

## **Strategies for the Control of Respiratory Disease**

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### **Introduction**

This is a paper about common sense and respiratory disease using information that has been known for some time i.e. historical, and which is not accessed when a literary search is supplied by a computer.

Respiratory disease in pigs has its most serious effects in the finishing stages, when the sequelae to progressive atrophic rhinitis, enzootic pneumonia and Actinobacillosis are most severe. Nowadays this relatively simple position is even more complicated by the worldwide occurrence of porcine reproductive and respiratory syndrome (PRRS) and porcine circovirus type 2 (PCV-2)-associated diseases principally PMWS where no vaccination is practised. Up to 80% of lungs may show the lesions of enzootic pneumonia, 20% may show the lesions of acute or chronic pleurisy, and up to 20% of the pigs may have snout lesions due to progressive atrophic rhinitis.

### **Risks of respiratory disease:**

The risk of respiratory disease is always great when we concentrate on animals in huge numbers in a restricted airspace. We have to do two things about it. Firstly, we have to reduce the probability of its occurring. Secondly, when it has occurred we have to reduce the impact that is going to have. Many of the things that we might do, influence both. For example the attention to the climate and management methods described in this paper on

strategies will undoubtedly help in the prevention and control of the disease described in the next paper.

Animals that are healthy (proper nutrition and management ) will not succumb to infection very easily. The importance of these diseases during the finishing stages is associated with intensive systems of housing, which change the relationship between the microorganisms and the pig and its environment. Modern housing systems may also be of little psychological benefit to pigs because they increase stress and restrict the natural behaviour and social interactions of the animals.

### **Strategies for control**

In this paper the strategies for the control of respiratory disease are described. There are basically three approaches to strategies for control of respiratory disease. The first is to provide biosecurity and we shall discuss biosecurity in its widest sense. The probability of occurrence will be reduced by effective biosecurity (careful quarantine, no staff with pigs, transport control, personnel hygiene, pest and rodent and possibly bird control). The second is to provide a healthy pig with the normal homeostatic mechanisms with which to defend itself against the respiratory pathogens i.e. the normal functioning of the pig respiratory tract and how it can be defended. Thirdly, there are the environmental and managerial techniques to control the huge numbers of non-pathogenic organisms in the aerial environment so that

the normal functioning of the pig respiratory tract is not compromised.

### **Biosecurity:**

Biosecurity is a state of mind. It should occupy the waking senses of anybody connected with protection of an

animal population. It should also work at many levels. Outlines are shown in Table 1. This is not a paper on biosecurity (There are many recent publications on the science of this subject) so these main messages are outlined only in detail.

**Table 1** The essence of biosecurity

INTERNATIONAL	WHO and FAO should attempt to eradicate the major epizootics?
LARGE COUNTRIES WITH STATES AND PROVINCES (USA, EU, CHINA)	National policies to keep out the major epizootics. Some areas will need special derogations. May have regional freedoms. Requires inspection of all entry points, regularly and effectively.
NATIONWIDE	Special status may require extra vigilance e.g. meat importing countries, countries with ethnic populations, ethnic catering.
COMPANIES	Specific policies to protect the company, particularly suppliers of gilts and fully integrated companies.
THE INDIVIDUAL FARM	All of the above are fine but the individual unit is the only one that carries the ultimate responsibility for protection.  You have to assume that all the above do not exist and most probably they do not. No nation can protect a population of animals against the random habits of its human population e.g. tourists.
WITHIN THE FARM	New genetic material enters the farm and may carry disease and therefore the existing herd has to be protected; the pig, its products, its ova and semen and embryos may be sources of infection. We also do not want to transmit disease within the unit, usually backwards from the finishing unit to the growers and farrowing quarters. Therefore internal biosecurity may be as important.

The general factors that affect biosecurity are shown in Table 2.

**Table 2** Factors affecting biosecurity

Factor	Explanation
Location	Pig rich area, ideally no others within 2km
Site	Near roads, abattoirs, processing plants, sewage works, crossed by footpaths, outdoor unit
Proximity to other units	Yours is SPF, theirs is low health
New or old, specialist site or conversion	The former has all the necessary trappings of a modern unit but the latter will not have a proper office block, car park, shower unit
Cleansing and disinfection	Cleansing and disinfection and drying on the unit
Fencing	Must be wild animal proof and preferably predator proof
Minimal disease	Minimal disease unit needs bird proof and vermin proof
Pest control	Vermin, rodent and pest control policy
Hygiene	Internal (workforce) and external (visitors and vets) hygiene

The sorts of hazard that present to the average pig farm on a daily basis are shown in Table 3. You can see the complexity of the movements on an individual farm that have to be carefully policed to prevent any failures of biosecurity.

