

Ammonia in Poultry Houses: A Literature Review

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Introduction

With rising costs of both labour and materials in the past few years a number of poultry farmers are re-using old litter (Caveny *et al.*, 1980), sometimes for as many as four or five flocks. There are risks attached to this practice including the increased potential for disease spread (Parkhurst *et al.*, 1974; Lovett *et al.*, 1971; Bacon and Burdick, 1977), and the production of unacceptably high levels of ammonia (Reece *et al.*, 1979).

High concentrations of ammonia can also result from reduced ventilation which with increases in heating costs is becoming more common (Reece *et al.*, 1979). In practice poultry are often exposed to 50 ppm ammonia or more and in fact in poorly ventilated houses the ammonia concentration may be as high as 200 ppm. This poses a problem for poultry farmers during the winter when ventilation is reduced to avoid excessive heat loss, and therefore build-up of ammonia tends to be more severe (Anderson *et al.*, 1964b). Also, condensation, which can be greater in poorly insulated houses in winter, results in wet litter which favours ammonia release (Ivos *et al.*, 1966). See figure 1.

Although ammonia has been shown to have a bactericidal effect in litter (Turnbull and Snoeyenbos, 1973), which may be important in preventing the carry over of disease from one flock to the next, ammonia can adversely affect bird performance and profit. For this reason Deaton and Reece (1980) suggested that a level of 25 ppm ammonia should not be exceeded. This can be done by careful litter management (Anderson *et al.*, 1964b) and ensuring adequate ventilation (Quarles and Kling, 1974).

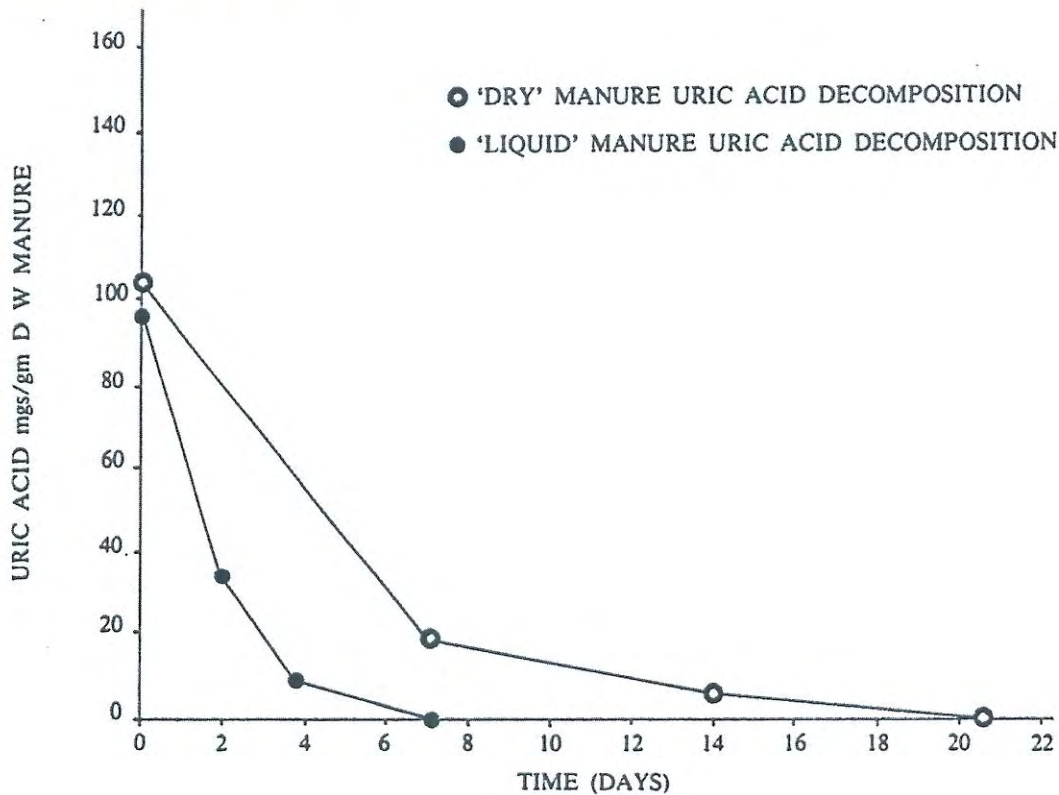
Effects of Ammonia

Ammonia is a colourless, irritant gas produced from the nitrogenous fraction of animal wastes by microbial activity. It is detectable by humans at a concentration of 25 ppm or more, while the maximum concentration that humans can withstand is 100 ppm for eight hours (Moum *et al.*, 1969). The Health and Safety Executive give a Threshold Limit Value (TLV) of 25 ppm ammonia for an eight hour exposure or 35 ppm for exposure up to fifteen minutes. However, poultry can develop a variety of disorders when exposed for long periods to levels as low as 20 ppm (Anderson *et al.*, 1964a).

Although the condition known as keratoconjunctivitis has been attributed to vitamin A deficiency (Valentine, 1964), high levels of ammonia are probably a major cause of this condition, which, although mortality is generally low, can result in considerable financial loss. The typical symptoms of keratoconjunctivitis include huddling of birds in a group, eye rubbing with the wings, the eyes becoming closed

and sensitive to light (Bullis *et al.*, 1950). Affected birds frequently have matted, damp feathers on the back between the wings. Bullis *et al.* (1950) observed that the highest incidence of keratoconjunctivitis occurred where litter was less satisfactory and ammonia concentrations highest. They also observed the condition typically in young birds. This is supported by Carnaghan (1958) who found that the symptoms first appeared in birds of 2-3 weeks of age. Quarles and Caveny (1979) suggested that if further exposure of a flock to ammonia is prevented the birds will eventually recover.

