Measures of stress and growth suppression in surgically castrated bulls

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ABSTRACT

The effects of surgical castration of pre- and post-pubertal bulls on measures of stress and growth were investigated in two separate experiments. In Experiment 1, 17-month-old Friesian bulls (n = 6 per treatment) were either left untreated (Controls), handled and administered local anaesthetic (Handled), or handled, administered local anaesthetic and surgically castrated (Castrates). In Experiment 2, 5-month-old Friesian bulls (n = 10 per treatment) were either left untreated (Controls) or surgically castrated without local anaesthetic (Castrates). Castrates in Experiment 1 had higher ($P<0.05$) plasma cortisol concentrations 7 and 14 days after treatment compared with Handled bulls and lower ($P<0.01$) gains in body weight for 14 days compared with Controls. There were no effects of treatment on plasma non-esterified fatty acid, ß-hydroxybutyrate and urea concentrations. Castrates in Experiment 2 had higher ($P<0.05$) plasma concentrations of cortisol and the inflammatory protein haptoglobin for 7 days after treatment compared with Controls. The feed intake and weight gain of Castrates were also lower ($P<0.05$) than that of Controls during the week following treatment. It is concluded that the castration of cattle causes stress, resulting in a check in growth which is possibly mediated via a reduction in feed intake.

Keywords: growth suppression; castration; cortisol; testosterone; stress.

INTRODUCTION

Bulls grow 17% faster and convert feed to lean meat 13% more efficiently than steers (Field, 1971). Bulls, therefore, reach slaughter weights at an earlier age than steers. However, bull meat is often of lower quality, with less fat and marbling and is prone to dark cutting (Field, 1971; Purchas, 1990) Some beef farmers attempt to optimise bull performance and profitability by taking advantage of the better growth rate of bulls, then castrating them after puberty to improve meat quality.

Studies have been conducted to determine the efficacy of this approach in maximising meat quality and meat yield under New Zealand conditions (Cosgrove *et al.*, 1996). Data from these studies indicate that there is a prolonged period following castration during which the growth rate of recently castrated cattle is lower than that of animals castrated at a younger age. This phenomenon tends to negate one of the major advantages claimed for the management practice of post-pubertal castration of bulls. Similar growth checks following castration of cattle have been documented in other studies (Faulkner *et al.*, 1992; Chase *et al.*, 1995); however, the possible mechanisms involved are not well understood. Clearly, the elucidation of such mechanisms would allow the development of strategies for minimising the impact of post-pubertal castration on growth.

The welfare of surgically castrated bulls is also a potential concern, mainly due to the tissue trauma and probable pain associated with the procedure. In two separate studies, measures of stress of surgical castration were determined in 5-month-old calves and post-pubertal bulls. Physiological measurements were used to determine the stress and welfare implications of the procedure during the 24 to 96 h immediately after surgery. In addition, growth and other physiological variables were measured over the 3- to 4-week period following castration. These data are used to examine the various mechanisms that may be responsible for the suppression of growth and degree of stress in recently castrated cattle.

MATERIALS AND METHODS

Experiment 1

Eighteen bulls (17 months of age, mean bodyweight 385.8 ± 9.2 kg) kept at Flockhouse were divided into three groups and grazed on 5-ha pastures; all groups were yarded, weighed and blood sampled twice weekly by tail vein venipuncture. After 3 weeks, three of the bulls in each of two groups were surgically castrated by a veterinarian after administration of local anaesthetic (Castrates), the other three were given local anaesthetic, handled and restrained in the same manner, but not castrated (Handled). Local anaesthetic (Anecaine 2; lignocaine hydrochloride 20 mg/ml and epinephrine bitartrate 0.036 ng/ml; Phenix, Belgium) was administered by injections into the scrotal neck (15 ml) and sac (7.5 ml), and into both testes (7.5 ml). The remaining group was used as Controls. Twice-weekly

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weighing and blood sampling was maintained for a further 4 weeks post-surgery.

Experiment 2

Twenty Friesian bulls (5 months of age; 178 ± 2.0 kg) were either left untreated (Control) or surgically castrated without local anaesthetic (day of treatment = day 0). The bulls were housed in individual stalls with *ad libitum* access to a barley:soybean meal and feed intake was recorded daily and bodyweight measured weekly, up to 3 weeks after treatment. Intravenous jugular cannulae were fitted the day before treatment, and the calves were treated and blood sampled in their stalls to minimise the effects of handling. Blood samples were taken at 0, 0.25, 0.5, 1, 3 and 7 days for plasma cortisol determination, and at 0, 1, 3, 7, 14 and 21 days for assay of plasma haptoglobin, an acute phase protein that was used as a marker of inflammation. This study was a component of a larger experiment examining immunosuppression in response to an acutely-acting stressor (Fisher *et al.*, 1997).

Assays

Cortisol and testosterone were measured by radioimmunoassay (Ingram *et al.*, 1994; Jago *et al.*, 1995; Fisher et al, 1996). Plasma concentrations of non-esterified fatty acid (NEFA), ß-hydroxybutyrate (BOH) and urea (all Experiment 1 only), and haptoglobin (Experiment 2 only; Skinner and Roberts, 1994) were measured using a Hitachi autoanalyser.