**Table 3** Items entering and leaving a farm on a daily basis

Items arriving	Items leaving
Personnel daily	Personnel daily
Protective clothing	Protective clothing
Laundry vehicles	Laundry vehicles
Feed lorries	Feed lorries
Equipment lorries	Equipment lorries
	Manure removal
Pigs (gilts and boars)	Slaughter pigs
	Casualties to abattoir
	Disposals
Semen, embryos	Visitors
Visitors	
Veterinarians	Veterinarians
<Birds, rodents, vermin, wild pigs >	

People (visitors and workforce) should enter past the office. Imports or delivery items (pigs, semen, boars, feed supplies equipment) should be passed across the fence at a separate point to exports (slaughter pigs, disposals, casualties etc), should exit from a separate site and both should take place across the perimeter fence. The farms

own systems should be within the fence and the haulier or supplier should be outside the fence. It is even more important to attend to the abstract things like the habits of the workforce in the evenings, do they bring meat to work, how do they dispose of it? Every little item of the wrong sort can be a hazard.

**Biosecurity - the isolation unit:**

Luckily for us most pig diseases arrive with pigs or pig products. Bordetellae, Salmonellae, *L. intracellularis* and *B. hyodysenteriae* are the exceptions as they may arrive with rodents, cats, birds, etc as the case may be. This means that extra care is needed with imported ova,

semen or embryos (again beyond the scope of this paper).

The real source of infection is the pig because it has an anus (faecal contamination) and a nose (nose to nose contact). Therefore the isolation unit is at the heart of the breeding enterprise. The keys to success are shown in Table 4.

**Table 4** The keys to success of an isolation unit

1)	Know the source-tested, results to show continual monitoring
2)	One source only if possible and same nucleus
3)	Import larger numbers less frequently so that the unit can be run as all-in/all- out with cleaning, disinfection and drying
4)	Match health of imports with your own stock
5)	Isolation means isolation (at least 50 metres), preferably with separate ideal (e.g. lighting for inspection) facilities and staff
6)	Isolate for at least 3-4 weeks
7)	Can use sentinels to detect disease
8)	Bad policy to feed diseased remains to incoming stock
9)	Can buy weaner gilts at a much earlier age and rear on new unit

The other main aspect of biosecurity, which should be mentioned because it is often neglected, is cleaning and disinfection and equally important, drying. Infectious material lies in faeces, nasal sprays, mucus drops, semen drips, urine milk and blood and, under the right circumstances, can be aerosolised from all the above sources and most important of all resides in the organic matter in pig units.

The key point is that no disinfectant works effectively in the microbiological cleansing of pig accommodation unless all the organic matter is removed first. Only then will the disinfectant do its job. The effects of cleaning are shown in Tables 5, 6 and 7.

**Table 5** Effects on bacterial numbers after each stage of the cleaning process

STAGE	BACTERIA/CM <sup>2</sup> (tvc)
Immediately after pigs removed	50,000,000
After plain washing	20,000,000
After hot wash/heavy duty detergent	100,000
Target after disinfection	< 500

(Quoted in Castiglia, A et al. (2006) Proc.Am.Assoc. Swine Vet. p111)

**Table 6** Effects of partial (to head height) or total cleaning (all room) and percentage improvement (%)

	<b>PARTIALLY CLEANED</b>	<b>TOTALLY CLEANED</b>	<b>% Improvement</b>
Batches	13	13	
Weight at exit (kg)	25.4	26.4	3.9
Daily gain (gms)	693	744	7.4
% Treated	6.9	4.1	40.6
% Mortality	3.5	2.1	40.0

(Danish National Committee for Pig Production Review, 1996)

**Table 7** Daily growth rates in cleaned and uncleaned buildings

<b>Class of pig</b>	<b>Cleaned</b>	<b>Uncleaned</b>	<b>% Improvement</b>
Weaners	595	515	15.5
Growers 1	643	597	7.7
Growers 2	736	712	6.3
Finishers	671	621	8.1
Wean to sale	658	619	5.9

(From Cargill, C. and Banhazi, T. (1998) Proc. 15th IPVS.,3,15.)

**The healthy pig respiratory tract components of respiratory defence:**

The components of respiratory defence are shown in Table 8 and can be divided into three: 1) the mechanical, 2) cellular and 3) humoral mechanisms.

**Table 8** The components of respiratory defence

(A) Mechanical	Hairs in nostrils	
	Turbulence of air - deposition by impaction and sedimentation	
	warming, humidification of air	
	Competition from other microorganisms	
	Mucus properties	
	Mucociliary clearance	
	Mucosal structure	
(B) Cellular Defence	Normally	Alveolar macrophage (Airway macrophage, interstitial macrophages, blood monocyte reserves).
	Inflammation	Blood monocytes Eosinophils Neutrophil
(C) Humoral Defence	Non-specific responses	
	Specific humoral response	
	Cell mediated responses	

