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Empirical and theoretical evidence for herd size as a risk factor for swine diseases

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Abstract

Herd size is frequently studied as a risk factor for swine diseases, yet the biological rationale for a reported association with herd size (whether positive or negative) is rarely adequately discussed in published epidemiological studies. Biologically plausible reasons for a positive association between herd size and disease include a greater risk of introduction of pathogens from outside the herd, greater risk of transmission of pathogens within and among herds when the herd is large, and effects of management and environmental factors that are related to herd size. However, compared with owners of small herds, owners of large herds might more frequently adopt management and housing practices that mitigate this theoretically increased risk. We used studies of pleuritis, pneumonia and pseudorabies to describe the epidemiological issues involved in evaluations of the relationship between management factors, herd size and disease. In future studies, we recommend that (i) herd size be measured in a way that best characterizes the true population at risk; (ii) studies that evaluate management-related risk factors should account for herd size wherever possible; (iii) population-based studies of the interrelationships among management factors and between management factors, herd size, herd density and pig density be done; (iv) likely biological reasons for any herd-size effect be postulated; and (v) the distribution of herd sizes in the source population and the study sample be described.

Introduction

Management and environmental factors are considered important determinants of the risk of the introduction and maintenance of an infectious agent in swine herds and also affect many measures of herd productivity. Among factors influencing pig health, herd size is considered to be an important correlate of disease and has been studied since the early 1970s (Bäckström, 1973; Lindqvist, 1974; Aalund *et al*., 1976). However, the biological basis for the herd-size effect (whether positive or negative) is not frequently known or specified in published studies.

In most epidemiological studies, some measure of herd size can be determined and this is often used as a surrogate for management variables that are herd size-related, such as all-in, all-out and multisite production, biosecurity and vaccination practices, and the use of hired employees (United States Department of Agriculture: Animal and Plant Health Inspection Service: Veterinary Services, 1995). Although the demonstration of an association between herd size and disease might be considered interesting by epidemiologists and provide direction for future studies, such a finding has little immediate relevance to swine producers. For example, producers with large herds often realize economies of scale and are therefore unlikely to reduce herd size voluntarily because such a change would usually result in increased production costs and a lower gross margin per pig sold. In addition, large herds have greater capital investment in facilities that might also preclude them from decreasing herd size voluntarily. In some countries, however, animal welfare, environmental and political considerations may ultimately result in limits on the maximal size of swine herds. Also, public-health concerns about foodborne disease associated with pig meat will increase interest in the effects of herd size on

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pathogen prevalence because larger herds contribute the greatest number of slaughter pigs to the market.

Interest in studying herd size associations in epidemiological studies has probably developed partly because estimates of size are frequently available in existing databases or can be obtained readily by questionnaire. In many studies, however, an unanswered question is whether the primary factor that produces the herd-size effect is the size of the population at risk, management and environmental factors that are related to herd size, or some other unmeasured factor, such as herd and pig density in an area.

We believe that one goal of future epidemiological studies of swine disease is to better characterize the nature of the effect, if any, of herd size, so that potential adverse (beneficial) effects of increases in herd size can be minimized (maximized). Accordingly, it is important to differentiate real from spurious herd-size effects because a change in herd size would have no benefit if the effect was attributable to management or environmental factors that were not modified. Also, determination of the reason for any herd-size effect would allow more specific advice to be given to farmers and decision-makers on appropriate preventive measures.

In this paper, we review epidemiological studies of herd size and swine diseases, describe measures of herd size, present biological explanations for possible effects of herd size on disease occurrence, and give examples of the relationships between herd size and disease risk based on our experience. Most of our examples are studies of pleuritis, pneumonia and pseudorabies because these are the diseases that have been studied most frequently with regard to herd-size effects and positive associations have often been found. In the final section, we make recommendations for future epidemiological studies of swine diseases that involve the evaluation of the effects of herd size and management and environmental factors.

Many definitions of 'herd' are possible, reflecting physical contact, physical proximity or shared inputs (e.g. common feed source, genetics, and the source of pigs in a production pyramid). Some definitions are more appropriate to diseases transmitted by direct contact (e.g. mange) than diseases transmitted by indirect contact (e.g. *Actinobacillus pleuropneumoniae*). For this paper, we broadly define a herd to be a cluster or aggregate of pigs in a single building or multiple physically separated buildings or outdoor lots located on a single site or on multiple sites. Pigs in the same building or site can be of different age and production classes. This is similar to the definition used in a prior study by one of us (Christensen and Gardner, 2000).

Published studies and measures of herd size

We reviewed published studies of swine diseases in which herd size was evaluated as a risk factor either individually or in addition to other management and environmental factors. Our literature review was restricted to manuscripts published in English up to and including the year 2001. The search was done using the keywords 'herd size', 'herd size and disease risk', 'risk factor', 'pig management', 'management factor', 'stocking density' and 'herd density'. We read the papers to determine how herd size was measured and then categorized studies according to the body system studied and evaluated associations with larger herd size as being 'positive', 'negative' or 'none' (Table 1). These classifications were often subjective because in some papers there were no statistical analyses or description of how herd size was classified, and multivariable analyses were not used consistently. Because of these limitations, most of our evaluations were qualitative. We considered the use of meta-analysis for a more formal comparison and summary of the results into a single effect measure, but the lack of comparability of studies precluded this approach.

Our evaluation of the published papers indicated that the relationship between herd size and disease varied with the disease studied (Table 1); however, it is interesting to us that for respiratory diseases, including pseudorabies, the relationship between large herd size and disease was typically positive. However, we are unable to determine whether bias against publication of negative results might have influenced this conclusion. In contrast, we found evidence that large herd size was 'protective' for some diseases, such as toxoplasmosis and liver milk spots. Presumably, this protective effect was in part attributable to more frequent use of confinement systems in large herds, which reduced exposure to the parasites.

