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# Dietary adipic acid reduces ammonia emission from swine excreta

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**ABSTRACT:** Adipic acid is only partially catabolized when it is fed to animals, and a portion of it is excreted in urine. The excreted portion may lower urinary pH and, as a result, ammonia emission. The present study tested this hypothesis. In Exp. 1, nursery pigs (n = 14) were fed (for a period of 7 d) either a standard nursery diet or the same diet supplemented with 1% adipic acid to assess effects on urinary pH (collected on d 5 or 6) and in vitro ammonia emission from the collected urine samples that were mixed with control feces. In Exp. 2, grower pigs housed 10 each in one of two chambers were fed a control diet or the same diet supplemented with 1% adipic acid. Ventilated air was quantified and analyzed for ammonia using Fourier transform infrared spectroscopy to determine the effects of feeding 1% adi-

pic acid on ammonia emission. The results from Exp. 1 showed that adipic acid strongly reduced urinary pH (from 7.7 to 5.5,  $P < 0.05$ ). In vitro ammonia emission from these urine samples was significantly reduced at all the time points evaluated (1, 3, 18, and 46 h with reductions of 94, 93, 70, and 39%, respectively,  $P < 0.05$ ). Experiment 2 showed that adipic acid supplementation reduced ammonia emission by 25% ( $P < 0.05$ ), which corresponded to the predicted reduction in ammonia emission based on the reduction in manure pH observed. In conclusion, feeding adipic acid lowers urinary pH and reduces ammonia emission. The reduction in ammonia emission, though, does not correspond to the reduction in urinary pH but corresponds to the reduction in fecal pH as a result of mixing the urine and feces, in which feces act as a strong buffer.

Key Words: Adipic Acid, Ammonia, pH, Pigs, Urine

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## Introduction

Ammonia ( $\text{NH}_3$ ) in livestock facilities originates predominantly from urea. Urea in urine is relatively stable. However, when the urea comes in contact with urease,  $\text{NH}_3$  is produced, which can be volatilized. Urease is ubiquitous in feces, and this contact between urea and urease thus occurs easily in production facilities (Aarnink et al., 1993).

The rate at which  $\text{NH}_3$  is emitted from a livestock facility is dependent on many factors: the mixing of urine with feces, urea content, temperature, airflow, surface area, and so on. Most of these factors are determined by building or pen design and are difficult to affect in existing buildings. Another factor influencing ammonia emission is pH. Ammonia in solution is in equilibrium with ammonium ( $\text{NH}_4^+$ ) in a pH-dependent manner, and only  $\text{NH}_3$  can be emitted. Thus, lowering the pH of manure will result in a conversion of  $\text{NH}_3$  to  $\text{NH}_4^+$  and, therefore, a lowered  $\text{NH}_3$  emission

(Canh, 1998). This principle has been proven in situations in which the manure in the pit has been acidified (Stevens, 1989). However, because a major portion of the  $\text{NH}_3$  is emitted from the surface of slats, this method of acidification is only partially effective (Rom, 1995). An alternative approach for reducing  $\text{NH}_3$  emission is to reduce the pH of urine, which would reduce  $\text{NH}_3$  emission from the surface of the slats as well.

Adipic acid is a dicarboxylic acid that can be metabolized. However, feeding studies in infants have shown that a portion of the adipic acid is excreted in urine (Mingrone et al., 1989). This fact led us to believe that adipic acid may provide a means to reduce  $\text{NH}_3$  emission through a reduction in urinary pH (van Kempen, 1998). The objective of the present research was to test the hypothesis that adipic acid, when included in the diet at 1%, reduced urinary pH and thus ammonia emission.

## Materials and Methods

### General

Animals used in these experiments were commercial, crossbred pigs obtained from the North Carolina State University swine farm. During each of the experiments, animals were observed twice daily (a.m. and

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p.m.) for visible health problems by the animal care staff. All experiments were approved by the Institutional Animal Care and Use Committee of North Carolina State University.

### Experiment 1

The first experiment extended observations from an experiment described in a companion paper (van Kempen et al., 2001). It was designed as a randomized complete block design and observations were made to evaluate whether urinary pH is reduced by feeding adipic acid. A total of 14 barrows of approximately 10 kg each (blocked by BW) were assigned to either a control nursery diet or this same diet supplemented with 1% adipic acid (Solutia, St. Louis, MO). The 1% level was chosen on the basis of extrapolations from benzoic acid research (Brok et al., 1997). The control diet contained 65.5% corn, 22% soybean meal (48% CP), and 12.5% pig launcher mix (a base-mix manufactured by Furst-McNess, Freeport, IL). The calculated composition of this diet was as follows: ME 3,339 kcal/kg, lysine 1.15%, threonine 0.76%, sulfur amino acids 0.66%, tryptophan 0.24%, Ca 1%, and P 0.72%.

