CONTRACT REVIEW

THE INFLUENCE OF STRESS ON ANIMAL PRODUCTIVITY

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There is little doubt that the aim of every primary producer is to maximise the efficiency of production, while at the same time maintaining a high quality of product.

In domesticating animals man has systematically selected animals that produce the greatest quantity of a product meeting a specific functional need with the greatest efficiency. These animals have invariably demonstrated the greatest ability to assimilate nutrient substrate into specific target tissues such as wool, muscle, milk or eggs. This greater efficiency may, for example, result from greater responsiveness of cells to anabolic hormones, by lowering the activity of specific enzymes required for growth processes or simply by increasing the efficiency of transport mechanisms for nutrients into cells. All of these factors are designed to preferentially partition nutrients to commercially important tissues. This greater efficiency, however, has not come without cost to the primary producer.

Since contemporary breeds and strains of animals are so responsive to metabolic cues, it is not surprising that they may also have developed a heightened sensitivity to the adverse stimuli that animals are exposed to on a daily basis. These reactions comprise the counter-regulatory mechanisms or stress responses that enable animals to survive when confronted with adverse environments or noxious stimuli. Therefore, although modern day genotypes are highly productive, they may be more responsive to stressful stimuli.

How do animals cope with stress?

There are 3 basic responses that an animal can make in order to alleviate the adverse effects of stress.

1. The simplest responses are behavioural, for example, by moving to another part of the paddock to avoid stressors such as socially dominant animals, or to seek shade. However, in many intensive production systems this is not possible, and therefore the animal is forced to adjust its metabolism.
2. Long term adjustment in response to a stress is dependent on the ability of the brain to receive stress inputs and transmit messages to the adrenal gland via a specific hormonal messenger, adrenocorticotrophic hormone (ACTH), which coordinates the release of glucocorticoids (eg. cortisol). These hormones redirect nutrients to the functionally most important tissues of the body including the brain, heart, liver and kidneys, at the expense of commercially important organs such as the mammary gland, wool follicle, skeletal muscle and the ovary or testis. Some of these tissues may be mobilised to meet the shortfall.
3. The short term responses to stress such as the sudden reaction of an animal to a barking dog, require the activation of the autonomic nervous system controlling adrenaline and noradrenaline secretion from the adrenal medulla, and noradrenaline from sympathetic nerve endings. These hormones are unlikely to influence long term productivity in view of the short term nature of their actions. However, they may exert a dramatic influence on the quality rather than quantity of animal products. For example, their roles in the formation of wool breaks, "dark cutting" meat, anoestrus in breeding animals and the deterioration of milk quality late in lactation are all unknown.

Are all effects of stress hormones necessarily bad?

There is probably no animal in commercial animal production that is not continuously exposed to stress during its productive life. Hormones of the stress axis fine tune the animals metabolism to enable it to function as "normally" as possible within the constraints of these stressors. If the source of these hormones is removed then animals simply die. Therefore there is a "window" of circulating levels necessary to maintain productive processes in commercially important tissues.

*What is the cost **of stress to** the grazier?*

It is the changes in biological function directed by the stress hormones to ensure its survival that comprises the "cost" of stress to the farmer. For example, the extra glucose supplied to tissues such as the brain from the liver and musculature in response to a stress through the actions of the glucocorticoids and catecholamines, is no longer available for production. If the stress is so severe that the animal's adaptive mechanisms are unable to cope, a pathological state develops which, if untreated, will result in the death of the animal.

In this contract we outline problems that have been encountered in various production systems which may be attributed to stress phenomena. Firstly we look at meat quality and problems associated with feedlotting: the next 2 papers address the issue of reproductive inefficiency in high producing dairy herds and in the sheep industry, while the last 2 report on the problems of tender wool and of heat stress in the pig industry. The review has been directed at a range of unrelated production systems in order to demonstrate that similar physiological principles apply across species.

