# Welfare, stress, behaviour and pathophysiology

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## 1. Introduction

Welfare is a term which is restricted to animals including man. It is regarded as particularly important by many people but requires strict definition if it is to be used effectively and consistently. A clearly defined concept of welfare is needed for use in precise scientific measurements, in legal documents and in public statements or discussion. If animal welfare is to be compared in different situations or evaluated in a specific situation, it must be assessed in an objective way. The assessment of welfare should be quite separate from any ethical judgement but, once an assessment is completed, it should provide information which can be used to take decisions about the ethics of a situation.

An essential criterion for a useful definition of animal welfare is that it must refer to a characteristic of the individual animal rather than something given to the animal by man. The welfare of an individual may well improve as a result of something given to it, but the thing given is not itself welfare. The loose use of welfare with reference to payments to poor people is irrelevant to the scientific or legal meaning. However, it is accurate to refer to changes in the welfare of an initially hungry person who uses a payment to obtain food and then eats the food. We can use the word welfare in relation to a person, as above, or an animal which is wild or is captive on a farm, in a zoo, in a laboratory, or in a human home. Effects on welfare which can be described include those of disease, injury, starvation, beneficial stimulation, social interactions, housing conditions, deliberate ill treatment, human handling, transport, laboratory procedures, various mutilations, veterinary treatment or genetic change by conventional breeding or genetic engineering.

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We have to define welfare in such a way that it can be readily related to other concepts such as: needs, freedoms, happiness, coping, control, predictability, feelings, suffering, pain, anxiety, fear, boredom, stress and health.

## 2. Welfare definition

If, at some particular time, an individual has no problems to deal with, that individual is likely to be in a good state including good feelings and indicated by body physiology, brain state and behaviour. Another individual may face problems in life which are such that it is unable to cope with them. Coping implies having control of mental and bodily stability and prolonged failure to cope results in failure to grow, failure to reproduce or death. A third individual might face problems but, using its array of coping mechanisms, be able to cope but only with difficulty. The second and third individuals are likely to show some direct signs of their potential failure to cope or difficulty in coping and they are also likely to have had bad feelings associated with their situations. **The welfare of an individual is its state as regards its attempts to cope with its environment** (Broom, 1986). This definition refers to a characteristic of the individual at the time. The origin of the concept is how well the individual is faring or travelling through life and the definition refers to state at a particular time (for further discussion, see Broom, 1991a,b, 1993, 1996a; Broom & Johnson, 1993). The concept refers to the state of the individual on a scale from very good to very poor. This is a measurable state and any measurement should be independent of ethical considerations. When considering how to assess the welfare of an individual, it is necessary to start with knowledge of the biology of the animal. The state may be good or poor, however, in either case, in addition to direct measures of the state, attempts should be made to measure those feelings which are a part of the state of the individual.

This definition of welfare has several implications (Broom and Johnson 1993), some of which are discussed in more detail later.

1.  Welfare is a characteristic of an animal, not something given to it. In recent American usage, welfare can refer to a service or other resource given to an individual, but that is entirely different from this scientific usage. Human action may improve animal welfare, but an action or resource provided should not be referred to as welfare.
2.  If welfare were viewed as an absolute state which either existed or did not exist then the concept of welfare would be of little use when discussing the effects on individuals of various conditions in life or of potentially harmful or beneficial procedures. It is essential that the concept be defined in such a way that welfare is amenable to measurement. Once the possibility of measurement is accepted, welfare has to vary over a range. If there is a scale of welfare and the welfare of

an individual might improve on this scale, it must also be possible for it to go down the scale. There are many scientists assessing the welfare of animals who accept that welfare can get better or can get poorer. It is therefore illogical to try to use welfare as an absolute state or to limit the term to the good end of the scale. Welfare can be poor as well as good.

Good welfare with associated pleasure or happiness, is an essential part of the welfare concept but the view of welfare as referring only to something good or "conducive to a good or preferable life" (Tannenbaum, 1991) is not tenable if the concept is to be practically and scientifically useful. Fraser (1993), referring to well-being as the state of the animal, advocates assessing it in terms of level of biological functioning such as injury or malnutrition, extent of suffering and amount of positive experience. However, despite using well-being to refer to scales of how good the animal's condition is, some of his statements explaining well-being imply only a good state of the animal, a limitation which is neither logical nor desirable.

3.  Welfare can be measured in a scientific way that is independent of moral considerations. Welfare measurements should be based on a knowledge of the biology of the species and, in particular, on what is known of the methods used by animals to try to cope with difficulties and of signs that coping attempts are failing. The measurement and its interpretation should be objective. Once the welfare has been described, moral decisions can be taken.

4.  An animal's welfare is poor when it is having difficulty in coping or is failing to cope. Failure to cope implies fitness reduction and hence stress. However, there are many circumstances in which welfare is poor without there being any effect on biological fitness. This occurs if, for example, animals are in pain, they feel fear, or they have difficulty controlling their interactions with their environment because of (a) frustration, (b) absence of some important stimulus, (c) insufficient stimulation, (d) overstimulation or (e) too much unpredictability (Wiepkema 1987).

    If two situations are compared, and individuals in one situation are in slight pain but those in the other situation are in severe pain, then welfare is poorer in the second situation even if the pain or its cause does not result in any long-term consequences, such as a reduction in fitness. Pain, or other effects listed above, may not affect growth, reproduction, pathology or life expectancy, but it does mean poor welfare.

5.  Fraser (1993) follows Broom (1986) and Broom & Johnson (1993) in drawing a conceptual parallel with the term "health" which is encompassed within the term welfare. Like welfare, health can refer to a range of states and can be qualified as either "good" or "poor".

6. Animals may use a variety of methods when trying to cope, and there are various consequences of failure to cope. Any one of a variety of measurements can therefore indicate that welfare is poor, and the fact that a measure, such as growth, is normal does not mean that welfare is good.

7. Pain and suffering are important aspects of poor welfare. Pain is a sensation which is very aversive and suffering is an array of unpleasant subjective feelings which are also aversive and avoided where possible. Even though some pain and suffering may be tolerated in order that some important objective be attained, both of these involve increased difficulty in coping with the environment and hence poorer welfare. The relationship between welfare and feelings is considered again later in this chapter.

8. Welfare is affected by what freedoms are given to individuals and by the needs of individuals, but it is not necessary to refer to these concepts when specifying welfare.

The term "well-being" is often used interchangeably with "welfare", but well-being is often used in a looser, less precise way. Welfare is the word used in English versions of modern European legislation.

### 3. Welfare and feelings

The feelings of an animal are an extremely important part of its welfare (Broom 1991b) . Suffering is a negative unpleasant feeling which should be recognised and prevented wherever possible. However, whilst we have many measures which give us some information about injury, disease and both behavioural and physiological attempts to cope with the individual's environment, fewer studies tell us about the feelings of the animal. Information can be obtained about feelings using preference studies and other information giving indirect information about feelings can be obtained from studies of physiological and behavioural responses of animals.

As discussed above, feelings are aspects of an individual's biology which must have evolved to help in survival (Broom 1998), just as aspects of anatomy, physiology and behaviour have evolved. They are used in order to maximise its fitness, often by helping it to cope with its environment. It is also possible, as with any other aspect of the biology of an individual, that some feelings do not confer any advantage on the animal but are epiphenomena of neural activity (Broom & Johnson, 1993). The coping systems used by animals operate on different time scales. Some must operate during a few seconds in order to be effectual, others take hours or months. Optimal decision-making depends not only on an evaluation of energetic costs and benefits but on the urgency of action, in other words the costs associated with injury, death or failure to find a mate (Broom 1981, p.80). In the fastest acting urgent coping

responses, such as avoidance of predator attack or risk of immediate injury, fear and pain play an important role. In longer time-scale coping procedures, where various risks to the fitness of the individual are involved, feelings rather than just intellectual calculations are amongst the causal factors affecting what decisions are taken. In attempts to deal with very long-term problems which may harm the individual, aspects of suffering contribute significantly to how the individual tries to cope. In the organisation of behaviour so as to achieve important objectives, pleasurable feelings and the expectation that these will occur have a substantial influence. The general hypothesis advanced is that whenever a situation exists where decisions are taken which have a big effect on the survival or potential reproductive output of the individual, it is likely that feelings will be involved. This argument applies to all animals with complex nervous systems, such as vertebrates and cephalopods, and not just to humans. Feelings are not just a minor influence on coping systems, they are a very important part of them.

In circumstances where individuals are starting to lose control and fail to cope, feelings may exist. These feelings might have a role in damage limitation which is functional. However they might also occur when the individual is not coping at all and the feelings have no survival function then. Extreme suffering or despair are probably not adaptive feelings but an observer of the same species might benefit and a scientist might use indications of such feelings to deduce that the animal is not coping.

If the definition of welfare were limited to the feelings of the individual as has been proposed by Duncan & Petherick (1991), it would not be possible to refer to the welfare of a person or an individual of another species which had no feelings because it was asleep, or anaesthetised, or drugged, or suffering from a disease which affects awareness. A further problem, if only feelings were considered, is that a great deal of evidence about welfare like the presence of neuromas, extreme physiological responses or various abnormalities of behaviour, immunosuppression, disease, inability to grow and reproduce, or reduced life expectancy would not be taken as evidence of poor welfare unless bad feelings could be demonstrated to be associated with them. Evidence about feelings must be considered for it is important in welfare assessment but to neglect so many other measures is illogical and harmful to the assessment of welfare, and hence to attempts to improve welfare.

In some areas of animal welfare research it is difficult to identify the subjective experiences of an animal experimentally. For example it would be difficult to assess the effects of different stunning procedures using preference tests. Disease effects are also difficult to assess using preference tests. There are also problems in interpreting

strong preferences for harmful foods or drugs.   However, research on the best housing conditions and handling procedures for animals can benefit greatly from studies of preferences which give information about the subjective experiences of animals.  Both preference studies and direct monitoring of welfare have an important role in animal welfare research.  Welfare assessment should involve a combination of studies and of other factors providing information about coping.

## 4. Welfare and stress

The word stress should be used for that part of poor welfare which involves failure to cope .  If the control systems regulating body state and responding to dangers are not able to prevent displacement of state outside the tolerable range, a situation of different biological importance is reached.   The use of the term stress should be restricted to the common public use of the word to refer to a deleterious effect on an individual (see Broom & Johnson, 1993 for more detailed information on this subject).  A definition of stress as just a stimulation or an event which elicits adrenal cortex activity is of no scientific or practical value.   A precise criterion for what is adverse for an animal is difficult to find but one indicator is whether there is, or is likely to be, an effect on biological fitness. **Stress can be defined as an environmental effect on an individual which over-taxes its control systems and reduces its fitness or seems likely to do so.** (Broom & Johnson, 1993, see also Broom 1983, Fraser & Broom 1990, Broom 2001).   Using this definition , the relationship between stress and welfare is very clear.  Firstly, whilst welfare refers to a range in the state of the animal from very good to very poor, whenever there is stress, welfare is poor.  Secondly, stress refers only to situations where there is failure to cope but poor welfare refers to the state of the animal both when there is failure to cope and when the individual is having difficulty in coping.  It is very important that this latter kind of poor welfare,  as well as the occasions when an animal is stressed, is included as part of poor welfare.  For instance, if a person is severely depressed  or if an individual has a debilitating disease but there is complete recovery with no long term effects on fitness then it would still be appropriate to say that the welfare of the individuals was poor at the time of the depression or disease.

## 5. Welfare assessment.

The general methods for assessing welfare are summarised in Table 1 and a list of measures of poor welfare is presented in Table 2. Most indicators will help to pinpoint the state of the animal wherever it is on the scale from very good to very poor. Some measures are most relevant to short-term problems, such as those associated with human handling or a brief period of adverse physical conditions, whereas others are more appropriate to long-term problems. (For a detailed discussion of measures of welfare, see Broom 1988; Fraser and Broom 1990; and Broom and Johnson 1993).

Table 1 - Summary of Welfare Assessment

| General Methods | Assessment |
|---|---|
| Direct indicators of poor welfare | How poor |
| Tests of (a) avoidance and (b) positive preference | (a) Extent to which animals have to live with avoided situations or stimuli (b) Extent to which that which is strongly preferred is available |
| Measures of ability to carry out normal behaviour and other biological functions. | How much important normal behaviour or physiological or anatomical development cannot occur |
| Other direct indicators of good welfare | How good |

(modified after Broom 1999a)

Table 2 - Measures of welfare

Physiological indicators of pleasure
Behavioural indicators of pleasure
Extent to which strongly preferred behaviours can be shown
Variety of normal behaviours shown or suppressed
Extent to which normal physiological processes and anatomical development are possible.
Extent of behavioural aversion shown
Physiological attempts to cope
Immunosuppression
Disease prevalence
Behavioural attempts to cope
Behaviour pathology
Brain changes, e.g. those indicating self narcotization
Body damage prevalence
Reduced ability to grow or breed
Reduced life expectancy

(after Broom 2000)

Some signs of poor welfare arise from physiological measurements. For instance increased heart-rate, adrenal activity, adrenal activity following ACTH challenge, or reduced immunological response following a challenge, can all indicate that welfare

is poorer than in individuals which do not show such changes. Care must be taken when interpreting such results, as with many other measures described here. The impaired immune system function and some of the physiological changes can indicate what has been termed a pre-pathological state (Moberg, 1985).

Behavioural measures are also of particular value in welfare assessment. The fact that an animal avoids an object or event strongly gives information about its feelings and hence about its welfare. The stronger the avoidance the worse the welfare whilst the object is present or the event is occurring. An individual which is completely unable to adopt a preferred lying posture despite repeated attempts will be assessed as having poorer welfare than one which can adopt the preferred posture. Other abnormal behaviour such as stereotypies, self mutilation, tail-biting in pigs, feather-pecking in hens or excessively aggressive behaviour indicates that the perpetrator's welfare is poor.

In some of these physiological and behavioural measures it is clear that the individual is trying to cope with adversity and the extent of the attempts to cope can be measured. In other cases, however, some responses are solely pathological and the individual is failing to cope. In either case the measure indicates poor welfare.