Figure 1: Uric acid Decomposition in 'Dry' and 'Liquid' Poultry Manure (Burnett and Dondero, 1969)



Another important consequence of high levels of ammonia in the atmosphere is the effect on the respiratory tract. Dalhamm (1956) suggested, from observations made on rats, that irritant gases such as ammonia impaired mucus flow and ciliary action in the trachea, resulting in lowered resistance to respiratory infection. This had been observed earlier by Moum *et al.* (1969) in poultry subjected to continuous exposure of 20 ppm ammonia, which exhibited increased susceptibility to Newcastle disease and air sacculitis. Nagaraja (1982) exposed turkeys to an aerosol of *Escherichia coli* and ammonia at a concentration of 10-40 ppm. As well as significant damage to the tracheal mucous membranes, turkeys exposed to ammonia had larger numbers of *E coli* in their lungs than those not exposed to ammonia. Valentine (1964) suggested that ammonia concentrations of 60-70 ppm predisposed

affected birds to respiratory disease and increased the risk of secondary infections. However, Quarles and Kling (1974) noticed that when the ammonia concentration was low, damage to the respiratory tract only became obvious when birds were subjected to infectious micro-organisms. This apparent decrease in resistance to infection due to ammonia appears to be a factor in vaccination stress. Kling and Quarles (1974) noticed that the bursa of Fabricius, part of the avian immune system, of birds exposed to 25 and 50 ppm ammonia from 4-8 weeks of age weighed less after infectious bronchitis vaccination than those not exposed to ammonia. They suggested that ammonia stress may have resulted in a more severe reaction to the vaccine so eliciting a greater response from the bursa. Caveny *et al.* (1981) also noticed this effect of ammonia on vaccine stressed broilers. They pointed out that ammonia at 25-50 ppm and vaccination with Newcastle disease virus at 21 days adversely affected broiler cockerel feed efficiency.

As well as the physical damage to the respiratory tract, respiration rate itself may be reduced in atmospheres containing high concentrations of ammonia. Charles and Payne (1966a) observed that 100 ppm ammonia caused a reduction in respiration rate and depth, which they attributed to a change in blood pH due to ammonia by-products from the lungs. This in turn was thought to affect the pH sensitive centre of respiratory control in the brain so causing a reduction in respiration rate. Although they have observed small but significant changes in blood pH after exposure to 75 ppm ammonia for 15 minutes the significance of these pH differences in relation to reduced respiration is still uncertain. They also suggested that, as body heat loss was lowered by reduced respiration rate, energy requirements were less and appetite reduced. Reduced appetite and the resulting decreased body weight gains on exposure to ammonia have been observed by several workers. For example Kling and Quarles (1974) observed that by 8 weeks of age ammonia stressed birds had significantly lower body weights than unstressed birds. Caveny *et al.* (1981) observed a significant reduction in feed efficiency in broilers exposed to 50 ppm ammonia from 1-49 days of age. Feed efficiency was found to improve with lower levels of ammonia. Charles and Payne (1966b) noted that birds exposed to 102 ppm ammonia had symptoms similar to calcium deficiency at post mortem.

Ammonia also has an effect on the laying capacity of layers and on egg quality. Charles and Payne (1966b) reared pullets in an ammoniated atmosphere from 11-18 weeks of age and observed that these birds came into lay later and tended to lay larger but fewer eggs. They did not record any difference in albumen quality, shell thickness or yolk colour between eggs produced by birds exposed to ammonia and those which were not. They also suggested that the effects on egg production could be prevented if the birds were given a high protein and vitamin diet.

Cotterill and Nordsog (1954) studied the effect of ammonia on egg white and suggested that, even at low concentrations, significant quantities of ammonia could be absorbed into the egg. The pH of the egg white was found to increase and this in turn enhanced the deterioration of the albumen. The yolks of eggs treated with high levels of ammonium hydroxide became translucent and developed a deep orange colour which resulted in lowered candle grade of the egg.

Other effects of ammonia on poultry include a significant increase in breast blisters on broilers grown in ammonia chambers, and delay in reaching sexual maturity (Charles and Payne, 1966). Exposure to ammonia may also increase the severity of coccidiosis (Quarles and Caveny, 1979), while long time exposure can result in changes to the spleen, liver, kidneys and suprarenal glands. (Moum *et al.*, 1969).