Stress and meat quality

One of the most difficult parameters to assess is the effect of chronic stress on growth and carcase composition of meat animals. Long term stimulation of glucocorticoids is associated with slower growth rates and greater fat deposition, while conversely chronic active immunization of prime lambs against ACTH has decreased the fat content of carcases (Wynn *et al.* 1994). It is difficult, however, to attribute this decrease directly to the fall in circulating cortisol levels since this procedure results in increases in circulating levels of a range of other hormones such as the opiate, β-endorphin and α-melanocyte-stimulating hormone (α-MSH) that are derived from the same large precursor hormone.

How can the prime lamb producer minimize the influence of stress on these parameters? Maintaining the lamb on the ewe undoubtedly buffers the lamb from variation in nutrient availability and protects it from the psychosocial stress of establishing its dominance ranking within the flock. Mixing of different flocks of lambs and ewes should be avoided, while the frequent rotation of flocks between paddocks may result in the persistence of the process of familiarization with a new environment. The movement of stock on hot days and the infrequent use of dogs are also factors that may contribute to heightened activity of the adrenal gland.

Of particular importance is the finding that the persistent exposure of animals to any particular type of stressor which may lead to the animal becoming familiar with that stress provides no protection from the stimulatory activity of other unrelated stressors. Therefore stressors experienced by animals "on farm" will not protect them against the adverse influence of trucking of animals prior to marketing.

Two conditions prevalent in the meat industry and which cost the meat industry $40 million annually are "dark cutting" in beef and lamb carcases, and the pale soft and exudative (PSE), and dark, firm and dry (DFD) carcases found in the pig industry. Both of these syndromes are related to the ultimate pH of meat which is the result of the acidification of meat during rigor development. The degree of acidification is dependent on the conversion of muscle glycogen stores to lactic acid and therefore any stressor that induces glycogen depletion to provide a ready source of energy for the animal will result in dark cutting or DFD pork. The most effective mobilizers of glycogen are adrenaline and strenuous muscle activity, while long term undernutrition will also slowly deplete muscle stores.

However, it is unlikely that there is a simple solution to this problem, since the blockade of muscle adrenergic receptors through which the adrenaline acts has little effect on the incidence of dark cutting (Tarrant 1988). A number of factors including the nutritional status and husbandry and handling practices on farm, transportation, handling and mixing of animals through saleyards, rapid changes in weather patterns and the preslaughter management of animals in abattoirs are thought to contribute to this condition. However, like so many stress induced conditions, it is very difficult to predict its occurrence and therefore difficult for graziers to take precautions to prevent it.

Genotype, however, plays an important role in the incidence of PSE pork, being closely associated with the expression of the halothane gene. These pigs yield carcases with a high lean content, however, the quality of the pork is inferior. The prevalence of this gene provides a classical example of what persistent selection for growth and carcase characteristics can lead to.

The feedlotting of cattle provides an opportunity to control a number of the variables known to be important in the regulation of growth, carcase composition and meat quality. Some of the problems associated with this intensive husbandry system are now outlined.

STRESSORS AFFECTING CATTLE IN COMMERCIAL **FEEDLOTS**

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The management of cattle in commercial feedlots in Australia, particularly those producing high quality beef for export, must be maintained at a high standard because there is little margin for error. In

considering potential stressors in feedlots it is convenient to distinguish the adaptation phase (during the first 30 days in the feedlot) from the routine finishing phase (the remaining 60-270 days in the feedlot). Stressors during the adaptation phase may include transport to the feedlot, mixing with animals from other locations, increased human handling, change of diet, confinement and crowding, and exposure to disease, particularly respiratory pathogens. Stressors which can apply generally are crowding (as it affects personal space requirements), competition for feed, water or shelter, other social interactions (including behavioural anomalies such as the buller steer syndrome), muddy ground (as it affects the animal's ability to rest by lying), dust and climatic variation, particularly excessive heat load due to a combination of high radiation, ambient temperature, humidity and low air movement.

The minimum space requirement for feedlot cattle for optimum production varies from 9-20 m^2/hd in different publications and with size of animals and the prevailing weather conditions. Little is known, however, about the interaction of stocking density with social behaviour of cattle and immune competence (see Fell and Clarke 1993).