Disease, injury, movement difficulties and growth abnormality all indicate poor welfare. If two housing systems are compared in a carefully controlled experiment and the incidence of any of the above is significantly increased in one of them, the welfare of the animals is worse in that system. The welfare of any diseased animal is worse than that of an animal which is not diseased but much remains to be discovered about the magnitude of the effects of disease on welfare. Little is known about how much suffering is associated with different diseases. A specific example of an effect on housing conditions which leads to poor welfare is the consequence of severely reduced exercise for bone strength. In studies of hens (Knowles & Broom, 1990, Norgaard Nielsen, 1990 ) those birds which could not sufficiently exercise their wings and legs because they were housed in battery cages had considerably weaker bones than those birds in percheries which could exercise. Similarly, Marchant & Broom (1996) found that sows in stalls had leg bones only 65% as strong as sows in group-housing systems. The actual weakness of bones means that the animals are coping less well with their environment so welfare is poorer in the confined housing. If such an animal's bones are broken there will be considerable pain and the welfare will be worse. Pain may be assessed by aversion, physiological measures, the effects of analgesics (e.g. Duncan et al (1991) or by the existence of neuromas (Gentle, 1986). Whatever the measurement, data collected in studies of animal welfare gives

information about the position of the animal on a scale of welfare from very good to very poor.

The majority of indicators of good welfare which we can use are obtained by studies demonstrating positive preferences by animals. Early studies of this kind included that by Hughes & Black (1973) showing that hens given a choice of different kinds of floor to stand on did not choose what biologists had expected them to choose. As techniques of preference tests developed, it became apparent that good measures of strength of preference were needed. Taking advantage of the fact that gilts preferred to lie in a pen adjacent to other gilts, van Rooijen (1980) offered them the choice of different kinds of floors which were either in pens next to another gilt or in pens further away. With the floor preference titrated against the social preference he was able to get better information about strength of preference. A further example of preference tests, in which operant conditioning with different fixed ratios of reinforcement were used, is the work of Arey (1992). Pre-parturient sows would press a panel for access to a room containing straw or one containing food. Up to two days before parturition they pressed, at ratios of 50-300 per reinforcement, much more often for access to, food than for access to straw. At this time, food was more important to the sow than straw for manipulation or nest-building. However on the day before parturition, at which time a nest would normally be built, sows pressed just as often, at fixed ratio 50-300, for straw as for food. Another indicator of the effort which an individual is willing to use to obtain a resource is the weight of door which is lifted. Manser et al (1996), studying floor preferences of laboratory rats, found that rats would lift a heavier door to reach a solid floor on which they could rest than to reach a grid floor.

The third general method of welfare assessment listed in Table 4 involves measuring what behaviour and other functions cannot be carried out in particular living conditions. Hens prefer to flap their wings at intervals but cannot in a battery cage whilst veal calves and some caged laboratory animals try hard to groom themselves thoroughly but cannot in a small crate, cage or restraining apparatus.

In all welfare assessment it is necessary to take account of individual variation in attempts to cope with adversity and in the effects which adversity has on the animal. When pigs have been confined in stalls or tethers for some time, a proportion of individuals show high levels of stereotypies whilst others are very inactive and unresponsive (Broom, 1987). There may also be a change with time spent in the condition in the amount and type of abnormal behaviour shown (Cronin & Wiepkema, 1984). In rats, mice and tree shrews it is known that different physiological and behavioural responses are shown by an individual confined with an

aggressor and these responses have been categorised as active and passive coping (von Holst, 1986 , Koolhaas et al, 1983 , Benus, 1988). Active animals fight vigorously whereas passive animals submit. A study of the strategies adopted by gilts in a competitive social situation showed that some sows were aggressive and successful, a second category of animals defended vigorously if attacked whilst a third category of sows avoided social confrontation if possible. These categories of animals differed in their adrenal responses and in reproductive success (Mendl et al, 1992). As a result of differences in the extent of different physiological and behavioural responses to problems it is necessary that any assessment of welfare should include a wide range of measures. Our knowledge of how the various measurements combine to indicate the severity of the problem must also be improved.

## 6. Health, Disease and Pathology

Health may be defined as "*an animal's state as regards its attempts to cope with pathology*" (Broom 2000). In this statement, animals include humans.

In their veterinary dictionary, Blood & Studdert (1999) define pathology as:

"*1. the branch of veterinary science treating of the essential nature of disease, especially of the changes in body tissues and organs which cause or are caused by disease.*

*2. the structural and functional manifestations of disease.*"

This is almost identical to the definition of pathology in Dorland's (1988) dictionary of human medicine.

Thus, pathology refers both to a scientific discipline and to the object of its study. The second definition is the relevant one in the present context. However, it is not satisfactory. Whilst this definition is faithful to the etymology of the term 'pathology', which literally means the study of disease, it does not get us any closer to an understanding of the subject, since it begs the question "what is disease?". Rather than taking the circuitous route of answering this question and deducing from it what pathology must actually mean, it is simpler to refer to several veterinary pathology textbooks which have advanced definitions without invoking disease. For example,

"*Pathology is the study of the derangement of molecules, cells, tissues and function that occur in living organisms in response to injurious agents or deprivations*" (Jones et al. 1997);

*"Pathology, in the broadest sense, is abnormal biology. As a science it encompasses all abnormalities of structure and function. It involves the study of cells, tissues, organs, and body fluids .... Pathology is essentially the search for and study of lesions, the abnormal structural and functional changes which occur in the body."* (Cheville 1988).

These definitions refer to the discipline of pathology, not to its object of study. Nevertheless, the object of study is made clear. The above definitions suggest that pathology is *"the derangement of molecules, cells, tissues and function that occur in living organisms in response to injurious agents or deprivations"*, or *"the abnormal structural and functional changes which occur in the body."*

One shortcoming of these definitions of pathology is that they imply, but do not explicitly state, that pathology is always detrimental to the organism. The terms 'derangement' and 'abnormal' are loaded, in that in common usage they usually refer to undesirable changes or states, but they need not do so. In practice, pathologists study detrimental changes of structure and function, not beneficial ones, and the definition of pathology should reflect this. It is suggested that the terms 'derangement' and 'abnormal' be qualified by the word 'detrimental'.

Cheville's use of the term 'lesion' is also somewhat problematic. In veterinary medicine, lesions are generally thought of as gross abnormalities, occurring at the level of the organs or tissues, not at the level of the cell. Hence, there can be pathology in the absence of lesions. Blood and Studdert's (1999) definition of a lesion, as *"any pathological .... discontinuity of tissue or loss of function of a part"* reflects its general usage. There is also a syntactic difference between the terms 'pathology' and 'lesion', which Cheville's usage reflects. 'Pathology' can be, and most frequently is, employed as a collective noun, whereas 'lesion' is a particular noun. Unless there is only one lesion present, pathology describes a collection of lesions. The plural, 'pathologies', is sometimes used to refer to the existence of pathology in more than one animal.

The distinction which pathologists make between structure and function is essentially one between the morphology of a cell, tissue or organ and its operation. Functional abnormalities include physiological changes, which are the subject of a subdiscipline known as pathophysiology. These physiological changes are seen as departures from the normal day-to-day balance or steady state. Functional abnormalities also include more obvious changes, such as loss of appetite and diarrhoea, which are often employed as clinical signs. The term 'lesion' usually refers to a structural abnormality, but is also applied to functional abnormalities, which may or may not have morphological counterparts. The term 'pathogenesis' refers to the way in which a lesion develops over time (Slauson & Cooper 1990).

The veterinary definition of the term 'disease' is in fact very similar to that of 'pathology'. Blood & Studdert (1999) begin by stating that disease is "*traditionally defined as a finite abnormality of structure or function with an identifiable pathological or clinopathological basis, and with a recognizable syndrome or constellation of clinical signs*", but go on to add that "*the definition has long since been widened to embrace subclinical diseases in which there is no tangible clinical syndrome but which are identifiable by chemical, hematological, biophysical, microbiological or immunological means.*" Slauson & Cooper's (1990) definition is "*the culmination of those various defects, abnormalities, excesses, deficiencies, and injuries occurring at the cell and tissue level which ultimately result in clinically apparent dysfunction*". This usage of the term 'disease', like the widespread veterinary usage of 'pathology', refers to injuries as well as to the effects of pathogens, although for many people, injury would not initially come under the heading of disease. Furthermore, these definitions of disease, like the definitions of pathology above, are too inclusive, describing diseases as "abnormalities", whereas the study of disease is exclusively concerned with changes which are detrimental to the organism.

Pathology or disease is classified in three ways: 1. according to its causes; 2. according to the type of tissue changes which are involved; and 3. according to the identity of the tissue or organ which is affected. The classification of pathology according to the type of tissue changes involved is probably the least ambiguous approach. Five types of tissue changes have been identified: cellular degeneration and death; circulatory disturbances common to all tissues; inflammation and repair; immunopathology; and disturbances of growth, including neoplasia (Cheville 1988; Slauson & Cooper 1990). However, the classification of pathology according to causes is more useful for the purposes of present review, since this approach is compatible with the classification of welfare measures already discussed. Because so many diverse causes exist, and because most pathology is multifactorial, it is not easy to devise a rigorous system of classification on this basis. In practice, most veterinarians employ a mixture of categories, relating not only to cause, but also to the identity of the affected tissue, when they make diagnoses. Nevertherless, attempts have been made to classify pathology by its causes (eg. Cheville 1988; Slauson & Cooper 1990). Slauson & Cooper's (1990) system is presented in Table 3.


**Table 3** Classification of pathology according to its causes

Genetic abnormalities.

Physical injury.

Thermal injury.

Chemical injury.

Infections or infestations.

Metabolic abnormalities.

Nutritional injury.

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Slauson & Cooper (1990) give examples of the pathologies which would fall into these categories, but do not offer an exhaustive list.

Thermal injury should include not only direct tissue damage, but also consequences of hyperthermia and hypothermia (Cheville 1988).

Infections and infestations can be subclassified, according to the pathogen, into those caused by viruses, mycoplasmatales/rickettsiales/ chlamydiales, bacteria, protozoa (eg. coccidia) and parasitic helminths and arthropods (Jones et al. 1997). Prions should be added to this list.

The category of metabolic abnormalities is quite difficult to define, owing to the complex aetiology of many metabolic disorders. Slauson & Cooper (1990) list hormonal imbalance, enzyme defects, membrane defects and structural protein defects as examples of metabolic abnormalities. This list emphasises pathology in which metabolic dysfunction is the sole cause. It would not include many conditions normally classed as metabolic diseases, particularly those associated with nutritional deficiency. Blood & Studdert (1999) define metabolic disease as:

*"diseases in which normal metabolic processes are disturbed and a resulting absence or shortfall of a normal metabolite causes disease, eg. hypocalcaemia in cows, or an accumulation of the end products of metabolism causes a clinical illness. eg. acetonaemia of dairy cows. Many diseases in this group really have their beginnings in a nutritional deficiency state."*

In fact, very few disorders of domestic animals are caused solely by metabolic dysfunction (Payne 1989). Even parturient hypocalcaemia and ketosis (acetonaemia) in dairy cows are associated with nutrition, arising from an imbalance between nutrient intake and the excessive metabolic demands of lactation (qv. Section 5.3.2.1). Slauman & Cooper's (1990) perspective reflects the human medical usage of the term 'metabolic disease' more than the veterinary usage. In man, 'metabolic disease' implies some inherent defect, such as the congenital absence of an enzyme (eg. 'storage diseases', in which metabolites slowly accumulate), or an endocrinological failure (eg.

diabetes mellitis). The veterinary usage is looser, admitting nutritional deficiencies and more complex disorders which result from a breakdown in the animal's capacity to meet the physiological demands of high productivity (Payne 1989).

There is substantial overlap between metabolic disease and the so-called 'production diseases', defined by Blood & Studdert (1999) as:

"*diseases caused by systems of management, especially feeding and the breeding of high-producing strains of animals and birds, in which production exceeds dietary and thermal input. Includes the group of diseases known in the veterinary literature as 'metabolic diseases'. They differ from nutritional deficiencies in which it is the nutritional supply which falls short of normal production.*"

Production diseases are effectively man-made, being caused by an inability to meet the demands of high production (Payne 1989). This category does not include nutritional deficiencies, but does include many other metabolic diseases, such as parturient hypocalcaemia and ketosis in dairy cattle. It is somewhat broader in general usage than Blood & Studdert's definition suggests, since it also includes acidosis and laminitis in dairy cows (Payne 1989; Webster 1993), which are caused by the overfeeding of concentrates. These conditions are closely associated with high productivity, because high-yielding cows require large quantities of concentrate to meet the demands of lactation. Production diseases will be discussed in more detail later.

In the category of 'nutritional injury', Slauson & Cooper (1990) include deficiency, imbalance, undernutrition and overnutrition. This agrees with Blood & Studdert's (1999) definition of 'nutritionally related disease', as: "*disease caused by deficiencies or excesses of specific feed nutrients or of a total ration*". The overlap between metabolic disease and nutritional disease in ordinary veterinary usage is clear from a comparison of Blood & Studdert's definitions of these terms. Also, some production diseases, including acidosis and laminitis in dairy cattle, would be classed as nutritional diseases (Fig. 1).

In discussing the causes of pathology, the effects of environmental factors upon an animal's resistance to infection is also generally acknowledged (eg. Thomson 1984; Slauson & Cooper 1990). For example, cold air and atmospheric pollutants have been shown to impair bacterial clearance from the lung in pigs, by interfering with the mucociliary elevator. This may predispose them to respiratory infections. Crowding, weaning, changes of feed and transportation are other environmental factors which are believed to reduce resistance to infection. Such factors may change subclinical or latent infection into acute or chronic disease (Fig. 2).

It should be noted that some veterinarians would define animal health more broadly than Broom (2000). For example, Blood & Studdert's (1999) definition is "*a state of*

*physical and psychological wellbeing and of productivity including reproduction"*. This definition is inadequate, partly because 'wellbeing' is not defined and partly because it is far too inclusive. It does not reflect the practice of veterinary medicine, which is primarily concerned with physical abnormalities. In reality, neither behavioural disturbances, nor psychological stressors are considered in the classification of pathology. This review will employ Broom's definition of health, because it reflects the *de facto* meaning of the term in veterinary medicine and because it makes clear the distinction between health and welfare.