All are delivered by virtue of the structure of the respiratory tract. The head of the piglet is adapted to sucking whereas the adult conformation is more adapted to rooting. This aspect of behaviour is in fact made possible only by the presence of the alar cartilages, which support the nostrils and snout. This change in shape is achieved by rapid post-natal growth of the facial and nasal bones, which may explain the sensitivity of these bones to the effects of bacterial toxins which can exert influences on bone. The pig is the only one of the domestic animal species to be affected endemically by rhinitis. A cross-section of the head highlights the huge impact that the presence of the conchal (turbinates) bones has on the capacity to warm and humidify the air and trap inhaled debris. The trachea is primarily just a conducting channel to the lung. The basic airway structure is that of a tree which is continually reduced in size down to the alveolar level through a series of bronchial and bronchiolar generations. The reduction of size is also achieved by the loss of wall components so that the normal 4-layered structure shown is lost by the level of the alveolus. Even the cell structures comprising the epithelium itself are reduced towards the alveolus. In the bronchioles the epithelial height is first columnar and then attenuated to cuboidal and by the alveolus there is only an attenuated pavement epithelium between the air outside the body and the intra-capillary haemoglobin of the red blood cell. The most important thing in the study of the anatomy and pathology of the lung is to remember that probably 90% of the nuclei of the cells of the lung seen in a microscopic section are nuclei of the endothelial cells of the capillaries. The other 10% under normal circumstances are probably alveolar wall type 1 (epithelial), or type 2 (surfactant-producing) cells and only the minority alveolar macrophages.

Under normal circumstances the nostril to the terminal bronchiole is cleared by mucociliary clearance, a function of the pseudostratified epithelium. As long as the basement membrane remains intact and the bronchiolar cells (Clara cell) and alveolar epithelial cell type 2 remains intact, the airway will retain the capability of

repair. However, at the alveolar level there is only room for the alveolar macrophage as a mechanism of clearance. There are only a few reserve intra- and inter- alveolar macrophages so all the components of defence against both inhaled and blood-borne foreign antigens in the alveolar region of the lung are derived from and delivered by the blood in response to chemical messengers from the macrophages. The initial response is the drawing of extra phagocytic cells in the form of neutrophils power for the ciliary clearance or they provide an epithelial lining to the airway. The goblet cells and the mucous glands produce secretions known as mucins, which is one of the major components of mucus which is the material that actually lines the surface of the airways from the bronchiole to the nares. All the airway cells function in one of two ways. They either provide motive upper respiratory tract mucus, which would not be movable in the bronchioles so these have a watery mucin produced by the Clara cells and the alveolar fluid is an even more fluid version produced by transudate from the capillaries with addition of surfactant from the alveolar type 2 cells. The cilia are easily damaged. Negative ions increase their beat, positive ions slow them down. They beat at about 1300 beats per minute and produce a flow rate of the mucus of 4-15 mm per minute. Thus, the nasal cavity can be cleared within 15-20 minutes and the alveoli largely within 24 hours.

Almost all foreign substances slow ciliary action e.g. smoke, formic acid, salts, tobacco but inhaled gases particularly,  $\text{NH}_3$ ,  $\text{NO}_2$ , and  $\text{SO}_2$  are damaging. They cause physical and chemical damage directly and produce a mucinous response in an attempt to dissolve the irritating material. In those cases where ciliated epithelial cells are severely damaged, e.g. influenza virus infections or in high levels of ammonia, the effectiveness of ciliary clearance is greatly reduced if large numbers of cells are desquamated. Once the process of hypersecretion of mucins from both goblet cells and mucous glands commences, it takes some time for the process to be reversed. The defence of the alveolus depends entirely on the alveolar macrophage. Together the mucociliary

clearance mechanisms and the alveolar macrophage provide an efficient way of clearing the respiratory tract of debris very quickly.

The basic function of the immune system is the detection and elimination of foreign substances from the body; in the respiratory tract this removal is aided by the phagocytic system and the mucociliary clearance mechanism, a large number of cells and a huge variety of cell products, which interaction are used in this process, which is usually efficient and successful and rarely involves the initiation of an inflammatory condition. Of course, if there is endothelial cell damage or alveolar wall damage then the process almost invariably involves pulmonary oedema, which will subsequently trigger an inflammatory process.

If the basic system of mucociliary clearance and alveolar macrophage phagocytosis fail then inflammation results. It is at this point that the neutrophil is called in and the potential for self damage from released neutrophil enzymes becomes a real possibility.

### **The host response:**

To produce disease a pathogen must 1) enter the epithelial surface or produce damaging diffusion products, 2) attach and multiply in the host's tissues and then 3) resist but if possible not stimulate the host defences and then finally 4) by virtue of tissue damage caused or toxin production, it produces often a self-damaging response that has resulted from neutrophil attraction and necrosis.

