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Airborne Infection and Livestock Disease Control: A Meteorological Appreciation [and Discussion]

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Airborne infection and livestock disease control: a meteorological appreciation

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The range of collaborative activities between meteorologist and veterinarian seen as necessary to ensure appropriate advice in epizootics is discussed briefly.

Non-specific advice relevant to commercial and advisory practice is examined under two headings, the nature and scale of the disease challenge and the success of that challenge.

The first of these areas of interest explores host–microparasite contact as measured by the time integral of ambient aerosol concentration and air sampling rate. This leads to comment on herd size and the siting of animal units, both locally and regionally.

The success of the disease challenge is seen to require examination of the impact of environmental factors on immune function. The importance of the thermal environment in this respect is emphasized, but the view is noted that advances in understanding and technology for control of the thermal environment in animal housing may have done little to advance biological performance. An extension of house design ventilation criteria is suggested so that air hygiene and air contaminant load may be considered. The new ventilation requirements are measured against existing design requirement in examples.

1. Introduction

Airborne infection is taken to include respiratory infection. 'Control' is defined simply as a reduction in incidence of disease. The term 'meteorological' is taken to embrace most physical environmental factors.

2. The background to specific advice in epizootics

Given a disease of intermittent occurrence, for which airborne spread is accepted as a significant component in transmission, and a policy that requires that foci of disease be actively sought out and treated, then the meteorological services are necessarily involved in any national operational control programme, if only to provide data from the national weather monitoring networks.

If, in addition, interpretation of meteorological data is required by veterinarians, so that the significance of disease at a named livestock unit becomes clear for periods before and after confirmation of infection, then separate *ad hoc* arrangements for necessary liaison and advice in each outbreak are unlikely to prove adequate in practice. The interval between outbreaks may be measured in months or years and yet the interval set by veterinarians for active control measures in the field may be measured in hours rather than days after the recognition of the disease. There is little scope for new learning either by meteorologists or veterinarians on this timescale, and formal standing arrangements for cooperation between their respective services are clearly needed. Logic might suggest that there should be a pool of staff members of the

meteorological service who are familiar in detail with the nature of the problem faced by veterinarians, who have the same commitment to a solution, and who have facilities and resources available to contribute to a solution.

Given that veterinarians and meteorologists alike may have something to learn from previous epizootics, and that case studies can provide for validation of epidemiological concepts, as well as of data handling and predictive techniques, then there is a further strong argument for a continuing collaboration on background research and development. There is some seasonality in disease outbreaks and some regional and inter-annual variability of disease incidence. Recognition of this general experience may be of limited value unless it is accompanied by appropriate environmental monitoring and prediction programmes that provide operational advice on the timing and severity of the disease challenge and on the occurrence of weather episodes that will stress the animals and affect immune response.

It is pleasing to report that in the U.K. organizational arrangements exist for continuing collaboration between the Meteorological Office and the State Veterinary Service and indeed the whole of the agricultural advisory service (Agricultural Development and Advisory Service, A.D.A.S.). These arrangements provide for cover in operational emergencies, for routine environmental monitoring programmes, for joint disease warning programmes and for basic background studies, led sometimes by meteorologists and sometimes by agriculturalists. Each of these activities provides an essential contribution to effective control measures in specific disease outbreaks.

3. Non-specific advice

There are two problem areas: the first is the nature and scale of disease challenge; the second is the success of that challenge.

Long-term trends in the impact of infectious disease are considered to be linked to advances in veterinary medicine, hygiene and nutrition. Associations between airborne disease in livestock and the weather need to consider the impact of weather on animal nutrition and hygiene, the impact of direct weather insults on metabolic and physiological control functions, together, of course, with the effect of weather on the pathogen itself. In such a listing we see factors that are to be manipulated or modified by management, in addition to factors that turn on the fundamental biology of the animal host and microparasite.

(*a*) *The nature and scale of the airborne disease challenge*

Factors of concern here are pathogen concentration (numbers and dispersion), pathogen survival and virulence, and host–microparasite contact, as measured by the time integral of the ambient aerosol concentration and the air sampling rate of the herd, that is primarily by herd size.

The situation to be considered is one in which each animal is an actual or potential emitter of infectious airborne particles (though not necessarily through respiration); each animal, however, samples the atmosphere in its respiratory function, and disease entry is effected essentially by inhalation.

Given that a range of size of infectious particles is to be found in the atmosphere around a primary source, we may come to a number of somewhat obvious, general conclusions, as follows:

(i) some host species are likely to be potentially greater emitters of infectious aerosol than others;

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(ii) for a given species of animal host, large herds or units are potentially greater emitters of infectious aerosol than small units;

(iii) large animal units have a greater risk of acquiring airborne infection than small animal units;

(iv) individual, large animals with their greater air sampling rate are at a greater risk of infection than smaller animals;

(v) the greater the separation of animal units, the greater the probable effects of air movements to reduce aerosol concentration, and the smaller the risk of infection;

(vi) intensive, as opposed to extensive livestock systems, provide for additional modes of contact and disease spread.

