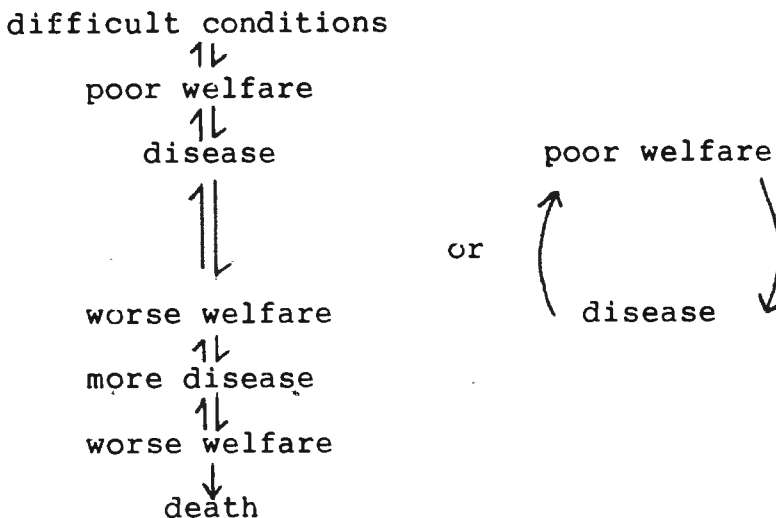


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RELATIONSHIP BETWEEN WELFARE & DISEASE SUSCEPTIBILITY IN FARM ANIMALS

The general theme of this paper is that if the welfare of an animal is poor, the chances that it will be susceptible to disease are often increased. If such a relationship between welfare and disease susceptibility exists, then it is important both to those attempting to reduce disease and to those looking for scientific indicators of welfare. Where the term welfare means the state of the individual with regard to its attempts to cope with its environment (Broom 1986), the incidence of disease might often be reduced by improving welfare and an inadequate response to disease challenge might imply that welfare is poor. The relationship would also account for the downward spiral towards death which has often been described for individuals which are initially affected mildly by disease or difficult conditions. Since disease itself usually means that welfare is poor, a positive feedback loop can be initiated with the sequence below (Table 1).

Table 1. The interaction between poor welfare and disease over time:



The sequence could, of course, start with an infectious disease which attacks an individual whose welfare is not poor until that time.

The evidence presented in this paper is of three kinds, each of which will be discussed in turn. Firstly, clinical data concerning which individuals show signs of disease. Secondly, experimental studies and surveys which compare levels of disease incidence in different husbandry systems or after different treatments. Thirdly, studies of immune system function after different treatments.

Which individuals show signs of disease?

Every veterinary clinician can give examples of situations in which a number of animals live in apparently similar conditions but only one or two show signs of disease, or most show signs of disease but only one or two died. The individuals which are affected more by disease are those which, using physical or behavioural signs, had looked weaker and less well able to cope with the environment, for example in calves (Morisse 1982). In group housing, the more susceptible animals are often those which are obviously at the bottom of a social hierarchy with the consequence that they are chased a lot, injured by others, excluded from favoured places and sometimes prevented from obtaining an adequate diet. There is little well documented scientific evidence concerning this effect in farm animals but much clinical evidence suggests that research is desirable in this area. For example, what is the reason why runt piglets are more likely to develop chronic enteritis than their larger siblings? Studies of man and of laboratory animals have concentrated much more on the question of why it is that certain individuals succumb to disease whilst others do not.

Effects of housing system or treatment on disease incidence

Some housing systems for farm animals or treatments such as handling, transport or farm operations lead to more welfare problems than do others. Hence there is the possibility of relating variation in welfare to variation in disease incidence. In some studies, an experimental treatment can be related directly to disease effects, for example Pasteur (Nichol 1974) found that chickens whose legs were immersed in cold water became more susceptible to anthrax. In other studies, it is noticed that changes in husbandry methods are associated with changes in disease incidence, for example Sainsbury (1974) reported a gradual increase in chronic infections of poultry over a period when the frequency of intensive production practices was increasing. Direct comparisons of disease incidence levels in different housing systems are also possible but any apparent relationship between poor welfare and disease incidence must be interpreted with care as other factors which vary with conditions may affect disease incidence. Ekesbo (1981) has emphasised that environmentally evoked diseases are caused by a combination of factors. In a discussion of the aetiology of clinically manifested pig herd diseases, e.g. abscesses in sows or piglets, Ekesbo lists the following factors which contribute to subclinical herd disease and interact in the production of clinical disease states:

Table 2.**Factors contributing to subclinical herd disease (e.g. abscesses) in sows and piglets (after Ekesbo 1981)**

- Large herd or group size
- Slatted floor in pen
- Rough concrete floor surface
- Unsuitable pen devices
- Sows kept confined
- No straw
- Infection

Each of these factors may, to a greater or lesser extent, lead to poor welfare and the effect on the manifestation of disease may be mediated in this way. The multifactorial causation of many farm animal diseases is also clearly stated by Vannier et al (1983).