### **Managemental and environmental controls:**

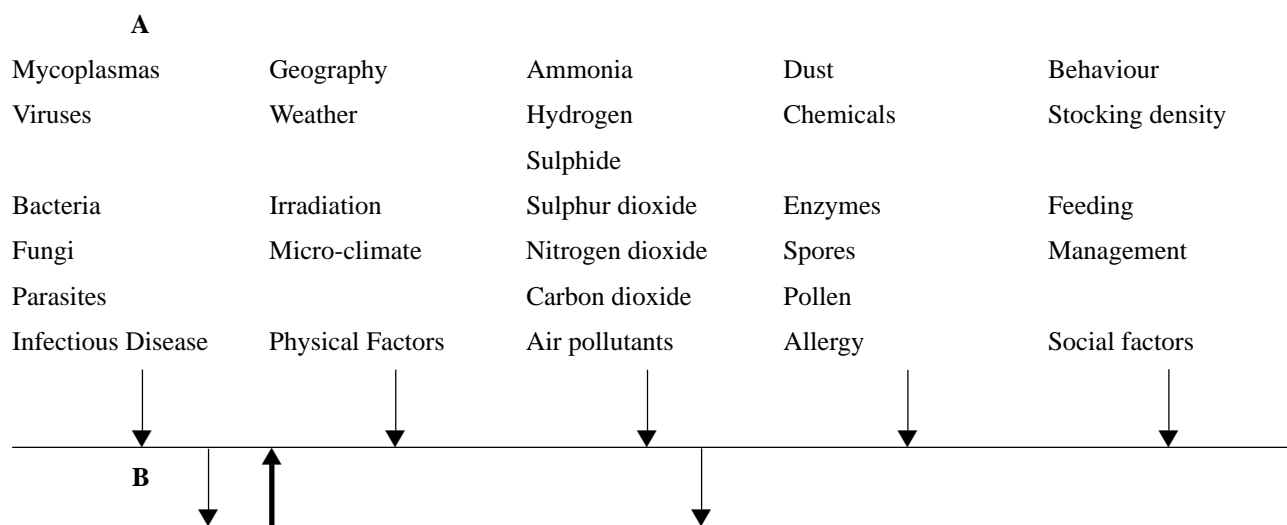
The complex interactions between the host and the environment in general are shown in Figure 1. It should be remembered, first, that the environment of each pig house comprises many components which are often difficult to define and measure. Many of them are likely to remain unappreciated, and some may affect the host as a whole, as well as its respiratory defence systems, and in addition, affect the microorganisms in the environment. Secondly, it should be remembered that these components may determine whether disease occurs, and its course, severity

and outcome. Thirdly, it is extremely difficult to relate lesions measured at slaughter to environmental factors experienced over a life-time, because it is often impossible, and usually inaccurate, to age the lesions. As a result authors have concentrated on clinical signs. However, monitoring individual animals for respiratory disease is time-consuming and prone to inaccuracy because of the difficulty of identifying the aetiological agents.

The scene was set in 1983, by Stanley Curtis who described "a pig in a box".

"Assume this box was a 1.5m cube, the sides were impervious to all except heat, water vapour and oxygen, and it has mechanisms to maintain standard conditions of pressure and temperature. Assume further that this pig weighs 80 kg and consumes 3.5 kg of a 13% crude protein (2.1% N) ration (corn/soya bean meal) daily. This diet would have 22% S. If we assume that 70% of the nitrogen and sulphur is excreted then the pig excretes 5 kg of volatile solids daily, of which 40% eventually shows up as CO<sub>2</sub> and 60% as methane, and this is contained in the box to further decompose. If we assume that about 50% is microbially decomposed each day, then it will become ammonia, hydrogen disulphide, carbon dioxide and methane. Also assume the pig expires 1000 litres of carbon dioxide each day. Assuming what we have stated then 40 litres of NH<sub>3</sub>, 2 litres of H<sub>2</sub>S, 85 litres of CO<sub>2</sub>, and 125 litres of methane will be formed each day in addition to the 1000 L of CO<sub>2</sub> respired. This means an increase in the pig's atmosphere from 3.375 L to 4,617 L. This is equivalent to 8,700 ppm of ammonia, 435 ppm of H<sub>2</sub>S, 235,000 ppm of carbon dioxide and 27,000 ppm of methane. In humans ammonia at around 700 ppm irritates eyes and nose, hydrogen sulphide at 500 ppm causes nausea, and carbon dioxide at 40,000 ppm causes drowsiness. Methane is explosive at 50,000 ppm.

If the pig survived the day it would be a pitiable, wet-eyed, wet-nosed, nauseated, dizzy individual in potentially explosive surroundings. This model explains the situation for 1 pig, imagine 1000 of them in one shed with continuous production.

**Figure 1** Schematic diagram of the relationship between the environment and the host defence mechanisms

Possible factors involved in the pathogenesis of respiratory disease

### Resistance

Agents modifying the relationships between the host and the disease producing factors