In the reviewed papers, herd size was measured in many different ways, although we assumed that one of the underlying goals of the authors was to use the chosen herd size measure to characterize the true population at risk for the infection or disease of interest. For diseases of sows, herd size was usually given as the number of breeding females (gilts and sows). It is important to note that the number of females may be based on the current inventory or the number of sows farrowed, and many definitions of a sow are possible. Female herd size estimates may be adjusted by excluding those not of breeding age (though already marked as replacement gilts) or cull sows that are present on a farm. For diseases in finishing pigs, the choice of a herd size measure tended to be more variable (and included the number of pigs slaughtered per annum, the number of pigs in the herd between weaning and market age, the total number of pigs in the herd, and the number of pig places per barn or building compartment). Another herd size measure used in Danish studies of possible airborne transmission of pseudorabies virus (PRV) (Christensen *et al.*, 1990) and porcine respiratory coronavirus pseudorabies virus (PRV) (Henningsen *et al.*, 1988; Flori *et al.*,

Table 1. Observational studies of swine diseases in which herd size has been evaluated as a risk factor

Disease grouping	Detection method	Herd size associations			References
		Positive	None	Negative	
Respiratory					
Pleuritis	Lesions	8	3	0	Lindqvist, 1974; Aalund *et al.*, 1976; Bäckström and Bremer, 1978, Andersen, 1981; Flesjå and Solberg, 1981; Flesjå *et al.*, 1982; Willeberg, 1984/85; Martinsson and Lundeheim, 1988[1]; Mousing *et al.*, 1990; Elbers, 1991; Maes *et al.*, 2001
Pneumonia	Lesions	8	2	0	Lindqvist, 1974; Bäckström and Bremer,1978; Willeberg, 1979; Flesjå and Solberg, 1981; Flesjå *et al.*, 1982; Pointon *et al.*, 1985; Martinsson and Lundeheim, 1988[1]; Elbers, 1991; Hurnik *et al.*, 1994[2]; Maes *et al.*, 2001
PRCV	Serology	1	0	0	Flori *et al.*, 1995[3]
PRRS	Serology	0	1	0	Mousing *et al.*, 1997
Pseudorabies	Serology/virus isolation	9	5	0	Andersen *et al.*, 1990; Christensen *et al.*, 1990; Morrison *et al.*, 1991; Duffy *et al.*, 1991a, b; Weigel *et al.*, 1992; Austin *et al.*, 1993; Siegel *et al.*, 1993[4]; Leontides *et al.*, 1994a, b; Leontides *et al.*, 1995; Boelaert *et al.*, 1999; Maes *et al.*, 1999; Maes *et al.*, 2000
Gastrointestinal					
Preweaning diarrhea	Clinical	1	2	1	Svensmark *et al.*, 1989a; Lingaas, 1991; Dewey *et al.*, 1995; Wittum *et al.*, 1995
Postweaning diarrhea	Clinical	0	0	1	Svensmark *et al.*, 1989b
Salmonella	Serology	1	1	1	Carstensen and Christensen, 1998[5]; Stege *et al.*, 2001; van der Wolf *et al.*, 2001
TGE	Serology/virus isolation	3	0	0	Pritchard, 1982; Pritchard, 1987; Yanga *et al.*, 1995
Reproductive					
Encephalomyocarditis	Serology	0	1	0	Zimmerman *et al.*, 1991
Mastitis-metritis-agalactia	Clinical	0	1	1	Bäckström *et al.*, 1984; Lingaas, 1991
Parvovirus	Antigen detection	0	1	0	Culter *et al.*, 1983
Parasitic					
Endoparasites	Egg counts	1	1	0	Roepstorff and Jorsal, 1990; Dangolla *et al.*, 1996
Liver milk spots	Lesions	0	2	1	Bäckström and Bremer, 1978; Bäckström and Bremer, 1978; Flesjå *et al.*, 1982; Martinsson and Lundeheim, 1988
Toxoplasmosis	Serology	0	0	3	Zimmerman *et al.*, 1990[6]; Assadi-Rad *et al.*, 1995; Weigel *et al.*, 1995
Other infections and conditions					
Abscesses/arthritis[7]	Lesions	2	1	0	Flesjå and Solberg, 1981; Martinsson and Lundeheim., 1988; Tuovinen *et al.*, 1992
Arthritis/lameness	Clinical	0	2	0	Lingaas, 1991; Christensen, 1996[8]
Classical swine fever	Serology/virus detection	2	0	0	Benard *et al.*, 1999; Elbers *et al.*, 2001
Sow mortality	Records/necropsy	1	1	0	Christensen *et al.*, 1995; Abiver *et al.*, 1998; Koketsu, 2000

PRRS, porcine reproductive and respiratory syndrome; TGE, transmissible gastroenteritis.
[1]Authors found no association with number of pigs delivered per year but a positive association with size of batch or barn.
[2]Herd size included as part of farm type description.
[3]Includes data previously reported by Henningsen *et al.*, 1988.
[4]Time under quarantine used as proxy for time to eradication.
[5]Subclinical infections. Significant statistical difference was not considered by authors to be biologically important.
[6]Negative association in sows only.
[7]Usually assessed together as partial or total condemnations.
[8]No consistent relationship with increasing herd size.

1995) was the number of 'heat-producing units' (HPU). The HPU (1 HPU = 1000 watts at 20°C) is an estimate of the heat loss (and hence the ventilation requirement) at different temperatures for different age classes of pigs (Strom, 1978): 1 sow and her litter = 0.4 HPU; 1 non-lactating sow, boar or gilt = 0.2 HPU; 1 grower/finisher = 0.17 HPU. This measure accounts for differences in size and weight of different age classes of pigs in farrow-to-finish herds and facilitates the comparison of sizes of breeding and finishing herds. The relative advantage of this method for accounting for pigs of different types in a single measure has not been evaluated, but such an approach seems intuitively appealing for studies of respiratory diseases transmitted by aerosols in intensively housed pigs where risk is constant across all ages. An assumption of age-independent risk might be realistic for first epidemics of many viral and bacterial respiratory agents before the risk starts to be influenced by passive and active immunity. In contrast, where transmission occurs primarily by nose-to-nose contact it would be more appropriate to give greater weight to finishing pigs in pens rather than breeding animals in crates. In the latter case, the use of HPU as a herd size measure would be inappropriate.

