLER et al., 1982; FENWICK et al., 1984). The depressive effect of rapeseed can also be related to the presence of (pro)goitrins (PEARSON and BUTLER, 1979; FENWICK and CURTIS, 1980; WILLEKE, 1980; BUTLER et al., 1982; PEARSON et al., 1983b; HENKEL and MOSENTHIN, 1989; MAWSON et al., 1995).

Egg taint may occur even if no known disposing factor is contained and when dietary levels of choline are normal (ZENTEK and KAMPHUES, 2000). When 10 tainter hens were selected from an affected flock and egg taint was scored on a daily base, the development as presented in Figure 3 was observed, indicating, that egg taint can persist over weeks without the requirement for disposing factors in the diet. After microscopical investigation, the estimated diet composition was wheat (50 %), maize (20 %), soya meal (20 %), mineral premix (5 %), and some minor compounds. Choline chloride (added concentration 1000 mg/kg) was reduced to 500 mg/kg after egg taint problems became obvious (total choline concentration from ingredients and additive was analysed to be 1090 mg/kg).

Figure 3: Egg taint (means ± sd) in hens (n=10) selected from a herd with tainting eggs (scores: normal egg quality (0); weak malodour (1), malodour (2); fishy taint (3))



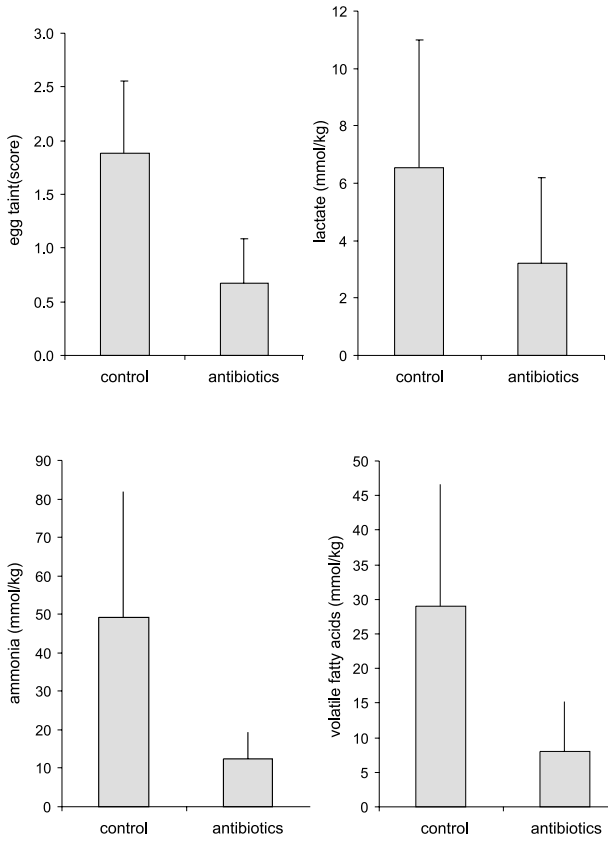
In another group of 10 hens producing tainting eggs and subjected to different dietary treatments it could be demonstrated, that taint was not reduced by feeding a diet free of the known disposing factors. The diet was based on wheat (25.6 %), maize (26 %), barley (20.3 %), soya meal (18 %), methionine (0.15 %), lysine (0.1 %), calcium carbonate (9 %), sodium phosphate (0.8 %), and sodium chloride (0.1 %). Egg taint score was 2.2 (scale 0-3, see legend to Figure 3) with the cereal soya based diet compared to 2.0 with the commercial diet as used on the farm where the problem firstly occurred and with the composition as described above. The addition of substances with potential impact on the metabolic activity of the gut flora (lactulose, herbal additive) did not have reducing effects on the egg taint (ZENTEK and KAMPHUES, 2002). Significant reduction of egg taint was observed only after oral application of broad spectrum antibiotics that were shown not only to reduce egg taint but also the levels of some microbial metabolites in the intestinal tract (volatile fatty acids, lactate, ammonia; Figure 4).

From these experiments it was concluded, that egg taint can occur without the presence of the known dietary factors in tainter hens and that the intestinal microflora plays an important role in the generation of malodour substances. Whether or not TMA was actually present in the eggs was not determined and it has to remain open, if tainting eggs are always related to this substance or if there are other factors, that should be considered in addition. This cannot be excluded for egg taint is in many cases not typically fishy and characteristic for TMA.

Conclusion

In conclusion, the problem of tainting eggs has been known and has been subject of many studies for many years, but is still of practical relevance today. It occurs despite the efforts to reduce the prevalence by specific breeding procedures and the avoidance of known disposing factors in diet formulation. This underlines, that breeding strategies are important and have to be optimised and that further investigations into the origin of egg taint and the potential influences of other substances are needed. This could be especially important in those cases, where egg taint is not the typical 'fishy' type.

Figure 4: Egg taint and levels of volatile fatty acids, lactate and ammonia in the excreta of tainter hens fed without and with addition of antibiotics*



* 3.3 g neomycine sulfate 25 % (Lohmann Cuxhaven, Germany), 0.3 g metronidazole (Sigma, Deisenhofen, Germany) per kg diet and 5 ml enrofloxacin 10 % (Bayer, Leverkusen, Germany per 10 l drinking water

Summary

Fishy or undesired egg taint occurs in certain strains of brown layers. The problem can be caused by dietary trimethylamine or by precursors for TMA formation like trimethylamine oxide, choline, sinapine, or betaine. It was demonstrated, that the metabolic activities of the small intestinal and caecal microflora release trimethylamine. Among the increased intake of TMA precursors, inhibitors of the acitivity of hepatic TMA oxidase have been identified as factors leading to fishy or crabby egg taint. Rapeseed is of specific importance and several factors in rapeseed (glucosinolates, tannins, goitrins) have been investigated in detail. Egg taint has been described in some cases to occur without detection of the presence of the known disposing factors. This could be explained either by a specifically low activity of TMA oxidase or due to the presence of unknown substances or metabolites transported into the egg. Due to the demonstrated persistence of egg taint in problem herds and the lack of scientifically proven dietary treatments it has to be emphasised, that selection against egg taint is most important for the prevention.

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