Evidence for the effects on disease incidence of some of the factors listed by Ekesbo is provided by large scale surveys of sow and piglet disease such as that of Backstrom (1973). For example, Backstrom showed that piglet health was better if straw bedding was provided than if there was no bedding and sows on partly slatted floors had more foot lesions (6.3%) than did sows on solid floors (3.3%, $p < 0.001$). Sows confined in a stall or tether during pregnancy had more illness at farrowing than did sows which were free to move around (Table 3.)

Table 3. Sow morbidity at farrowing in relation to housing conditions (after Backstrom 1973)

Sow housing during pregnancy and farrowing	Free to walk	Confined in crate	
Number of sows	654.0	1283.0	
% mastitis metritisagalactia	6.7	11.2	$p < 0.01$
% >8h farrowing time	2.3	5.4	$p < 0.01$
% total morbidity	12.8	24.1	$p < 0.001$
% veterinary treatment	10.41	16.8	$p < 0.001$

More recent survey work on sow diseases has included that of Tillon and Madec. Madec (1982) and Tillon and Madec (1984) found that urinary infections are the major cause of death in sows and that mastitis-melovitis-agalactia is related to urinary infection. Of 3% of sows which died in tether houses, 58.3% had serious urinary tract lesions whilst 31% had respiratory problems leading to pulmonary oedema. In a survey of urine from sows in a tether house, most had bacteria in the urine and 13.5% had a significant number of bacteria, 10^6 ml⁻¹ (Madec 1984). Locomotory disorders in some sows occurred on 64% of farm and in one quarter of tethered sow units more than 20% of the animals were lame (Tillon and Madec 1984). An abattoir survey of previously confined sows showed that 80% had pododermatitis and 40% had cracked hooves (Le Denmat, Saulnier and Le Meur 1982). The incidences of these diseases are higher on these tethered sow units than would be expected on units where sows are housed in groups but, as Tillon and Madec point out, the tether units differ from other units in being enclosed, in the absence of straw and in their larger size as well as in the use of tethers. In these studies and in that of Backstrom it is not possible to be certain which of the many factors which vary are important in leading to increased disease incidence. The likelihood exists, however, that poor welfare which is a consequence of the housing conditions, is making the pigs more susceptible to disease.

Relatively brief exposure to adverse conditions can affect the animal in such a way that resistance to disease is reduced. Armstrong and Cline (1977) reported that if three-week old weaned piglets were exposed to cold air there was an increase in the incidence of diarrhoea following Escherichia coli challenge. Similarly, Shimizu, Shimizu and Kodama (1978) demonstrated that a sudden drop in ambient temperature, either before or after transmissible gastroenteritis virus inoculation, induced severe disease in those fattening pigs exposed to the virus. The effects of cold appear to reduce the ability of the pig to combat the pathogens. Many other studies of this kind have been carried out (Kelley 1983, Siegel 1985).

The most detailed and precisely controlled experiments comparing the effects of disease challenge on farm animals subjected to different treatments have been carried out by Gross, Colmano and P. B. Siegel. In this series of studies, chickens treated in ways which increased plasma corticosterone levels were subjected to pathogen challenge and the relationships between the treatment and disease susceptibility elucidated. When chickens are injected by man, the injection procedure itself has an effect on the chicken, since as far as most chickens are concerned, man is a large dangerous predator. Gross and Siegel (1979) compared chickens which had been handled slowly and deliberately for 1½ - 2 minutes per day for several weeks, with chickens which had received minimal handling. When injected with standard quantities of Mycoplasma gallisepticum, regularly handled birds developed fewer lesions than unhandled birds. When chickens were introduced to strange birds they displayed, fought and showed increased adrenal cortex activity. Frequent social mixing of this kind resulted in reduced resistance to Mycoplasma gallisepticum, Newcastle disease, haemorrhagic enteritis or

Marek's disease (Gross 1962, Gross and Colmano 1965, Gross and Siegel 1981). In contrast, such social mixing led to increased resistance to E.coli and Staphylococcus aureus (Gross and Colmano 1965, Gross and Siegel 1981). When antibody activity was measured it was clear that chickens subjected to social mixing showed less activity against both viral antigens such as Marek's disease and particulate antigens such as E.coli (Gross and Siegel 1975, Thompson et al 1980). The social mixing leads to increased adrenal cortex activity and this can help in counteracting inflammatory responses. Hence the pathological effects are reduced following high adrenal activity when there is invasion by organisms like E.coli or Staphylococcus aureus which induce local or general inflammation and endotoxin formation. Where the principal means of defence against the pathogen is immunological, treatment such as social mixing, which leads to high adrenal activity and impaired immune system function, results in greater susceptibility to pathogen attack (H. S. Siegel 1985).