## 7. Relationship between welfare and health

Health is a part of welfare. When an animal's health is poor, so is its welfare, but poor welfare does not always imply poor health. There are many circumstances where behavioural or physiological coping mechanisms are activated, indicating that welfare is poor, but the animal's health remains good. These include: situations where the coping mechanisms are successful, such as when body temperature is maintained despite extreme ambient temperatures; circumstances where failure to cope has consequences for psychological, but not physical, stability, such as in the development of non-injurious pathological behaviours; and where detrimental effects upon physical stability are compensated for by management practices, such as the routine use of antibiotics.

A comparison of Table 1 with Table 3 indicates that there are some indicators of poor welfare which are classified as pathology and, as such, will also indicate poor health. These include body damage and 'disease', which refers in this case to infectious disease. The prevention of normal physiological processes and anatomical development will also indicate poor health, where these phenomena can be shown to be symptoms of an infectious, metabolic or nutritional disease. Mortality rate is also an indicator of welfare in general and health in particular in many circumstances. When animals are close to death, their welfare and their health will often be very poor.

Other indicators of poor welfare, whilst not being signs of poor health at that time, may indicate a risk of poor health in the future. These include: immunosuppression, which renders the animal susceptible to infection; the chronic activation of physiological coping mechanisms, which may cause immunosuppression; and certain behavioural pathologies and redirected behaviours, which can result in serious injury or predisposition to infection, either in the animal itself or in others. It is these measures which the review will focus upon, since poor welfare precedes poor health and is instrumental in its deterioration.

Two pathways can be identified, linking poor welfare to poor health:

1. chronic activation of physiological coping mechanisms ---> immuno-suppression ---> infectious disease;

2. behavioural coping mechanisms ---> injurious abnormal behaviour ---> physical injury.

The review will also consider metabolic 'production diseases', in which poor welfare also causes poor health. In some domestic breeds, a combination of nutrition and genetic selection for high productivity has produced a situation in which the animal is barely able to cope with the demands of its own physiology.

A third pathway, linking poor welfare to poor health, therefore corresponds to the development of metabolic 'production diseases':

3. genetic selection for high productivity + nutrition ---> metabolic stress ---> metabolic 'production disease'.

The remainder of the review will be concerned with the description of these three pathways. Examples will be given, several of which will be discussed in detail.

## 8.1. Physiological coping mechanisms and infectious disease

### 8.1.1 General principles

The relationship between the chronic activation of physiological coping mechanisms, immunomodulation and susceptibility to infectious disease has been explored in the field of psychoneuroimmunology. It is important, because all environmental challenges which lead to poor welfare, whether they threaten mental or bodily stability, activate these coping mechanisms. However, the relationship is not a simple one. The response of the neuroendocrine system is not the same for all environmental challenges (Mason 1968a,b, 1975). It may also vary between species (Griffin 1989) and between individuals, depending on how they perceive the challenge (Corley et al. 1975). Glucocorticoids and other hormones modulate the immune system in various ways, which have been studied in vitro and are relatively well understood, at least in the case of glucocorticoids. But, a given change in the immune system may affect an animal's susceptibility to different pathogens in different ways (Gross & Colmano 1969). These complications mean that it is often necessary to consider one challenge, one species and one pathogen at a time.

Glucocorticoids have certain, relatively uniform, effects on the immune system (Griffin 1989). They reduce the number of circulating lymphocytes (lymphopaenia) and increase the number of neutrophils (neutrophilia). In many species, they also

reduce the number of eosinophils (eosinopaenia). Lymphocytes, neutrophils and eosinophils are all types of white blood cell, or leucocyte. In species with relatively high numbers of lymphocytes, such as chickens, these changes result in a reduction in the total number of circulating leucocytes (leucopaenia), whereas in species with relatively low numbers of lymphocytes, including cattle, sheep and pigs, the net result is an increased leucocyte count (leucocytosis).

Lymphocytes include: B cells, T cells and natural killer (NK) cells. B cells synthesise antibodies, the humoral immune response. These bind to circulating antigens, facilitating their ingestion by granulocytes and macrophages, neutralising them, or otherwise assisting the host's defences. T cells are subdivided into cytotoxic T cells, which destroy host cells presenting foreign (eg. viral) antigens on their surface, the cell-mediated immune response, and helper T cells, which facilitate the humoral and cell-mediated immune responses. NK cells destroy host cells which do not present antigens on their surface, a characteristic of tumour cells and some cells infected by viruses. B cells and T cells are sensitive to specific antigens. In addition to performing their characteristic functions, they respond by proliferating. In the case of B cells and cytotoxic T cells, memory cells are produced which greatly increase the humoral and cell-mediated immune responses to the antigen if the host is exposed to it again, the secondary immune response. Neutrophils and eosinophils are two varieties of granulocytes, a cell population distinct from the lymphocytes, which also includes basophils. Neutrophils are the most numerous of the granulocytes. They are attracted by chemical signals to damaged tissues, where they capture and destroy foreign material by phagocytosis. Eosinophils can also phagocytose small particles, but are better suited to the destruction of large parasites by extruding enzymes into the surrounding fluid. Basophils are not phagocytic, but release vasoactive amines in damaged tissues, provoking acute inflammation. A third distinct population of leucocytes is the macrophages, known as monocytes when immature, which reach damaged tissues after the neutrophils and destroy not only foreign material but also dead and dying host cells. They also assist in wound healing and secrete cytokines, which activate lymphocytes and produce feelings of illness.

The differential effects of glucocorticoids upon different leucocyte populations may explain the observation that a given stressor can increase the susceptibility of chickens to some pathogens whilst reducing their susceptibility to others (Gross & Siegel 1965; Gross & Colmano 1969, 1971; Gross 1972, 1976). The rapid, non-specific immune response mediated by neutrophils is of considerable importance in dealing with certain bacterial and coccidial infections, but is of little use against some agents, such as *Mycoplasma gallisepticum* and Newcastle disease virus, which can only be tackled effectively by lymphocytes (Gross 1962; Gross & Siegel 1975; Siegel 1980).

Not only do glucocorticoids reduce the number of circulating lymphocytes, they also suppress the activity of B cells and cytotoxic T cells, by interacting with macrophages and helper T cells. For example, glucocorticoids decrease the synthesis of interleukin 1 (IL-1) by macrophages (MacDermott & Stacey 1981) and the synthesis of interleukin 2 (IL-2) by helper T cells (Gillis et al. 1979). These cytokines increase the activity of B cells and cytotoxic T cells, as well as that of other leucocytes, including macrophages and helper T cells.

Glucocorticoids are very important mediators of the immune system (Biondi & Zannino 1997). However, they are not the only means by which stressors influence immunocompetence (Griffin 1989; Biondi & Zannino 1997; Yang & Glaser 2000). For example, both the synthesis of β-endorphin by the anterior pituitary gland (Haynes & Timms 1987) and the release of vasopressin and oxytocin from the neurohypophysis (Wideman & Murphy 1985; Williams et al. 1985) are increased in response to environmental challenges. β-endorphin enhances T cell proliferation *in vitro* (Gilman et al. 1982), while vasopressin and oxytocin both stimulate helper T cells to produce more interferon-γ (Johnson & Torres 1985), a cytokine which activates macrophages and NK cells. In humans at least, catecholamines suppress the cell-mediated immune response whilst enhancing the humoral immune response (Yang & Glaser 2000). Furthermore, the lymphoid organs, including the bone marrow, thymus, spleen and lymph nodes, where lymphocytes are produced and stored, are all innervated (Felten & Felten 1991; Schorr & Arnason 1999), permitting the CNS to influence lymphocytes directly. The situation is further complicated by the fact that vasopressin and oxytocin (Gibbs 1986a,b; Gaillard & Al-Damluji 1987) and catecholamines (Axelrod 1984) also stimulate the secretion of ACTH, whilst β-endorphin is secreted in parallel with ACTH, from their mutual precursor pro-opiomelanocortin (Guillemin et al. 1977; Rossier et al. 1977). With so many pathways modulating the effects which environmental challenges have upon the immune system, it is not possible to make generalised predictions concerning the effects of stress upon immunocompetence.

What is clear from the study of psychoneuroimmunology is that environmental conditions which elicit physiological coping responses in animals, and which can therefore be said to compromise their welfare, alter their susceptibility to infectious agents and hence their health status (Biondi & Zannino 1997). It is also apparent that, despite the complexity of the physiological processes which mediate between the exposure of an animal to an environmental challenge and its health status, the majority of experimental studies to date have reported an increased susceptibility to pathogens (Peterson et al. 1991; Biondi & Zannino 1997). Notwithstanding this, it is essential that the effect of a given challenge upon the susceptibility of a given species to a given disease should be investigated individually.

## 5.1.2. Examples of effects of environmental challenges on pathology

In Tables 4 to 6, lists have been made of environmental challenges which have been shown to alter the susceptibility of farm animal species to specific pathogens. The studies cited are of two kinds. The majority have proceeded by innoculating experimental subjects with a pathogen, subjecting them to an environmental challenge, either before or after innoculation, and comparing the clinical course of the disease in these animals with its course in controls, which have been subjected to the environmental challenge but not innoculated. In a second, much smaller group of studies, researchers have investigated the ability of environmental challenges to reactivate pathogens whose clinical effects are either much reduced or no longer apparent, but which remain present in the body. Studies which have simply observed the effect of an environmental challenge upon the spontaneous incidence of disease have not been included, since it is often unclear which pathogen or combination of pathogens is involved.

**Table 4.** Effects of environmental challenges upon bacterial (including mycoplasmal) pathology

| Study | Species | Challenge | Bacteria | Effects on clinical course |
|-------|---------|-----------|----------|---------------------------|
| Gross & Siegel (1965) | Chicken | Mixing<br>Transportation | *Escherichia coli*<br><br>*Mycoplasma gallisepticum* | ↓ susceptibility<br><br>= susceptibility |
| Gross & Colmano (1969) | Chicken | Mixing | *Staphylococcus aureus*<br><br>*Escherichia coli*<br><br>*Mycoplasma gallisepticum* | ↓ susceptibility<br><br><br>↓ susceptibility<br><br>↑ susceptibility |
| Gross & Siegel (1981) | Chicken | Mixing | *Escherichia coli*<br><br>*Mycoplasma gallisepticum* | ↓ susceptibility<br><br>↑ susceptibility |
| Gross (1984) | Chicken | Various degrees of mixing stress | *Escherichia coli* | As stress increased, susceptibility ↓ then ↑ |
| Larson et al. (1985) | Chicken | Mixing | *Staphylococcus aureus* | ↓ susceptibility |
| Gross & Siegel (1981) | Chicken | Isolation | *Mycoplasma gallisepticum* | ↑ susceptibility |
| Juszkiewicz et al. (1967) | Chicken | Heat | *Pasteurella multocida* | ↑ speed of mortality |

| Study | Species | Challenge | Bacteria | Effects on clinical course |
|---|---|---|---|---|
| Juszkiewicz (1967) | Chicken | Cold | *Pasteurella multocida* | ↓ speed of mortality |
| Moore et al. (1934) | Chick | Cold | *Salmonella pullorum* | ↑ susceptibility |
| Ram & Hutt (1955) | Chick | Cold | *Salmonella pullorum* | ↑ mortality |
| **Study** | **Species** | **Challenge** | **Bacteria** | **Effects on clinical course** |
| Bierer (1961) | Chick | Cold | *Salmonella typhimurium* | ↑ mortality |
| Thaxton et al. (1974) | Chick | Cold | *Salmonella worthington* | ↑ mortality |
| Soerjadi et al. (1979) | Chick | Cold | *Salmonella typhimurium* | ↑ shedding |
| Larson et al. (1985) | Pig | Isolation | *Staphylococcus aureus* | ↓ susceptibility |
| Armstrong & Cline (1977) | Pig | Cold | *Escherichia coli* | ↑ gastroenteritis |
| Filion et al. (1984) | Calf | Transportation + marketing | *Pasteurella haemolytica* | = susceptibility |
| Binkhorst et al. (1990) | Calf | Forced exercise | *Pasteurella haemolytica* | = severity |
| Anderson et al. (1991) | Calf | Forced exercise | *Pasteurella haemolytica* | ↑ severity |
| Hamdy et al. (1963) | Calf | Heat + cold | *Pasteurella haemolytica* + *Pasteurella multocida* + bovine parainfluenza-3 virus | ↑ severity |
| Diesel et al. (1991) | Calf | Cold | *Pasteurella haemolytica* | ↑ susceptibility |
| Owen et al. (1983) | Horse | Transportation | *Salmonella typhimurium* | Reactivated |

Table 4 is concerned with the effects of environmental challenges on bacterial pathology; Table 5 with viral pathology; and Table 6 with parasitic pathology. It can be seen from all three tables that the number of studies which have been conducted using domestic livestock species is small. Furthermore, the majority of studies have reported an increased susceptibility to disease following environmental challenge.

It can be seen from Table 4 that mixing with unfamiliar animals generally reduces the susceptibility of chickens to *Escherichia coli* and *Staphylococcus aureus* (Gross & Siegel 1965, 1981; Gross & Colmano 1969; Larson et al. 1985) although when the mixing procedure evoked a very substantial coping response (the neutrophil: lymphocyte ratio was measured), their susceptibility to *Escherichia coli* was increased (Gross 1984). In pigs, isolation also reduces susceptibility to *Staphylococcus aureus* (Larson et al. 1985). Both mixing and isolation increase the susceptibility of chickens to *Mycoplasma gallisepticum* (Gross & Siegel 1965, 1981; Gross & Colmano 1969). Exposure to low environmental temperatures increases the susceptibility of recently hatched chicks to *Salmonella spp*. (Moore et al. 1934; Ram & Hutt 1955; Bierer 1961;

Thaxton et al. 1974; Soerjadi et al. 1979). However, the survival time of adult chickens innoculated with *Pasteurella multocida* is increased by low temperatures (Juszkiewicz 1967), whilst being reduced by high temperatures (Juszkiewicz et al. 1967). In pigs, low temperatures increase susceptibility to *Escherichia coli* (Armstrong & Cline 1977). Attempts to experimentally induce pneumonic pasteurellosis ('shipping fever') in calves have been unsuccessful when transportationwas used as a stressor (Filion et al. 1984) and have had mixed findings with forced exercise (Binkhorst et al. 1990; Anderson et al. 1991), but have been successful when using low temperatures (Diesel et al. 1991) and abrupt temperature changes (Hamdy et al. 1963). Transportation has been shown to reactivate *Salmonella typhimurium* in horses (Owen et al. 1983).