One is led to look for additional information that might help to quantify some of these statements.

TABLE 1. CATTLE HERD SIZE WITH HIGH RISK OF AIRBORNE FMD INFECTION

(Source strength 4000 cattle or 1 pig (peak emissions); assumed windspeed: 4 m s^{-1}.)

height of mixing layer	herd size† at downwind distance from source		
m	10 km	50 km	100km
100	> 30	—	—
200	> 60	> 300	—
300	> 100	—	> 1000
500	> 150		
1000	> 300		

† Number of animals.

(*b*) *Size of unit and infection risk from external sources*

Comments made here on the size of livestock units have to be read in the context of the situation in the U.K. where for cattle, for example, the commonest class of holding has a total of 100–200 cattle (or a dairy herd of 70–100 cows) on 50–100 ha. The corresponding figures for pigs is for units of 200–400 animals, again on holdings of 50–100 ha. In broiler production the largest class is of units of 100000 to 250000 birds, though some units with over a million birds are reported (Anon. 1981).

In the 1981 foot-and-mouth disease (FMD) outbreak, after the primary occurrence in pigs in France, disease was first confirmed in cows in the U.K. in Jersey, and subsequently on the Isle of Wight. The herd sizes involved were representative of the U.K. as a whole. The source strength of aerosol emission in France at any one time was that to be associated with perhaps 35 pigs. Given the acceptance of the idea of the emission of an infectious aerosol plume from a source many kilometres upwind (*ca.* 200 km) and its subsequent travel (at 6 m s^{-1}) under stable atmospheric conditions that effectively limited the height of the plume (to about 500 m), then a reasonable proposition in the circumstances of the 1981 FMD outbreak might be that a herd of 100 cows, each with an air sampling rate (in round figures) of 5 m^3 h^{-1}, has a probability approaching unity of taking up sufficient aerosol to initiate infection.

If we use as scaling factors the source strength, the height of the atmospheric layer through which aerosol emitted at ground level is diffused by convection and forced turbulent mixing, windspeed, and distance from the source (Pasquill 1961), then the size of the herd for which the probability of airborne infection is great is shown in table 1.

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Table 2 shows the outcome when a similar calculation is carried out for peak emission of Newcastle disease (ND) by unvaccinated poultry (for which infectious aerosol output is scaled down by a factor 10^3 compared with FMD output from pigs and where the respiratory air sampling rate is scaled down by a factor of 50 for birds compared with cattle). Such an analysis offers little comfort on the typical holding in those weather situations where convective (buoyancy) forces on parcels of air in the lowest layers are not dominant.

Because high-risk weather situations for airborne disease spread will always be a feature of the farming scene in the U.K. and because the present scale of farm enterprises (herd or flock size) is not readily reduced, it does appear that non-specific strategies to minimize host–microparasite contact from a source outside the farm have to be directed at avoidance of large ambient aerosol concentrations.

TABLE 2. POULTRY FLOCK SIZE WITH HIGH RISK OF AIRBORNE ND INFECTION

(Source strength 100 birds; assumed wind speed 4 m s^{-1}.)

height of mixing layer	flock size† at downwind distance from source	
m	10 km	50 km
100	> 15000	—
200	> 30000	> 150000
300	> 50000	> 250000
500	> 75000	
1000	> 150000	

† Number of birds.

(c) *The avoidance of large aerosol concentrations*

(i) *Source strength*

In the interest of all, there is something to be said for not carrying good receptors of infection (cattle in FMD) and good emitters of infection (pigs in FMD) on the same holding.

(ii) *Regional advantage*

For those enterprises able to consider the disposition of livestock units nationally and not just locally, the direct advantages of the physical isolation of key stock may be reinforced by an appropriate choice of area.

For specific pathogens, for which the life expectancy or virulence has been established in relation to combinations of ambient air temperature and humidity (or other environmental factors), there is the possibility of establishing occurrence probability distributions for the relevant meteorological variables and of establishing the relative advantage of the climate in different regions.

A regional climate analysis should include some assessment of the probability of air trajectories arriving from likely source regions of infection. Work on wind fetch has to be linked with simultaneous analyses of temperature lapse rates (or stability) in the atmospheric boundary layer, because it is the probability of carriage under conditions that limit vertical dispersion that assumes importance. Diurnal wind effects need to be written into such regional climate analyses.

The effect of regional vegetation, its vertical dimensions and the extent of ground cover is

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perhaps better examined not so much in terms of the effect of associated or relative surface roughness on the depth of vertical mixing (the consequential increase in aerosol cloud height over rough terrain is thought to be marginal) as through consequences of the scale of eddying motions over and within the vegetation canopy and therefore on aerosol capture and removal. Comment on the processes of dry deposition (sedimentation, impaction, interception, Brownian motion) is provided by Chamberlain & Little (1981). Regional advantage in aerosol depletion can be assigned to the presence of forested areas as against grassland. This advantage will be reinforced in areas where the frequency of days with rain is larger than elsewhere and the canopy wet, i.e. sticky.