A discussion of herd size measures would be incomplete without reference either to herd (and pig) density in an area, which might also be positively correlated with herd size, or to pig stocking density within herds. Greater herd (and pig) density may be confounded with larger herd size at least in some countries, such as Denmark (Flori *et al*., 1995), and therefore might in part explain the reported herd-size effects. The importance of considering the density of infected herds or pig density on an area basis in addition to herd size as risk factors for pseudorabies was recognized in at least five studies (Austin *et al*., 1993; Leontides *et al*., 1994a, 1995; Boelaert *et al*., 1999; Maes *et al*., 2000). Indeed, in one study (Leontides *et al*., 1994b), the effect of larger herd size was no longer evident after related management variables and density of pigs had been included in the statistical models.

Pen and air-space stocking density have been investigated infrequently as risk factors for swine diseases, and there a number of possible reasons for this. First, pig density is difficult and tedious to measure accurately, and the value changes with the stage of production in farrow-to-finish herds. Secondly, even if a good measure exists, measurement is usually done at the pen level and herd-level analysis is complicated by the need to correct for clustering or over-dispersion. Thirdly, there is collinearity between herd size and pig density. Of the 14 unique reviewed studies of pleuritis or pneumonia lesions (Table 1; seven authors studied both lesion types), only seven (50%) evaluated a stocking density measure of herd size, yet standard recommendations for the control of pneumonia in problem herds (Done, 1991) emphasize alteration of up to 20 environmental variables, including density, air space, and pigs per building. The importance of air-space stocking density and floor-space stocking density as important predictors of respiratory health in finisher pigs has been reaffirmed in recent studies (Buddle *et al*., 1997; Stark *et al*., 1998; Maes *et al*., 2001).

Another consideration relevant to the measurement of floor and air-space density and measures of herd size warrants comment. The possible collinearity of these measures in multivariable models may have dissuaded researchers from recording more than one or two measures of herd size. In practice, the most easily acquired measure of herd size (e.g. the number of breeding females or the number of finishers slaughtered per year) is generally used and, for whatever measure is selected, authors probably assume that the variables are sufficiently highly correlated to be interchangeable. The latter assumption may not necessarily be correct. For example, we estimated pairwise correlation coefficients between three frequently used measures of herd size: the number of breeding females, the number of pigs sold per year and the total inventory of pigs for 1306 US farrow-to-finish farms studied as part of the National Swine Survey (United States Department of Agriculture: Animal and Plant Health Inspection Service: Veterinary Services, 1992). The estimates ranged from 0.20 to 0.61, which indicated only low to moderate linear relationships. Hence, multiple measures of herd size might be included in multivariable models.

Biological reasons for an effect of herd size on disease occurrence

In this section, we describe plausible biological reasons for increased risk of introduction and spread of infectious agents with increasing herd size. We note that large herds might be able to mitigate many of these increased risks by the use of management procedures (e.g. biosecurity practices, all-in, all-out production and age-segregated production) which decrease the risk of introduction of pathogens into herds or their within-herd transmission. In addition, owners of smaller herds in the USA frequently use riskier management practices, including swapping boars with neighbors, increased likelihood of attending fairs and livestock shows, and more frequent use of trucks for a single purpose, which counteract the benefits of small herd size.

Greater risk of introducing infectious agents from outside the herd

The number of direct and indirect contacts that a swine herd has with potential outside sources of infection may increase as the herds expand in size. These contacts include introduction of breeding and feeder pigs, transportation of feed and of slaughter animals, and visitors. Consequently, unless protective management practices are used as outlined above, the risk of introduction of

pathogens will also usually increase. We describe the interrelationships between management factors and herd size in more detail below (see Influence of management-related factors that are associated with herd size).

Introduction of infectious agents with carrier pigs

To demonstrate the effects of herd size, consider herds that replace a fixed proportion of the female breeding herd with gilts purchased from outside herds. Assuming a binomial distribution, Marchevsky *et al.* (1989) showed that the risk (P) of introducing at least 1 infected female is

$$P = 1 - (1 - p)^n \quad (1)$$

where n is the number of gilts introduced and p is the prevalence of the infectious agent in the source population. For example, assuming that $p = 0.01$ and no testing for the agent was done prior to the introduction, then a herd of 500 sows replacing 20% of females per annum would have a risk (P) of 0.64 of introducing an infectious agent compared with a risk of 0.18 for a herd of 100 sows with the same replacement rate. A similar herd size-related phenomenon occurs when herds are initially established from the same source without mitigations. One possible strategy for owners of larger herds to mitigate this theoretically increased risk is to purchase pigs from non-infected herds or to only introduce test-negative pigs into their herds.

Thorburn *et al.* (1991) suggested a modification of equation 1 to include sampling without replacement, and the equation could be further modified to include sampling of variable numbers of pigs from herds with different prevalences of infection. For simplicity, we do not present these modifications here. Also, to allow direct comparison with the model in the following section, we assume that, once an infectious agent is introduced into a herd with an infected pig, it is transmitted to other pigs in the same herd.

Introduction of infectious agents transmitted by airborne routes

Even for herds not introducing pigs and with limited contacts with other herds, there is empirical evidence that large swine herds are at increased risk of airborne introduction of viral diseases. For example, for infections such as PRCV and PRV (for which airborne transmission in Denmark has been reported), an increased risk of infection was evident in large herds (Henningsen *et al.*, 1988; Christensen *et al.*, 1990; Flori *et al.*, 1995). In addition, the authors of these three studies found that a natural logarithmic transformation of herd size was the best formulation of the independent effect of herd size on the odds of positive PRCV or PRV status.