**Table 5.** Effects of environmental challenges upon viral pathology

| Study | Species | Challenge | Virus | Effects on clinical course |
|---|---|---|---|---|
| Gross & Colmano (1969) | Chicken | Mixing | Newcastle disease virus | ↑ susceptibility |
| Gross & Colmano (1971) | Chicken | Mixing | Marek's disease virus | ↑ susceptibility |
| Gross (1972) | Chicken | Mixing | Marek's disease virus | ↑ susceptibility |
| Mohamed & Hanson (1980) | Chicken | Mixing | Newcastle disease virus | ↑ susceptibility |
| Lin et al. (1998) | Pig | Mixing | Latent swine vesicular disease virus | Reactivated |
| Furuuchi & Shimizu (1976) | Pig | Heat | Transmissible gastroenteritis virus | ↓ gastroenteritis |
| Shimizu et al. (1978) | Pig | Heat | Transmissible gastroenteritis virus | ↓ gastroenteritis |
| Shope (1955) | Pig | Cold | Latent swine influenza virus | Reactivated |
| Furuuchi & Shimizu (1976) | Pig | Cold | Transmissible gastroenteritis virus | ↑ gastroenteritis |
| Shimizu et al. (1978) | Pig | Cold | Transmissible gastroenteritis virus | ↑ gastroenteritis |
| Filion et al. (1984) | Calf | Transportation + marketing | Bovine herpesvirus-1 | ↑ mortality and pneumonia |

In Table 5, it can be seen that mixing chickens increases their susceptibility to Newcastle disease virus (Gross & Colmano 1969; Mohamed & Hanson 1980) and Mareks disease virus (Gross & Colmano 1971; Gross 1972). In pigs, mixing also reactivates latent swine vesicular disease virus (Lin et al. 1998). Low temperatures increase the susceptibility of pigs to transmissible gastroenteritis virus, whilst high temperatures make them more resistant (Furuuchi & Shimizu 1976; Shimizu et al. 1978). Low temperatures also recativate latent swine influenza virus in pigs (Shope

1955). The transportation of calves has been shown to increase the incidence of pneumonia caused by bovine herpesvirus-1 (Filion et al. 1984).

**Table 6.** Effects of environmental challenges upon parasitic pathology

| Study | Species | Challenge | Parasite | Effects on clinical course |
|---|---|---|---|---|
| Hall & Gross (1975) | Chicken | Mixing | *Ornithonyssus sylvarium* (northern fowl mite) | ↓ susceptibility |
| Gross (1976) | Chicken | Mixing | *Eimeria necatrix* | ↓ susceptibility |
| Hall et al. (1979) | Chicken | Mixing | *Ornithonyssus sylvarium* | ↓ susceptibility |
| Gross (1985) | Chicken | Mixing | *Eimeria tenella* | ↓ susceptibility |
| Pierson et al. (1997) | Chicken | Mixing | *Eimeria tenella* | ↓ susceptibility |

Table 6 indicates that mixing chickens makes them more resistant to the northern fowl mite (Hall & Gross 1975; Hall et al. 1979) and to coccidia of *Eimeria spp*. (Gross 1976, 1985; Pierson et al. 1997).

### 5.1.3. Examples in detail

Two examples are discussed in more detail below: the transportation of cattle and the transportation of sheep. An account is given of how the various environmental challenges which occur during transportation activate physiological coping mechanisms, modulate the immune system and increase disease incidence in these species. The effects of these challenges upon the incidence of physical injury and mortality are also described.

The experimental studies which have assessed the effects of these challenges upon the immune system have employed a variety of assays, which require some explanation beforehand. These assays have been designed to measure either humoral or cell-mediated immune responsiveness, following exposure to the putative stressor.

Assays of humoral immune responsiveness measure the production of specific antibodies following an experimental challenge (usually an injection) with an antigen. The antigen should be injected either during exposure to the stressor, shortly before, or shortly afterwards. Sometimes, the primary antibody response is assessed, by estimating the amount of antibody present in blood samples obtained five or more days following the injection. A common technique is agglutination, in which blood serum is added to a suspension of antigen particles and the level of clumping or agglutination which occurs reveals the concentration of the antibody. Alternatively, the secondary antibody response may be assessed, either by administering a second antigen challenge to the animal and estimating the number of antibodies in blood samples obtained subsequently, or by means of a plaque-forming cell assay. The plaque-forming cell assay employs foreign red blood cells (erythrocytes) as the antigen. Five or more days following injection of the erythrocytes, lymphocytes are taken from the spleen and incubated, with the erythrocyte antigen and complement, between two microscope slides. Complement is the name given to a series of enzymes which destroy cells presenting antigen bound to antibody. A plaque, clear of erythrocytes, forms around each B cell, its diameter being proportional to the level of antibody synthesis.

Assays of cell-mediated immune responsiveness measure the activity of cytotoxic T cells. They can be classified into *in vitro* and *in vivo* techniques. *In vitro* techniques include measures of the proliferation of T cells, and of their production of cytokines, in response to antigen. For these tests, blood samples are obtained either during exposure to the stressor, shortly before, or shortly afterwards. In order to measure T cell proliferation, the cells are mixed with antigen and incubated, sometimes in the presence of a mitogen, such as phytohaemagglutinin (PHA), concanavalin A (conA) or pokeweed mitogen (PWM), which stimulates proliferation. The rate of proliferation is estimated by measuring the uptake of radio-labelled thymidine, which dividing cells incorporate into new DNA. In order to measure the production of cytokines, blood samples are incubated with antigen and the resulting concentrations of cytokines are measured using a standard assay, such as the enzyme-linked immunosorbent assay (ELISA). A third *in vitro* technique estimates T cell cytotoxicity following exposure to antigen. In this case, the animal is injected with the antigen around the time of exposure to the stressor and blood samples are obtained later. T cells are then incubated with radio-labelled cells presenting the antigen. Cytotoxic action of the T cells releases the radio-label into solution, so the final concentration of the label yields an estimate of T cell activity. *In vivo* techniques are quite different. They measure a cell-mediated inflammatory response to antigen challenge on or below the skin surface, known as a delayed hypersensitivity reaction because it develops gradually over the course of several days. The inflammation is

caused primarily by basophils, which are attracted to the site by T cells. An inflammatory response to most antigens only occurs when an animal has had previous exposure to them, in other words reactions of this kind are generally secondary cell-mediated immune responses. The antigen must therefore be injected intradermally on two occasions, the inflammatory response following the second injection being assessed by a measurement of skin thickness. However, certain chemical agents, including dinitrochlorobenzene (DNCB) and PHA, elicit a delayed hypersensitivity-like reaction on the first exposure, known as a contact sensitivity response. This allergic cell-mediated response is peculiar, in that it does not require previous sensitisation to an antigen. PHA is injected intradermally, while DNCB is painted onto the skin surface. The inflammatory response is assessed by measuring skin thickness.

### 5.1.3.1. Transportation of cattle

**General**

Cattle are transported by road, rail, sea and air, for breeding, fattening and slaughter. In Europe, the biggest trade is by road and most research has been conducted on road transport (Tarrant & Grandin 2000).

If dairy calves are to be sold, they are normally transported to market within the first two weeks of life. If purchased, they are transported twice more, first to the dealer's premises and then to a beef rearing unit (Webster 1984). Most calves exported from the UK for veal are just over a week old (Knowles 1995). Beef calves from suckler herds are considerably older when transported for finishing, usually between six and twelve months of age (Allen & Kilkenny 1984). Beef calves are normally weaned at the same time as they are transported (MacKenzie et al. 1997), whilst dairy calves are weaned at the rearing unit, at four or five weeks of age (Allen & Kilkenny 1984). Weaning consists of removal from the dam in suckled beef production systems, or withdrawal of milk substitute in systems which rear dairy calves for beef. In the USA, journey distances for beef calves are typically much greater than in Europe: calves often travel between 1000 and 3000 km to the feedlot (Tarrant & Grandin 2000).

Most older cattle, transported to slaughter, also pass through live auction markets (Knowles 1999). In the USA, distances between feedlots and slaughterhouses are relatively short (Tarrant & Grandin 2000), but transport times are increasing in Europe, owing to a steady decline in the number of abattoirs (Knowles 1999; Tarrant & Grandin 2000).

The transportation of cattle involves exposure to a number of environmental challenges, other than the journey itself. These may include: withdrawal of feed and water; mixing; introduction to a novel environment; loading and unloading; and extremes of temperature, light, noise and vibration. Some or all of these factors may contribute to the overall levels of stress and injury observed during commercial transportation. Some experimental studies have attempted to separate out these factors, while others have simulated commercial procedures.

**Mortality**

Young calves, less than four weeks of age, are not well adapted to cope with transport and marketing, often suffering very high rates of mortality (Knowles 1995). The younger the calves are, the higher their mortality (Staples & Haugse 1974; Barnes et al. 1975; Mormède et al. 1982). Comparatively few calves actually die during the process of transportation, but many more succumb to secondary disease, mostly with symptoms of pneumonia or scouring, within four weeks of arrival at the rearing unit (Staples & Haugse 1974).

Mortality is very low in adult cattle transported by road (Knowles 1999). In South Africa, Henning (1993) reported a mortality rate of 0.01% in slaughter cattle in 1980 and no mortality amongst 22,000 cattle in the early 1990's. Mortality rates during sea journeys can be considerably higher (Hails 1978; Connell 1984).

**Physical injury**

Transportation of cattle results in bruising. Jarvis et al. (1995) found bruising on 97% of carcases, with a mean of 1.4 bruises per carcase (Knowles 1999). Bruising is caused by rough handling and poor road driving techniques (Grandin 1983), and exacerbated by high stocking densities, long journey times and repeated loading and unloading.

Cattle which are handled roughly show much more bruising than those which are handled gently (Grandin 1981). In particular, the use of a stick for driving cattle increases bruising (Jarvis et al. 1995). Observations made during the journey indicate that minor losses of balance, where cattle quickly shift their footing to regain stability, are nearly all caused by driving events including cornering, gear changes and braking (Kenny & Tarrant 1987a,b; Tarrant et al. 1988).

Falls were rarely observed at low stocking densities (200 or 300 kg/m$^2$ in 600 kg steers), but were much more frequent when stocking density was high (600 kg/m$^2$) (Tarrant et al. 1992). They were triggered by driving events and by animals struggling to change position. Tarrant et al. (1992) observed that shifts of footing, to regain

balance, were inhibited at the high stocking rate. After falling, cattle were unable to get up again. Fallen animals were trampled and this caused other animals to lose their footing. The result was a high incidence of carcase bruising.

For typical commercial driving, during which accelerations causing loss of balance are frequent (Broom 1999), there appears to be an optimum stocking density at which bruising is minimised. Eldridge & Winfield (1988) transported 360 kg steers for six hours at three stocking rates. Animals transported at the intermediate density, of about 310 kg/m$^2$, showed the least bruising. The incidence of bruising was twice as high at a density of about 260 kg/m$^2$ and four times as high at a density of about 405 kg/m$^2$. The Farm Animal Welfare Council (1991) and Randall (1993) have proposed guidelines, in the form of equations relating space allowance to body weight. Both recommend a stocking density of 360 kg/m$^2$ for 360 kg animals. For cattle weighing 600 kg, the recommendations are in the region of 400 kg/m$^2$.

The extent of bruising has also been shown to increase with distance travelled and with time spent in the lairage (McNally & Warriss 1996). In lairage, mounting behaviour, seen especially in groups of unfamiliar bulls, is known to cause bruising (Kenny & Tarrant 1987c). More bruising was also seen in cattle arriving for slaughter from an auction market (Jarvis et al. 1995; McNally & Warriss 1996). This may have been due either to rough handling or to social interactions such as mounting.

**Physiological measures of welfare**

Welfare problems arising during the transportation of cattle include not only the physical injury and psychological stress associated with mixing, handling and driving, but also fatigue, hunger, dehydration and thermal stress.

A variety of different physiological measures of welfare have been employed, some of which assess particular problems, whilst others are more general. Measures which are specific include the plasma concentration of creatine kinase (CK), an enzyme released into the blood from heart and skeletal muscle after damage or vigorous exercise. High levels of this enzyme after transportation are associated with physical fatigue. Dehydration also has specific physiological correlates, including the packed cell volume or haematocrit, defined as the percentage, by volume, of the blood which is occupied by erythrocytes, and the osmolality, a measure of the concentration of solutes in the blood. A more general measure of welfare in transit is the depletion of muscle glycogen levels, which may be a consequence either of sustained physical exertion, or of chronic activation of the SAM axis (Lacourt & Tarrant 1985;

Shackleford et al. 1994), since adrenaline mediates the conversion of stored glycogen to glucose. An allied measure is the incidence of dark, firm, dry (DFD) beef, also known as dark cutting beef. This meat has a characteristically high pH, a result of the depletion of muscle glycogen before death (Tarrant & Grandin 2000). Another relatively general measure of welfare is plasma cortisol concentration, although this measure is not completely general, since glucocorticoid levels are not elevated by fasting or high temperatures (Mason 1968a,b).

Cattle from intensive production systems, which are accustomed to human contact, can be loaded and unloaded without poor welfare, if rough handling is avoided (Kenny & Tarrant 1987a). However, extensively raised beef cattle exhibit elevated heart rates and cortisol levels when handled (Lay et al. 1992a,b). The physiological responses of range-reared beef cattle to handling are almost as great as their responses to hot-iron branding (Lay et al. 1992a,b), and this may be the most stressful component of transport for these animals.