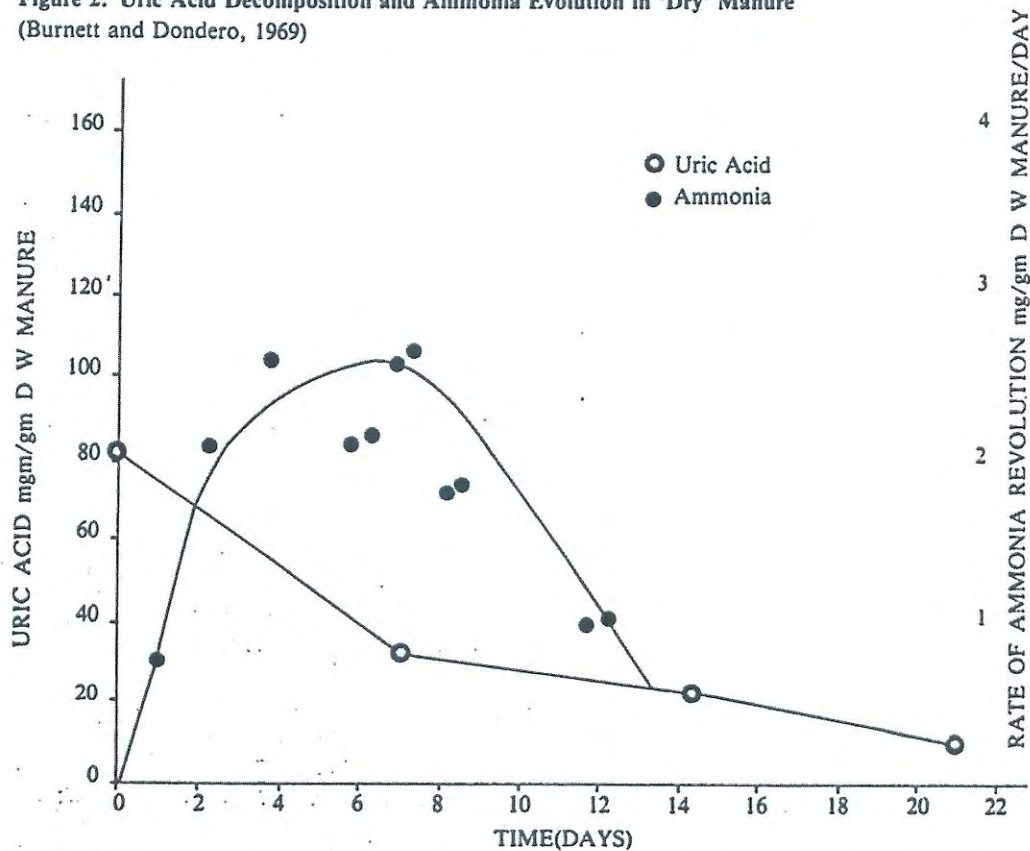
Microbiology of Litter and Ammonia Production

The formation of ammonia in poultry houses has been attributed by several workers (Figure 1) to microbial decomposition of uric acid in the manure (Burnett and Dondero, 1969; Bacharach, 1957; Schefferle, 1965).

Kitai and Arakawa (1979) demonstrated the role of micro-organisms in ammonia release by sterilizing broiler excreta at 121°C for 20 minutes. When this material was incubated at 33°C for 24 hours little ammonia gas was released.

It can be seen in Figure 2 that as the uric acid concentration of manure decreases, the amount of ammonia released increases. This decomposition and the resulting ammonia concentration in the air is thought to be dependent on a number of factors such as litter moisture content, temperature and pH (Ivos *et al.*, 1966), although Schefferle (1965) did not observe any correlation between these factors and the microbial population. Lovett *et al.* (1971) stated that litter age was the only factor affecting microbial densities.

Figure 2: Uric Acid Decomposition and Ammonia Evolution in 'Dry' Manure (Burnett and Dondero, 1969)



Ivos *et al.* (1966) observed that litter retained all the qualities of the original material in the first month of occupation of a house. They recorded high numbers of micro-organisms in litter in the first month with a rapid increase in the second month. Thereafter microbial numbers declined and remained low until the end of rearing.

It would appear that there is a succession of different bacteria and fungi in poultry litter and manure during a crop of birds. Dennis and Gee (1973) observed, in

their study of the microbial floras before and after houses had been used for a single crop of birds, that *Paecilomyces* sp, *Trichoderma* spp, *Aureobasidium pullulans* and *Hyalodendron lignicola* were predominant in fresh litter while *Scopulariopsis brevicaulis* and *Aspergillus* spp were predominant in used litter. It was suggested that dust could act as a potential source of inoculum. They also observed that the total bacterial counts of final litter samples were consistently higher than initial litter samples. Bacon and Burdick (1977) found 18 species of fungi capable of growing in broiler litter during rearing, while Lovett *et al.* (1971) isolated 17 species from litter including *Penicillium* spp, *Scopulariopsis* sp, and *Candida* sp. They found that *Penicillium* spp were dominant in litter until it became alkaline, then *Scopulariopsis* sp, became dominant.

This succession of micro-organisms is also seen in the uricolytic bacterial population. Schefferle (1965) suggested that the uric acid decomposing bacteria may comprise as much as a quarter of the total bacterial population. She observed that unused litter was strongly acid and contained few uricolytic organisms while used litter was alkaline and had high numbers of uric acid decomposers. She also suggested that the aerobic population of uric acid decomposers was of more significance in ammonia production than the anaerobic population. Burnett and Dondero (1969) observed that there were higher numbers of aerobic uricolytic bacteria than anaerobes in poultry manure (See Figs. 3 and 4).