There are obvious differences between feedlot and range cattle in feeding behaviour, the amount of walking and the time spent idling, but analysis of the essential elements of maintenance behaviours does not suggest that feedlot cattle are unduly stressed once a stable dominance order has been established. Natural photoperiodic effects on behaviour have been described (Stricklin 1988), and time spent lying and feeding were found to be positively correlated with growth rate (Hicks *et al.* 1989). To date there has been little, if any, measurement of hormonal or immunological parameters which could indicate relationships between more subtle stressors and health and production.

Feedlot cattle cope with the normal range of climatic variation without exhibiting signs of stress. However, the areas in which feedlots are located in Australia have relatively high levels of solar radiation and ambient temperature, and when these are combined with high humidity and low air movement (which only happens occasionally), serious heat stress can occur. Behavioural signs of increasing heat load include alignment with the sun, seeking shade, reduced food intake, crowding near water troughs and crowding to seek shade from other animals. Physiological changes include an initial increase in respiration rate followed, under more severe heat load, by open-mouthed and laboured breathing, indicating a deterioration in the animal's ability to cope and the possibility of irreversible hyperthermia and death.

Treatment in severe cases of excessive heat load is not easy. Water spraying increases evaporative cooling, but also increases humidity. Increased air movement and cooler drinking water are certainly helpful. Preventative measures are recommended such as the use of heat-tolerant breeds, well designed solar shades which promote air movement, cooled drinking water and close monitoring of climatic changes. In some Australian feedlots this now includes automatic monitoring of black globe temperature and humidity to calculate the heat load experienced by the cattle (Fell *et al.* 1993).

During the 1992/93 summer it was found that shade had a demonstrable, but not a dramatic effect on cattle performance (Fell *et al.* 1993), while there was no great effect on the animals welfare. There were improvements in average daily gain and feed conversion efficiency, but no significant effects that could be extrapolated across the industry. Economic analysis of the most industry-applicable responses showed that there were only 2 in which the financial return was sufficient to justify the capital costs involved.

The normal responses to heat in unshaded cattle, such as panting and standing close to water, were significantly reduced in cattle provided with shade indicating that they were able to adjust their heat load simply by seeking shade. These were assessed to be natural adaptive responses (not signs of stress), and therefore not associated with the development of any pathological state, although the energy utilised could be considered as a cost of stress. However, the animal's clear choice of seeking shade suggested that the comfort of the cattle was improved in the shaded pens, particularly in summer.

Further work is needed on the effects of shade under more extreme climatic conditions. However, shade is only one aspect of the management of heat load in feedlot cattle and other factors such as cattle size, condition, coat colour and acclimatisation and site pen surface and air flow characteristics, also need to be considered.

One important aspect of animal production which has been influenced markedly by selection for production traits such as growth rate or yield of meat, milk or wool is reproduction. When highly selected genotypes are subjected to a stressor, often the first and presumably the least essential physiological function for survival that is affected is reproductive performance. The next 2 contributions address these problems in the dairy and sheep industries.

METABOLIC STRESS AND REPRODUCTIVE PERFORMANCE OF DAIRY CATTLE

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The dairy cow provides an extreme example of persistent selection for a single production trait. Over the last 30 years, the widespread adoption of artificial breeding has allowed genetic gains in lactational performance of approximately 1% per annum, resulting in cows capable of producing more than 10,000 L of milk in a 300 day lactation. However, these gains have been made at a cost in reproductive performance within the herd, with major decreases in conception rates being recorded in high producing cows (Lean *et al.* 1989). The challenge is to identify the metabolic/hormonal lesions and compensate for them without compromising lactational performance.