On the basis of this empirical evidence and the assumption that the herd-size effect was not confounded by herd density, we proposed that the model specified for the risk of disease introduction associated with carrier pigs (equation 1) could be applied to the airborne spread of viral diseases with modification of the meanings of p and n (Willeberg *et al.*, 1994). In the context of airborne spread, P remains the probability of a herd becoming infected, p is the probability of an individual pig in the herd becoming infected if exposed to droplet nuclei containing virus of constant concentration (virus/m^3) and n is the herd size at the time of exposure to the virus (Willeberg *et al.*, 1994). One way to conceptualize the meaning of the herd-size effect is to consider that larger herds occupy a greater area of land. For example, assuming a fixed density of pigs per m^2 of floor space, a herd of 500 fattening pigs would occupy an area five times greater than a herd of 100 fattening pigs.

The probability of the herd becoming infected (P) increases non-linearly with n and p, but when p is small it can be shown that the relationship is approximately linear, i.e. $P = 1 - (1 - p)^n \cong np$ (Fig. 1; e.g. when $p \leqslant 0.001$). For two herds of size n_1 and n_2 with constant risk p of infection on a per-pig basis, the odds ratio (OR) of becoming infected is the ratio of the two herd sizes, i.e. OR = n_1/n_2 (OR = 5 for the two fattening herds referred to above). In a logistic model of the ln(OR) of infection, logarithmic transformation yields

$$\ln(\text{OR}) = \ln(n_1) - \ln(n_2) \quad (2)$$

Field data are used in logistic regression analysis to model ln(OR) as a function of the herd-size variable (X) as follows:

$$\begin{aligned} \ln(\text{OR}) &= \beta X \\ &= \beta(x_1 - x_2) \end{aligned} \quad (3)$$

where x_1 and x_2 are two levels of the herd-size variable (X), measured on a continuous scale, and β is the logistic regression coefficient for a 1-unit change in herd size. We note that equation 2 is a special case of equation 3 in which $\beta = 1$ and $x_1 = \ln(n_1)$ and $x_2 = \ln(n_2)$.

Evidence in support of this simple model is provided by results of the following two analyses. Christensen *et al.* (1990) analysed data from outbreaks of pseudorabies in southern Denmark from 1987/88. Cases were pseudorabies-positive herds for which no known contact with other infected herds had occurred. Control herds were non-infected herds from the same general geographical area as the cases. In the analysis, herd size was expressed as HPU to reflect the relative metabolic sizes of the pigs. Because the distribution of HPU was positively skewed, a logarithmic transformation of HPU was used to model the effect of herd size. The final model (which adjusted for whether the herd was a conventional or SPF herd and whether the herd was a fattener or sow herd) included a statistically significant effect of ln(HPU) with β estimated as 1.26. In a subsequent paper co-authored by one of us (Mousing *et al.*, 1991), however, the control sample was restricted to 11 postal districts for which complete data were available for outbreak herds in both the 1987/88 and the 1989/90 epidemics. Here

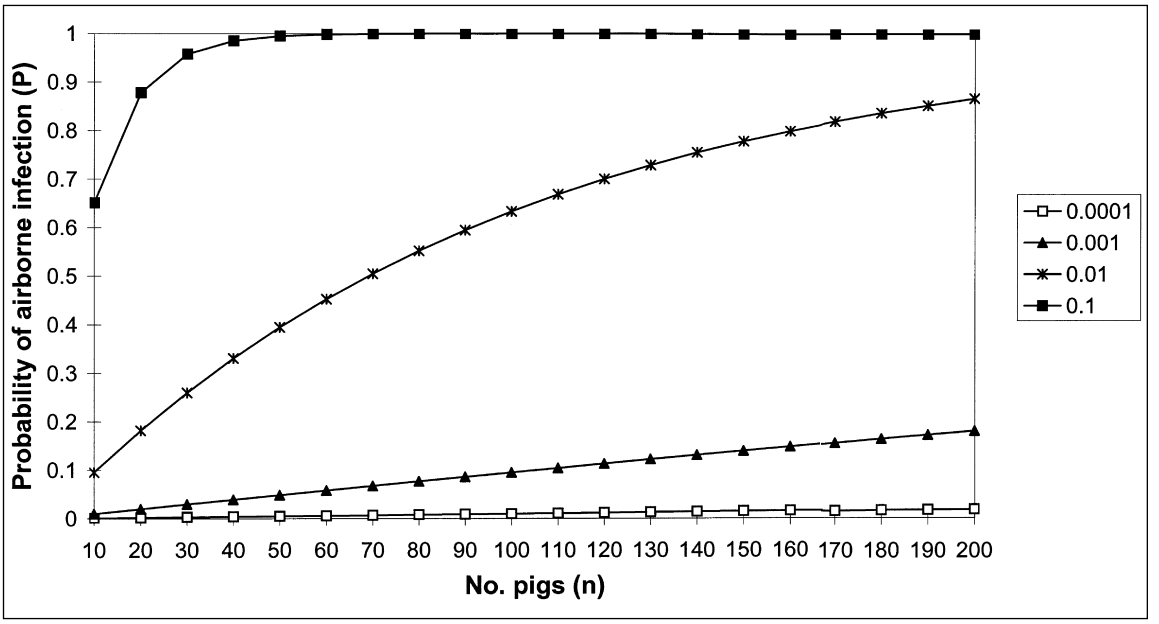

Fig. 1. Probability of airborne infection of swine herds (P = at least 1 pig becoming infected) as a function of the number of pigs in the herd (n) and the probability of an individual pig becoming infected (p). Calculations are based on equation 1 in the text: $P = 1 - (1 - p)^n$.

also, only fattener/sow management was accounted for, and the β estimate for ln(HPU) was 0.86. Leontides *et al.* (1994b) studied herd risk factors for PRV infection of breeding sows in northern Germany and estimated β for ln(herd size) to be 0.89 after controlling for possible introduction of infection with carrier pigs (purchase of replacement gilts versus use of own inventory). In a study of risk factors for herd-level PRCV seropositivity, Flori *et al.* (1995) analysed data from a subset of 125 swine herds with a seropositive neighboring herd and found a ln(HPU of own herd) estimate of 0.97 for PRCV infection.