Figure 3: Aerobic Uricolytic Bacteria
(Burnett and Dondero, 1969)

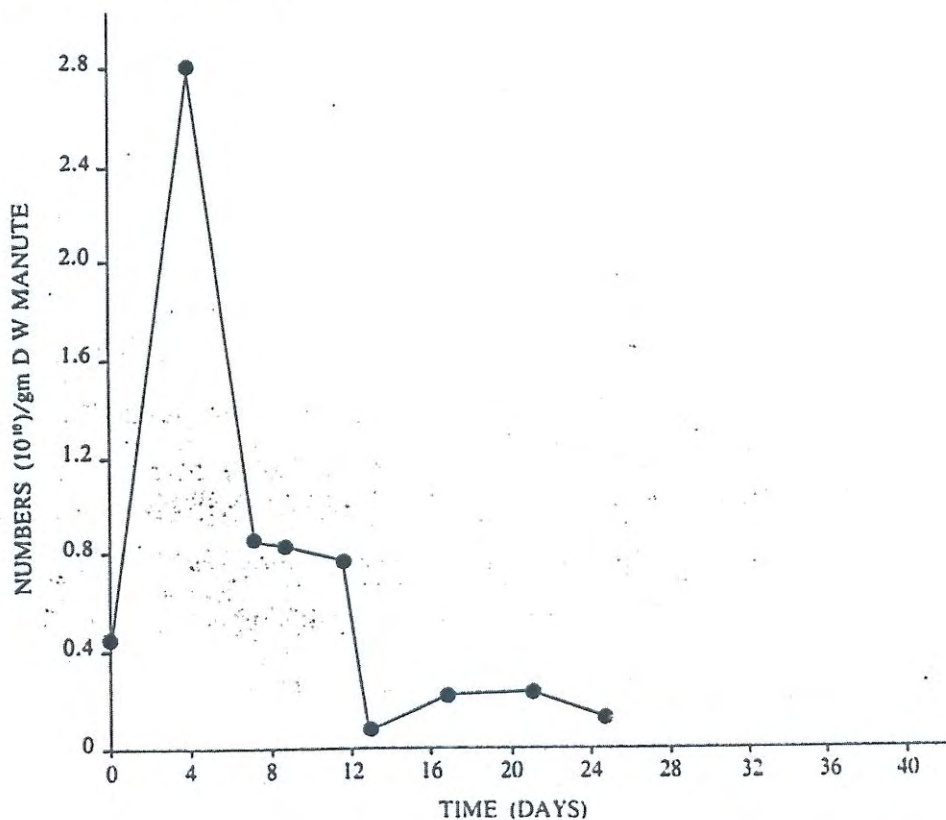
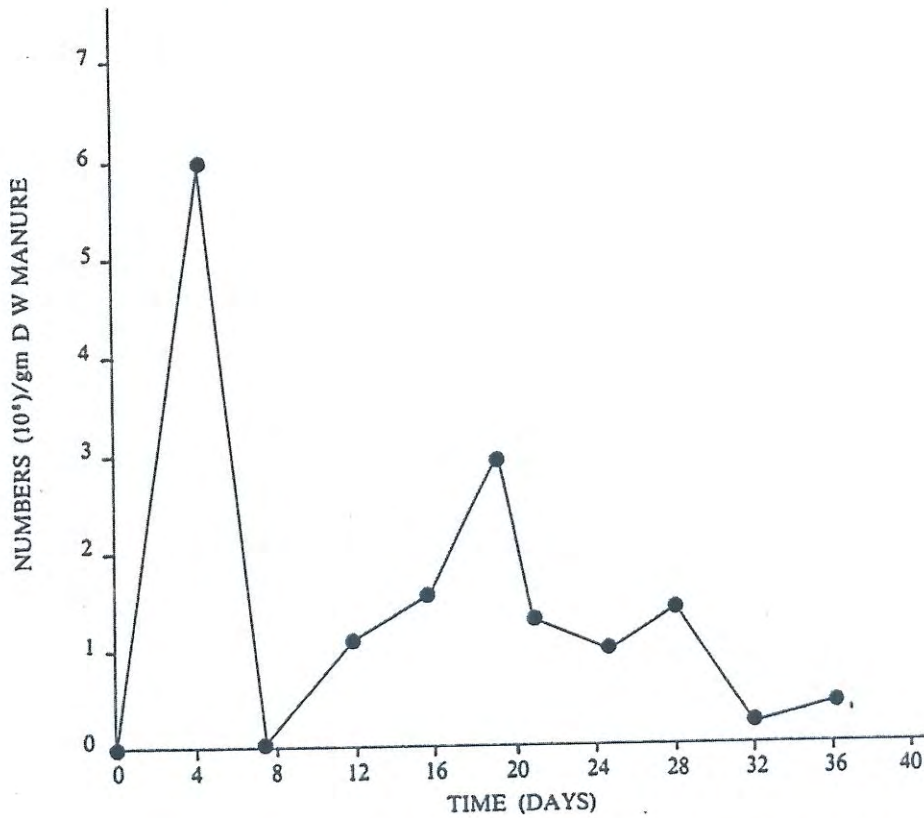


Figure 4: Anaerobic Uricolytic Bacteria
(Burnett and Dondero, 1969)



The decomposition of uric acid and subsequent production of ammonia are the result of a series of reactions in which urea is formed from allantoin which in turn is a product of uric acid breakdown, as shown in Figure 5.