The effect which the process of transportation has upon plasma cortisol concentration is somewhat variable: in many studies cortisol levels were elevated during or after transportation (Crookshank et al. 1979; Simensen et al. 1980; Kent & Ewbank 1983; 1986b; Murata et al. 1987; Kegley et al. 1997), but in several studies they were not (Blecha et al. 1984; Kegley et al. 1997). This is probably due to differences in journey time and in a wide range of factors which influence the quality of a journey. These may include stocking density, ventilation, vehicle design, the standard of driving and the quality of the roads (Tarrant & Grandin 2000). Plasma cortisol concentration is greater at high stocking densities (Tarrant et al. 1988; 1992).

When weaning coincides with transportation, this will also make the journey more difficult for the animals. Calves which are weaned and transported simulaneously exhibit higher cortisol levels than calves weaned two weeks before transportation (Crookshank et al. 1979).

The cortisol response of young calves, of less than 4 weeks of age, to transportation is reduced as compared with older cattle (Mormède et al. 1982; Fell & Shutt 1986; Kent & Ewbank 1986a; Knowles et al. 1997), but this does not indicate that they cope better with transport. On the contrary, it reflects the immaturity of their physiological coping mechanisms (Mormède et al. 1982; Knowles et al. 1997). Hartmann et al. (1973) have demonstrated that the reactivity of the adrenals to an ACTH challenge increases with age and is not yet fully developed in one-week-old calves. An inability to mount an effective glucocorticoid response, which is adaptive in the short term, may be a contributing factor to the high levels of morbidity and mortality which occur in young calves. It has also been observed that young calves are unable to closely regulate their body temperature during transportation (Knowles et al. 1997).

DFD beef is generally caused by mixing and by prolonged transportation, including time spent in the lairage. Long road journeys result in a significant elevation of meat pH, between 0.1 and 0.2 pH units, and a corresponding increase in the incidence of DFD beef (Wythes et al. 1981; Tarrant et al. 1992). The prevalence of DFD meat increases substantially with time spent in the lairage (Augustini et al. 1980; Fabiansson et al. 1984; Wajda & Wichlacz 1984). The mixing of unfamiliar animals, especially bulls, is a major cause of DFD meat. Mounting behaviour has been implicated (Bartos et al. 1993). Mixing also causes fighting in adult male cattle (Kenny & Tarrant 1987b) and six-month-old calves (Trunkfield & Broom 1991), which may increase the incidence of DFD beef (Knowles 1999). DFD meat is also seen in cattle which fall during transportation (Warnock et al. 1978; Tarrant et al. 1992). In addition, sharp temperature fluctuations or temperature extremes 24 to 72 hours before slaughter have been shown to increase the incidence of DFD meat (Scanga et al. 1998).

Plasma CK has been found to be elevated after journeys of 15 hours (Warriss et al. (1995) and 24 hours (Tarrant et al. 1992), indicating physical fatigue. Cattle remain standing during short journeys (Kenny & Tarrant 1987a,b; Tarrant et al. 1988), but begin to lie down after about 16 hours of transportation, if the stocking density permits them to do so (Tarrant et al. 1992).

Weight loss and dehydration also occur during long journeys. Weight loss during the first 12 hours is primarily due to the loss of gut contents. Thereafter, weight loss continues, but at a slower rate (Knowles 1999). Dehydration has been observed after 18 hour (Lambooij & Hulsegge 1988) and 24 hour (Tarrant et al. 1992; Warriss et al. 1995) journeys. On arrival, animals drank water at the first opportunity (Lambooij & Hulsegge 1988; Tarrant et al. 1992).


**Immunomodulation**

The transportation of calves usually produces a neutrophilia (Simensen et al. 1980; Blecha et al. 1984; Kent & Ewbank 1986b; Murata et al. 1987; Kegley et al. 1997), characteristic of a glucocorticoid response. It sometimes also results in a lymphopaenia (Kent & Ewbank 1986b; Kegley et al. 1997), but this is not always the case (Simensen et al. 1980; Blecha et al. 1984; Murata et al. 1987). Some studies have also found that ACTH injection produces a neutrophilia without lymphopaenia in cattle (Simensen et al. 1980; Roth et al. 1982). The reason for this apparent lack of lymphopaenia is unclear, but may be due to a difficulty in distinguishing lymphocytes from monocytes under light microscopy (Hammer & Weber 1974).

Transportation generally suppresses the cell mediated immune response. Several studies have found a reduction in the mitogen-induced proliferation of lymphocytes

following transportation (Kelley et al. 1981; Blecha et al. 1984). Murata et al. (1987) found a reduction in the spontaneous proliferation of lymphocytes, but no change in mitogen-induced proliferation. Only Murata et al. (1985) have observed an enhanced rate of proliferation, and this followed a very short journey of one hour's duration. The contact sensitivity response to agents applied to the skin has been more variable. Transportation followed by simulated marketing, in which cattle were penned and fasted for 17 hours, reduced the inflammatory response to dinitrochlorobenzene (DNCB) (Kegley et al. 1997), but transportation alone had no effect upon the response to phytohaemagglutinin (PHA) (Blecha et al. 1984).

The effects of transportation upon the humoral immune response have been very variable. Transportation has been found to reduce the synthesis of antibodies to *Salmonella dublin* and to horse red blood cells (HRBC) (Hartmann et al. 1976). However, the transportation of weaned beef calves, followed by 17 hours of simulated marketing, had no effect upon antibody responses to infectious bovine rhinotracheitis virus (IBRV) or porcine red blood cells (PRBC) (Kegley et al. 1997), whilst the transportation of weaned and unweaned beef calves, followed by 24 hours of simulated marketing, actually increased the immunoglobulin G (IgG) antibody response to keyhole limpet haemocyanin (KLH) (MacKenzie et al. 1997).

The weaning of beef calves, which often occurs at the same time as transportation, has been shown to reduce the antibody response to PRBC and HRBC, compared with pre-weaning responses (Gwazdauskas et al. 1978). However, MacKenzie et al. (1997) found that weaning calves on the day of transportation had no clear effect upon the IgG response to KLH after transportation, compared with calves weaned two weeks before being transported, and actually increased the immunoglobulin A (IgA) response.

Several studies have investigated the effects of extreme temperatures upon immunocompetence in calves, although most have been concerned with chronic exposure, lasting for a week or more. Such studies cannot elucidate the effects of thermal stress during transportation, but may have a bearing upon the development of shipping fever in an outdoor feedlot.

Kelley (1980) cited one unpublished study in which calves were exposed to a low temperature for 12 hours. The surprising result was an increased lymphocyte count and an enhanced delayed-type hypersensitivity response to *Mycoplasma tuberculosis*. However, the chronic exposure of three-week-old calves to -5°C, for two weeks, reduced the delayed-type hypersensitivity response to *Mycoplasma tuberculosis* and the contact sensitivity responses to DNFB and PHA (Kelley et al. 1982b). The mitogen-induced proliferation of lymphocytes was unaffected (Kelley et al. 1982a).

When dairy cows were exposed to very high temperatures, between 40 and 48°C, for 24 hours in an environmental chamber, the ratio of neutrophils to lymphocytes in their blood was increased (Wegner et al. 1976). Chronic exposure to high temperatures during hot summers does not have this effect, because the cattle have time to acclimatise (Lee et al. 1976; Wegner et al. 1976). The exposure of three-week-old calves to a temperature of 35°C for two weeks, had effects upon cell mediated immunity which were similar to low temperatures. There were reductions in the delayed-type hypersensitivity response to *Mycoplasma tuberculosis* and in the contact sensitivity responses to DNFB and PHA (Kelley et al. 1982b), whilst the mitogen-induced proliferation of lymphocytes was unaffected (Kelley et al. 1982a).


**Disease incidence**

Shipping fever, also known pneumonic pasteurellosis, is a common cause of morbidity and death in calves following transportation, particularly in beef calves transported to feedlots in North America. In the USA, it has been estimated that 1% of cattle die from shipping fever (Irwin et al. 1979). It may be responsible for 50% of mortalities in feedlots and 75% of cases of sickness (Edwards 1996). Shipping fever is characterised by fever, dyspnoea and fibrous pneumonia, less often by gastroenteritis and occasionally by internal haemorrhage (Tarrant & Grandin 2000). The disease usually occurs within 10 to 14 days after arrival at the feedlot (Radostits et al. 2000).

Shipping fever is caused primarily by the bacterium *Pasteurella haemolytica*, or occasionally *Pasteurella multocida*, but infection with this organism alone is not sufficient to produce the disease in the laboratory (Shoo 1989). It is thought that viruses, such as bovine parainfluenza-1 virus and infectious bovine rhinotracheitis virus, or mycoplasmas may act synergistically to render *Pasteurella* sp. pathogenic and that environmental stressors may also be involved (Hoerlein 1980; Radostits et al. 2000). It has often been proposed that stress caused by transportation renders calves susceptible to shipping fever (Hails 1978; Irwin et al. 1979; Yates 1982; Tarrant & Grandin 2000). The evidence for this is largely circumstantial: in the first place, outbreaks commonly follow transportation (Radostits et al. 2000); and in the second place, transportation may cause physiological stress and immunosuppression (see above). Frank & Smith (1983) have shown that the prevalence of Pasteurella haemolytica in the nasal passages of calves is increased by transportation and marketing, but the presence of *Pasteurella haemolytica* in the nasal passages is not sufficient to cause pneumonia. It must first invade the lungs, a process which is probably facilitated by a simultaneous viral infection.

Several large-scale field studies have monitored the incidence of mortality from shipping fever in beef calves arriving at North American feedlots, over a three year (Martin et al. 1980; 1981; 1982) or a four year (Ribble et al. 1995a) period, and related this to the distance travelled from market. Journey distances ranged from 90 km to 1,326 km in Ribble et al.'s study and from several hundred kilometres to about 3200 km in Martin et al.'s studies. In both cases there was no relationship between mortality and journey distance. The studies by Martin et al. also monitored the incidence of morbidity from shipping fever and again found no relationship with journey time. These findings demonstrate that neither long journeys *per se*., nor factors which which increase with journey time, such as fatigue, hunger and dehydration, play a significant role in the aetiology of shipping fever.

A number of studies have identified mixing as an important factor, although it remains unclear whether this is due to the stress associated with agonistic interactions, or simply to the increased risk of exposure to pathogens. Martin et al. (1981), Martin et al. (1982) and Alexander et al. (1989) measured the mixing of calves from various truckloads after arrival at the feedlot. They found that increased mixing at the feedlot increased the incidence of treatment for respiratory tract disease. Ribble et al. (1995b) traced calves arriving at the feedlot to their farms of origin, in order to investigate the relationship between the level of mixing before arrival and mortality from shipping fever at the feedlot. They found that the level of mixing, given by the mean number of calves per farm in a given truckload, varied depending on the geographical origin of the animals and that feedlot mortality increased with the level of mixing.

However, mixing is not the only factor affecting the incidence of shipping fever. This was apparent in Ribble et al.'s (1995b) study from the fact that mixing could not account for the wide variation in mortality which occurred over time, both within and between years. Ribble et al. (1995c) observed that the mortality rate peaked each year in November, several weeks after the number of calves passing through the markets had peaked. This could have been due to a number of factors, including: crowding in the market lairage, associated with stress or exposure to pathogens; reduced efficiency in the processing of calves arriving at the feedlot; or cold and wet weather. An additional factor, which may have contributed to the downturn in mortality after November, is that many of the calves arriving in December had been weaned before leaving the farm of origin.

There is some evidence that low temperatures and high humidity, both during transportation and on the rearing unit, may increase the incidence of shipping fever (Radostits et al. 2000). Staples & Haugse (1974) observed that mortality in calves of less than 6 months of age, during the weeks following their arrival at the rearing unit, was higher in the winter months than at other times of year. The principle causes of death were pneumonia and scours. These are also the primary causes of mortality in

calves of this age which are not transported (Oxender et al. 1973) The on-farm mortality of such calves is highest in the winter (Martin et al 1975b) and is associated with cold, wet and windy weather and with temperature fluctuations (Martin et al. 1975a,c). Jennings & Glover (1952) have demonstrated that the wetting of calves to create a chill increases the severity of pneumonic lesions in animals experimentally infected with tissues taken from calves exhibiting pneumonia. Another study, by Diesel et al. (1991), has shown that exposure to low temperatures increases the colonisation of the lung by *Pasteurella haemolytica* introduced intranasally. Furthermore, Hambdy et al. (1963) showed that acute exposure to a high temperature followed by a low temperature increased the severity of pneumonia in calves innoculated with *Pasteurella* spp. and bovine parainfluenza-1 virus. The immunomodulation caused by exposure to extreme temperatures (see above) may play an important role in the development of shipping fever.

### 8.1.3.2. Transportation of sheep

**General**

Within Europe, most sheep are transported by road and are destined for slaughter, whether as meat lambs or as adults (Figs. 3,4). Lambs are subjected to a series of potentially stressful events, sometimes within the space of a few days. They are weaned from the dam and transported first to market and then possibly to an exporter's premises; they are mixed with lambs from other farms, weighed, handled for assessment of conformation, ear tagged and clipped; and finally transported again, either for immediate slaughter or for several weeks fattening before slaughter (Hall & Bradshaw 1998). Research has mostly been concerned with assessing the effects of road journeys. The combination of events which occur during the commercial transportation of lambs has not been investigated (Hall & Bradshaw 1998).

A number of studies have also focused upon the transportation of sheep by sea, from Australia and New Zealand to the Middle East. Approximately 7 million sheep are transported from Australia to the Middle East each year (Bailey & Fortune 1992). A significant proportion of these are adult Merino sheep, between three and five years of age, although the number of younger animals is increasing (Kelly 1988). In contrast, most of the sheep exported from New Zealand are lambs, less than a year old (Black et al. 1994). In both countries, sheep are transported by road from their farms of origin before assembly at the port feedlot.

**Mortality**

The mortality rate of sheep transported by road is relatively low. Within the UK, Knowles et al. (1994a) observed a mortality rate of 0.018% in sheep transported to slaughter.