One possible contributory factor is the high circulating levels of growth hormone or bovine somatotropin (bST) observed in high producing cows, which results in elevated plasma ratios of bST to insulin. Many studies have identified the potential for exogenous bST to increase milk production, however, this treatment is also often associated with decreased conception rates. The bST stimulates insulin like growth factor (IGF) synthesis in the liver and peripheral tissues including the ovary which in turn stimulates the growth of small to medium ovarian follicles at the expense of large follicles. The bST also inhibits directly pituitary responsiveness to gonadotropin-releasing hormone (GnRH) and luteinizing hormone (LH) secretion. However, it is difficult to determine whether the actions of bST result from a depletion of energy substrate for the ovary or from the direct actions of either bST or IGF-1 on gonadotropin secretion or on ovarian follicle development. These hormones may be particularly important in limiting ovarian function in early lactation in view of the calf's demand for nutrients. Tissue mobilisation at this time is associated with risk of reproductive failure (Butler *et al.* 1981) and increased risk of disease (Shanks *et al.* 1981), both of which may be more prevalent in high producers. Dietary factors such as energy density, the protein content, degradability and availability play important roles in determining the extent of body tissue mobilisation. The inability to mobilise tissues later in lactation also limits subsequent reproductive performance including return to cyclicity and conception, the timing of which coincide with the phase of rapid follicle development (60-80 days). Various metabolites (glucose and 3-hydroxybutyrate) and amino acids have been associated with the re-establishment of cyclicity although we have been unable to establish such a relationship with glucose and 3-hydroxybutyrate (Abe *et al.* unpublished results).

Evidence that energy status may be operating through the expression of IGF-1 is provided by the positive correlations between these factors, follicular growth and the oestrogen:progesterone ratio in dominant follicles (Lucy *et al.* 1992). However, it is difficult to establish cause and effect relationships in such studies since homeorrhetic adaptations involve the coordinated adaptation of a number of time dependent variables (Lean *et al.* 1992). A range of other nutritional factors may also improve fertility at either mating or in the periparturient period such as antioxidant vitamins and metallo-enzymes.

Hormones of the stress axis may also play an important role in inhibiting ovarian activity post-partum in the high producing cow. Stressors such as restraint and transport undoubtedly inhibit LH secretion, however, the relative contribution of ACTH, cortisol and β-endorphin on this parameter is difficult to quantify (Moberg 1991). For example, the administration of ACTH blocks the pre-ovulatory release of LH as well as lowering the basal concentrations of LH, whereas cortisol blocks the pre-ovulatory surge without influencing basal levels. Glucocorticoids may also limit glucose uptake in the ovary while β-endorphin inhibits the pulsatile secretion of the hypothalamic releasing hormone, GnRH (see Moberg 1991).

Reproductive failure in ruminant genotypes is not confined to the cow and in the following paper some aspects of the influence of stress on reproduction in the ewe are reviewed.

THE IMPACT OF STRESS ON THE REPRODUCTIVE PERFORMANCE OF THE BREEDING EWE

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The impact of stressors on reproductive function in the ewe is no less significant than that observed in modern dairy cow genotypes. Both environmental and psychological stressors have been implicated

in reproductive failure in the ewe: extremes of ambient temperature, husbandry practices such as the mixing of flocks and transportation add to a recurring theme in this contract. As with the cow the effects of stress on reproduction in the ewe are thought to be mediated by the stress-induced hormones of the hypothalamic-pituitary-adrenal axis. It is likely that hormones from each of the 3 levels of regulation, corticotropin-releasing hormone (CRH) from the hypothalamus, ACTH from the pituitary and cortisol from the adrenal are involved in this inhibition. It is conceivable that some of the effects of ACTH and CRH are direct and not mediated by the secretion of the glucocorticoids: CRH, for example, has been identified in the testis and in the placenta where it may have inhibitory actions independent of its central role of suppressing the secretion of GnRH.

Heat stress has been shown to increase oestrous cycle length when the stress was experienced late in the year (Sawyer *et al.* 1979) in the Merino ewe. Undernutrition can also induce longer oestrus cycles and increase the incidence of silent heat (Allan and Lamming 1961). The stress of shearing has also been reported to inhibit oestrus (Parr *et al.* 1988).

Despite these marked effects on the oestrus cycle, the influence of stress on ovulation rate is equivocal. Whereas fasting did not alter ovulation rate (MacKenzie *et al.* 1975), Doney *et al.* (1976) have shown the complete suppression of LH release in ewes treated with ACTH. Other studies have shown that cortisol, decreases the sensitivity of the pituitary to the actions of GnRH on LH secretion (Fuquay and Moberg 1983).