The first enzyme in the pathway, uricase, is a metalloenzyme containing Cu^{2+} or Fe^{3+} and in most cases has a pH optimum about pH 9.0 (Vogels and van der Drift, 1976). The enzyme appears to be highly specific, with oxygen being the only known electron acceptor in the reaction. The precise mechanism of uricase action is not known but the enzyme appears to be present in a number of organisms. However, because O_2 is the sole electron acceptor, uricase is not present in anaerobes, which have developed a different degradative pathway. Bacteria capable of anaerobic uric acid decomposition are widely distributed in soils.

Other systems appear to be able to oxidize uric acid, such as lactoperoxidase— H_2O_2 , verdoperoxidase— H_2O_2 , and uricase—like reactions have been observed to occur via the cytochrome-oxidase system. (Vogels and van der Drift, 1976).

The ability to decompose uric acid may be adaptive rather than constitutive as Rouf and Lomprey (1968) observed when studying aerobic urate decomposition. They recorded a lag in disappearance of uric acid with cultures of *Aerobacter aerogenes*, *Klebsiella pneumoniae*, *Serratia kiliensis*, *Pseudomonas fluorescens*, *P. aeruginosa* and *Bacillus* sp.

Figure 5: Aerobic Degradation of Uric Acid

Bongaerts and Vogels, (1976)
 Bachrach, (1957)

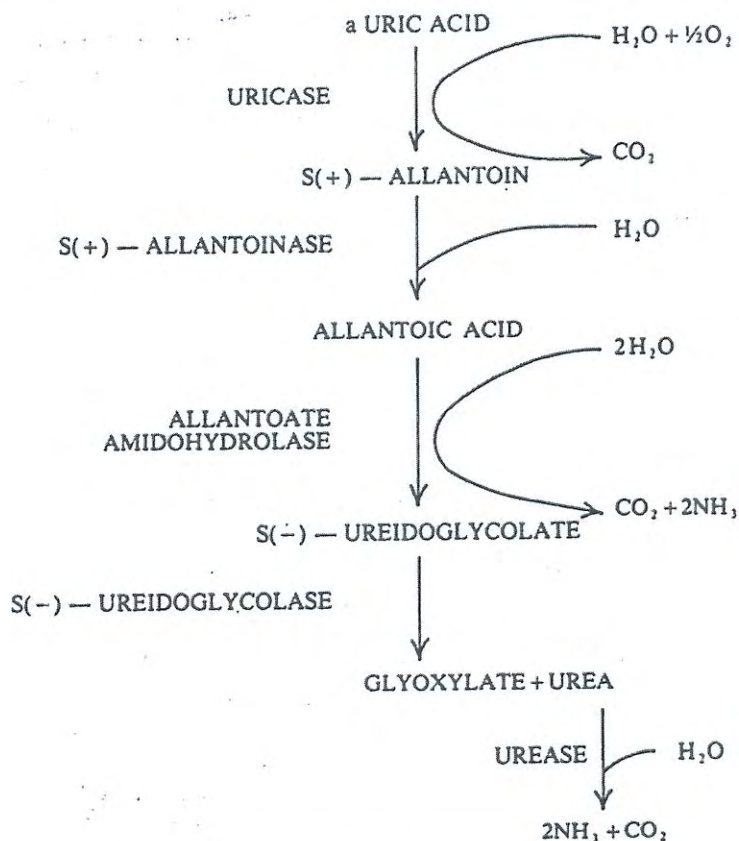


Table 1 lists a few of the organisms reported to be capable of degrading uric acid, some of which are able to use it as the sole source of carbon and nitrogen.

Not all the organisms capable of decomposing uric acid convert it completely to ammonia. Some are only able to degrade uric acid to urea or other intermediates and lack the enzymes necessary for the conversion of these intermediates to ammonia. Therefore, within poultry litter and manure, groups of organisms must exist, their combined effect being the complete degradation of uric acid to ammonia and carbon dioxide.

Methods of Control of Ammonia Production

Several chemicals have been tested for their ability to control or reduce ammonia release from poultry litter and manure. They act by either inhibiting microbial growth, and hence uric acid decomposition, or by combining with the released ammonia thereby neutralizing it. The latter form of control is probably more satisfactory as it will not upset the natural composting of the litter or manure which is important in disease control and general management.

Paraformaldehyde

Paraformaldehyde is commonly used as a fumigant in the disinfection of hatching eggs and incubators so its anti-microbial effect is well documented

TABLE 1: Organisms Capable of Degrading Uric Acid
(Vogels and van der Drift, 1976)

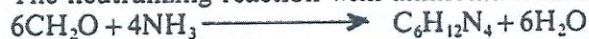
BACTERIA	FUNGI	ACTINOMYCETES
Alcaligenes eutrophus	Penicillium brevicaulis	Nocardia otilis—caviarum
Micrococcus denitrificans	P. chrysogenum	N. polychromogenes
Pseudomonas aeruginosa	P. citres-viride	N. opaca
Arthrobacter globiformis	P. frequentans	
Aerobacter aerogenes	P. glaucum	
Klebsiella pneumoniae	P. notatum	
Enterobacter cloacae	Aspergillus flavus	
Serratia marcescens	A. nidulans	
Proteus vulgaris	A. niger	
Erwinia herbicola	A. glaucus	
Paracolobactrum aerogenoides	A. oryzae	
Proteus mirabilis	Botrytis bassiana	
P. inconstans	Geotrichum candidum	
P. morganii	Gliocladium sp	
Bacillus megaterium	Neurospora crassa	
B. guano	Sporotrichum gougeroti	
B. subtilis var niger	Trichophyta violaceum	
B. fastidiosus	Phytophthora infestans	
Pseudomonas acidovorans	Mucor spinosus	
	Rhizopus nigricans	
	Cunninghamella elegans	
	N. sitophila	

(Lancaster and Crabb, 1953; Lancaster *et al.*, 1954). Veloso *et al.* (1974) also used formaldehyde flakes as an anti-microbial agent in built-up litter, as a method of preventing spread of disease.