The occurrence of rainfall events that will give rise to wet deposition or 'washout' of the aerosol plume is a further consideration. For a plume of small particles (*ca.* 2μm) of fixed vertical extent, travelling in moderate winds (5 m s^{-1}), travel distances of the order 100 km can be required before airborne concentrations are reduced to 50% of initial values by washout; for larger particles (more than 10 μm) travel distances before significant depletion by washout will only be a few kilometres or at most a few tens of kilometres (Gloster & Field 1981).

(iii) *Local advantage*

For particular sites in difficult terrain, general regional advantage has to be set against the risk of high local concentrations of aerosol. These may be associated with local sources of aerosol, particularly in those weather situations where a stable atmosphere in the lowest surface layers leads to an effective decoupling of the surface and upper flows.

It would seem prudent to keep away from locations subject to the mechanical channelling of airflow by features of the terrain, locations that are low down on sloping ground (and so likely to experience gravity driven downslope and drainage flows that may simply collect in stagnant 'cold pools') and locations that, in an advective situation, remain within the lower layer that moves around (rather than over) hills and higher ground. These considerations are reinforced by a concern for the good, natural (wind) ventilation of animal houses and for the avoidance of large, diurnal, ambient air temperature changes, with their attendant and potentially stressful changes in air movement patterns within animal houses (that follow on the temperature difference of the incoming air jet from the bulk air temperature in the house).

Although there may be no unique quantitative answer to questions on the airflow, likely dispersion and aerosol concentration from identified sources in every meteorological situation over complex terrain, work reported, for example, by Rowe *et al.* (1982), Reible *et al.* (1981), Hunt & Snyder (1980) and Scorer (1968) draws together information from case studies; in agricultural extension work, the qualitative understanding of factors influencing airflow and carriage of aerosol should enable the worst of errors in the location of animal units to be avoided.

(iv) *Protection afforded by housing.*

It is perhaps necessary to consider briefly a role for housing in the protection of animals. Given that ventilation of the house with the ambient air takes place at all times, we may represent the situation by that shown schematically in figure 1*a* and assume for simplicity a rectangular perturbation in the external aerosol concentration C_0. Assuming perfect mixing of the internal air, we may establish expressions for the instantaneous rate of change of concentration of aerosol in the house and for the concentration C_1 itself as a function of C_0 and time. For a perturbation in C_0 of duration 1 h and for reasonable ventilation rates (a few house-volume

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air changes an hour) then there is no significant difference in host–microparasite contact (as measured by the time integral of the ambient aerosol concentration) encountered by housed and unhoused animals. The practical experience of FMD epizootics in the U.K. has been that the housing of animals does not materially influence the occurrence of secondary outbreaks.

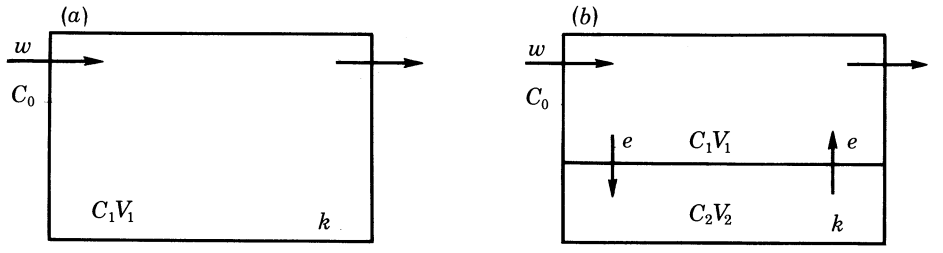

FIGURE 1. Representation of the ventilation of an animal house at a rate of w m^3 h^{-1} with air having an aerosol concentration C_0 g m^{-3}. In (a) the house has a volume V_1 m^3, in which the aerosol concentration is C_1 and in which aerosol is generated at a rate k g h^{-1}. In (b) the house is divided into compartments of volumes V_1 and V_2 and aerosol concentrations C_1 and C_2 respectively. Air is exchanged between the compartments at rate e and aerosol is generated at rate k in the lower compartment only.

Although there is some evidence of protection from being indoors against inhalation of suspended particulate matter of outdoor origin (Cohen & Cohen 1980) it is open to question whether such studies involving human housing are strictly relevant to intensive animal housing with their greater ventilation requirements, air velocities and internal activity.

(v) *Local obstruction to airflow and building separation*

The disturbance to the low-level airflow posed by bluff obstacles such as farm buildings is complex, but certain generalizations can be made about the resulting patterns of air movement. These are shown schematically by Hosker (1980). Where there are close arrays of farm buildings (separation of the same order as their linear dimensions) then one can visualize reattachment of the low level flow to the surface represented by the roofs of the buildings rather than to the ground itself and the extension of the wake bubble to encompass the entire group of buildings. The resulting relatively stagnant air circulation enclosing the buildings has serious implications for the air removal rate by natural (wind) ventilation within the houses, and this will commonly be a cause for concern in its own right (see §4). However, the fluctuating pressures (both in sense and magnitude) on the external surfaces of buildings due to the eddying and wake flows arising from the buildings themselves, as well as to changes in the free wind, will result in air re-entry. In the present context, farm buildings in close juxtaposition can be expected to perform much as a single building.