For these examples of probable among-herd airborne transmission, we speculate that the agreement between observed and expected data [i.e. β estimates for ln(HPU) close to 1] may be explained by the fact that airborne transmission of the virus closely follows assumptions inherent in the model: equal, independent and low risk of initial infection of individuals. In our opinion, if the model were inappropriate or confounding or other biases were present in the data, then one would expect to find β estimates substantially different from 1.

The use of equation 1 for a probabilistic assessment of virus airborne transmission has also been proposed by Manuel-León and Casal (2001) to explain patterns of herd infection with pseudorabies in 1981/82 in Yorkshire, England.

Greater risk of transmission of infectious agents within and among herds

Factors that affect the transmission of infectious agents among individuals have been described by Koopman and Longini (1994). Briefly, these factors are the infectiousness of infected individuals, the quantity and quality of the agent transferred by the different routes of transmission, the number of contacts per unit time (for the different routes), the susceptibility of non-infected individuals, and the number of different individuals for which there is contact. Analogous factors affect the transmission of pathogens among herds: infectiousness of infected herds, the susceptibility of non-infected herds, the amount of viable pathogen that is transferred during a contact, the rate at which contacts occur and the number of herds that make contact (Stegeman *et al.*, 1999).

To help explain and predict disease patterns, many models of disease transmission have been proposed. For brevity we mention only one: the Reed–Frost model (Fox *et al.*, 1971; Frost, 1976; Yorke *et al.*, 1979; Anderson and May, 1982). The Reed–Frost model predicts that the effective contact rate and the number of susceptible and infected pigs will affect the spread of an agent in a herd:

$$C_{t+T} = S_t(1 - q^{Ct}) \quad (4)$$

where C_t is the number of cases at time t, S_t is the number of susceptible pigs at time t, and q is the probability that a susceptible pig will not become infected during the next time period of duration T. This equation indicates that the occurrence of an epidemic is largely a function of the number of susceptible pigs and the contact rate. The number of susceptible pigs is usually greater in large herds unless their risk of infection is reduced by vaccination. Moreover, the probability of an effective contact might also be greater because of hous-

ing in common areas or air spaces, and the increased likelihood of direct and indirect contact with susceptible, non-immune pigs. Direct contacts between pigs in the same space often increases with increasing stocking density. In the Reed–Frost model, changes in these factors will result in different patterns of incident cases with time (Fig. 2) and in the total number of affected pigs before a given age or at slaughter (as is evident by comparing Fig. 2a and b).

Although q in equation 4 could be independent of population or herd size, a herd-size effect implies that herd size (denoted N) should be incorporated explicitly in the model. Accordingly, De Jong (1995) has recommended the following form with a herd size-dependent transmission rate (SI/N, where S is the number of susceptible animals, I is the number of infectious animals and N is the total number of animals; for a more detailed discussion see De Jong, 1995):

$$C_{t+T} = S_t(1 - e^{-\beta CtT/N}) \quad (5)$$

For pneumonia and pleuritis, direct comparison of empirical data with the theoretical predictions of the Reed–Frost model at an individual animal level is difficult because of complex interrelationships among host, agent and environment factors, the involvement of multiple etiological agents, and because prevalence rather than incidence is the usual outcome measure in studies of slaughter pigs. The prevalence of pneumonic and pleuritic lesions in slaughter pigs is determined by the incidence rate of lesions during the grower/finisher phase, the distribution of age at infection and the rate of lesion resolution. There is evidence that many pneumonic lesions heal partly or completely before slaughter; resolution of chronic pleuritis lesions is more limited (Christensen and Mousing, 1992).

For PRV, evaluation of the effects of herd size on within-herd transmission is easier because a single infectious agent is involved and pigs remain chronically infected (or at least serologically positive for life). Duffy *et al.* (1991a) proposed that more replacement gilts are introduced in large breeding herds than in small herds; therefore, large herds will usually have more susceptible females than small herds—provided that replacement gilts are PRV-negative at introduction. A deterministic mathematical model of PRV showed also that the most important determinants of viral persistence were herd size and the density at which sows were maintained. A threshold herd size of 66 sows was identified, below which virus would be eliminated from the herd even when no specific control measures were implemented (Smith and Grenfell, 1990). In contrast, Bouma *et al.* (1995), using a susceptible–infectious–recovered (SIR) model, failed to show that the PRV transmission in groups of 10 and 40 vaccinated pigs was dependent on population size.

Influence of management-related factors that are associated with herd size

In swine herds, management and environmental factors are interrelated and often associated with herd size. Large herds are different from small herds in many ways other than merely the number of pigs or stocking density. Thus, any herd-size effect (positive or negative) is often mixed with the effects of other factors which may not be recorded *per se* but which are associated with herd size. These factors vary from herd to herd and from country to