Statistical Analysis

Data were analysed by ANOVA for the effects of treatment. All data are shown as untransformed means ± SEM. Differences are shown when P<0.05.

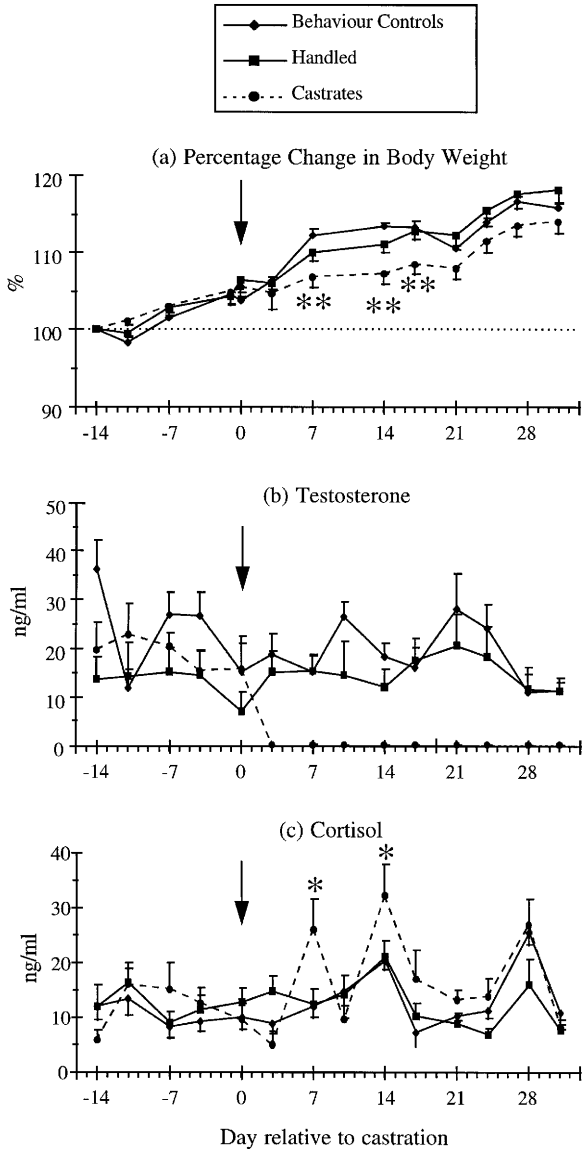
RESULTS

Experiment 1

Changes in body weight as a proportion of initial weight are presented in Figure 1a. There were significant differences in bodyweight change between Castrates and Controls for more than 2 weeks post-surgery (P<0.01), but after this time the differences between groups were not statistically significant. A short period when two groups of animals (one of which was the Controls) were accidentally mixed adversely affected growth rates during the third week after castration. At the end of the study period, 4 weeks after castration, the total weight increases (kg) in each treatment group were: Controls 58.5 ± 0.9; Handled 65.5 ± 5.0; Castrates 51.8 ± 5.3 (P<0.10).

Mean plasma testosterone concentrations are presented in Figure 1b. The mean testosterone concentration of intact bulls at pasture was 19.8 ± 1.6 ng/ml, and there was considerable variability between animals (range: 2 to 65 ng/ml) reflecting the pulsatile nature of testosterone secretion (Carragher *et al.*, 1997). Testosterone concentration in Castrated animals decreased to < 1.0 ng/ml within 1 h of surgery, and remained at undetectable levels for the remainder of the experiment. Handled animals showed a non-significant suppression of testosterone on the day after treatment (~7 ng/ml), but thereafter concentrations were similar to Control bulls.

FIGURE 1: The effects of castration of 17-month-old bulls in Experiment 1 on (a) changes in body weight, (b) plasma testosterone, and (c) plasma cortisol concentrations. The arrow indicates the time of castration. *(P<0.05) **(P<0.01)



Mean plasma cortisol concentrations are presented in Figure 1c. The concentration of cortisol measured in intact bulls was 11.7 ± 0.9 ng/ml; this compares with 4.3 ± 0.8 ng/ml for samples collected remotely from animals at pasture (Carragher *et al.*, 1997). Castrated animals had higher (P<0.05) cortisol concentrations than Handled and Controls on days 7 and 14 following surgery.

Mean plasma concentrations of NEFA, BOH and urea are shown in Fig. 2a-c. Although there were changes in mean concentrations of NEFA and, to a lesser extent, urea over the course of the experiment, there were no significant differences between the three treatment groups at any one time. Plasma NEFA concentrations were

FIGURE 2: The effects of castration of 17-month-old bulls in Experiment 1 on plasma (a) non-esterified fatty acid, (b) ß-hydroxybutyrate, and (c) urea concentrations. The arrow indicates the time of castration.