It is common practice to use ammonia to neutralize the formaldehyde used for such fumigation and the same principle has been exploited to neutralize ammonia released from poultry droppings.

Paraformaldehyde, a mixture of polyoxymethylene glycols, is available as a powder, granules, or flakes. It decomposes fairly rapidly on exposure to air so releasing formaldehyde gas. Decomposition occurs more quickly at higher temperatures, and is speeded up by moisture in the manure and in the air (Seltzer *et al.* (1969).

The neutralizing reaction with ammonia occurs as follows:



Seltzer *et al.*, (1969), using atmospheric pH as a measure of ammonia concentration, observed that addition of 4.5 kg of paraformaldehyde to 26 square metres of litter reduced the atmospheric pH to 7 (equivalent to 5 ppm ammonia), at which level humans could no longer detect the ammonia. However, 21 days after treatment the ammonia concentration was greater than 100 ppm. Further treatment of the same area 4 weeks later resulted in a similar drop in ammonia concentration and after both treatments a significant rise in egg production was observed.

Veloso *et al.* (1974) did not detect any ill effects in broilers raised on treated litter but suggested that 3% (w/w) paraformaldehyde was close to the upper treatment rate to avoid possible toxic effects from gaseous formaldehyde. They noticed that, even at the highest treatment level (3%), no flakes were visible after 3 weeks and the faint formaldehyde odour was no longer detectable.

It would appear, therefore, that, while paraformaldehyde is effective in controlling ammonia, it decomposes quickly and loses its neutralizing ability within 3 weeks, suggesting that re-treatment may be necessary. Although Seltzer *et al.* (1969) envisage no problems of toxicity if birds are properly debeaked to avoid pecking at flakes and dispersal of the flakes is ensured, recent findings (Swenberg *et*

al., 1980) question the general safety of formaldehyde. As they have suggested that it is carcinogenic, the desirability of using formaldehyde for this task is in question.

Zeolites

Zeolites are crystalline, hydrated aluminosilicates of alkaline earth cations which are abundant and easily obtained. They have the ability to lose or gain water reversibly and can, without major structural changes, exchange constituent cations. In fact they are the most effective ion exchangers known to man. They have been used extensively in Japan for many years for such purposes as purifying fish hatchery waters in aqua-culture, reducing intestinal diseases in young pigs and ruminants, and controlling moisture and ammonia in manure. (Mumpton and Fishman, 1977).

It is because of the preference of some zeolites, eg clinoptilolite, for large cations such as ammonia that they are used to control its levels. Torii (1974) reported that the Japanese mixed clinoptilolite directly with droppings or used boxes containing clinoptilolite hanging from the ceilings of poultry houses to reduce ammonia levels. Apparently this led, in both cases, to an overall increase in egg production and healthier birds. Nakaue *et al.* (1981) carried out a series of experiments using clinoptilolite at different treatment levels. They suggested that surface application on clean wood shavings was more effective at reducing ammonia at 28 days than at 21 days. An application rate of 5 kg/m² on the 21st day reduced ammonia nitrogen by 15% while 5 kg/m² applied on the 28th day reduced ammonia nitrogen by 35%. It may be that application at an early stage in rearing resulted in the clinoptilolite being sifted through the shavings, therefore being less effective in controlling ammonia. It was also noted that litter moisture content was reduced and the incidence of foot pad burns was significantly lower when clinoptilolite was used (See Table 2). Problems were encountered with dust when clinoptilolite was used as the sole source of litter and mortality increased significantly.

TABLE 2: Effect of Direct Application of Clinoptilolite on Broiler Litter—Production Parameters

Clinoptilolite Application	Mean body Weight	Feed Conversion	Mortality	Foot pad burn	Litter Moisture
Production Quantity	(g)		%	%	%
(day) None	1814	2.08	3.2	53	46
28-49 None	1801	2.05	3.9	10	38
21-49 5.0	1778	2.12	3.6	29	(17.4)*
21-49 5.0	1836	2.04	3.2	29	42
21-49 2.5					(8.7)*
					41
					(10.9)*

* Per cent reduction from control group. Nakaue, Koelliker and Pierson (1981).

Ammonia levels were also significantly reduced in these trials when clinoptilolite was incorporated into the feed from 1-49 days of production (See Table 3), although Nakaue and Koelliker (1981) did not observe any such effect. They noticed an unpleasant fishy odour from manure of birds fed clinoptilolite and suggested that there was little value in adding it to rations.