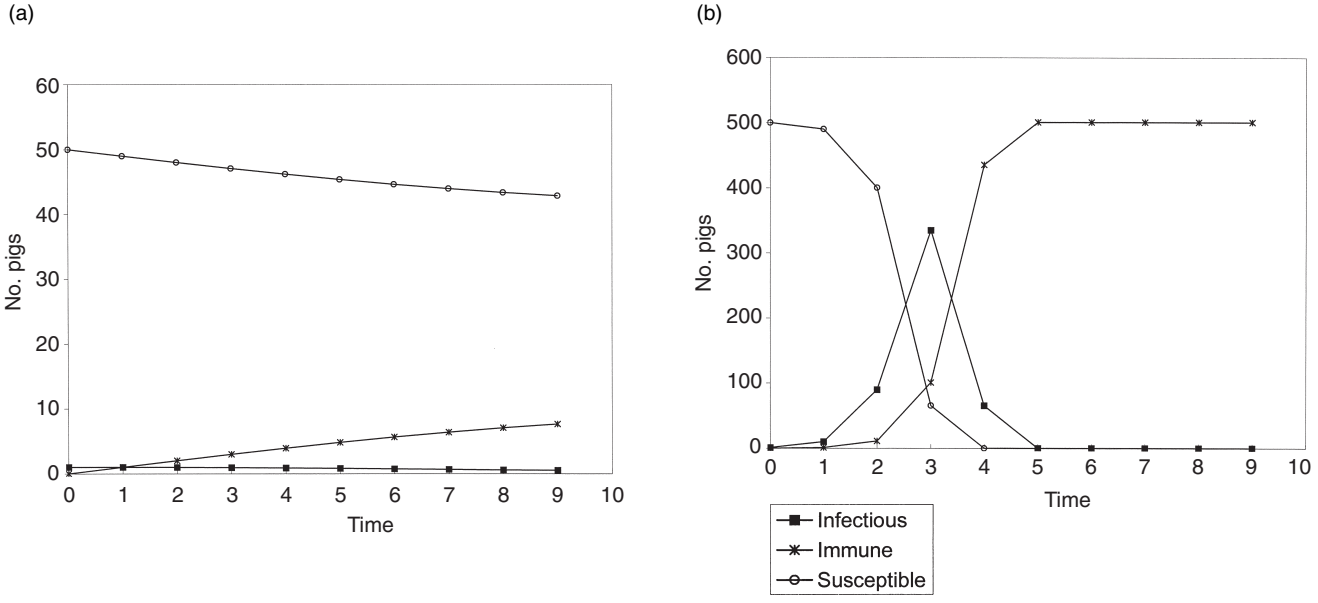

Fig. 2. Reed–Frost model estimates of the number of cases of an infectious disease for a contact rate = 0.02 and for initial population sizes of 50 (a) and 500 susceptible pigs (b). The category 'immune' includes pigs at risk of being chronically affected.

country, but many factors, including confinement housing (Anderson *et al*., 1990), all-in, all-out and multisite production systems, automation and the use of hired workers, probably occur more frequently in large herds, while others, such as the purchase of pigs for finishing, are more frequent in small herds in some countries (Pointon *et al*., 1985). Specialized large herds often incorporate improved environmental features in new buildings, including small compartments (Elbers, 1991), but there are few recent population-based studies which have described the relative frequency of practices in herds of different size or in herds with buildings of different ages.

Disease-control practices such as vaccination and medication policies are also likely to differ by herd size (Svensmark *et al*., 1989a, b; Siegel and Weigel, 1999). In the USA, for example, the use of vaccines to control reproductive and neonatal diseases increases with increasing number of breeding females in the herd (United States Department of Agriculture; Animal and Plant Inspection Service; Veterinary Services, 1995). If large herds use vaccine more frequently than small herds and vaccination is effective, then vaccination might counteract any increased risk of transmission in large herds. Although vaccination can alter both the susceptibility of non-infected pigs and the shedding of pathogens by infected pigs, the assumption that vaccines for all diseases are effective is inappropriate. For example, vaccines for agents such as *Actinobacillus pleuropneumoniae* have often failed to be protective (Hunneman, 1986).

The skill and experience of people interacting with the pigs can influence neonatal mortality, reproductive performance and other productivity measures (Wilson *et al*., 1986). Whether there is also an influence on infectious disease has not been demonstrated clearly, but the astute herdsperson or manager may be capable of early detection and intervention in clinical illness and potentially influence the course of disease. Such an influence might be reflected in a decreased frequency and severity of lesions at slaughter. The skill and knowledge of the herdsperson or manager is especially difficult to quantify in an objective manner and, hence, this effect usually will be included with any residual herd effect.

Substantial scope probably exists to identify important management variables that influence disease occurrence in large herds. Willeberg (1979) and Martinsson and Lundeheim (1988) showed that for pleuritis lesions at slaughter, prevalence increased with the number of pigs slaughtered per year, but there were some large herds that had prevalences similar to those in small herds. Explanations for these differences among large herds were not determined but were probably related to management.

During test-and-removal programs for diseases, time to eradicate infection (approximated by time under quarantine) could be greater for large herds even if no spread of infection occurs during the eradication process. This might be attributable to management and housing factors, a higher initial prevalence of infection, or to the use of tests with a sensitivity of less than 100% (discussed in the following section). For example, Siegel *et al*. (1993) studied factors associated with time under quarantine for swine herds in the voluntary phase of pseudorabies eradication in Illinois, USA. Larger herds (>80 versus 30–80 sows) had an increased quarantine time; however, large herd size, initial seroprevalence, confinement housing and delay in initiating a herd clean-up plan were positively interrelated. After inclusion of all factors in a Cox proportional hazards model, the positive association with larger herd size was reduced but was still marginally significant.

Herd size effects attributable to imperfect diagnostic tests and sampling strategies

Time to eradicate infection

When disease-eradication programs are based on tests of imperfect sensitivity, the expected number of false-negative test results is directly proportional to herd size (assuming that the entire population is tested and that test sensitivity and prevalence are constant from herd to herd) (Martin *et al*., 1992). Assuming that a herd with n pigs with a prevalence of infection p is tested with a test of sensitivity s, the expected number E of infected pigs that will test negative is

$$E = np(1 - s) \tag{6}$$

For example, a test of 90% sensitivity used in a herd of 100 pigs with a prevalence of infection of 10% would be expected to yield one false-negative test result, while in a herd of 1000 pigs with the same prevalence it would yield 10 false-negative results. Usually, pigs testing negative on the first test in an infected herd are retested again until a predetermined number of negative herd tests are recorded that provide sufficient evidence that the herd is free of infection. The number of rounds of testing to eliminate infection should therefore depend on herd size, the correlation of the test–retest results of pigs that initially tested negative yet were truly infected, and the transmission rate of the pathogen between infectious and susceptible pigs. Similar considerations apply when combinations of tests or pooled tests are used (Christensen and Gardner, 2000). In the study by Siegel *et al*. (1993), only nine of 164 PRV-infected herds were on a test-and-removal program, so we are unable to determine whether this theoretical consideration truly applied to the study.