We are led to ask what is the separation of buildings that in the general situation will allow some significant reduction in the risk of airborne disease being spread between them.

The representation of an animal house as a point source of aerosol generated at ground level is somewhat unrealistic, because wake effects may be detected for downwind distances of perhaps ten times building height. There are consequences for the detailed application of the aerosol plume dispersion equation (Pasquill 1961) in this situation, and Miller (1981) examines various alternative procedures in the light of some field trials over downwind distances up to 800 m. One should perhaps represent an animal house as an elevated rather than a ground-level source of particles. The result is to modify the curves of downwind aerosol concentration against

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distance associated with a low-level source (figure 2), from which very well defined recommendations for building separation in excess of 100–200 m follow. For an elevated source the aerosol concentration peak at low level (1–2 m above the ground) is reduced numerically by an order of magnitude (figure 2*b*) and the distance from source to one half peak concentration is stretched (in stable or neutral atmospheres) to distances of the order 500 m or more.

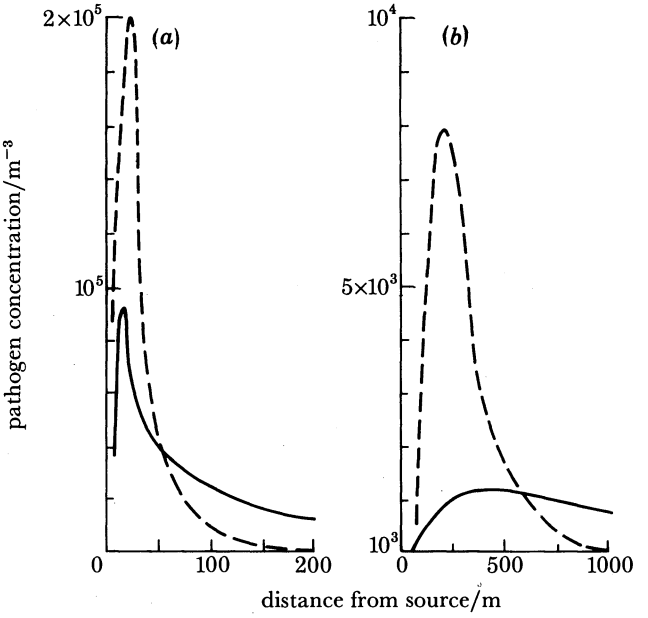

FIGURE 2. Low-level concentration of airborne pathogen as a function of downwind distance from source in a stable atmosphere. Source strength 10^6 pathogens per second. Emission height in (*a*), 3 m; in (*b*) 10 m. (– – –), strongly sedimenting particles; (——), weakly sedimenting particles. (After Müller *et al.* (1978).)

The outcome of an alternative 'random walk' model of particle diffusion (table 3) also reflects the high proportion of particles that remain airborne at distances up to 1 km downwind with elevated sources. It should be noted that large (50 μm) droplets emitted by animals will be expected to evaporate rapidly to form smaller particles (less than 10 μm).

Overall the conclusion is that the physical separation of animals on the scale permitted by individual holdings is not likely to be great enough to avoid significant airborne contact, although attention to the spacing and topographical exposure of units and to hygiene generally should not be neglected on this account.

4. Environmental factors and immune function

In so far as extensive, free-ranging farming systems may periodically involve the bringing together and housing of substantial numbers of animals, it is appropriate to concentrate comment largely on intensive animal units, primarily those in the U.K.

In the incidence of respiratory disease there is support for the concept of synergism, in which one species of microorganism is though to act by reducing the effectiveness of non-specific defence mechanisms, resulting in colonization by other species; there is also support for the idea that stress may act as an agency that reduces the effectiveness of non-specific defence mechanisms and disposes the host towards invasion by disease. Success in inducing certain

respiratory diseases experimentally has been found to require both social and environmental stress as well as exposure to the pathogens (Harkness 1977).

(*a*) *The thermal environment*

A conventional representation of thermal energy balance relations in a homeotherm is shown in figure 3 (after Mount 1973).

TABLE 3. OUTCOME OF A 'RANDOM WALK' MODEL SIMULATION OF PARTICLE DIFFUSION (AFTER THOMPSON 1982)

(Assumptions: near neutral stability; wind speed 5 m s^{-1}.)

release height / m	drop size / μm	probability of absorption on first impact at ground	percentage of particles airborne at downwind distance from source: 50 m	100 m	200 m	500 m	1000 m
1	50	1	38.8	25.2	17.5	11.4	7.9
5	50	1	93.7	81.3	62.6	42.7	31.6
10	50	1	99.6	95.6	82.6	62.4	47.1
1	10	1	67.5	56.4	46.9	38.5	34.5
5	10	1	97.5	91.5	84.5	72.5	65.3
10	10	1	100.0	99.1	94.1	84.7	78.6
1	10	0.05	89.7	83.5	76.8	68.8	63.2
5	10	0.05	99.3	97.0	92.3	85.7	80.5
10	10	0.05	100.0	99.7	98.1	92.3	88.1

For ruminants (excluding neonates), adaptation enables both the upper and lower critical temperatures to be seen as somewhat disposable constants, and it is the departure from thermal environments to which the animals have become accustomed that will provide measures of thermal stress, rather than absolute measures of the thermal environment *per se*.