A considerably higher rate of mortality has been estimated to occur at port feedlots in Australia, following road transportation. Feedlot managers estimated a mortality rate of around 1% for an average 7-day assembly period (Richards et al. 1989). The higher rate of mortality may be largely due to the fact that the animals are not slaughtered on arrival, giving diseases time to develop. Richards et al. (1989) found that the the most common causes of death at the feedlot, in descending order, were salmonellosis (53.4%), miscellaneous diseases (23.8%), physical injury (12.6%) and inanition (10.2%).

Transportation by sea is also associated with a high rate of mortality. The average mortality rate on voyages from Australia to the Middle East is 2% (Kelly 1988). Richards et al. (1989) found that the main causes of death, in descending order, were inanition (43.4%), salmonellosis (20.2%) and physical injury (10.6%). An unpublished survey by Ryan, cited in Black et al. (1994) indicated that the average mortality rate for voyages from New Zealand was also 2%, with the mortality on individual voyages reaching 5 to 10%. Black et al. (1994), observed that the most frequent causes of death, in descending order, were suffocation from smothering (31%), inanition (28%), pneumonia (25%) and dehydration (9%). Salmonellosis was not observed in this study, but was seen in a subsequent study by Black (1996) and is not uncommon (Ryan, cited in Black et al. 1994).

**Physical injury**

In Richards et al.'s (1989) study, physical injuries which caused mortality on board ship were mostly acute injuries, associated with splaying of the hind limbs on slippery floors during loading. Most of the injuries which caused mortality in the feedlot were acute or subacute and were probably sustained during transportation from the farms of origin.

"Trauma and other causes" accounted for 7% of deaths in Black et al.'s (1994) study. However, the commonest cause of death in lambs was suffocation from smothering. This was a consequence of competition for resources, such as food and water, which were not provided *ad libitum*. Pushing and plunging (lifting of the forefeet from the ground and lunging forward) would sometimes cause a loss of footing, resulting in suffocation beneath other animals.

**Inanition**

In sheep transported long distances by sea, inanition is an important cause of mortality. Inanition occurs for different reasons in adult sheep and lambs. In lambs transported from New Zealand, it is a consequence of competition for feed (Black et al. 1994), which is not provided *ad libitum* during sea voyages to the Middle East. A minority of animals stop feeding altogether under these circumstances. If moved to less crowded pens, feeding normally resumes.

In adult Merino sheep transported from Australia, deaths from inanition are instead a consequence of inappetance (Norris et al. 1990). Failure to eat is seen first in the feedlot and, in a minority of animals, persists during the voyage. The separate penning of non-feeders on board ship makes no difference to their mortality. Furthermore, when Norris et al. (1990) took sheep from the port feedlot and housed them for closer observation, they found that providing groups of non-feeders with extra trough space or *ad libitum* feed made no difference to their feeding, growth or mortality. The reason for inappetance is unclear, but it has been observed that the mortality rate is much higher in sheep originating on some farms than on others. In two studies, half of all deaths aboard ship occurred in sheep from 25% (Bailey & Fortune 1992) and 14.2% (Higgs et al. 1999) of the farms of origin. Since inanition and salmonellosis are the most common causes of death on such voyages (Richards et al. 1989) and salmonellosis is generally a consequence of failure to eat (see below), it follows that inappetance is more prevalent among sheep from some farms than others. Bailey & Fortune (1992) suggested that inappetance could be related to prior handling and feeding experience. Merinos are reared extensively, with minimal handling and usually no supplementary feeding except when young. Hence, they may find handling and transportation very stressful and may also be reluctant to eat the pelleted feed which is offered to them at the feedlot and on board ship. With respect to feeding experience, it is well established that the exposure of sheep to a particular type of feed while young is an effective method of ensuring that they will accept it later in life (Mottershead et al. 1985; Burritt & Provenza 1989). Higgs et al. (1999) observed that mortality rate was higher amongst sheep originating in zones with a longer pasture-growing season, where animals might have had less experience of supplementary feeding.

**Physiological measures of welfare**

Loading sheep onto a lorry will, to a greater or lesser extent, result in poor welfare. It has been found in several studies that loading caused an elevation in plasma cortisol concentration (Broom et al. 1996; Parrott et al. 1998b), although this was not observed by Cockram et al. (1996). Loading has also been reported to increase the

plasma creatine kinase (CK) concentration in fleeced sheep (Cockram et al. 1996; Parrott et al. 1998b), but not in shorn sheep (Parrott et al. 1998b). The release of CK into the blood is associated with tissue damage or vigourous exercise.

Mixing with unfamiliar animals leads to an increased heart rate (Baldock & Sibly 1990), but is probably not a prolonged problem for sheep during transportation (Hall & Bradshaw 1998).

Plasma cortisol concentration also increases at the onset of driving (Broom et al. 1996; Cockram et al. 1996; Parrott et al. 1998a,b), but declines during the course of the journey (Broom et al. 1996; Cockram et al. 1996; Parrott et al. 1998a), at least when road conditions are good (Parrott et al. 1998b). Parrott et al. (1998a) observed an elevation for only the first 2 hours of a 3.25 hour journey and Broom et al. (1996) observed an elevation for the first 3 hours of a 15 hour journey. This suggests that sheep readily adapt to motion in a carefully driven vehicle. However, when the lorry moved from major roads onto minor roads during the course of the journey, this caused cortisol levels to increase (Parrott et al. 1998b). Bradshaw et al. (1996) have also observed a higher plasma cortisol concentration in sheep during rough journeys compared with smooth journeys.

Extensively raised sheep have been observed to exhibit greater cortisol responses to transportation than sheep housed indoors (Reid & Mills 1962). This may be due to the fact that extensively raised animals are less accustomed to handling. However, breed differences also exist. Hall et al. (1998) have shown that the plasma cortisol concentration, following 45 to 90 minutes of transportation, is greater in sheep of predominantly upland genotype, which are traditionally raised extensively, than in sheep of predominantly lowland genotype, which are normally reared with more human contact.

Sheep lose weight during road transportation, most of which is probably due to the loss of gut contents associated with feed and water deprivation. Experiments which have compared the weight loss in sheep during transportation with the weight loss caused by food and water deprivation alone have had variable findings. Knowles et al. (1995) found an 8% weight loss after 24 hours of transportation, mainly incurred during the first 15 hours, and the same loss in sheep held in pens for 24 hours without food and water. In contrast, Knowles et al. (1993) found a 6.7% loss in sheep transported for 14 or 24 hours, but only a 1.5% loss in animals held in pens for 24 hours, suggesting that much of the weight loss observed during transportation could not be accounted for by feed and water deprivation. However, it is not clear what other factors might have been responsible. Catabolic activity associated with physical exertion and physiological stress could not have resulted in such a substantial difference in weight loss. Even during a 12 day period of starvation, the daily loss of

body tissue through catabolism is inconsequential in relation to total body weight (Smith et al. 1938). Other studies have observed weight losses of 4% after 18 hours transportation (Knowles et al. 1994b) and 5.7% after 14 hours confinement in a stationary vehicle (Hall et al. 1997).

Sheep do not suffer from dehydration during road journeys of up to 31 hours duration. Packed cell volume (PCV) has been observed to decrease, not increase, during the course of most journeys (Knowles et al. 1993; Broom et al. 1996; Parrott et al. 1998b). Similarly, plasma osmolality declines or stays the same (Knowles et al. 1993; 1994b; 1995; Broom et al. 1996; Parrott et al. 1998b). After unloading, most sheep eat before drinking (Knowles et al. 1994b; 1995; Cockram et al. 1996; Parrott et al. 1998b). In fact, Parrott et al. (1996) have shown that plasma osmolality is unaffected by 48 hours of food and water deprivation. This is probably due to the fact that sheep have a sizable reserve of water in the rumen (Silanikove 1994).

Dehydration is an important cause of mortality in lambs transported by sea from New Zealand to the Middle East. Dehydration is associated with competition for water, which is not provided *ad libitum*, and with heat stress. Heat stress is exacerbated by: poor air movement (reduced convective cooling); high salt content of feed and water, which increases urination, resulting in a wet floor and hence a high humidity (reduced evaporative cooling); and high stocking density (increased heat production and floor wetting) (Black et al. 1994).


**Immunomodulation**

No experimental studies have investigated the effects of transportation upon the immune system of sheep.


**Disease incidence**

Salmonellosis causes mortalities in both adult sheep and lambs transported by sea, but is particularly important in adult sheep. It is caused by bacteria of *Salmonella sp.*. Richards et al. (1993) reported that the pathogen most commonly isolated at necropsy from salmonellosis mortalities on board ship was *Salmonella typhimurium*. Other common isolates were *Salmonella bovis-morbificans* and *Salmonella havana*. However, epidemiological studies have revealed that *Salmonella* challenge alone is generally insufficient for the development of salmonellosis during transportation and that it is usually caused by a combination of *Salmonella* challenge and failure to eat (Norris et al. 1990; Higgs et al. 1993). Higgs et al. (1993) observed that deaths from salmonellosis were not spatially or temporally clustered on board ship, as would have been expected if pathogen challenge had been a sufficient cause. Furthermore,

*Salmonella* excretion in the faeces was sufficiently widespread on some journeys that most animals would have been exposed, yet only a small minority of animals were infected with salmonellosis. In Norris et al.'s (1990) study, mortality from salmonellosis during the voyage was very much higher in sheep which had failed to eat at the feedlot (8.8%) than in those which had eaten (0.6%), suggesting that failure to eat predisposed animals to the disease. This was confirmed by Higgs et al. (1993), who removed sheep from the port feedlot and housed them for closer observation. Only those animals who failed to feed developed salmonellosis.

In lambs transported by sea from New Zealand, salmonellosis also appears to be caused by a combination of *Salmonella* challenge and failure to eat. Black (1996) observed that almost all lambs exhibiting salmonellosis lesions at necropsy also exhibited signs of inanition. The incidence of primary salmonellosis was very low.

The connection between salmonellosis and failure to eat in adult sheep means that the factors responsible for inappetence in these animals are also risk factors for the development of salmonellosis. Thus, either the stress of handling road transportation, or the response to a novel feedstuff, or both contribute to the incidence of salmonellosis at the port feedlot and at sea.

Pneumonia in sheep often involves *Pasteurella haemolytica*. As in cattle, pneumonic pasteurellosis in sheep is an important disease worldwide and is frequently seen in ranches and feedlots in the USA (Brogden et al. 1998), not just during long sea voyages.

The aetiology of pneumonic pasteurellosis in sheep is similar to that of the condition in cattle. Attempts to produce pneumonia experimentally by *Pasteurella haemolytica* challenge alone have had inconsistent results (Gilmour et al. 1980). It is possible that stress or initial infections with viral or primary bacterial agents break down innate pulmonary immune barriers. This may release *Pasteurella haemolytica* from its usual commensal status in the nasopharynx and allow it to colonize and proliferate throughout the upper respiratory tract and induce tissue damage in the lung (Brogden et al. 1998). Various environmental challenges have been reported to predispose sheep to respiratory infections, but experimental and epidemiological investigations have not been carried out. Respiratory viral infections are known to increase the susceptibility of sheep to secondary bacterial infection, usually by interfering with mucociliary clearance mechanisms, which remove organisms from the lower respiratory tract (Jakab 1982). Experimental studies infecting sheep first with virus and then with *Pasteurella haemolytica* have shown that parainfluenza-3 virus (Davies et al. 1981; Cutlip et al. 1993), bovine respiratory syncytial virus (Al-Darraji et al. 1982a,b,c; Sharma & Woldehiwet 1990) and an ovine adenovirus, OAV-6 (Lehmkuhl et al. 1989; Cutlip et al. 1996) can all increase the susceptibility of sheep to

*Pasteurella haemolytica*. Some respiratory bacterial infections also increase the susceptibility of sheep to secondary *Pasteurella haemolytica* infection, including *Mycoplasma ovipneumoniae* (Jones et al. 1982).

## 8.2. Abnormal behaviour and physical injury

### 8.2.1. General principles

Abnormal behaviours which are potentially injurious include redirected behaviours, stereotypies and heightened aggression.

#### 8.2.1.1. Redirected behaviours

Animals have evolved motivational mechanisms which govern their behavioural decisions, helping to ensure that their actions are consistent with biological priorities. The brain monitors changes in the animal's internal and external environments, interprets them in the light of experience and generates internal variables known as causal factors which predispose or motivate the animal to follow various courses of action. The animal's motivational state at a given time is determined by the levels of a combination of competing causal factors. Which course of action the animal takes will depend on the relative magnitude of these factors. By influencing the weighting which the brain gives to various changes in the animal's internal and external environments when it generates causal factors, natural selection and learning both hone the animal's behaviour to better meet its biological priorities. (Broom 1981; Fraser & Broom 1990)

Ultimately, an animal's biological objectives are to survive and reproduce. In order to achieve these objectives, the animal must procure many resources such as food, water, warmth, a mate and a nest. The satisfaction of some motivations appears to be solely or primarily dependent upon obtaining or consuming a resource. For example, pre-parturient sows, who show a strong motivation to build a nest from straw when housed on a bare concrete floor (Arey 1992), exhibit very little nest-building behaviour when they are given a comfortable water bed on which to farrow (Baxter & Robertson, cited by Dawkins 1990). Another example is thermoregulation. Thermoregulatory behaviour serves the objective of maintaining a constant body temperature and does not occur in thermoneutral environments.

However, species have also evolved to procure resources in specific ways, to the extent that the performance of species-specific appetitive behaviour patterns is often motivationally important. In fact, what is required to satisfy many motivations appears to be a combination of the behaviour itself and its outcome. What the animal

is motivated for in this case is best described as adequate feedback from the performance of a species-specific behaviour pattern.