TABLE 3: Effect of Direct Application of Clinoptilolite on Broiler Litter—Ammonia Levels

Production period	Clinoptilolite application Quantity	Mean NH ₃ -N	
		16-49 days	31-49 days
(days)	(kg/m ³)	(ppm)	(ppm)
None	None	9.0	7.5
28-49	5.0		4.9 (35)*
21-49	5.0	7.6 (15)*	
21-49	2.5	8.2 (9)*	

* Per cent reduction from untreated group in same sampling.
(Nakaue, Koelliker and Pierson 1981)

Koelliker *et al.* (1978) constructed a small air scrubbing device using two sizes of clinoptilolite, fine and coarse. Measurements of the ammonia—nitrogen entering and leaving the device were made periodically and showed that as much as one third of the NH₃-N was removed from the intake air.

Superphosphate and Phosphoric Acid

Reece *et al.* (1979) used monobasic calcium phosphate (Superphosphate) and phosphoric acid to suppress ammonia, not only because of the relatively low cost and availability but also because of their value as plant nutrients when poultry manure is used as fertilizer. Hardwood shavings litter, on which one crop of birds had been reared was treated with 2.5 molar phosphoric acid solution by spraying at a rate of 1.7 l/m² before mixing. Superphosphate was applied as a ground, prilled fertilizer at 0.5 kg/m² and 1.0 kg/m² before mixing with the litter. Ammonia concentrations were measured using a Matheson-Kitagawa gas detector. Results are shown in Table 4 from which it can be seen that phosphoric acid was more effective in controlling ammonia and reducing litter pH than either of the two superphosphate treatments. However, by 17 days all treatments were found to be relatively ineffective suggesting the need for retreatment, as in the case of paraformaldehyde.

Yucca Saponin

Yucca saponin is extracted from the stem of the yucca plant by drying and pulverising. The steroid saponins, of which there are several in the plant extract, have been found to enhance the growth of plants, animals and micro-organisms (Johnston *et al.* 1982).

Yucca saponin has been used, especially in the United States, as a growth promoter for beef animals and broilers (Johnston *et al.* 1981), and has been investigated as a method of controlling ammonia in poultry litter. These workers used pine shavings litter and male broilers to record the effect of performance and ammonia suppression of the addition of yucca saponin (63 ppm) to the ration. Ammonia levels were measured using a Kitawara Precision gas detector held at approximately 2 cm above the litter. Although at 28 days of age broilers receiving yucca saponin were significantly heavier than controls, there were no significant differences in ammonia levels.

However, Smith (1980), reporting on work carried out at Minnesota University, stated that the use of 40% yucca saponin mixed with the ration of turkeys reduced ammonia levels. He reported that this reduction could be particularly useful in older, used litter. Average ammonia readings were found to be 118 ppm, 70 ppm and 55 ppm for zero, one ounce and two ounce treatments respectively.

TABLE 4: Effect of superphosphate and phosphoric acid on ammonia release and litter pH during brooding of broiler chickens

Days after start of brooding	Treatment														
	0.5 kg/m ² Superphosphate				1.0 kg/m ² Superphosphate				0.4 kg/m ² Phosphoric Acid				Control		
	NH ₃ (ppm) ^a	pH	Moisture %	NH ₃ (ppm) ^a	pH	Moisture %	NH ₃ (ppm) ^a	pH	Moisture %	NH ₃ (ppm) ^a	pH	Moisture %	NH ₃ (ppm) ^a	pH	Moisture %
0	0	7.1	13	0	6.7	13	0	5.4	13	0	5.4	13	0	7.6	13
3	15	7.1	19	9	6.6	24	0	5.9	21	0	5.9	21	5	7.5	18
7	128			88			26			26			180		
10	140	7.5	30	93	7.6	31	37	7.2	30	118	7.2	30	118	7.9	26
15	125			83			138			138			152		
17	190	8.2	22	253	8.1	27	268	8.1	27	337	8.1	27	337	8.3	24

Reece, Bates and Lott (1979)

^a: Parts per million on volume basis using a Matheson-Kitagawa gas detector sampled from closed container inverted over litter after 2 h.

The lack of information and the few positive results available to date suggest that further work with yucca saponin may be worthwhile.

Acetic and Propionic Acids

In 1974, Parkhurst *et al.* treated pine sawdust litter with 60% acetic and 40% propionic acids at rates of 1% and 3% (w/w) in each case.

Although no measurements were made of ammonia levels, a significant reduction in litter pH was observed for 2 weeks at the 1% treatment level and for 3 weeks at the 3% level. This reduction in litter pH suggests that ammonia release is being reduced, possibly as a result of reduced microbial numbers due to treatment. It would appear however, that retreatment would be necessary, as suggested by a rise in litter pH by week 6.

Analysis of total nitrogen content of the litter showed that treatment did not interfere with the microbial oxidation and digestion of the manure.

Further investigations may reveal that volatile fatty acids such as acetic and propionic acids are effective in controlling ammonia.

Antibiotics

The use of antibiotics as growth promoters in animal production has been a routine practice for many years particularly in the poultry industry. Alvares *et al.* (1964), reported that under certain dietary conditions antibiotics reduced the ureolytic activity and the ammonia concentration of the gut of poultry. For this reason Kitai and Arakawa (1979), tested the ability of antibiotics to control ammonia release from litter by additions to the diet and by mixing with fresh excreta. They measured ammonia release using 50 g excreta in 250 ml flasks plugged with cotton wool, which were incubated at 33°C for 24 hours. Ammonia concentrations were measured using a Kitagawa gas detector. The results, indicate that addition of thiopeptin at a rate of 100 mg/kg fresh excreta decreased ammonia release significantly while 100 mg/kg caprylohydroxamic acid appeared to have no effect on ammonia release.