With this proviso, the range of ambient environments associated with minimal metabolic activity (the thermoneutral zone, CE) is determined primarily by levels of food intake for a given age and species of stock. Within this zone there is a ready balance between heat loss to the environment and that arising from energy taken up in maintenance processes, together with that arising from the inefficiency of food (energy) conversion in growth and productive processes.

Departures from the thermoneutral zone place demands on metabolic and physiological control systems; even within the thermoneutral zone, departures from the zone of minimal thermoregulator effort, CD, nevertheless demand physiological adjustments (commonly associated with increased respiration rate or increased tidal volume).

Weather may indirectly affect host resistance to disease in farm animals if it leads to changes in food energy intake, or to changes in their behaviour or management.

Exposure to heat or cold stress is linked to changes in the immune system. Specific resistance to infection through passively acquired antibody immunity is likely to be reduced; attendant changes in antibody and cell-mediated immune responses have been shown both to enhance and to suppress susceptibility to a variety of pathogens. The timing and degree of exposure to heat or cold stress are important discriminants in this (Bletcha & Kelley 1983; Kelley & Mertschinger 1983). Non-specific resistance to infection is also modified by heat and cold.

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Phagocytosis can be reduced in cool environments, and ambient temperatures (or perhaps rather ambient air temperature and moisture contents in combination) affect mucin production, ciliary action and the 'mechanical' clearance of mucus, and attached inhaled aerosol, from the pulmonary system.

Such a summary identifies a significant role for the thermal environment (together with food energy intake) in the control of disease incidence. Information is widely available on the critical temperatures of various classes of stock under various feeding régimes (see, for example, Close 1981) and is not repeated here.

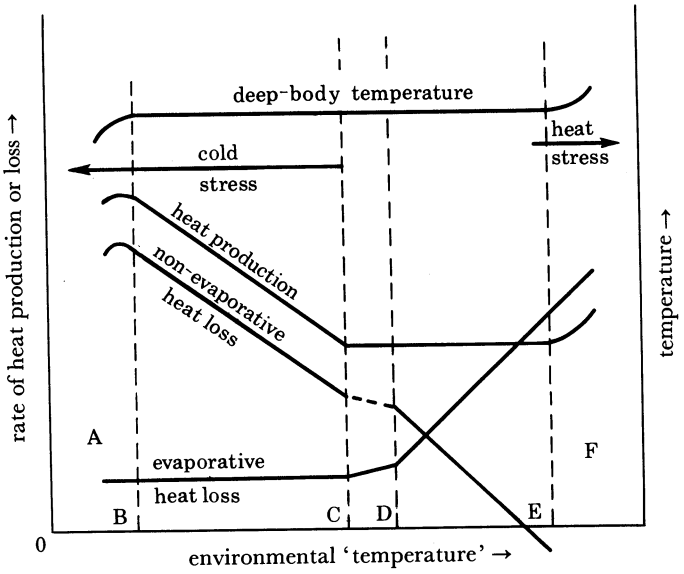

FIGURE 3. Diagrammatic representation of components of the energy balance of a homeotherm (after Mount 1973). A, zone of hypothermia; B, 'temperature' of summit metabolism; C, lower critical 'temperature'; D, 'temperature' associated with marked increase of evaporative loss; CD, zone of least thermoregulatory effort; E, upper critical 'temperature'; F, zone of hyperthermia; CE, zone of minimal metabolism (Thermoneutral zone).

(*b*) *Ambient air temperature and humidity*

The mucous membranes provide the initial, physical defence against airborne infection. If dehydration or other processes impair the action of the cilia, then retention of attached aerosol *in situ* provides a site for potential infection.

The saturation vapour pressure of air at body temperature is of the order 65 mbar (6.5 kPa). Typically in the U.K., the ambient outside air with a temperature of 10 °C or less will have a moisture vapour pressure of less than 10 mbar. The implications of breathing such air are met by the (low) temperature stimulation of a reflex-controlled secretion process for watery mucus. Dehydration arises with inspired air of high temperature (above 20 °C) but of low moisture vapour pressure (less than 10–15 mbar), and this is associated with an increased incidence of respiratory infection or response to airborne allergens (cf. Jericho & Magwood (1977) on calves).

Intensive livestock units in the U.K. in which considerations of 'air conditioning' are restricted solely to the achievement of a large temperature 'lift' over outside temperatures, to promote economic aspects of production other than animal health, have to face an increased probability of infection due to impaired cilial action, as well as a decrease in hygiene (air quality) at low ventilation rates.

