THE EFFECTS OF AERIAL AMMONIA AND STREPTOCOCCAL ORGANISMS ON THE FEED INTAKE, IMMUNE FUNCTION AND PHYSIOLOGY OF THE PIG

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Introduction
Growth rates and feed efficiency of pigs raised under commercial conditions in Australia are well below their genetic potential, and the values that could be achieved if the animals were housed under ‘ideal’ conditions (1). This difference has a significant impact on the potential profitability of a pig enterprise. There are many factors within a commercial piggery environment that can contribute to the depression in feed intake, growth rate and efficiency of feed use and act to increase the stress level of the pigs. The stressors can be divided broadly into three categories; social, climatic and hygiene. In most commercial environments several stressors are acting simultaneously. Hygiene and air quality in intensive animal housing is a major concern to producers, employees, housing and farming specialists, and veterinarians involved in the intensive livestock farming industries. In recent years a number of reports have highlighted the negative effects of sub-optimal air quality and hygiene on the health and production of animals, as well as the health of workers (3,7).

Material and Methods
A series of experiments were conducted to quantify the effects of varying concentrations of aerial ammonia and viable Streptococcal organisms on the feed intake, immune function and physiology of 16 week old gilts. The experiments were conducted in the Research Facility of the PPPI Roseworthy Piggery, University of Adelaide. The experimental room (24°C, 55% RH), was cleaned and disinfected between each trial, and hosed three times daily during cleaning and the pit was flushed every second day. Pigs with faecal matter on their skin were washed and dried and returned to their stall. Other stressors, such as stocking rate, background ammonia and bacterial levels were minimised.

Large White x Landrace respiratory disease-free gilts, housed individually on a partially slatted floor, were used in each experiment. Pigs were weighed daily and given access to water at all times. They were fed 2.5 kg of a commercial diet daily, divided into equal portions (morning and afternoon). Twenty two pigs were used in each experiment with 5 pigs per treatment, replicated 4 times. Ammonia gas in nitrogen was pumped into the feed bin at a rate of 12L/min for 20 minutes while pigs were eating, and each pig was observed to ensure maximum exposure. Concentrations of ammonia used were 0, 10, 25 and 50 ppm. After feeding, pigs were dosed intranasally with a solution of Viridans streptococcus suspended in buffered saline at a concentration of 2x10^5 CFU/ml. The organisms used were collected from the airspace of a shed housing growing pigs, using a 6-stage Andersen Sampler loaded with Columbia Horse Blood Agar plates.

Pigs were weighed daily and any uneaten food was collected and weighed.

Pigs were bled prior to pollutant exposure and again 14 days later, and lymphocyte proliferation, phagocytosis (as a measure of neutrophil function), and surface markers CD4, CD8, CD21 measured. Blood analysis included T cell proliferation assays - expressed as CCPM measuring the incorporation of 3HT into actively dividing cells in response to a mitogen and Phagocytosis assay - expressed as % granulocytes (eosinophils and neutrophils) with phagocytic potential. Pigs were slaughtered at the end of each experiment and lungs examined grossly for lesions. Data were analysed by ANOVA (Statistix 7.1)

Results
No lesions were observed in lungs examined macroscopically after slaughter. There was a significant reduction in the growth rate of pigs exposed to ammonia compared with untreated controls (0 ppm) and the reduction increased as levels increased from 10 to 50ppm (Table 1). Growth rate suppression was further increased when pigs were exposed to bacteria as well. Similar reductions were also recorded in feed efficiency (Table 1).

Table 1. Mean growth rate (ADG) and feed efficiency (FCR) pre- and post- exposure with NH₃ or NH₃ and bacteria (2x10⁵ CFU/ml) (NH₃ + B).

<table>
<thead>
<tr>
<th>NH₃</th>
<th>ADG</th>
<th>FCR</th>
<th>NH₃ + B</th>
<th>ADG</th>
<th>FCR</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>796 ± 25.8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.12 ± 0.11&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0</td>
<td>691 ± 24.5&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.61 ± 0.14&lt;sup&gt;h&lt;/sup&gt;</td>
</tr>
<tr>
<td>10</td>
<td>754 ± 27.4&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>3.41 ± 0.13&lt;sup&gt;d&lt;/sup&gt;</td>
<td>10</td>
<td>555 ± 20.8&lt;sup&gt;c&lt;/sup&gt;</td>
<td>4.24 ± 0.17&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>25</td>
<td>713 ± 27.2&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.01 ± 0.14&lt;sup&gt;d&lt;/sup&gt;</td>
<td>25</td>
<td>464 ± 23.6&lt;sup&gt;c&lt;/sup&gt;</td>
<td>5.76 ± 0.16&lt;sup&gt;h&lt;/sup&gt;</td>
</tr>
<tr>
<td>50</td>
<td>590 ± 40.3&lt;sup&gt;c&lt;/sup&gt;</td>
<td>5.85 ± 0.45&lt;sup&gt;c&lt;/sup&gt;</td>
<td>50</td>
<td>264 ± 32.8&lt;sup&gt;c&lt;/sup&gt;</td>
<td>10.97 ± 1.27&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Although lymphocyte proliferation (as measured by the stimulation index) was not consistently increased in pigs exposed to ammonia, a significant increase was recorded in pigs exposed to both ammonia and bacteria (Table 2). In the latter groups, the stimulation index increased as concentrations of ammonia increased.

