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Welfare in relation to feelings, stress and health

D.M. Broom. Department of Veterinary Medicine.- University of Cambridge.- Madingley Road Cambridge CB3 0ES UK

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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.

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.

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.

Introduction

Welfare is a term that refers to animals including man. It 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 that can be used to take decisions about the ethics of a situation.

Welfare refers 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.

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.

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 that 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 El bienestar de un individuo es su estado con relación a sus intentos por afrontar su ambiente** (Broom, 1986). This definition refers to a characteristic of the individual at the time. The question is how well the individual is at a

particular time (for further discussion, see Broom, 1991a,b, 1993, 1996a; Broom & Johnson, 1993, Broom 2001, Broom and Kirkden,2004). 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 that 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.

1. 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.

Welfare and feelings

The feelings of an animal are an extremely important part of its welfare (Broom 1991b,1998). Suffering is a negative unpleasant feeling which should be recognised and prevented wherever possible. However, whilst we have many measures that 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.

Welfare and stress

The word stress should be used for that part of poor welfare that 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.

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.

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. Nevertheless, 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.

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.

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 (Broom in press). 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.

It is important to 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'.

Physiological coping mechanisms and infectious disease

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 □-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. □-endorphin enhances T cell

proliferation *in vitro* (Gilman et al. 1982), while vasopressin and oxytocin both stimulate helper T cells to produce more interferon-□ (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 □-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.

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