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5. Respiratory disease and design criteria for livestock housing

The ability to manipulate internal (house) environments (primarily air temperature and air velocities) and to exploit waste metabolic heat through control of house ventilation (and insulation) has advanced significantly in recent decades. Both understanding and technology are sophisticated and can be seen as adequate in principle for control of the thermal environment. They have been applied most extensively to livestock enterprises in which food costs are the largest component of the cost of production (poultry and pigs) and for which experimentation and theory together have led to the identification of target thermal environments in which production is apparently achieved most efficiently in economic terms.

Despite these advances, the advantages have been measured in economy of fuel and labour costs and not in animal productivity and health (Sainsbury 1981). This might suggest that the principles underlying housing design need some extension if airborne disease and respiratory infection are to be controlled (reduced in incidence) by environmental measures.

Currently, livestock requirements are denoted primarily by a nominated thermal environment and since ventilation of the house is the key determinant in this, by criteria that note:

(*a*) the concentration of carbon dioxide;

(*b*) the maintenance of air temperatures between the lower and upper critical temperatures of the animals;

(*c*) the relative humidity and the occurrence of condensation on the fabric of the building (Bruce 1981).

The proposal made here is that the requirement specification for livestock housing should include both the thermal environment and air quality. Criteria for house ventilation and environment control would now also establish:

(*d*) A target level for the dosage arising from infectious (and other) aerosol generated by respiration or other processes; a convenient base from which to measure would be the dosage associated with an air clearance rate for the house equal to the rate of air use by the animals (i.e. the product of the number of animals in the house and their individual pulmonary ventilation rate);

(*e*) acceptable combinations of house air temperature and relative humidity that do not inhibit mucal clearance of inhaled particulates.

(*a*) *House ventilation and air hygiene*

In this section I explore, in simple terms, how the operation of ventilation systems and resultant patterns of internal air movement affect the air contaminant load and microparasite–host contact (as measured by the time integral of the ambient aerosol concentration in the private environment experienced by the individual animal).

Consider the case shown schematically in figure 1*a*. Assuming perfect mixing and no re-entry of contaminated air, a general expression for C_1, the concentration of aerosol contaminant may be derived which implies that at the steady state (as time $t \rightarrow \infty$)

$$C_1 = C_0 + k/w. \tag{1}$$

Consider now the same house where the ventilation system effectively divides the house into two compartments (as in figure 1*b*); this may well occur where air movements at animal level arise essentially from secondary circulations linked by entrainment to the primary ventilation

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jet or from free convection over the backs of the animals. If there is a local air exchange rate, e, between the compartment containing the animals and in which aerosol is again generated at rate k, general expressions for C_1 and C_2 may now be derived that suggest that at the steady state

$$C_1 = C_0 + k/w \tag{2}$$

and

$$C_2 = C_0 + k/w + k/e. \tag{3}$$

The local air exchange rate, e, rather than the overall house exchange rate will primarily determine C_2, the local concentration of aerosol at animal level when e is smaller than w by an order of magnitude (as may well occur in buildings with a poorly commissioned fan-assisted ventilation system or in naturally ventilated houses). Having made this point, I shall employ the house ventilation rate, w, in further discussion on ventilation rates, house volume allocated per animal and aerosol contaminant dosages but the consequences of the replacement of w by the smaller quantity e are readily seen.

If in (2), $C_0 = 0$, $k = k'\ (Np)$, where N is the number of animals in the house and p is the individual pulmonary ventilation rate (cubic metres per hour), then

$$C_1 = k'(Np)/w. \tag{4}$$

Let

$$w = RV_1 = RNv, \tag{5}$$

where R is the house ventilation rate (volume changes per hour) and v is the house volume allocated per animal (cubic metres) (and both are quantities readily visualized in advisory practice). The dosage, D, of aerosol experienced by each animal in 1 h is then

$$D = C_1 p = k'(Np^2)/w = kp^2/Rv. \tag{6}$$

Let $D = D^*$ when $w = Np$, the base air clearance rate; then generally

$$D/D^* = p/Rv. \tag{7}$$

The ratio D/D^* is independent of the rate of mode of production of aerosol (we might equally have written $k = k'\ (NA)$, where A is the surface area of the individual animal). Primarily it provides a measure of the relative challenge within a given house under various régimes in which clearance of aerosol (including biologically active aerosol) by ventilation, and house volume allocated per animal, are seen as the main variables. Unless one is in a position to quantify and differentiate between the rate of production of aerosol in different houses, the quantity D/D^* can be seen as offering some more general measure of air hygiene levels.

We may explore the consequences of (7), assuming that capital costs restrict the house volume allocated per animal, v, to a few time the volume taken up by the standing animal. The outcome is shown in table 4.

Some feel for the values of D/D^* that follow from good current practice (where the emphasis is on the thermal environment provided) may be obtained by comparison with the nomograms of Bruce (1981). These nomograms relate house ventilation rate for thermoneutrality of various classes of pigs to internal and external air temperatures. In addition to the minimum ventilation rate linked with a CO_2 concentration level of 0.3%, recommended maximum and minimum ventilation rates culled from the literature are also indicated.