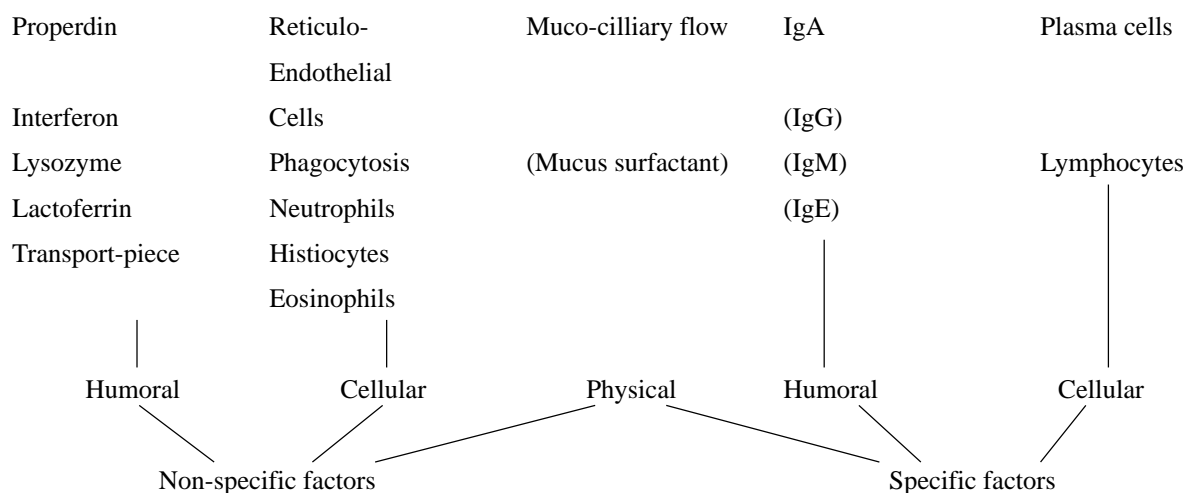
- |                       |              |                          |
|-----------------------|--------------|--------------------------|
| 1) Metabolism         | 2) Nutrition | 3) Inter-current disease |
| 4) Genetic influences | 5) Drugs     | 6) Management            |

### Disease



**C**

Factors involved in the defence of the host (as far as they are known)





## Meteorological factors:

**Temperature** - Most pig housing systems are principally concerned with the maintenance of adequate temperatures in winter. Failure to maintain a high enough temperature may be responsible for respiratory disease in a variety of ways; chills due to draughts and poor floors causing chilling may reduce resistance to infection. Wide temperature variations may reduce natural immunity and a 12°C variation or more within 24 hours also increase the incidence of pneumonia. Dry conditions increase the dustiness of food. Finally, the high temperature of the sweathouse, when combined with high humidity, reduces the incidence of pneumonia by increasing the size of the particles and therefore the rapidity and degree to which they sediment.

The author's studies on the prevalence of clinical pneumonia have shown that in pigs kept outdoors with access to shelter, low temperatures, particularly in winter, are associated with increased incidence of pneumonia; low minimum temperatures and wide daily variations are also associated with clinical pneumonia. By contrast, in animals housed all the time increasing outdoor temperatures are associated with an increased incidence of pneumonia. As the temperature rises, the ventilation is often insufficient to maintain the recommended indoor temperatures for fattening pigs, because of the heat production by the pigs themselves. Pneumonia levels also increase as the minimum temperature falls and the temperature variations widen. The prevalence of pneumonia increases with increasing maximum temperature and with decreasing minimum temperature.

**Humidity** - there has been much less work on the relationship between humidity and respiratory disease. Deviations from the mid-range may be detrimental to health. Outdoors, high humidities may increase the incidence of pneumonia, but indoors a wide variation in humidity levels seems to be the only significant association with the prevalence of pneumonia. As mentioned above, increasing outdoor humidity may help to reduce pneumonia indoors by increasing the rate of

sedimentation of particulate matter.

**Ventilation** - There are only two to four air changes per hour in most pig houses (a rate which is unlikely to reduce the aerial hazards due to high stocking densities). In buildings which have a high prevalence of pneumonia, there is often much poorer ventilation than in buildings in which the pigs have a low prevalence of pneumonia. Irregular ventilation may also increase the prevalence of pneumonia but a high air exchange rate of 60 m<sup>3</sup>/hour/pig may reduce pneumonia.

**Season** - Winter housed animals appear to have a higher prevalence of pneumonia, both in terms of mortality and from the results of abattoir studies probably because in winter ventilation is reduced to help maintain temperature and both pollution of the atmosphere and variations in temperature and humidity are increased.

## Population and social factors:

**Sex** - Both sexes appear to be at equal risk from pneumonia.

**Age** - The peak incidence of pneumonia occurs at 16 to 19 weeks of age, a time which is probably related to stocking density. There is a suggestion that sows carry fewer pathogenic organisms and provide higher levels of maternal antibody as they get older but these potential advantages are not much used in practice because the rate of genetic improvement is paramount.

**Genetics** - It has been suggested that Landrace pigs are less predisposed to pneumonia than Yorkshire pigs.

**Herd size** - As herd size increases there is an increased prevalence of pneumonia. Herds which had less than 200 pigs slaughtered had 64 percent of the pigs clear of pneumonia, in herds with between 200 and 400 pigs slaughtered 59 percent of the pigs were clear, but that in herds which had more than 400 pigs slaughtered only 55 percent of the pigs were clear of pneumonia. In small herds a high prevalence of pneumonia may be due to high sow culling rates and hence a younger average age of sows but as herd size increases there is a continual addition of highly susceptible individuals.

**Shed populations** - The larger the number of pigs in one air space the greater the incidence of pneumonia. More than 500 pigs is too many, 250 to 300 may be better and Muirhead (1979) suggested that 150 pigs in one air space is ideal. Pointon and others (1985) counted the pigs in the sheds on small and large farms with high and low prevalences of pneumonia and showed that on average there were fewer pigs in the sheds on the farms with a low prevalence of pneumonia.