Conception rate and/or embryo loss appear to be influenced by stress. Thwaites (1971) reported increased ovum wastage in ewes exposed to high temperatures at days l-4 of the oestrous cycle. Likewise Edey (1966) found that embryo loss increased when ewes were exposed to submaintenance nutrition levels or fasting during the first week post mating. We have conducted studies recently to further quantify the effects of nutritional and parasite stressors on reproductive efficiency of ewes.

Submaintenance nutrition

Our recent studies have shown that weight loss of 200 g/day over 25 days has only a minor effect on the responsiveness of ovaries to pregnant mare serum gonadotropin (PMSG) in Merino ewes when compared with animals held at maintenance, yet chronic undernutrition, particularly if it is combined with exogenous cortisol treatment during the 5 day period prior to ovulation, can increase the number of abnormal ovarian structures. These effects could be minimized by treatment with human chorionic gonadotropin (HCG) which implies that the stress effects are mediated via negative effects on GnRH release. Similar increases in abnormal ovarian structures have also been observed in heat stressed ewes (Hinch and Wynn unpublished results).

Parasite infection

The influence of chronic parasitism and immunization against ACTH to suppress cortisol secretion has been investigated recently. Trickle infection with *Trichostrongulus* sp. had no significant effects on ovarian function (Table 1) but immunization against ACTH increased conception rates ($P < 0.01$) in ewes treated with 400 iu of PMSG in both stressed and non-stressed ewes.

Further experiments have confirmed that ACTH treatment, but not cortisol, can reduce peripheral progesterone levels during the first 3 days post-oestrus which suggests that ACTH can increase embryo loss independently of cortisol. Our observations have suggested that chronic stress influences reproductive function in superovulated ewes and that these effects may be mediated via ACTH acting

Table 1. The effects of adrenocorticotrophic hormone immunisation (I are immune and NI are non-immune) and parasitism (S were parasitised and NS were controls) on ovulation rate (ovulations/ewe ovulating), follicle number (number of follicles > 5 mm/ewe ovulating) and conception rate (% of ewes mated that conceived) (means ± S.E.M.)

Immune stress	Parasite stress	Ovul. rate	Foll. no.	Concept.
I	NS	1.88(0.15)	0.59(0.16)	80.8
I	S	2.00(0.16)	1.21(0.18)	73.9
NI	NS	2.04(0.16)	0.91(0.17)	52.2
NI	S	1.88(0.14)	0.84(0.16)	44.0

either on gonadotrophin release or corpus luteum function. Therefore infertility is likely to be caused by stress induced hormones, although our data suggest that ovarian function in the Merino ewe is most resilient in times of nutritional deprivation.

In the same way that reproduction is considered to be relatively expendable in the hierarchy of importance of physiological functions when animals are subjected to chronic stress, the maintenance of wool growth in the wool follicle rates at a similar low level. The influence of stress on wool growth is now considered.

THE INFLUENCE OF STRESS ON WOOL PRODUCTION AND THE OCCURRENCE OF WOOL BREAKS/TENDERNESS IN MERINO SHEEP

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A major problem of the Australian wool industry is that a high proportion of the wool clip has a fibre tensile strength that is below that required by manufacturers. Approximately 30% of the wool clip in Western Australia has been classified as "tender" or below 25 N/ktex (Ralph 1986), costing Western Australian producers approximately $13 million dollars in 1992. The position of break is another parameter that can attract a major price discount.

The production of high quality wool in Australia is limited by climate, nutrition, parasites, genotype/phenotype, physiological state (eg. pregnancy, parturition and lactation) and other environmental variables. Poor nutrition in the autumn period, particularly in Mediterranean climates of southern Australia, is associated with the production of tender fleeces. As a result much effort has been focussed on identifying the appropriate supplementary feeding regime to prevent the problem, often with mixed success. Since wool breaks also occur in times of ample feed supply in animals without any apparent parasitic burden, it is highly likely that nutritional status is not the only wool growth inhibitory mechanism.