Addition of 100 mg/kg of thiopeptin or zinc bacitracin to the diet was found to reduce ammonia production but caprylohydroxamic acid did not. 2.5 mg/kg thiopeptin or 20 mg/kg zinc bacitracin also reduced ammonia but to a lesser extent (see Table 5).

TABLE 5: The concentration (ul/1) of ammonia from fresh excreta of chickens receiving dietary supplements after incubating at 33°C for 24 hours.

Experiment	Duration of feeding (weeks)	Basal Diet	Basal + 100 mg/kg Thiopeptin	Basal + 100 mg/kg CHA	Basal + 100 mg/kg Zinc Bacitracin	Basal + 2.5 mg/kg Thiopeptin	Basal + 20 mg/kg Zinc Bacitracin
1	3	400 ± 104	65 ± 20	760 ± 73	—	—	—
	4	330 ± 87	33 ± 14	770 ± 196	—	—	—
	5	580 ± 231	510 ± 149	770 ± 92	—	—	—
2	3	590 ± 106	240 ± 37	—	210 ± 58	—	—
	4	710 ± 116	290 ± 80	—	300 ± 31	—	—
	5	310 ± 37	50 ± 15	—	160 ± 37	—	—
3	3	610 ± 66	—	—	—	580 ± 116	500 ± 83
	4	740 ± 106	—	—	—	590 ± 48	570 ± 102
	5	780 ± 92	—	—	—	380 ± 140	490 ± 139
	6	650 ± 70	—	—	—	410 ± 86	270 ± 50
	7	280 ± 50	—	—	—	240 ± 58	240 ± 58

Kitai and Arakawa (1979)

It would appear, therefore, that antibiotics could be a useful method of controlling ammonia release from poultry litter considering the added advantage of their growth promoting properties and it is possible that the widespread use of antibiotics in broiler feeds may be a factor in reducing the damage which might otherwise be occurring from the build-up of ammonia in broiler litter.

Other Chemicals

A number of other chemicals, such as sorbic acid, gentian violet and calcium propionate (Arafa *et al.*, 1979; Dilworth *et al.*, 1979) have been used to reduce microbial numbers and improve the physical condition of litter. Further work may show them to be effective in controlling ammonia release from litter.

Conclusion

Although the addition of chemicals to litter, either to neutralize the ammonia or to reduce microbial breakdown has in certain cases been effective in controlling ammonia levels, the best method of control is by the provision of adequate ventilation and careful litter management. Because of the cost, chemical control can only be regarded as a method of reducing ammonia when ventilation and litter management have been poor, thus creating a problem.

Summary

AMMONIA IN POULTRY HOUSES: A LITERATURE REVIEW

With changes in management practices the incidence of exposure of poultry to air pollutants is increasing. Ammonia, produced by breakdown of faecal material, can adversely affect bird performance and profit. Ammonia levels are particularly important during winter in cooler climates where ventilation may be reduced to conserve heat.

Typical symptoms of ammonia stress include irritation to the eyes which, in severe cases may develop into keratoconjunctivitis, and the mucous membranes of the respiratory tract. Exposure to ammonia may also predispose affected birds to respiratory disease by lowering the resistance to infection. Feed intake and therefore live weight gain are reduced, as well as the keeping quality of eggs produced in ammoniated atmospheres. Other effects include increased incidence of breast blisters, delay in reaching sexual maturity and changes to the spleen, liver, kidneys and suprarenal glands.

Ammonia is formed by the microbial breakdown of uric acid in the poultry manure, and a variety of moulds and bacteria have been identified which cause these reactions.

A number of chemical methods have been used to control ammonia production including the addition of paraformaldehyde, zeolites, superphosphate, phosphoric acid, yucca saponin, and antibiotics either to the manure or in the feed. However, these only provide a measure of control once the ammonia concentration has increased and should not be used as an alternative to good litter management and provision of adequate ventilation.

Résumé

L'AMONIAQUE DANS LES BATIMENTS POUR VOLAILLES: REVUE DE LA LITTERATURE. (Fiona S. Carlile)

Avec les changements dans les pratiques d'élevage, la fréquence de l'exposition des volailles à des agents polluants de l'air augmente. L'ammoniaque, produit par la dégradation des matières fécales, peut affecter défavorablement les performances des animaux et le profit de l'éleveur. Le taux d'ammoniaque est particulièrement important l'hiver en climat froid, où la ventilation peut être réduite pour conserver la chaleur.

Les symptômes typiques du stress dû à l'ammoniaque comprennent une irritation des yeux qui, dans les cas les plus sévères, peut dégénérer en une kératoconjunctivite, ainsi que des muqueuses du tractus respiratoire. L'exposition à l'ammoniaque peut aussi prédisposer aux maladies respiratoires en abaissant la résistance à l'infection. L'ingestion d'aliment et par suite le gain de poids vif sont réduits, de même que la qualité des oeufs produits en atmosphère chargée d'ammoniaque. D'autres effets sont une augmentation de la fréquence des ampoules du brichet, des reins et des surrénales.

L'ammoniaque est formé par la décomposition microbienne de l'acide urique dans les déjections des volailles, et une variété de moisissures et de bactéries causant ces réactions ont été identifiées.

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