**Pen populations** - There was a greater prevalence of pneumonia among pigs kept in pens holding more than 12 pigs, but other authors have found no difference between the pen populations in herds with high and low prevalences of pneumonia mainly because most of the pens on the units examined contained 10 or 11 pigs.

**Air space** - In herds with a high incidence of pneumonia the pigs had less than 3 m<sup>3</sup>/pig of air space and providing more than 3 m<sup>3</sup>/pig appeared to reduce the rate of incidence of pneumonia. The pen stocking rate, in terms of kg pig meat/m<sup>3</sup>, was not very different in small and large herds with high and low prevalences of pneumonia, ranging from 17 to 24 kg/m<sup>3</sup>.

**Floor space** - Less than 0.5m<sup>2</sup> of floor space per pig in the lying area is associated with high levels of pneumonia. It has been suggested that more than 0.5m<sup>2</sup>/pig in the lying area and 0.7 m<sup>2</sup>/pig of total area, including the dunging area, reduced the incidence of pneumonia. In the study of Pointon and others (1985) weight of pig meat (kg/m<sup>2</sup>) in herds with a high prevalence of pneumonia ranged from 93 to 132 kg/m<sup>2</sup> compared with a range from 83 to 120 kg/m<sup>2</sup> in herds with a low prevalence of the disease.

## Management factors:

Muirhead (1979) suggested that stocking density, group size, temperature and ventilation are the most important factors to manage. All management practices influence the microclimate (Done, 1990) and the quality of housing and management strongly influence the incidence of pneumonic lesions at slaughter so that

houses with good environmental conditions rarely have diseased pigs (the production and management system is generally more important than herd size) and the hygiene and husbandry are better, and the farmer's interest in preventive measures is much greater in herds with a low incidence of pneumonia. A poor management system will involve the frequent introduction of new animals and frequent climatic changes but if such deficiencies can be corrected then moderate and severe pneumonia should be reduced, and the herd's production may improve by up to 0.09 kg/pig/day.

**Method of production** - The method of production is probably the most important factor affecting the prevalence of pneumonia. Breeders and fatteners have less respiratory disease in their herds than fatteners who buy in pigs. Purchasing weaners is a significant factor but purchasing from one source of imported pigs is only a marginally greater risk than home producing.

**Finishing house organisation** - Table 9 shows the results of Lindqvist's (1974) study which emphasised the value of all-in/all-out systems compared with continuous systems. Similar conclusions have been drawn by Flesja and Solberg (1981). All-in/all-out systems greatly improve cleanliness, which was the only important factor in the multivariate model described by Tuovinen and others (1990).

**Table 9** Relationships between management system and the incidence of disease in pig herds (Lindqvist, 1974)

Method	Number of pigs	Pneumonia (%)	Pleurisy (%)	Pericarditis (%)
Batches all-in/all-out	39, 000	22.1	4.4	2.3
Batches but continuous	43, 000	28.5	6.0	2.3
Continuous	83, 000	28.5	13.4	4.5

**Manure systems** - Systems which manage manure as a solid appear to have less pneumonia (Lindqvist, 1974), particularly if continuous methods of production are being used. Tielen (1978) reported a prevalence of 23.3 percent pneumonia in pigs kept in fully slatted accommodation, 20.1 percent in pigs kept in partially slatted accommodation and 15.8 percent in pigs kept on solid floors.

**Moving** - Regrouping pigs is usually associated with moving them, and at weaning to two weeks after weaning it is an acute stressor. Tielen and others (1978) showed that among pigs which were moved once 15 percent had pneumonia, among those which were moved twice 17 per cent were affected but that among those which were moved more than twice the prevalence rose to more than 21 percent.

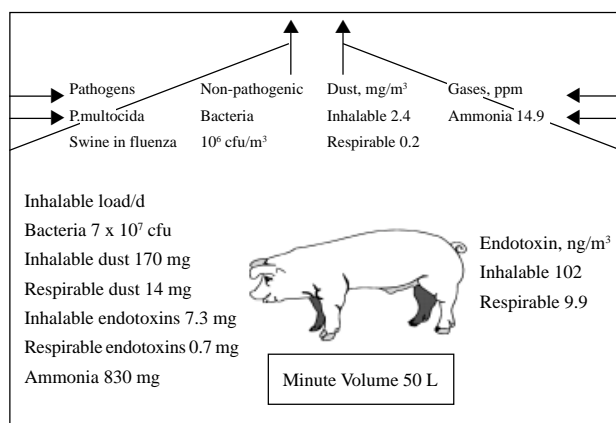
**Feeding and pen structure** - Trough feeding was more often associated with pneumonia than floor feeding (Lindqvist, 1974) and less than 0.3 m trough space per pig was also associated with an increase in the incidence of pneumonia (Flesja and others, 1982). Solid partitions, although they restrict the free circulation of air, may prevent cross-infection and thereby reduce the incidence of pneumonia (Flesja and Ulvesaeter, 1980).

endotoxins or other organic substances. There are no recommendations for the pigs, probably because they are slaughtered after only short periods of exposure. However, the levels of all these pollutants can be high and they may act synergistically. If experimental work shows a link between the levels of pollutants and the levels of respiratory disease it may be necessary to introduce safeguards on the grounds of animal welfare.