In the second case discussed by Bruce (1981) (500 pigs at 60 kg live weight, in groups of

15, fed at three times maintenance), the lower limit set for ventilation (0.3% CO_2) is 1.44 $m^3 s^{-1}$. Equation (7) indicates, with p set at 1.0 $m^3 h^{-1}$ and D/D^* set at 10^{-1}, R equal to 6.7 house volumes per hour, i.e. 5000 $m^3 h^{-1}$ or 1.4 $m^3 s^{-1}$.

In the other cases also discussed in detail by Bruce (1981), a value for $D/D^* = 10^{-1}$ identifies equally closely with the lower limit set for ventilation on other grounds.

TABLE 4. HOUSE VENTILATION RATE TO ACHIEVE VARIOUS DESIGN RATIOS OF DOSAGE FOR INTERNALLY GENERATED (RESPIRATORY) AEROSOL

stock	pulmonary ventilation rate, p / $m^3 m^{-1}$	house volume per animal, v / m^3	aerosol dosage ratio, D/D^*	house ventilation rate, R (air changes per hour)
cattle	5.0	10	10^{-1}	5
			10^{-2}	50
pigs	1.0	1.5	10^{-1}	7
			10^{-2}	67
	0.5	1.5	10^{-1}	20
			10^{-2}	200
poultry	0.1	0.15	10^{-1}	7
			10^{-2}	67

If in these cases D/D^* is now set at 10^{-2}, then we find that the ventilation rate implied by (7) covers the bulk (*ca.* 90%) of the permissible temperature and ventilation rate combinations considered by Bruce (1981), where the ventilation ranges from the minimum determined by CO_2 levels to the highest ventilation rates intended to limit the temperature lift within the house at high external temperatures.

The quantity D/D^* provides one simple measure of air hygiene and it appears that values of D/D^* in the range 10^{-1} to 10^{-2} would be achieved in the best commercial practice with its emphasis on control of the thermal environment (provided that the assumptions underlying the derivation of D/D^* are met, i.e. no compartmentation of the air distribution, perfect mixing of the air, and an efficient control system for ventilation).

Nevertheless, within this range of values respiratory health problems are accepted and subclinical and chronic infection can go largely unrecognized. Such problems are accentuated in the winter quarter of the year when mean daily (external) temperatures are somewhat below 5 °C over the greater part of the British Isles. Ventilation rates in the winter season in the best commercial practice (cf. Bruce's (1981) nomograms) are likely to be no more than twice the minimum determined by acceptable CO_2 thresholds and imply values for D/D^* in the range 10^{-1} to $10^{-1.3}$ (D/D^*, 0.1–0.05). If we note the seasonality of respiratory disease incidence and if a target threshold for D/D^* were to be set by the ventilation rate to be associated with mean minimum (external) temperatures in the summer months (*ca.* 10 °C), the implied design minimum ventilation rates (of the order three times the minimum set by acceptable CO_2 thresholds) in turn imply design values for $D/D^* \simeq 10^{-1.5}$ ($D/D^* \approx 0.03$).

Clearly the raising of design standards has to be paid for; running costs are increased if a limit is set to the temperature lift that may be supplied by waste metabolic heat, and, alternatively, capital costs are increased if the design volume allocated per animal is increased. However, the problem is to demonstrate and quantify the improvement in animal health and performance that occurs if air hygiene standards such as have been suggested are adopted.

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Pritchard *et al.* (1981) have examined the effect of air filtration on respiratory disease in intensively housed veal calves. Assuming that it is legitimate to treat the effects of a reduction in aerosol dosage independently of the way in which this reduction is achieved (i.e. by air filtration rather than by increased ventilation), we may examine these results in the context of the measure of air hygiene, D/D^*. Pritchard *et al.* (1981) indicate three ventilation rates, and calculation on the basis of the highest of these (after setting $p = 1.0\ m^3\ h^{-1}$) gives, for the control calf houses, $D/D^* = 10^{-1}$ ($D/D^* = 0.09$). The effect of air filtration is to reduce the mean aerial concentration of bacteria by 45% and this may be taken to imply $D/D^* = 10^{-1.4}$ ($D/D^* = 0.04$). In the units where a reduction in aerosol was achieved, there was a reduction in both incidence and severity of clinical and subclinical disease. A comparison of units on a paired basis showed that the number of animals requiring antibiotic treatment for respiratory disease was reduced by about 19%, the number of repeat courses of treatment was reduced by about 29% and the total use of antibiotic was reduced by about 30%. At slaughter, the average area of lung consolidation in calves from units with reduced air contaminant load was reduced by about 38%.

Viewing the work of Butler & Egan (1974) in the same way, we may point to the considerable and highly significant reduction in the synthesis of immunoglobulins (together with increased food conversion ratios) for specific pathogen free chicks raised in isolators compared with conventional accommodation. The filtration of air entering isolators eliminated overtly pathogenic agents, but the specific pathogen free animal can have a normal microflora and therefore be viewed as similar to the conventionally kept health animal. A value for D/D^* may be derived for the birds in isolators from the details provided by Butler & Egan (1974). Assigning a value to p of $0.05\ m^3\ h^{-1}$, it is found that $D/D^* = 10^{-1.6}$ ($D/D^* = 0.03$). In intensive animal units, direct contact must remain a potent mechanism for the transmission of disease, but at the same time the adoption and implementation of appropriate design criteria for air hygiene has considerable merit. A simple and readily understandable scale that provides a measure of air quality has been proposed

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Discussion

G. C. Pritchard (*Veterinary Investigation Centre, Norwich, U.K.*). In the face of an airborne virus plume, is there any difference in the probability of encountering the virus if the animal is outside in a field, in an open, naturally ventilated building, or inside a controlled environment house with minimal air inlets and outlets?