The diets were provided for ad libitum consumption for a period of 7 d, and feed intake and weight gain were measured over this 7-d period. Animals were housed in individual pens measuring 0.6 × 1.5 m. On d 5 or 6, animals were temporarily moved to stainless steel cages that had a stainless steel collection pan under the wire floor that allowed for urine collection. Upon being voided, urine was immediately transferred (as-is) to a test tube and the pH of the collected urine was determined with an Orion pH electrode (Fisher Scientific, Pittsburgh, PA).

In vitro NH<sub>3</sub> release from these urine samples was determined after mixing 1 mL of urine (that was stored frozen) with 1 mL of a 10% mixture of fresh feces (from control animals in a different experiment) in water. This urine and feces mixture was placed in an organ culture plate; the urine and feces mixture was placed in the outer well, and the center well was filled with one mL of a 10% sulfuric acid solution (serving as a trapping agent for the released ammonia). The culture plate was subsequently sealed airtight and left to incubate at room temperature. The sulfuric acid solution was sampled at 1, 3, 18, and 46 h after the start of the incubation, and the sampled solution was analyzed for NH<sub>4</sub><sup>+</sup> as described by Willis et al. (1996). Ammonia emission data were expressed relative to urinary urea values determined with a Sigma kit (Sigma, St. Louis, MO).

Performance results from Exp. 1 were analyzed using analysis of variance (GLM) with block and treatment in the model. Ammonia emission data were analyzed using analysis of variance (GLM) for each time point with block in the model. For illustrative purposes, sigmoidal regression curves were fitted to these data using nonlinear regression. SPSS version 8.0

(SPSS Inc., Chicago, IL) was used for all data analyses.

### Experiment 2

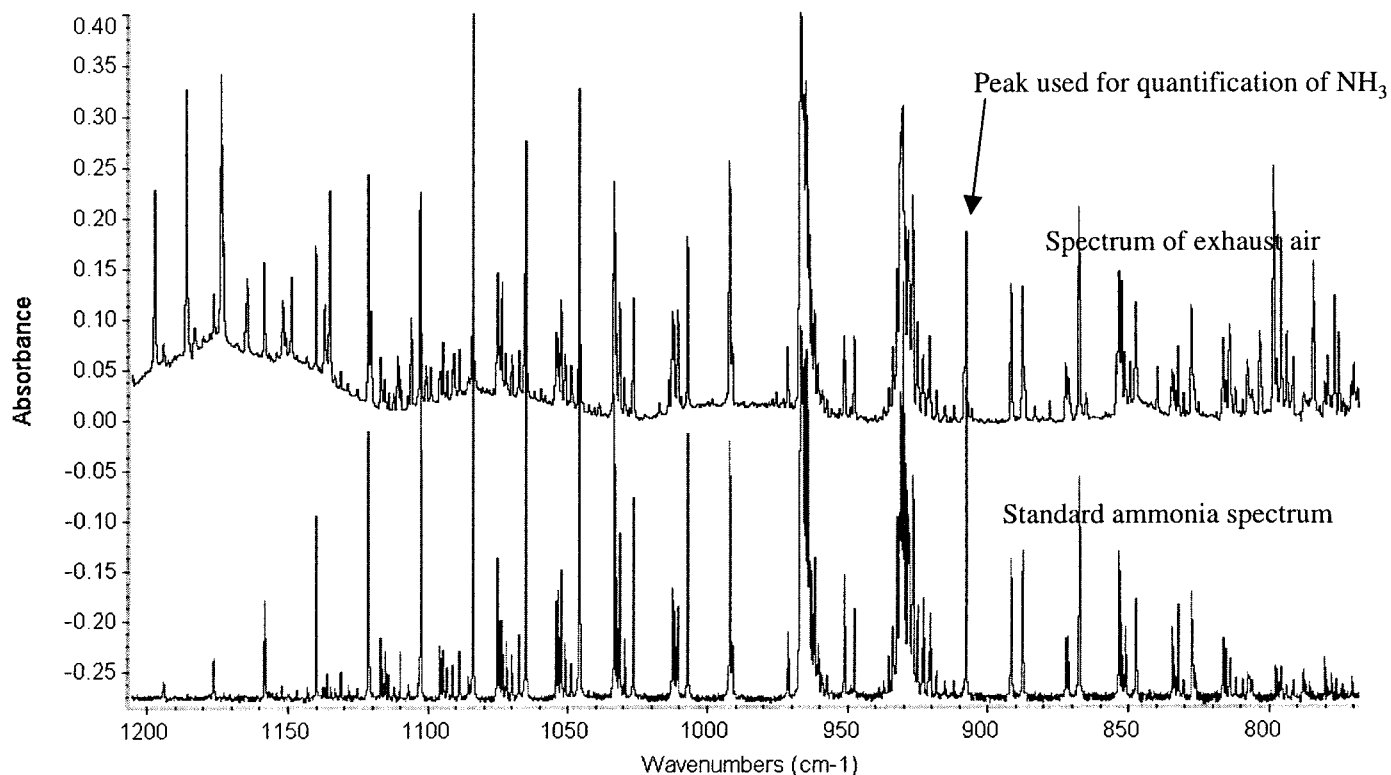
To determine whether adipic acid reduced NH<sub>3</sub> emission, pigs were housed in two chambers designed for determining NH<sub>3</sub> emission (the Swine Malodor Emission Laboratory; van Kempen, 1999). Each chamber housed 10 pigs, weighing 25 kg, in a pen measuring 2.1 × 2.4 m. The flooring in each chamber consisted of standard concrete slats, each 15 cm wide and 2.4 m long (Hog Slats, Newton Grove, NC). The opening between slats was approximately 2 cm. Water was provided through a bite nipple (one per chamber), and feed was provided with a two-hole stainless steel feeder (Hog Slats). Manure from the animals was collected in a pit-recharge pit that was emptied weekly and recharged with 120 L of water. During emptying, manure was sampled, and the pH of the sample (as-is) was determined using an Orion pH electrode (Fisher Scientific, Pittsburgh, PA).