There are only two ways of reducing aerial pollution. First, it can be removed by good ventilation, although the adequacy of ventilation is usually related only to the indoor concentration of carbon dioxide and occasionally to moisture levels when there is the threat of condensation. In practice, ventilation is often controlled only in relation to the outside temperature. If one can smell toxic gases such as ammonia and hydrogen sulphide the ventilation is probably inefficient and insufficient. Secondly, and much more importantly, stocking density is the major controller of pollution, because each extra pig within the air space increases the levels of carbon dioxide, ammonia, hydrogen sulphide, water, dust and bacteria, thus imposing an extra strain on the ventilation system and decreasing its efficiency. The overall levels of pollution in a typical finishing house are shown in Figure 2.

### Airborne pollution:

Much recent work on the levels of pollution in pig houses has been concerned with the hazards faced by the workers in these environments. Safety recommendations usually refer only to the concentrations of carbon dioxide, ammonia and hydrogen sulphide, and the total and respirable dust and not to other hazards such as bacteria,



**Figure 2** Typical concentrations of aerial pollutants found in a finishing house.

**Gases** - Acute exposures to ammonia at 2000 to 5000 ppm, to hydrogen sulphide at 500 ppm after the agitation of a slurry-pit or slurry, and to nitrogen dioxide (silo-gas poisoning) have all been reported to cause the sudden death of pigs. However, of greater importance is the chronic inhalation of low levels of noxious gases which may interfere with mucociliary clearance or alveolar phagocytosis and facilitate further colonisation and damage by pathogenic bacteria (Curtis, 1983). Nevertheless, there has been little practical work to assess the relevance of ammonia in the pathogenesis of enzootic pneumonia.

**Dust** - The pig fattening house has the highest levels of dust of all the types of pig housing, and the activity of the pigs causes these high levels. Moreover, it is possible that a high proportion of the dust may be respirable - 50 percent was suggested by Donham et al. (1977) and 90 to 95 percent by Honey and McQuitty (1967). Dust is noxious because it may aid the transmission of infectious diseases by being heavily laden with bacteria (Curtis et al., 1975). Much work has been done to measure the levels of dust inside buildings but little to assess its importance in relation to pneumonia. Kovacs et al., (1967) reported that 81 percent of pneumonia cases came from the dustiest pens, but this is the only practical report. Dust has four potential toxic effects: it may be a physical irritant, it may

carry toxic chemicals, it may carry pathogens and it may carry commensal organisms. In addition, if the dust particles were entirely organic, they might pose an immunological threat to the pig's lung. It is only recently that there has been an appreciation of the high levels of endotoxin which may be found in the atmosphere of pig buildings.

**Bacteria** - As with the investigations of dust, much work has been done to measure the levels of bacteria found in pig houses and to identify them, but little has been done to relate these data with the incidence of pneumonia. Usually between  $10^4$  and  $10^6$  bacterial colony forming units (bcfu)/m<sup>3</sup> of air are found in pig houses (Curtis et al. 1975), but they are mostly non-pathogens including micrococci, haemolytic-streptococci and a few faecal coliforms (Underdahl et al., 1982). Of the total bacteria, some 65,000 to 110,000 bcfu/m<sup>3</sup> were found to be Gram-negative organisms (Clark et al., 1983). These authors suggested that 95 to 410 bcfu/m<sup>3</sup> fungi and up to 220 bcfu/m<sup>3</sup> of *Aspergillus* were also found. These levels of bacteria were associated with 1.76 to 5.17 mg/m<sup>3</sup> of dust and, more importantly, with 0.04 to 0.28 endotoxin units/m<sup>3</sup> air. The close association of bacteria with dust was shown by Buhatel (1978); each grain of dust contained  $5.6 \times 10^6$  aerobic bacteria,  $7.4 \times 10^4$  coliforms,  $1.3 \times 10^6$  haemolytic streptococci,  $6 \times 10^5$  staphylococci,  $1.6 \times 10^5$  anaerobes and  $7.3 \times 10^5$  fungal spores.

Underdahl et al. (1982) have shown that high levels of dust and high levels of bacteria may be associated with a higher prevalence of pneumonia, and Baekbo (1990) has shown that they are implicated in the development of progressive atrophic rhinitis (Table 10). The units with atrophic rhinitis had higher levels of dust, bacteria endotoxin and ammonia. As Tuovinen et al. (1990) showed in their study it may be that cleanliness is the most important factor.