A role for the hormones of the stress axis in the formation of wool breaks was established first by the observation that the administration of pituitary ACTH to sheep suppressed wool growth (Lindner and Ferguson 1956). Subsequent investigations have shown that glucocorticoids exert a dramatic inhibitory effect on the rate of wool fibre synthesis particularly if circulating levels are raised for several days.

It is possible that the inhibitory influences of cortisol may be mediated by activating local paracrine/autocrine factors within the follicle which act as inhibitors of cell division or wool protein synthesis. Epidermal growth factor (EGF), which transiently inhibits the growth of the fibre when administered to sheep, and therefore acts as a wool harvesting agent, is one potential candidate to fill this role. Both EGF and its specific target receptor have been identified in the outer root sheath of the follicle and in the epidermis, pilary canals and sebaceous glands in mature Merino skin (Wynn *et al.* 1989; du Cros *et al.* 1992).

In order to decrease the circulating levels of cortisol in the grazing animal, we have actively immunised animals against their own ACTH. We investigated the effect of the psychosocial stress of the infrequent disruption of the social hierarchy and ACTH immunisation on wool growth and showed that the immunisation when applied with the stress stimulated fibre length by 9% without altering fibre diameter (Behrendt *et al.* 1992). Staple strength and wool yield were not altered even though the stressed group were subjected to 2 periods of fasting for 48 hours. There were, however, differences in the position of break between unstressed and stressed groups possibly pointing to a stress response which was not altered by the ACTH immunisation. As we were unable to induce marked changes in wool growth rate or induce wool breaks in this trial, we were not able to assess the effectiveness of ACTH immunisation to prevent wool breaks. Clearly an experimental model has to be established in which wool breaks can be reproduced reliably in order to establish the cause of the phenomenon.

In view of our hypothesis that the formation of wool breaks due to stress induced cortisol levels was mediated by the expression of EGF, we investigated if ACTH immunisation influenced the efficiency of EGF as a wool harvesting agent in the same experiment. Both the imposition of the psychosocial stress and the ACTH immunisation increased the efficacy of the EGF with the combination of both treatments resulting in 60% weaker staples than the control unstressed non-immune group (Behrendt *et al.* 1993b). The apparently confounding effects of both stress and ACTH immunisation suggest that changes in the

expression of other hormones related to ACTH such as -endorphin and -MSH may also be modifying this response, since the circulating levels of these hormones are increased dramatically by immunisation. A potential role for -MSH and -endorphin in the control of wool growth is supported by the localisation of these peptides in the outer root sheath of the follicle (Behrendt *et al.* 1993a).

In determining the stress susceptibility of different genotypes, we have compared the circulating levels of stress hormones in modern fine wool Saxon Merinos with the primitive unselected genotype, the Camden Park Merino. The levels of cc-MSH, β-endorphin and cortisol were all higher in blood samples collected by venipuncture in the modern genotype, suggesting that they are more stress responsive than the unselected genotype.

Although research into the role of hormones of the stress axis on wool growth is still in its infancy, it is likely to be a rewarding area in solving the major problem of low tensile strength wool in the Australian clip.

Thus far this contract has focussed on problems associated with the ruminant industries. The pig provides a model species which has been adapted to intensive production, and some of the genetic stock displaying superior growth rates currently being introduced into the Australian industry have displayed poor adaptability to commercial production systems in Australia.

THE IMPACT OF HEAT ON THE PRODUCTIVITY OF THE COMMERCIAL PIG

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Grower pig accommodation in Australia is characterised by partly-slatted floor construction, with an iron roof and side walls consisting of adjustable shutters or blinds. Thus animals are frequently exposed to cold, draughty conditions in winter and high temperatures in summer. A recent survey of 21 grower/finisher piggery environments in eastern Australia (Lemin *et al.* 1991) found that summer temperatures (December to February) exceeded 26°C for 32% of the time surveyed, with maximum internal temperatures in these piggeries during summer ranging from 34-39°C.