The evidence that animals are motivated to perform many species-specific behaviour patterns includes the persistence of these activities when the resources which they would normally procure are made available for free. For example, foraging behaviours persist in many species when food is freely available, indicating that they are motivated to forage as well as to eat. Thus, growing pigs, housed in concrete pens with food pellets available *ad libitum,* will direct additional rooting and chewing behaviour toward straw (Fraser et al. 1991) or a trough of earth (Wood-Gush & Beilharz 1983). Similarly, starlings prefer to search for mealworms than to eat them straight from a dish (Inglis & Ferguson 1986). Persistence is also seen in activities other than foraging. Unlike sows, chickens are motivated to perform nest-building behaviour as well as to lie in a comfortable nest. In fact, they exhibit as much pre-laying nest-building behaviour in the presence of a nest which they have built on a previous day as they do when building one from scratch (Hughes et al, 1989).

The importance of adequate feedback from most of these behaviour patterns is evident from the fact that few behaviours are performed in the absence of feedback of some kind. When this does occur, the behaviour is said to be expressed as a vacuum activity. For example, both chickens (G. Martin 1975; Vestergaard 1981) and junglefowl (Vestergaard et al. 1990), the ancestral stock of the domestic chicken, perform the movements of dustbathing when housed in wire cages, although it is possible that these birds were obtaining some limited feedback from their feed or from the feathers of other birds (Vestergaard 1990).

In some cases, the motivation of animals to persist in performing such activities is very strong. Motivational strength is estimated using preference tests, in which animals are required either to choose between several alternatives, or to perform a task in order to obtain a desired outcome (Broom & Johnson 1993; Fraser & Matthews 1997). Experimental studies of this kind have revealed, for example, that caged hens are prepared to pay as much for access to a nest-box before laying, in which nesting behaviours can be performed with some feedback, as they are for access to food after 20 hours of food deprivation (Duncan & Kite 1987).

The environments of captive animals are often very different from the environments in which their species evolved. They may lack suitable substrates for the performance of important behaviour patterns, such as foraging and nest-building. Because many motivations are tied genetically to species-specific behaviour patterns, there is a limit to how much an animal's behaviour can adapt such environments. Despite domestication, many species of mammals and birds remain motivated to perform activities which, although important in the wild, are biologically redundant in the

captive environment. At the same time, the feedback which the captive environment provides from these activities is often inadequate. This means that in a captive environment, animals will often be unable to obtain adequate feedback from the performance of species-specific behaviour patterns, with negative consequences for their welfare (Dawkins 1990).

When the natural target or substrate of a behaviour pattern is absent from the environment, the behaviour will be redirected, if possible, toward a different target or substrate. The feedback which is obtained when a behaviour is redirected will generally fail to fully satisfy the animal's motivation, but is better than no feedback at all. For example, when growing pigs are housed in concrete pens without access to a natural foraging substrate such as earth or straw, chewing behaviour is often redirected toward their penmates in the form of tail-biting (van Putten 1969). In early-weaned dairy calves fed milk replacer from a bucket, sucking behaviour is redirected toward a non-nutritive teat if one is provided (Hammell et al. 1988), or toward pen fixtures and other calves if one is not (Metz 1984). Chickens housed in cages often exhibit feather-pecking, which Blokhuis (1986) has argued is a redirected foraging activity, although Vestergaard (1989) has suggested that it might instead by a redirection of dust-bathing.

The evidence that the feedback obtained when a behaviour is redirected is generally inadequate to satisfy an animal's motivation is that most redirected behaviours are hardly ever observed when the natural substrates of these behaviours are available in the environment. Thus, although there is nothing to prevent animals from performing activities such as tail-biting, cross-sucking, or feather pecking in the presence of straw, the dam, or litter, respectively, they very rarely do so. It follows that the feedback obtained from the redirected form of the behaviour must be less satisfying than the natural type of feedback. Provided that the animal in the barren environment does not compensate by performing the redirected form of the behaviour pattern more frequently than it would carry out the natural form in an enriched environment, it's welfare can be said to be poorer in the barren environment. Where frequencies have been compared, the frequency of performing the redirected form in a barren environment has generally been found to be lower (eg. Vestergaard et al. 1990, for dustbathing in junglefowl) and at most no higher (Signoret et al. 1995, for foraging in pigs) than that of the normal form in an enriched environment.

There is no *a priori* reason why natural substrates should be preferred to alternatives, and sometimes they are not. For example, deep litter appears to provide supernormal feedback to nesting chickens (Duncan & Kite 1989), which in feral conditions line their nests rather sparsely with vegetation (Duncan et al. 1978). Rather, a general preference for natural substrates over available alternatives is something which has been observed in practice. It arises from the fact that the available alternatives in

many captive environments are very poor. In many intensive housing systems, the only substrates which can provide any kind of feedback from activities such as foraging are the pen fixtures and other animals.

As well as being associated with poor welfare, some redirected behaviours put the health of animals at risk, either that of the individual performing the behaviour or that of other animals in the group. For example, tail-biting in pigs and cross-sucking in calves (Sambraus 1985a; Fraser & Broom 1990).


**8.2.1.2. Stereotypies**

Another type of behaviour associated with captive environments is stereotypic behaviour, a stereotypy is defined as "*a repeated, relatively invariate sequence of movements which has no obvious function*" (Broom & Johnson 1993) (Fig. 5). Despite this widely accepted definition, it is in fact unclear whether or not stereotypies are functional: they may or may not help animals to cope with their environments (Dantzer 1986; Mason 1991b; Rushen 1993). Like redirected behaviours, stereotypies are most often observed when animals are confined and when there are constraints on their ability to perform certain behaviour patterns (Mason 1991a). Stereotypies are believed to result from the frustration of specific motivations. In restrictive environments, strongly motivated behaviours gradually become modified or "channelled" into a few simple behavioural elements, which exhibit very little variability (Lawrence & Terlouw 1993; Rushen et al. 1993).

Stereotypies sometimes arise from the redirection of an appetitive behaviour pattern, such as foraging, and it may be difficult in such cases to distinguish stereotypic behaviour from redirected behaviour. For example, both spot-picking in caged canaries, which is a redirected foraging activity (Keiper 1969), and belly-nosing in weaner pigs, which is a redirection of the motivation to suckle (van Putten & Dammers 1976), are unvarying, repetitive behaviour patterns. Other stereotypies are a consequence of feed restriction and probably have more to do with hunger than with a need to perform foraging behaviour. For example, postprandial stereotypies in sows, including chain manipulation, sham chewing, drinker manipulation and excessive drinking (Terlouw et al. 1991; Lawrence & Terlouw 1993). A third group of stereotypies develop from frustrated escape attempts, such as the stereotypic pacing observed before laying in caged hens with no access to a nest-box (Mills & Wood-Gush 1985).

Whether stereotypic behaviour is a coping attempt or a behaviour pathology, its occurrence indicates that an animal has some difficulty coping with its environment and it is therefore an indicator of poor welfare (Broom & Johnson 1993). Some

stereotypies also increase the risk of injury or other disease in that individual or in others (Sambraus 1985b; Fraser & Broom 1990).

### 8.2.1.3. Heightened aggression

A third type of abnormal behaviour which is potentially injurious is heightened aggression. Like redirected behaviour and stereotypy, heightened aggression is characteristic of intensive husbandry systems. It is either a coping mechanism or a behavioural pathology and indicates that the welfare of the aggressive individual is poor. It has a number of different causes, including: inability to form a stable social group; inability to resolve conflicts; regrouping; and the frustration of specific motivations, especially feeding.

Many social species, including chickens, pigs and cattle, normally establish a dominance order within the group, which governs access to resources and minimises the incidence of fighting. However, when group size is large, so that individual recognition of all group members is not possible, a stable social hierarchy cannot develop. This may result in ongoing aggression, manifest as aggressive pecking (directed at the head) in laying hens (Appleby et al. 1992), as fighting in pigs (Petherick & Blackshaw 1987) and as mounting in steers (Fraser & Broom 1990). Estimates of the number of group members who can be recognised or remembered by an individual, based on observations of the point at which social disruption begins to occur, are about 80 in chickens (Guhl 1953), 50 to 70 in cattle and 20 to 30 in pigs (Fraser & Broom 1990).

In some housing systems, it may be difficult for animals to resolve conflicts, even when group size does not prevent individual recognition. This is most apparent in pigs, in whom submission is communicated not by means of a postural signal, but by fleeing. When groups of growing pigs are confined in small, bare pens, escape is not possible and this may prolong bouts of aggression. The problem is exacerbated by a high stocking density, since crowding makes it difficult for penmates to avoid encroaching on each others' individual space, leading to more agonistic encounters (Ewbank & Bryant 1972). The incidence of aggression can be reduced by construction of partitions within the pen, which permit fleeing animals to move out of sight (Nehring 1981). The provision of 'head-hides', where an animal can place its head out of sight, also reduces aggression, because the head is the focus of aggressive biting (McGlone & Curtis 1985). Unresolved conflict is also responsible for high levels of aggression between sows when they are housed individually, but in close proximity to one another, in tether- and crate-stalls (Barnett et al. 1989).

Regrouping results in vigorous fighting in pigs (Meese & Ewbank 1973), aggressive pecking in chickens (Guhl 1953) and fighting in male adult cattle (Kenny & Tarrant

1987b) and six-month-old calves (Trunkfield & Broom 1991). Fighting is seen less often in younger calves. The repeated regrouping of animals is a routine part of many systems of pig and cattle husbandry, so periods of intense fighting are endemic to these systems. Pigs are normally mixed first at weaning, then often again for finishing, and they are subsequently mixed both before and after transportation to the slaughterhouse. Calves from beef suckler herds which are not fattened on site are transported to a rearing unit or feedlot at more than six months of age. They are mixed several times during the process of transportation and marketing, and again on arrival. All cattle are mixed before and after transportation to the slaughterhouse.

Aggression also occurs in response to the frustration of specific motivations, particularly the motivation to feed. Duncan & Wood-Gush (1971) observed aggressive pecking in hens when their motivation to feed was thwarted by a perspex cover over the food container. Carlstead (1986) gave groups of growing pigs signals predicting the delivery of food and observed that the incidence of aggression was higher in groups which received unreliable signals than in groups which received reliable signals. In group-housed sows, vulva biting is associated with the use of a transponder-operated feeding station to feed animals sequentially. It develops when sows are queuing for access to the feeding station and is believed to result from the frustration of a motivation to feed when other sows are feeding. It can be reduced by the simultaneous provision of silage to all sows and by encouraging the formation of sub-groups, both of which reduce competition for the feeding station (Van Putten & Van der Burgwal 1990).

### 8.2.2. Examples listed

In Table 7, a list is presented of redirected behaviours and stereotypies which can result in poor health, as well as several forms of aggressive behaviour whose morphology is distinctive from normal aggression. Several other abnormal behaviours are included, whose causes are as yet unclear, but which are associated with indoor housing or intensive husbandry systems in which welfare is poor and which can result in poor health.

**Table 7.** Redirected behaviours, stereotypies and other abnormal behaviours associated with indoor housing or intensive husbandry systems where welfare is poor, which result in poor health.

| Behaviour | Description | Causes | Health risks |
|---|---|---|---|
| HORSES | | | |

| Wind-sucking and crib-biting | Mouth opened, pharynx contracted and air sucked into upper pharynx. In crib-biting, sucking faciliated by clamping teeth onto some solid object. | Redirected foraging: stabled horses fed concentrates eat their feed quickly. May then gnaw objects, which leads to crib-biting (Schäfer 1974). Boredom. Imitation. | Swallowing air can lead to stomach dilation, bloat, colic and chronic intestinal catarrh. Feed intake may be reduced, leading to nutrient deficiency (Eikmeier 1970). |
|---|---|---|---|
| **Behaviour** | **Description** | **Causes** | **Health risks** |
| Litter-eating | Increasingly indiscriminate consumption of litter, which may be soiled with manure or mouldy. | Redirected foraging: as for wind-sucking (Houpt et al. 1978). Insufficient or imbalanced feed. Worms. | Consumption of soiled or mouldy litter can lead to colic (Summerhays 1973). |
| Polydipsia | Excessive consumption of water. | Not known. Usually seen in confined and isolated animals. | Suspected cause of gastric or intestinal volvulus (twisting), which can be fatal. |
| Flank-biting | Biting at own sides, in the absence of any pathological skin condition, parasitism or gastrointestinal disorder. | Not known, but grazing or provision of a stall companion can ameliorate the disorder (Houpt 1981). | Usually only the hair is damaged, but occasionally results in self-mutilation. |
| Weaving | Head stretched forward and swayed from side to side, whilst body weight is shifted from one forelimb to the other. | Frustration of motivation to move forward. Commonly occurs when horse anticipates feeding or release to pasture. Boredom (Houpt 1981). Imitation. | Stereotyped motion puts great strain on tendons, joints and hooves and can lead to pathology in these tissues. Has resulted in luxation of elbow joint (Summerhays 1973). |
| Pacing | Pacing or circling around a horse box. | Frustration of attempts to escape from confinement. | Considerable spinal flexion is required in circling or turning and can lead to back conditions. |
| Pawing | Fore foot repeatedly moved along the ground | Not known. Usually seen in confined and isolated animals. | Continual pawing on a hard floor can result in leg injury. |
| Stall-kicking | Head lowered, ears pulled back, back arched and stall wall kicked forcefully with hind leg. | Not known. Possibly boredom. May be an attempt to attract attention. | Kicking can cause injury. Splintered wood may also cause injury. |
| CATTLE | | | |
| Self-licking | Long periods spent licking own body. Licking behaviour may be stereotyped in form. | Redirected activity associated with lack of straw (van Putten & Elshof 1978), social contact (Waterhouse 1978), or opportunity to explore (Webster et al. 1985). Also sodium deficiency (Phillips et al. 1999). | Hair balls may clog the rumen, leading to digestive problems (Groth 1978). |