The diet was a grower diet with or without 1% adipic acid. The ingredients in the control diet were corn 61.4%, soybean meal (48% CP) 31.3%, dicalcium phosphate 1.42%, limestone 0.81%, salt 0.5%, vitamin and mineral premix 0.25%, antibiotics 0.5%, and poultry fat 4%. The calculated composition was ME 3,479 cal/g, crude protein 19.9%, crude fat 7.29%, calcium 0.75%, phosphorus 0.65%, lysine 1.1%, sulfur amino acids 0.66%, and threonine 0.75%.

Chambers were maintained at approximately 25°C. Water was provided for ad libitum consumption, and water consumption was monitored (data not shown). The airflow through the chamber was 150 ± 1.5 m<sup>3</sup>/h per chamber and all air was vented from the top of the chamber. Air-flow was measured using Panametrics GM868 ultrasonic flow probes (Panametrics, Waltham, MA) and was kept purposefully low in order to concentrate NH<sub>3</sub>.

To determine NH<sub>3</sub> emission, an aliquot of the ventilated air (100 L/min) was drawn through a Saturn variable path-length gas cell (set to 84 m; Gemini, Anaheim, CA) connected to a Magna 760 Fourier Transform Infrared spectrometer equipped with a liquid nitrogen-cooled MCT-B detector (Mercury-Cadmium-Telluride; Nicolet, Madison, WI). Infrared spectra were obtained by scanning the sampled air from 740 to 4,000 cm<sup>-1</sup> at a resolution of 0.5 cm<sup>-1</sup>; the background used for these spectra was obtained by scanning air entering the chambers. Each spectrum consisted of 104 scans collected after an 80-s equilibration of the gas cell. Ammonia was quantified by measuring the area under the peak between 906.1 to 909.7 cm<sup>-1</sup> because this peak corresponds with NH<sub>3</sub> (based on reference spectra obtained from the EPA; see Figure 1).

The experiment was carried out using a cross-over design; the treatment schedule for Chamber 1 was A-



**Figure 1.** Part of the FTIR spectrum of exhaust air from the chambers (top spectrum) and of a standard ammonia spectrum (displaced downward to facilitate interpretation). The peaks in the ammonia spectrum are easily recognizable in the chamber spectrum, but the chamber spectrum has additional peaks and shifts in baseline indicating the presence of other compounds (e.g., *para*-cresol). The peak at  $908\text{ cm}^{-1}$  was used for the quantification of ammonia because it was a well-defined peak that could be cancelled out completely by subtracting the ammonia spectrum from the chamber spectrum, indicating that no confounding compounds were present at that wavelength.