In a recent study in Australia it was shown that most buildings have a relatively low level of pollution see table. However these Australian studies have shown

**Table 10** Mean concentration of key airborne pollutants in Australian piggeries

Pollutant	Max conc suggested	Actual result
Ammonia (ppm)	10	3.7
Inhalable particles (mg/m <sup>3</sup> )	2.4	1.74
Respirable particles (mg/m <sup>3</sup> )	0.23	0.26
Respirable endotoxins (EU/m <sup>3</sup> )	50	33
Total airborne bacteria (10(5)cfu/m <sup>3</sup> )	1.0	1.17

(From Banzani, T. Pig International, 35, 3, 26)

that the best way of obtaining cleaner air is by improving pen hygiene. This may be the way in which all-in/all-out works by giving a proper period for cleaning and disinfection.

In a set of experiments involving 560 pigs over a three year period, pigs were placed in experimental weaner accommodation for 6 weeks (3.5-9.5 weeks of age) and exposed to 0-20 ppm of ammonia and/or 0-10 mgdust /m<sup>3</sup>. Between 30-40 air changes an hour were used to remove background pollution. There was continual monitoring of health and production parameters. The performance of the pigs was depressed by the dust levels but not by the ammonia. In terms of health the ammonia levels appeared to have no deleterious effects but the dust was linked to an increase in lung score and pericarditis 2 weeks after the pigs emerged from the trial when they were placed in the conventional grower area.

There is strong circumstantial evidence for the transmission of pathogens within a farm e.g. APP, M. hyopneumoniae and PM but the evidence for farm to farm transmission is much less strong for PM. There are three key factors here: 1) quantities of pathogens released in aerosols 2) survival of the pathogens in the aerosol and 3) minimum infectious dose that is required for susceptible individuals in recipient herds. Don't forget that large particles sink to the ground, medium sized particles are trapped in the nose and small particles remain suspended for a long period.

### Conclusions:

If pig herds get larger, more pigs have to share an air space, and if the continuous throughput of pigs becomes a more common management system, the problem of respiratory disease will increase. However, the seriousness of the problem could be reduced by manipulating the environment, the husbandry or the management in a few simple ways.

Under normal circumstances the pig's respiratory tract may not be free of infection, or even of lesions, but the pig is usually free from clinical signs of disease. The primary agent may not be present, or it may be kept at bay at the level of the herd, group or individual pig. However, secondary environmental factors may tip the balance between the respiratory invaders and the host in favour of the invaders. It is possible to draw an arbitrary line at any point.

**Table 11** Relationships between the levels of dust, bacteria, endotoxin and ammonia in herds with and without progressive atrophic rhinitis

	<b>PAR +ve (15 units)</b>	<b>PAR -ve (29 units)</b>
Dust (mg/m <sup>3</sup> )	2.58	1.86
Bacteria (10 <sup>6</sup> /m <sup>3</sup> )	3.65	1.41
Endotoxin (10 <sup>4</sup> Eu/m <sup>3</sup> )	4.8	3.3
Ammonia (ppm)	9.27	8.36

PAR: Progressive atrophic rhinitis, Eu: Endotoxin units

If pneumonia becomes a major problem in a finishing house it may be possible to alter up to 20 environmental variables to try to reduce the incidence of the disease:

- (1) There should be a maximum of 250 to 300 pigs per shed and ideally 150
- (2) There should be 10 to 12 pigs in a pen, preferably with no mixing or moving, or at most one move not associated with weaning
- (3) There should be more than 3 m<sup>3</sup> of air space per pig
- (4) There should be at least 0.7 m<sup>2</sup> of lying and dunging area per pig with at least 0.5 m<sup>2</sup> for lying
- (5) An all-in/all-out policy should be operated
- (6) There should be one source of piglets to the finishing system, ideally breeder/finisher
- (7) The houses should be thoroughly cleaned and disinfected between groups.
- (8) The number of air changes per hour should be increased
- (9) A solid manure system is preferable
- (10) If a slurry system is used the slurry should ideally be remove daily to prevent accumulation of toxic gas
- (11) The temperature fluctuations should be kept to a minimum
- (12) Single span buildings are best because they help to prevent cross infection
- (13) Air should flow from the lying to the dunging area and then out of the building
- (14) Ammonia levels should be kept below 20 ppm and hydrogen sulphide below 10 ppm; other potential gas hazards should be reduced by segregating the pigs from the stored slurry
- (15) Dust levels should be kept as close as possible to outside levels (1 mg/m<sup>3</sup>) and not allowed to exceed 10 mg/m<sup>3</sup>
- (16) The level of respirable dust (0.5 to 2.5 µm) should be kept as low as possible by attention to feeding and bedding techniques i.e. pellets are better than meal.
- (17) Bacterial levels should be kept below 10<sup>4</sup>/m<sup>3</sup> by attention to stocking densities and cleanliness; filters may become necessary
- (18) Gram-negative organisms should be kept at low levels, to reduce the aerial concentration of endotoxin, probably by efficient manure removal and cleanliness
- (19) The pigs should have free access to drinking water
- (20) Most important, attention should be paid to stocking density, particularly during the winter

### Summary:

The adoption of a monitored biosecurity policy, an all in/all out policy with proper cleaning and disinfection and drying before restocking and a policy of producing a healthy pig through proper management and nutrition will be the major strategies in controlling the appearance of any disease and respiratory disease in particular.