C. V. Smith. Simple calculations would suggest that provided that the air in the house is well mixed and that the house undergoes several air changes each hour, then the dosage experienced by the animals is much the same whether they are housed or not. There is some evidence for protection from airborne material in human habitation. Animal houses have a greater stock density than human habitation, a greater ventilation requirement and more activity, so the two cases are not entirely analogous.

C. M. Wathes (*Department of Animal Husbandry, Langford House, Bristol, U.K.*). I should like to turn from the spread of disease by the direct transmission of airborne pathogens to the role that air hygiene plays in disease, especially respiratory diseases, of farm animals. When animals are brought indoors one inevitable consequence is a rise in the level of airborne contaminants that the animal must endure. This includes not only pathogens but also non-pathogenic viable organisms and inert dust. For example, the concentration of a heterogenous cocktail of bacteria in a calf house may exceed 10^5 organisms per cubic metre, two or three orders of magnitude above that outside.

Needless to say, such large burdens may not by themselves be damaging to the healthy animal, although this is not proven, but they probably exacerbate existing respiratory conditions.

Where does this leave the architect of animal houses? Although he can engineer almost any degree of aerial hygiene by filtration, he is hampered by the lack of any threshold limits for tolerable concentrations of airborne contaminants.

At Bristol we have approached this problem by examining the source of and sinks for airborne contaminants. In the simple case of a steady-state balance the concentration of contaminants reflects the differing rates of emission or release and of clearance. The two major sources of airborne bacteria in a calf house are (i) the calves themselves releasing bacteria resident on

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skin squames, and (ii) the building, primarily the bedding. Routes by which bacteria are cleared from the air are ventilation, inspiration and sedimentation, and microbial death occurs both during and after dispersal in an aerosol. First-order kinetics can be used to express quantitatively the effectiveness of each route and thus to examine their relative efficiency.

As an illustration of the convenience of this approach, we find a linear relation between the reciprocal of the concentration and the rate of ventilation, assuming that the rates of release and clearance by other mechanisms remain constant. The slope of the regression provides an estimate of the total rate of release from all sources; the ratio of the intercept to the slope gives the combined total rate constant for the other clearance routes, apart from ventilation:

$$C = \frac{(\bar{R}_a + \bar{R}_b)}{\{V(q_d + q_s + q_v + q_r)\}},$$

where C is the concentration of bacteria per cubic metre, $\bar{R}_a$ and $\bar{R}_b$ are the total hourly release rate of bacteria from the animals and building surfaces respectively, V is the volume of the building in cubic metres, and q_d, q_s, q_v and q_r are hourly clearance constants or extinction coefficients for microbial death, sedimentation, ventilation and inspiration respectively.

In this example of 29 calves housed on straw, the total release rate from all sources, the regression slope, is 1.2×10^9 colony-forming units per hour, whereas from other measurements we can estimate the animal's contribution at 4 % of this. Most of the scatter about the regression may be attributed to variation in other factors influencing microbial death rates, such as relative humidity. We also estimate the sum of all the remaining clearance constants at 30 h l, equivalent to a rapid ventilation rate.

In conclusion, the type of approach that I have just outlined can be used for any airborne contaminant, including dust. It suggests that a stragety for improving aerial hygiene should be based not just on physical removal by ventilation but should also consider the other pathways of clearance. Different tactics may even lead to contradictions: the manipulation of relative humidity to hasten microbial death rates may require a reduction in ventilation rate to maintain high air temperature.

Until animal scientists can provide threshold limits for the common airborne contaminants, the engineer can only view the problems of animal hygiene in animal houses through a murky haze of polluted air.

P. K. C. Austwick (*Aerobiology Unit, Cardiothoracic Institute, University of London, Frimley, U.K.*). Dr Wathes's figures for the airborne concentration of bacteria in calf houses of *ca.* 10^5 m^{-3} are low compared with Dr J. Lacey's maxima of 10^9 m^{-3} for actinomycetes and 10^8 m^{-3} for fungi. Does he think that the specially adapted airborne spores of these organisms from mouldy hay and straw might passively pick up droplets containing virus or bacteria and carry them outside, providing inoculum for other animals downwind?

C. M. Wathes. At Bristol the maximum concentration of airborne bacteria that we have observed in a calf house is 10^6 colony-forming units, per cubic metre, with a mean of about 10^5. I am surprised that Dr Lacey has recorded such high concentrations of actinomycetes and fungi; this suggests that the release rates must be very rapid. I do not know if spores from mouldy hay and straw can pick up viruses and bacteria and carry them on the same particle.

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