B-A-C and for Chamber 2 was B-A-C-A, with A being the control treatment, B being the adipic acid treatment, and C being phosphoric acid supplemented feed (results not reported). One group of animals was used for this entire sequence. Treatments were tested over 2-wk periods during which animals were allowed to adapt to the diet for 1 wk. During the following week,  $\text{NH}_3$  emission was monitored for three 24-h periods (spaced 1 d apart). Within each 24-h period, the  $\text{NH}_3$  level was determined 96 times at 15-min intervals, thus providing a semicontinuous measurement. During measuring days, approximately 0.5% of the ventilated air was analyzed for  $\text{NH}_3$ .

For the statistical analysis of Exp. 2, ammonia emission data were averaged within a period, after which data from all four periods were combined to provide adequate degrees of freedom for the analysis. The model included period, chamber, and treatment. Period effects were not significant and were, therefore, removed from the model for the final analysis.

## Results

### General

No problems were observed with animal health that could be linked to the consumption of the adipic acid-

fortified diet. Because adipic acid is considered GRAS (generally recognized as safe), health problems were not expected.

### Experiment 1

Animals fed the adipic acid-fortified feed had a greater feed intake (1.14 vs 0.94 kg/d, SEM = 0.05,  $P < 0.05$ ), and correspondingly, a higher weight gain (0.65 vs 0.54 kg/d, SEM = 0.04,  $P < 0.10$ ). Gain:feed, however, was not affected by treatment (0.57 for both groups, SEM = 0.03).

Urinary pH responded strongly to the addition of adipic acid to the diet; control pigs had a urinary pH of 7.7, and pigs fed adipic acid had a urinary pH of 5.5 (SEM = 0.02,  $P < 0.05$ ). Urea concentrations in urine were increased due to adipic acid addition (72.5 vs 49.3  $\mu\text{M}$ , SEM 5.8,  $P < 0.05$ ). However, the relative ratio of urea:creatinine was not affected (1.08 vs 1,  $P = 0.66$ ), suggesting that differences were caused by differences in urine volume rather than by a change in total urea excretion.

Ammonia release from these urine samples (as a percentage of the urea) is expressed as a function of time in Figure 2. At  $t = 1\text{ h}$ , the urine from animals fed adipic acid had released 94% less  $\text{NH}_3$  than the

urine from control animals (0.034 vs 0.62%); at  $t = 3$  h, the reduction in release was 93% (0.31 vs 4.48%); at  $t = 18$  h it was 70% (12.5 vs 42.4%); and at  $t = 46$  h it was only 39% (67.6 vs 112%, all differences  $P < 0.05$  as determined using ANOVA). At  $t = 46$  h, the urine samples from the control animals had released the equivalent of 100% of the urea as  $\text{NH}_3$ .

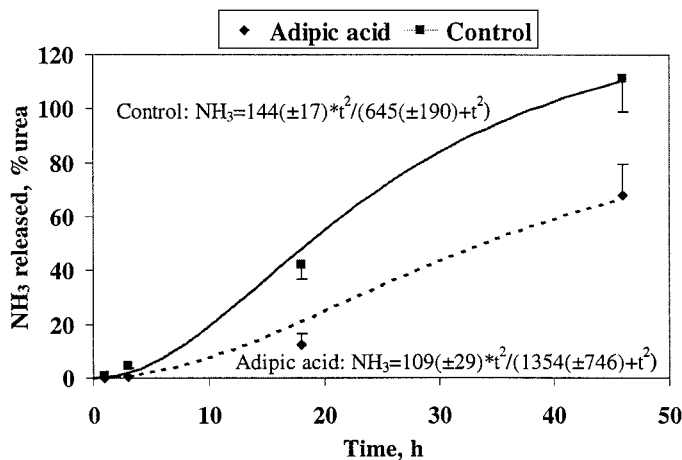
### Experiment 2

Pigs fed adipic acid and control pigs had a similar feed intake (1.45 vs 1.42 kg/d, SEM = 0.01,  $P > 0.1$ ), average daily gain (0.71 vs 0.70, SEM = 0.01,  $P > 0.1$ ), and gain:feed (2.04 vs 2.03, SEM = 0.03,  $P > 0.1$ ). These results do indicate that differences in feed intake and gain were not responsible for differences in  $\text{NH}_3$  emission.