Regulation of heat exchange at high temperatures

For an animal which is relatively hairless and unable to perspire, the pig is well adapted to a wide range of climatic conditions. This apparent paradox is possible because pigs have a number of behavioural and physiological mechanisms to control heat loss including seeking shelter, shivering, changing posture, huddling and changing blood flow to the skin. The pig is thus able to maintain normal body temperature (39 ± 05°C) within a specified range or "zone of thermal comfort".

As ambient temperature increases within this range the pig maintains normal body temperature by separation from their pen-mates, increasing body contact with the cool pen floor and increasing vasodilation. With further temperature increases the pig increases heat loss by evaporation from the lungs and skin, or by reducing food intake. The ambient temperature at which evaporative heat loss increases is defined as the evaporative critical temperature (ECT) and marks the upper end of the zone of thermal comfort.

If ECT is exceeded, respiration rate increases markedly and pigs resort to any form of skin wetting to provide evaporative cooling. Even on a slatted floor or wire mesh pigs are able to wet some skin with drinking water, dung, urine and saliva.

With each successive increase in ambient temperature beyond ECT, the pig will reduce heat production to achieve a new energy balance by reducing voluntary food intake and activity (Giles and Black 1991). These changes are accompanied by successive increases in respiration rate above 30 breaths/minute, an elevation in body temperature above 39.5°C and a transfer of blood flow to the body surface to maintain skin temperature at 2-3°C below body temperature, and thus maintain the transfer of heat from the body core to the skin surface.

Beyond that temperature that the pig is unable to lose heat, the upper critical temperature (UCT), body temperature will rise dramatically above 43°C, often causing death.

Extending the zone of thermal comfort in commercial pig production

The ECT is an important benchmark for production efficiency as voluntary food intake declines beyond this temperature. The range of ECT has been defined as 22-25°C in growing pigs (90 kg liveweight) fed *ad libitum*, housed separately on wire mesh floors at low humidity (< 50%) with still air

(0.15 m/second), and prevented from wetting their skin surface (Giles and Black 1991). However, this figure will vary under commercial conditions, where these parameters fluctuate markedly.

The pig producer is able to observe changes in ECT by monitoring respiration rate and body temperature above 39.5°C. Several management options are available to extend the ECT to maintain food intake, including spray cooling, increased air movement and reduced stocking rate. The ECT can be extended to at least 32°C when growing pigs are provided with free access to sprinklers (Giles *et al.* 1987), while they are unaffected by daily fluctuations between 35° and 22°C provided water is available from nipple drinkers or sprinklers. The fact that these techniques are now being readily adopted in the industry is testimony to the commercial effectiveness of the evaporative method of shedding heat loads in the low humidity Australian environment.

The impact of heat as a stressor in commercial pig production

Psychological reactions in the heat stressed animal, such as restlessness, anxiety and escape attempts, which are most likely initiated by stress related hormones, provide an accurate index of the stress status of animals. In addition to reducing food intake and activity, animals also lower their heat load by adjusting cardiac output, heart rate and oxygen consumption, all of which are regulated by the same hormones. These physiological adjustments above the ECT are merely the adaptation to stress involving no overt pathological state and therefore the associated decrease in feed intake and growth rates are merely the "cost" of stress.

Although intensification of the pig industry has removed the pig from its natural behavioural response to wallow during periods of high ambient temperatures, cost effective management options to ameliorate the environment are available in Australia. However, the introduction of new strains of pigs displaying superior growth rates and feed conversion efficiencies but which are less heat tolerant may increase the adverse impact of this source of stress on productivity in the future.

SUMMARY

In this contract we have focussed on specific instances where interactions between stressful stimuli and genotype within animal production systems have compromised the efficiency of production.

What can the farmer do to minimise the effect of stress on production efficiency? This is a very difficult question to answer since we know very little about the basic biological cause of a number of stress-related production losses. Until we identify these mechanisms the best advice to farmers is to minimise the adverse stimuli which are known currently to influence the secretion of the stress hormones, many of which have been identified throughout this review and which recur in many animal industries.

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