Effects of housing system or treatment on immune system function

A wide variety of studies have demonstrated that the efficiency of both antibody responses and cell-mediated immunity can be affected by exposing animals to difficult conditions (Kelley 1980). In young calves and piglets, immunoglobulin absorption in the absence of the mother (Selman, McEwan and Fisher 1971, Fallon 1978) and if exposed to cold (Olsen, Papasian and Ritter 1980, Blecha and Kelley 1981).

The work of Gross and collaborators on chickens, which has been quoted already, has included the assessment of antibody responses in parallel with the assessment of whether disease challenge resulted in clinical signs of disease. Another study in which an antibody response was measured was that of Metz and Oosterlee (1981) who investigated the differences between sows tethered in a farrowing crate and sows in a straw bedded pen. The antibody response to sheep red blood cells was greater, in tests on both sows and their piglets, when the sows had been kept in a straw pen.

Studies on laboratory animals have shown that cell mediated immunity can be impaired by adverse physical conditions and by experiences such as persistent losing of fights (Baillieux and Hejnen 1987). Most studies of farm animals, however, have involved assessing the effects of exposure to heat, cold or transport. Mitogenic responses to concanavalin A and phytohaemagglutinin were impaired after calves were transported (Kelley et al 1981). Exposure of calves to 35°C or -5°C resulted in reduced delayed-type hypersensitivity to protein and reduced mitogenic response to phytohaemagglutinin (Kelley et al 1982). Other studies of the effects of adverse temperature conditions are reviewed by Kelley (1983, 1985). The intermediary role of cortisol is suggested in some of these studies and also in that of Westly and Kelley (1984) who found that when young pigs were confined in a box for 2 hours on three successive days, there was an immediate increase in cortisol levels and a later reduction in response to the phytohaemagglutinin skin test.

The mechanisms of cell mediated responses to adverse conditions and consequent adrenal cortex activity are not entirely clear but theories have been advanced to explain the results described above. Siegel (1985, 1987) considers that, in chickens, increased levels of glucocorticoids, which result from treatments such as social mixing, have an effect on the immune system as follows. In lymphoid cells, corticosterone binds to specific protein receptors and the resulting complex passes from the cytoplasm into the nucleus where it alters enzyme activity and influences nucleic acid metabolism. The resulting suppression of glucose uptake and protein synthesis leads to a reduction in the cell proliferation factor, interleukin II which is produced by T-helper cells. Hence the primary immunological effect is on T-cell populations. It is important to emphasise, however, that adverse conditions can have effects on animals, perhaps including those on the immune system, which are not mediated via adrenal cortex activity. Freeman (1985) points out that in the domestic fowl, acute heat, water deprivation and Eimeria maxima infection may lead to no adrenal cortex response.

Kelley (1985) reviewing the effects of adverse temperatures on the immune system, concludes that thermal exposure can affect the function of T-cells and have little effect on antigen-specific B cells. Restraint of animals also had adverse effects on T-cell mediated immune events. The mode of action of gluco-corticoids on the immune system via the reduction in production of interleukin II, as proposed by Siegel (1985), is generally supported but the role of B-endorphin as a modulator of cell mediated immunity is also emphasised. The complex interactions between psychological events and immune system function are reviewed by Ader (1981), Kelley (1985), and Bailleux and Heijnen (1987). In man and in laboratory animals a variety of situations in which coping mechanisms are operating, e.g. preparing for examinations, bereavement, experiencing electric shocks, or being forced to live in unstable social groups, changed cell-mediated immune responses. Some awareness of these results is essential if the causation of disease is to be appreciated and if the optimum steps needed to prevent disease are to be taken. It is also clear that changes in the immune system may be the best indicator of prolonged or intermittently high adrenal cortex activity. Given the difficulties of using adrenal responses as an adequate indicator of long-term welfare problems (Broom 1986, 1988 in press) the possibility of using an immune system indicator of welfare is important. Any treatment of animals which makes them more vulnerable to disease is bad for their welfare, even if preventive use of antibiotics can occur.

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