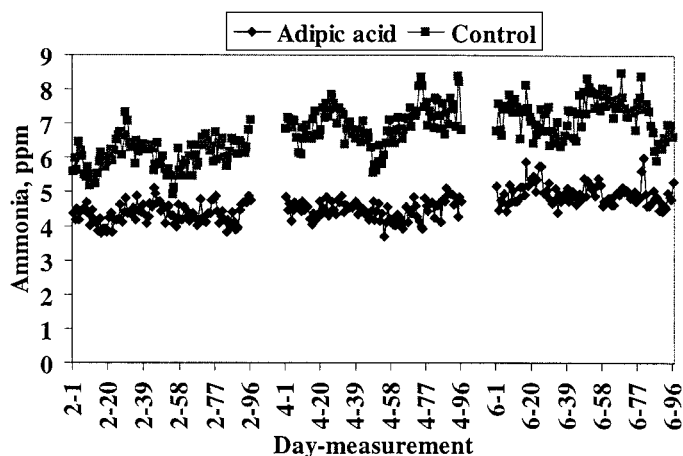
For illustrative purposes, Figure 3 shows ammonia concentrations measured in the exhaust air from the two chambers during wk 4 of the experiment. The results from both wk 2 and 4 show that adipic acid supplementation resulted in a decrease in  $\text{NH}_3$  emission by 25% ( $P < 0.05$ , 21% in wk 2 and 29% in wk 4). The average measured  $\text{NH}_3$  concentration in the exhaust air from the control group was  $7.1 \pm 0.3$  ppm, and the average concentration from the adipic acid group was  $5.3 \pm 0.4$  ppm. The ppm values have to be interpreted in light of the low airflow through the chambers; thus, concentration data should not be extrapolated to field conditions. Ammonia emission expressed as a percentage of the nitrogen consumed was 3.3% for the controls and 2.4% for the pigs fed adipic acid.

### Discussion

For the nursery pigs in Exp. 1, the addition of adipic acid at 1% to animal feeds decreased urinary pH. This



**Figure 2.** In vitro ammonia release, expressed as a percentage of the urea present in the sample at time 0, from urine samples ( $n = 7$  per point) incubated with 10% fecal material. The curves fitted to the data points are sigmoidal regression curves ( $r^2 = 0.81$ ) and only serve an illustrative purpose.



**Figure 3.** Ammonia concentrations measured in the exhaust air from the two chambers during wk 4 of the experiment. Measurements consisted of three 24-h periods (with a 1-d break in between) during which concentrations were measured every 15 min (for a 2-min period). Note that strong circadian rhythms are not observed because air flow and temperature were kept constant.

decrease was rather large, with urinary pH in the treated animals averaging 5.5 compared to 7.7 for the controls. Reducing urinary pH by this magnitude should have a major effect on  $\text{NH}_3$  emission from urine because the equilibrium between  $\text{NH}_3$  and  $\text{NH}_4^+$  shifts toward unvolatilizable  $\text{NH}_4^+$  (Zhang et al., 1994) and because  $\text{NH}_3$  emission is a linear function of the  $\text{NH}_3$  concentration in a solution (provided other factors are kept the same, Hashimoto, 1972).

The ratio of  $[\text{NH}_3]$  to  $[\text{NH}_4^+ \text{ plus } \text{NH}_3]$  in a solution can be described by the function  $10^{\text{pH}} / (10^{\text{pH}} + 5 \cdot 10^{(0.0897 + 2729/\text{T})})$  (Zhang et al., 1994), where T is the temperature in Kelvin. Zhang's equation indicates that pH and temperature are factors determining the ratio of  $[\text{NH}_3]$  to  $[\text{NH}_4^+ \text{ plus } \text{NH}_3]$ . Increasing either pH or temperature results in an exponential increase in  $\text{NH}_3$  concentration; a one-unit increase in pH results in a roughly 10-fold increase in  $\text{NH}_3$  (emission), whereas a  $1^\circ\text{C}$  increase in temperature results in a 7 to 9% increase in  $\text{NH}_3$  (dependent on the starting temperature, with higher starting temperatures resulting in a larger increase in  $\text{NH}_3$  concentration upon increasing temperature by  $1^\circ\text{C}$ ).

By the data obtained in Exp. 1, lowering the urinary pH from 7.7 to 5.5 should have led to a 155-fold reduction in  $\text{NH}_3$  concentration in urine and a similar reduction in  $\text{NH}_3$  emission. However, because feces were added to these urine samples (after which pH was not measured), the actual pH difference at the start of the experiment is not known. The reduction in ammonia emission at 1 h was 93%, and the reduction in ammonia emission actually decreased over time.