| Cross-sucking | Sucking of the mouth, ears, navel, prepuce, scrotum or udder of other calves. | Redirection of sucking at dam (Metz 1984). Also sodium deficiency (Phillips et al. 1999). | The part of the calf sucked may become inflamed, damaged and infected (Kiley-Worthington 1977). Sucking may lead to hair balls. Also to urine-drinking, which can cause liver disorders. May persist in adult cows as milk-sucking (Wood et al. 1967), which can cause mastitis and teat damage and udder damage. |
|---|---|---|---|
| Buller steer syndrome | Mounting in steers. The 'buller' stands to be mounted by the 'rider'. | Redirection of sexual activity in all-male herds, also related motivationally to aggression. Exacerbated by high stocking density, large group size and mixing. Also by use of female sex hormones (Schake et al. 1979). | Weight loss in 'riders'. Exhaustion, injury or collapse in 'bullers'. |
| SHEEP | | | |
| Tail-biting and hoof-biting | Parturient sheep bite at the tails and hooves of lambs. | Not known, but only occurs when animals are stabled (Hiepe 1970; Brummer 1978) and can be prevented by provision of more space and grazing. Nutritional deficiency may be a factor. | Biting can result in serious physical injury. Also, wounds may become infected. Severe biting of the tail can lead to rectal prolapse, as the affected lambs are apt to strain when defaecating. |
| Wool-eating in lambs | The lamb sucks, chews and ingests wool from the dam's stomach, udder and tail. | Not known, but occurs when animals are stabled. Possibly redirected foraging, or fibre deficiency. | Hair balls form in abomasum. Can result in severe colic and anaemia. Also obstruction, which can be fatal. Flock morbidity up to 10% (Hutyra et al. 1959; Behrens 1962; Hiepe 1970). |
| PIGS | | | |
| Tail-biting | Oral manipulation of the tails of other pigs. Gradually turns into harder biting. | Redirected foraging (van Putten 1969). Triggered by restlessness, associated with poor air quality (van Putten 1969), over-stocking, thermal discomfort and insufficient access to food or water. | Physical injury. Wounds may provoke cannibalism, which can be fatal (van Putten 1969). Bacterial infection may spread through-out the body, via the spinal cord, causing abscesses in the spinal canal, lungs, kidneys and other organs and joint |

| | | | inflammation. |
|---|---|---|---|
| Anal massage | Anus of other pigs massaged with snout. When this triggers defaecation, faeces ingested. | Not known, but may be linked motivationally with tail-biting, occurring when tail-biting is prevented by docking. Anal massage performed alternately with attempts to bite tails, ears or legs, with belly-nosing and with rooting and biting pen fixtures. | Can lead to swelling and wounds in the area of the anus. Afflicted pigs may become weak, with reduced food intake and growth. |
| Belly-nosing | Snout rubbed up and down against belly of other pigs and against the soft tissue between their hind legs or fore legs. | Redirection of udder massage (Fraser 1978). Also linked with foraging, since incidence reduced by providing straw (Schouten 1986). | May cause inflammation of nipples, umbilicus, penis or scrotum. |
| Cronism | Sows kill and eat viable piglets. | Associated with hyper-excitability. Suspected causes include: lack of straw for nest-building; insufficient time to acclimatise to a new environment; pain in uterus or udder (Boothroyd 1965). | Death of piglets. |
| Head-rubbing | Top of snout rubbed repetitively and vigorously along the underside of a bar across the front of the stall. | Not known. Only seen in sow stalls. | Rubbing may be sufficiently vigorous that the head is repeatedly bumped against side of stall at end of each rub. Can result in serious injury. |
| Vulva-biting | Vulva of another sow bitten with incisors. | Frustration of a motivation to feed at the same time as other sows. Develops when sows queue for access to a feeding station. (Van Putten & Van der Burgwal 1990.) | A single bite can cause considerable damage to vulva. Once damaged, it is likely to be bitten again. The wound usually becomes infected. (Van Putten & Van der Burgwal 1990.) |
| CHICKENS | | | |
| Feather-pecking | Feathers pulled out, from the back, stomach, breast, tail and wings of other birds, and consumed. | Redirected ground-pecking, which is a foraging activity (Blokhuis 1986) and a dust-bathing activity (Vestergaard 1989). Exacerbated by: over-stocking; insufficient space at troughs; too much light; presence of sick birds. | Physical injury. Wounds may provoke cannibalism (body-pecking), leading to death of the afflicted bird. |
| Toe-pecking | Pecking directed at toes | Redirected ground- | Physical injury. |

| | of other birds, or occasionally at own toes. | pecking. | Infection of wounds causes weakness, reduced food intake and growth. |
|---|---|---|---|

Statements in this table which are not referenced come from Sambraus (1985a), Sambraus (1985b) or Fraser & Broom (1990).

It can be seen from Table 7 that a large number of potentially injurious abnormal behaviours are observed in domestic animals when their welfare is poor.

### 8.2.3. Example in detail

One example is discussed in more detail, that of redirected sucking in calves.

### 8.2.3.1. Sucking in calves

**General**

Dairy calves reared for beef or veal are removed from the dam at one or two days of age and housed individually or in groups. They are fed colostrum, then whole milk, until six days of age, after which they receive milk replacer. Calves reared for beef are weaned at four to twelve weeks of age, usually between seven and nine weeks. Calves raised for white veal are not weaned.

Milk replacer is most commonly fed in restricted quantities from a bucket, twice a day. In some cases, the bucket is fitted with a nipple. Bucket-feeding is used for both individually housed and group housed calves. An alternative system involves feeding milk replacer *ad libitum*. An automatic feeder delivers the milk replacer under pressure to a series of teats. This system is only used for group housed calves.

Calves which are suckled by the dam consume their daily milk intake in around five bouts of ten to fifteen minutes duration each, the number of bouts declining with the age of the calf (Phillips 1993).

**Welfare**

Calves are motivated not only to consume milk, but also to suck. This is evident from the persistence of sucking behaviour when milk is available in an open bucket. For example, Hammell et al. (1988) observed that calves fed *ad libitum* from an open bucket spent 13 minutes per day on average sucking a non-nutritive teat. This was

substantially more than calves fed *ad libitum* from a teat system, which sucked the non-nutritive teat for just 1 minute per day on average. In both cases, use of the non-nutritive teat was temporally associated with bouts of feeding. It occurred before, during and after feeding bouts. The positive affective value of sucking is also evident from the fact that calves which were allowed to feed *ad libitum* from a teat system consumed more milk than calves which fed *ad libitum* from an open bucket.

The persistence of sucking behaviour may have its physiological basis in the enhanced secretion of digestive hormones. De Passillé et al. (1993) observed greater postprandial increases in the plasma concentrations of insulin and cholecystokinin (CCK) when calves were allowed to suck a non-nutritive teat after a meal than when they were not. The postprandial rise in these hormone levels has been implicated in satiety.

Calves are clearly sensitive to the quality of the feedback which a sucking substrate provides. When calves are bucket-fed a restricted quantity of milk replacer and the empty bucket is not removed after feeding, buckets fitted with nipples receive more non-nutritive sucking than open buckets, suggesting that the shape or texture of the sucked object is important. Furthermore, removal of the bucket after feeding results in less non-nutritive sucking, in this case directed at pen fixtures and other calves, than when the empty bucket remains available, suggesting that the taste or smell of milk provides important feedback (Metz 1984).

The sucking of a non-nutritive teat, the bucket, pen fixtures and other calves are all examples of redirected behaviour. The sucking of a non-nutritive teat (Hammell et al. 1988), pen fixtures (Metz 1984) and other calves (Alexander 1954; Geddes 1954; Hoyer & Larkin 1954; Metz 1984) are all very substantially reduced in calves fed *ad libitum* from a teat system. The sucking of pen fixtures and other calves are also greatly reduced in calves remaining with the dam (Metz 1984).

The sucking of other calves, known as cross-sucking, is directed at various parts of the body, including the mouth, ears, navel, prepuce, scrotum and udder. The position while sucking corresponds to that of the calf at the cow. The 'bunting' movements with which the calf prepares the cow's udder are also shown (Sambraus 1985a).


**Health**

Cross-sucking commonly results in the appearance of inflammations on the recipient animal (Sambraus 1985a). The sucked area sometimes becomes damaged and infected (Kiley-Worthington 1977). Recipients also show reduced weight gain (Illes 1964).

Calves which perform cross-sucking may swallow hair, leading to the formation of hair balls (Fraser & Broom 1990). These may clog the rumen, causing digestive

problems (Sambraus 1985a). Sucking of the prepuce may lead to urine drinking (Stephens 1982), which can cause liver disorders (Fraser & Broom 1990).

Cross-sucking is often reduced by tethering calves for an hour following feeding (Fraser & Broom 1990). A more satisfactory solution, from the point of view of the calves' welfare, would be to employ an *ad libitum* teat feeding system, or failing this to fit nipples to the feeding buckets and make non-nutritive teats available.

## 6. Summary

Animal welfare is the subject of rapidly increasing concern in most countries in the world and this concern is resulting in changes in the ways in which farmers and other animal users keep and treat animals. Welfare can be defined in a way which incorporates ideas about needs, feelings, stress and health. The scientific assessment of animal welfare has developed substantially and very many studies of different kinds of animals have been carried out. Information from such studies is used by legislators, food companies and the public with the consequence that the various kinds of regulation lead to real improvements in animal welfare.

Health is defined as *an animal's state as regards its attempts to cope with pathology*, where pathology is *a detrimental derangement of molecules, cells, tissues and functions that occur in living organisms in response to injurious agents or deprivations*. Pathology can be classified into: genetic abnormalities; physical, thermal and chemical injury; infections and infestations; metabolic abnormalities; and nutritional disorders.

Health is a part of welfare. When an animal's health is poor, so is its welfare, but poor welfare does not always imply poor health. There are some measures of poor welfare which are classified as pathology and will therefore also be indicators of poor health, including body damage and symptoms of infectious, metabolic and nutritional disease. Other measures of poor welfare, whilst not being signs of poor health at that time, indicate a risk of poor health in the future. They include immunosuppression and the occurrence of injurious abnormal behaviours. These are causal links between poor welfare and poor health. Two pathways can be identified:

A. chronic activation of physiological coping mechanisms ---> immuno-suppression ---> infectious disease;

B. behavioural coping mechanisms ---> injurious abnormal behaviour ---> physical injury.

The connection between physiological coping mechanisms, immune function and susceptibility to infectious disease is complex. Different environmental challenges elicit different neuroendocrine responses; and different species and individuals may respond differently to a given challenge. Furthermore, a given neuroendocrine response has different effects on different leucocyte populations, with the consequence that susceptibility to some pathogens is enhanced, whilst susceptibility to others is reduced. It is therefore necessary to consider one challenge, one species and one pathogen at a time.

A list is presented of experimental studies in which the effects of specific environmental challenges upon the susceptibility of domestic species to particular pathogens have been investigated. The number of studies which have been conducted is small. The majority of studies have reported an increased susceptibility to disease. This is in agreement with previous reviews, which have included laboratory species.

The transportation of cattle and sheep is reviewed in detail. The transportation of cattle by road usually elicits physiological coping responses and may also result in physical exhaustion, dehydration and injury. The environmental challenges which are responsible for these welfare problems include weaning, rough handling, mixing, poor driving, high stocking density, food and water deprivation and extreme temperatures. Transportation usually suppresses the cell-mediated immune response, but has variable effects on the humoral immune response. Pneumonic pasteurellosis ('shipping fever') is a common cause of mortality in calves which have undergone transportation and it is thought, although not yet proven, that environmental challenges during transportation may play a role. The incidence of pneumonia is increased by mixing and by acute or chronic exposure to cold and wet weather.

The transportation of sheep by road elicits physiological coping responses. Extensively raised breeds exhibit greater cortisol responses than those reared with more human contact. Australian Merino sheep held in port feedlots for one week after road transportation exhibit a mortality rate in the region of 1%, caused by salmonellosis and other diseases, injuries sustained during transportation, and inanition. When these animals are then transported by sea to the Middle East, the mean mortality rate is 2%, due to inanition, salmonellosis and physical injury. Inappetance is responsible for both inanition and salmonellosis in these animals. The cause of inappetance is not known, but may be a consequence either of the stress of handling and transportation, or of unfamiliarity with pelleted feed. A similar level of mortaility is seen in lambs transported by sea from New Zealand to the Middle East, but the causes of death are different. These animals do not show inappetance. Pneumonia is a more important cause of death than salmonellosis. Like 'shipping fever' in cattle, pneumonic pasteurellosis in sheep is thought to be precipitated by

environmental challenges, but there is as yet no experimental evidence for this in sheep.

Abnormal behaviours include redirected behaviours, stereotypies and heightened aggression. The redirection of behaviour is a coping mechanism and hence a sign of poor welfare. Stereotypies and heightened aggression are either coping mechanisms or behavioural pathologies and also indicate that welfare is poor. Some of these behaviours are injurious, either to the animal itself or to other animals in the group. They can therefore lead to poor health.

Abnormal behaviours associated with indoor housing or intensive husbandry systems where welfare is poor, which lead to increased risk of poor health, are listed. These include redirected behaviours, stereotypies and heightened aggression, as well as other abnormal behaviour patterns whose causes are as yet unclear. A large number of potentially injurious abnormal behaviour patterns have been identified.

Redirected sucking in calves is reviewed in detail. Dairy calves reared for beef or veal are separated from the dam at one or two weeks of age and fed milk replacer from a bucket, or, less frequently, from a teat system. Calves are motivated not only to consume milk, but also to suck. Sucking stimulates the secretion of digestive hormones implicated in satiety. When fed from a bucket, sucking is redirected towards other calves. This may lead to the formation of hair balls in the rumen, which can cause digestive problems, and to urine-drinking, which can cause liver disorders. The part of the recipient which is sucked may also become inflamed, damaged or infected.

It is concluded that there are several routes by which poor welfare results in an increase in disease. The pathophysiology of states typified by behavioural abnormalities and emergency physiological responses is a subject which is insufficiently investigated.

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**<u>Legends</u>**

Fig. 1   Sole ulcer in cattle occurs more often in metabolically challenged individuals.
(photograph F.A. Galindo).

Fig. 2   Crowding of pigs predisposes them to various clinical conditions.
(photograph R.H. Bradshaw).

Fig. 3   Transport affects sheep disease incidence.
(photograph S.J.G. Hall).

Fig. 4   Sheep at a transport staging point may encounter many new pathogens
(photograph S.G.J. Hall).

Fig. 5   The drinker-pressing stereotypy in a confined sow.
Stereotypies are indicators of poor welfare.
(photograph D.M. Broom).