Identification of the herd for intervention

On the other hand, because herd-level sensitivity (the probability that an infected herd will test positive, usually meaning that at least one pig in the herd tests

positive) is strongly determined by sample size, a larger infected herd would be more likely to be identified for an intervention if a fixed proportion of the herd (rather than a fixed number per herd) were tested (assuming other determinants of herd-level sensitivity were held constant) (Martin *et al.*, 1992).

Examples of the relationship between herd size and disease risk

Herd size might be non-causally (spuriously) associated with disease occurrence, because herd size and management systems are interrelated and management factors influence disease occurrence. In this section, we give some examples, based mainly on our experience in Danish swine production, to demonstrate how herd size might influence disease risk independently or in association with other management risk factors. We note that most of the studies are from Denmark, where the swine population is more homogeneous with respect to management factors than in many other countries. Hence, caution must be used in extrapolating findings from Danish studies of herd size and disease risk to production systems in other countries.

Confounding of management variable associations by herd size

Confounding occurs when the estimate of the effect of a factor of interest is in part attributable to other risk factor(s) for the disease. The extraneous risk factor must be associated with the exposure and also be an independent risk factor for disease. The net effect of failure to account for confounding is that the risk estimate (e.g. odds ratio, risk ratio) for the factor of interest is over- or underestimated. Statistical methods such as stratified analyses or multivariable modeling can be used to adjust risk estimates for confounding variables so that the estimates are unbiased. Detailed descriptions of criteria for confounding are covered in epidemiological texts.

To demonstrate confounding by herd size, we reanalysed data from pseudorabies outbreaks in 1987/88 and 1989/90 from 11 districts in southern Jutland adjacent to the Danish–German border to estimate the risk of pseudorabies according to herd health status [specific pathogen-free (SPF) or conventional]. Possible airborne transmission of PRV was evaluated by restricting analysis to those herds which neither purchased pigs from nor had other known contacts with infected herds. Case herds were those which had positive virus isolation or at least one seropositive pig, and controls were seronegative herds located in the same geographical area as the case herds. Approximately five control herds were sampled for each case herd. Three hundred and forty herds (58 cases, 282 controls) had complete data and were used in the subsequent analysis. Crude analysis indicated a higher risk (odds ratio = 2.75) of PRV outbreaks in conventional herds than in SPF herds. Because herd size was also strongly positively associated with pseudorabies risk and conventional herds (mean = 225 pigs) were significantly ($P < 0.0001$) smaller than SPF herds (mean = 484 pigs), adjustment for herd size in a logistic regression model increased the odds ratio for herd type (conventional versus SPF) to 10.8. In this analysis, the magnitude of the conventional herd effect was about four times greater than that reported by Christensen *et al.* (1990). We attribute this difference primarily to the different methods of selecting control herds.

Effect modification between herd size and management variables

Effect modification (also termed 'interaction' when used in a statistical context) occurs when the joint effect of two variables on disease risk is greater or less than that predicted from their individual effects. Interaction can be assessed on an additive or multiplicative scale depending on how the disease outcome is modeled. One manifestation of interaction is a difference in stratum-specific odds ratios, with the crude odds ratio intermediate between the values for the different categories (strata) of the risk factor.

We present two examples to demonstrate that interaction between herd size and management factors is possible. Aalund *et al.* (1976) showed that, in large herds (>400 fatteners slaughtered per year), a herd history of diarrhea seemed to protect (odds ratio = 0.6) against chronic pleuritis, whereas in small herds a history of diarrhea contributed to a two-fold increase in the odds of pleuritis. This difference in stratum-specific estimates was considered evidence of interaction between a herd history of diarrhea and herd size, and the odds of chronic pleuritis. Moreover, this interaction was independent of whether the pigs were purchased or raised in the study herd. Pointon *et al.* (1985) found that factors associated with high prevalences of pneumonia were different in herds of 20–70 sows (sow culling rate, purchase of pigs for finishing, pigs per shed section, and pigs per group) compared with herds of >100 sows (pen stocking rate and air-space stocking rate). Stratified analyses were not done by the authors to determine whether confounding and/or interaction with herd size occurred or whether the management factors were interrelated, but their findings confirm that the impact of environmental and management factors often depends on herd size.

Management and infectious agent associations that are dependent on herd size

In Denmark, complex interrelationships exist between herd size, health status (SPF or conventional), the occur-

rence of infectious agents, and production system (fattening herd or farrow-to-finish herd). The data used by Mousing *et al.* (1990) were reanalysed to demonstrate these interrelationships and to show that a residual herd size association can exist after controlling for management variables. Although few management factors were evaluated, the study was unique because it considered exposure to known infectious causes of pleuritis as risk factors for slaughter lesions. A subset of 2718 records [no missing values on any variables and no history of vaccination against *A. pleuropneumoniae* serotype 2 (AP2)] were reanalysed to demonstrate how the incorporation of serological data on exposure to infectious agents (specifically AP2) may explain at least part of the herd-size effect. For the analysis, herd sizes were grouped in quartiles [<620 (reference category), 620–1200, 1201–2000 and >2000 pigs slaughtered per year]. In bivariable analysis, seropositivity to AP2 increased the odds of pleuritis 9.9 times and the odds ratio for pleuritis was greater than 1 only for the largest herd-size quartile (odds ratio = 2.4). After inclusion of AP2 serostatus in the multiple logistic regression analysis, the odds of pleuritis were substantially reduced in the largest herds (odds ratio = 1.4), although the confidence interval for the estimate still excluded 1. Estimates for the two intermediate herd size categories were essentially unchanged. The residual effect of the largest herd size category could also not be explained by other factors examined (herd type and exposure to other known infectious causes of pleuritis), but since only a few factors were evaluated it is possible that unmeasured management factors were at least part of the explanation.