A possible reason for decreased reduction in  $\text{NH}_3$  emission over time is that the hydrolysis of urea by urease leads to the production of  $\text{NH}_3$  and pushes the

equilibrium between  $\text{NH}_3$  and  $\text{NH}_4^+$  toward  $\text{NH}_3$ . This effectively raises the pH of the urine because a portion of this ammonia, as shown by Zhang's equation, will combine with  $\text{H}^+$  to form  $\text{NH}_4^+$  (Elzing and Aarnink, 1996). The effect of this urea hydrolysis and the corresponding increase in pH is that lowering urinary pH serves to delay emissions; urease needs to hydrolyze more urea prior to being able to raise the  $\text{NH}_3$  concentration to the level at which it is easily volatilized. Our urine samples, on average, contained 4 g urea/L, or the equivalent of 0.1 M of  $\text{NH}_3$  (which can neutralize 0.1 M of acid), so only urinary pH of extreme low values could have prevented ammonia emission, and such low values are not physiological.

Under field conditions, the mixing of feces and urine further complicates the phenomenon. Feces have a very strong buffering effect on urine (Canh, 1998). The combination of urease raising pH and the buffering of fecal material on urine pH led to only a small difference in manure pH (measured after homogenization at manure collection): 6.93 for the controls and 6.82 for the pigs fed adipic acid. Using these values in Zhang's formula yields a reduction in ammonia concentration of 22%. This value is very close to the reduction in ammonia emission of 25% that was observed, suggesting that the above model is appropriate for the conditions described (note that these results are for one cycle of a pit recharge situation and thus are expected to be relevant for a pit-recharge setup; results for a pit flush or deep pit system are likely different, with pit flush expected to yield less ammonia emission and deep pit more).

Other studies of manure acidification through nutritional means have yielded reductions in ammonia emission that do not comply with Zhang's equation. For example, Brok et al. (1997) tested acidifiers fed to pigs housed in a deep-pit barn and noticed larger reductions in manure pH (0.5 to 0.8 compared to 0.11 units in our study) and a larger reduction in ammonia emission (40%), but this reduction is smaller than the 60 to 80% reduction predicted simply from manure pH. Canh et al. (1998) reported a decrease in *in vitro* ammonia emission of 5.4% for each 0.1-unit drop in manure pH, but according to Zhang's equation a drop of approximately 20% should have resulted. The reason why our data agree with Zhang et al. (1994) and other studies do not is not clear, but this suggests that factors other than manure pH (and temperature) play a major role. For example, Cahn did not study nutritional strategies that affect solely manure pH (in contrast to Brok and the work presented here), and his findings are confounded with changes in nitrogen excretion in urine vs feces.

Rom (1995) determined that  $\text{NH}_3$  emission is largely derived from urine wetting slats or floors. In these wet spots (floors or slats), the mixing of urine and feces is less extensive than that in the manure pit. Therefore, it was expected that urinary pH plays an important role in  $\text{NH}_3$  emission from these wet spots. However,

larger reductions in ammonia emission compared to those predicted by manure pH were not observed in the current study. A possible explanation may be that buffering of the urine pH by fecal material responds in parallel to the exposure of urea to urease. Thus, only wet spots in which adequate mixing of feces and urine has occurred play a role in ammonia emission. In such wet spots, the pH is strongly affected by this mixing, and thus likely to resemble manure pH and not urine pH.

Adipic acid is a flavor enhancer, and the data presented suggest that it may increase feed intake in nursery pigs. Adipic acid also lowers urinary pH and, as a result, ammonia emission. Currently, ammonia emission from livestock facilities is not penalized in the United States; therefore, adipic acid's economic benefits are limited to possibly increasing feed intake and improving animal and human health through lowering ammonia concentrations in the animal house.

In conclusion, adipic acid strongly reduces urinary pH and, as a result,  $\text{NH}_3$  emission. Under simulated field conditions, feeding 1% adipic acid in the diet led to a reduction in  $\text{NH}_3$  emission of 25% relative to controls.

## Implications

Adipic acid supplementation to the diet at 1% lowers urinary pH and, therefore, ammonia emission. In situations in which reducing ammonia emission has perceived monetary benefits, urine acidifiers such as adipic acid may be of interest as an easily implemented method for reducing ammonia emission.

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