Table 2. Mean lymphocyte proliferation (SI) pre- and post- exposure with ammonia (NH₃) or ammonia and bacteria (2x10⁵ CFU/ml) (NH₃ + B).

<table>
<thead>
<tr>
<th>NH₃</th>
<th>Before (SI)</th>
<th>After (SI)</th>
<th>NH₃ + B</th>
<th>Before (SI)</th>
<th>After (SI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>38.90 ± 4.66&lt;sup&gt;a&lt;/sup&gt;</td>
<td>46.40 ± 5.19&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0</td>
<td>45.20 ± 2.71&lt;sup&gt;b&lt;/sup&gt;</td>
<td>65.50 ± 4.09&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>10</td>
<td>47.40 ± 5.31&lt;sup&gt;a&lt;/sup&gt;</td>
<td>56.3 ± 6.96&lt;sup&gt;c&lt;/sup&gt;</td>
<td>10</td>
<td>47.00 ± 3.48&lt;sup&gt;c&lt;/sup&gt;</td>
<td>92.1 ± 7.15&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>25</td>
<td>38.80 ± 5.34&lt;sup&gt;a&lt;/sup&gt;</td>
<td>46.60 ± 6.23&lt;sup&gt;c&lt;/sup&gt;</td>
<td>25</td>
<td>50.60 ± 3.03&lt;sup&gt;d&lt;/sup&gt;</td>
<td>152.10 ± 8.23&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>50</td>
<td>35.50 ± 4.97&lt;sup&gt;a&lt;/sup&gt;</td>
<td>47.4 ± 5.89&lt;sup&gt;c&lt;/sup&gt;</td>
<td>50</td>
<td>42.80 ± 2.99&lt;sup&gt;a&lt;/sup&gt;</td>
<td>178 ± 13.37&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

a→h – ADG/FCR different superscripts significantly different (P<0.05)

a→e – Rows with different superscripts significantly different (P<0.05)
While phagocytic activity (expressed as % granulocytes - eosinophils and neutrophils with phagocytic potential) in pigs exposed to ammonia tended to increase, a significant increase was recorded in pigs exposed to both ammonia and bacteria (Table 3). In the latter groups, the neutrophil phagocytic activity increased as concentrations of ammonia increased.

Table 3. Mean phagocytosis activity pre- and post-exposure to NH3 and NH3 and bacteria (NH3 + B).

<table>
<thead>
<tr>
<th>NH3</th>
<th>Before</th>
<th>After</th>
<th>NH3 + B</th>
<th>Before</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>9.30 ± 1.03 a</td>
<td>11.25 ± 1.22 a</td>
<td>0</td>
<td>15.25 ± 1.30 a</td>
<td>28.25 ± 2.28 a</td>
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<tr>
<td>10</td>
<td>10.85 ± 0.81 a</td>
<td>13.10 ± 0.99 a</td>
<td>10</td>
<td>13.30 ± 0.80 a</td>
<td>33.20 ± 2.34 a</td>
</tr>
<tr>
<td>25</td>
<td>10.10 ± 1.07 a</td>
<td>14.65 ± 1.62 a</td>
<td>25</td>
<td>14.55 ± 0.94 a</td>
<td>51.00 ± 3.24 a</td>
</tr>
<tr>
<td>50</td>
<td>11.75 ± 1.04 a</td>
<td>17.63 ± 1.55 a</td>
<td>50</td>
<td>14.65 ± 0.96 a</td>
<td>66.50 ± 4.47 a</td>
</tr>
</tbody>
</table>

Discussion

While neither overt clinical signs, nor macroscopic lesions suggestive of respiratory disease, were observed in pigs throughout the experiments, significant production effects and immune changes were recorded. This finding is consistent with previous reports where immune responses and growth rate suppression was demonstrated in disease free pigs exposed to poor air quality and hygiene (5).

While exposure to bacteria appeared to have a greater effect than ammonia on growth rate and feed efficiency, as well as aspects of immune function, the most significant effects were observed in pigs exposed to high levels of ammonia followed by bacteria. This agrees with previous studies (8) where pleurisy prevalence was higher levels of ammonia followed by bacteria. This agrees with significant effects were observed in pigs exposed to high
effect than ammonia on growth rate and feed efficiency,
quality and hygiene (5).

This finding is consistent with previous reports where production effects and immune changes were recorded.

In pigs throughout the experiments, significant lesions suggestive of respiratory disease, were observed in pigs exposed to both ammonia exposure to NH3 and NH3 and bacteria (NH3 + B).

Conclusions


The effects of stocking density on air quality. Proceedings of the 16th International Pig Veterinary Society Congress, Melbourne, Australia, pp 326.


References