Spurious associations between herd size and disease

For some diseases, an apparent effect of herd size may be adequately explained when other important risk factors, such as herd density, pig density and management factors, are included in analyses. Leontides *et al.* (1994b) studied factors associated with PRV circulation in fattening herds in an intensively vaccinated area of northern Germany. Herd size increased the risk of PRV (odds ratio = 1.69 for a 100-pig increase in herd size) in crude analyses, but when the related demographic and management variables [farrow-to-finish or feeder-to-finish (herd type) and pigs/km^2] were included in logistic regression models, the herd size association was no longer significant (odds ratio = 1.02).

Recommendations for future herd size studies

In the last decade there have been substantial changes in swine production internationally, including the increased implementation of all-in, all-out, multisite and age-segregated production systems, and greater use of compartments for finishing pigs to reduce pathogen transmission. Many of these changes can be implemented more readily in large herds, and hence the historic positive relationships between larger herd size and disease risk may no longer exist.

Although many of the published studies and data sets that we reanalysed involved Danish swine production, the underlying issues are widely applicable to other countries and production systems. Accordingly, we believe that more structured approaches are necessary to determine how herd size, herd density, pig stocking density and other management and environmental factors affect disease risk. For disease control decisions, it is especially important to determine whether any herd-size effect is real or spurious so that appropriate advice about mitigations can be given.

To ensure the greatest utility of findings from future studies on the relationship between herd size and disease, we make the following recommendations.

(i) Herd size should be measured in ways that best characterize the population at risk for the disease or productivity measure of interest. For infectious agents, this would ideally be the number of susceptible pigs, but in practice only the total number of pigs and number per building or compartment will probably be available. In addition, we recommend measurement of pig density (in terms of floor and air space occupied) especially for diseases of weaned and grower/finisher pigs. Results of analyses of the herd-size associations should include evaluation of the joint and individual contributions of each type of herd size measure. Because standards for stocking density and air space are well accepted, relatively stable and less subject to arbitrary categorization than other herd size measures, we strongly recommend their evaluation in all studies of infectious disease. Also, improved characterization of herd size will help remove the rather arbitrary classification of herds as 'large' and allow better comparison of results among studies in different countries.

(ii) Studies that evaluate management-related risk factors at the level of herd, building or compartment should consider herd size as a potential confounder, unless there is adequate prior evidence to suggest that herd size is not a causal factor. If herd size is used as a surrogate for one or more unmeasured management risk factors, it is important to be aware that residual confounding may still exist after adjustment for herd size, unless herd size is a perfect correlate for these variables (Greenland and Neutra, 1980), a situation that we believe is most unlikely in swine production systems. In addition, the relationship between herd size and herd (and pig) density in an area should be clari-

fied since these two variables are often associated.

(iii) Population-based studies of the interrelationships among management factors and between management factors, herd size, herd density and pig density are needed. The availability of multivariable statistical techniques provides a powerful tool to better define these relationships, although no simple relationship exists between forms of the selected statistical models and the underlying biological processes (Greenland, 1989). Statistical techniques to deal with a large number of independent variables in epidemiological studies have been reviewed recently by Dohoo *et al*. (1997).

(iv) Likely biological reasons for any herd size association should be postulated. Moreover, there should be adequate discussion of the potential biases and limitations of the study that affect the causal interpretation of a positive or negative association or no association between herd size and disease risk (Savitz, 1992). Such candor should also facilitate improvements in study design and lead to a more critical assessment of the likelihood of successful interventions.

(v) Whenever the herd size distribution of study herds differs from that of the source population, authors should report at least the median herd size (and ideally the distribution of herd size) for the country or region of the study. For risk calculations, deviations from the median herd size (continuous variable) or the use of the category with the population median herd size as the reference group should be used as a standard way to report herd size associations. Such an approach will account for shifts in herd size distribution with time and the likely disappearance of herds smaller than a certain minimum herd size.

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This book, in three volumes, presents a detailed revision of the systematics and taxonomy of the platyhelminth class Trematoda, subclasses Aspidogastrea and Digenea, with keys for the identification of these parasites at the superfamily, family, subfamily and generic levels.

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V Porter, *Liphook, Hants, UK*

March 2002 400 pages Hardback
ISBN 0 85199 430 X
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This is a new edition of a standard reference text, and contains key breed information about cattle, sheep, pigs, goats, horses, asses and buffalo. It contains approximately 9000 entries and cross-references on breeds, sub-breeds, types, varieties, strains and lines of these species. The aim is to include all the livestock names that may be encountered in international literature.

Each entry includes the current recommended English name, region or country of origin, notes on usage, followed by a brief physical description of the breed, in terms of colour/markings, horns, and coat type. There is a description of the relationship with other breeds or types, and historical notes about the origin of the breed, plus details of breed societies, and herdbooks where relevant. Finally, foreign names and synonyms are listed, to aid identification.

The book has been thoroughly updated since the 4th edition was published in 1996. Revisions have been undertaken by Valerie Porter, a well-respected author of more popular works on animal breeds.

Review of the 4th Edition:

Ian Mason, the author, has made a prodigious effort to keep this dictionary up-to-date in an era when synthetic breeds are appearing with great frequency...The publication is a useful reference for any veterinarian interested in the development and history of livestock breeds. It should also be in all reference libraries which aim to serve veterinary and livestock interests.

(W J Pryor, Australian Veterinary Journal)

Selected entries

Ass
- Austro-Hungarian albino
- burro
- Common Spanish

Buffalo
- African buffalo
- Burmese

Cattle
- Aberdeen-Angus
- Charolais
- Galician Blond
- Humpless shorthorns
- Russian Black Pied

Goat
- Cashgora
- Gobi Wool Goat
- Moroccan Black
- Zinder Brown

Horse
- Arab
- Exmoor Pony
- Half Saddlebred
- Przewalski horse
- Swedish Warmblood

Pig
- Duroc
- Froxfield Pygmy
- Konstantinovo
- Penzhou Mountain
- Sulawesi warty pig

Sheep
- Barbados Black Belly
- Canary Woolless
- Carpetmaster
- Four-horned
- Manchega

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