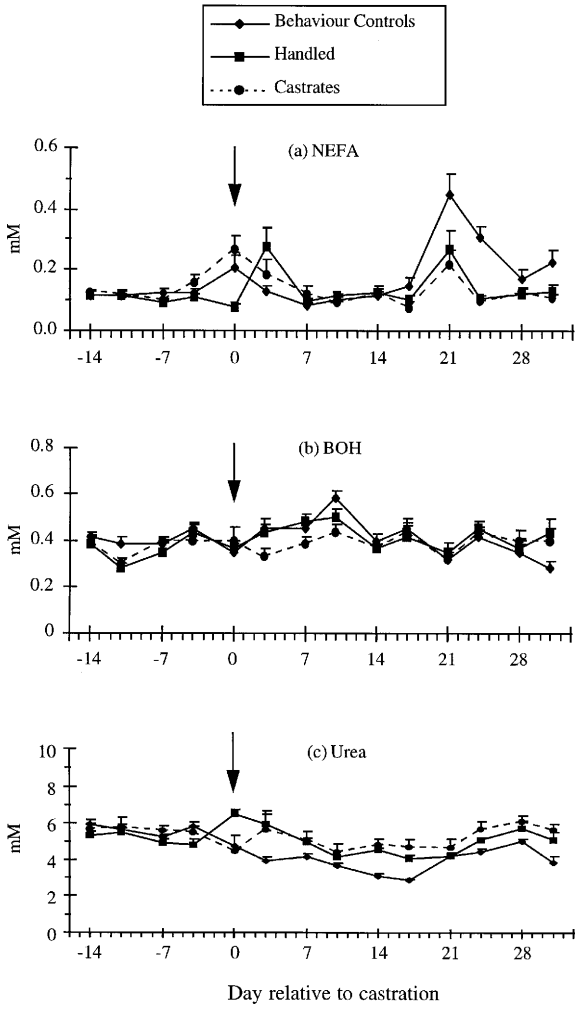


FIGURE 3: The effects of castration of 5-month-old bulls in Experiment 2 on mean weekly feed intake and average daily weight gain. *(P<0.05)

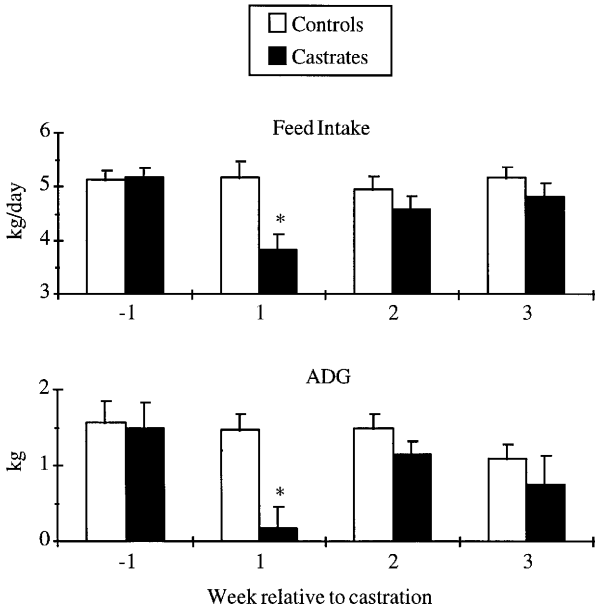
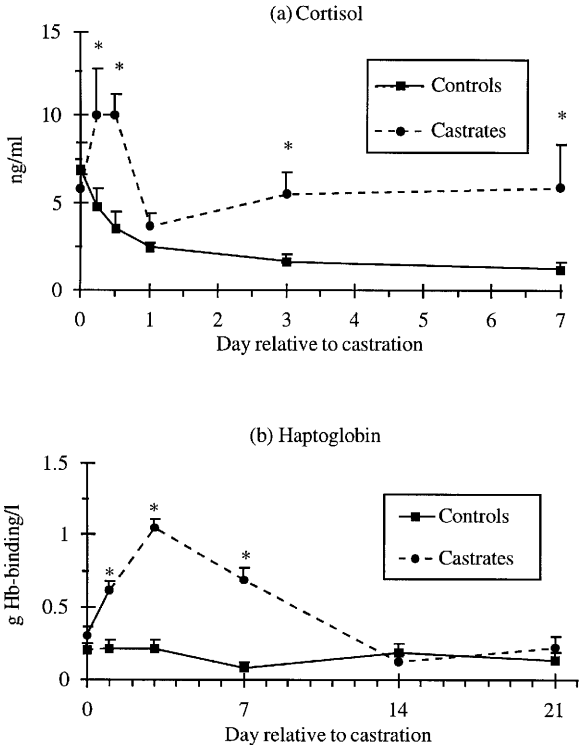


FIGURE 4: The effects of castration of 5-month-old bulls in Experiment 2 on plasma (a) cortisol, and (b) haptoglobin concentrations. *(P<0.05)



elevated in all groups around the time of treatment, and during the short period when two groups of animals were accidentally mixed. There was no change in plasma BOH concentrations during the study period.

Experiment 2

Feed intake and average daily weight gain (ADG) in castrated calves were depressed (P<0.05) compared with Controls for the first 7 days after surgery (Figure 3). There were no differences between treatments in either feed intake or ADG for the remainder of the study.

Mean plasma cortisol concentrations in castrated calves were elevated (P<0.05) compared with Controls on days 3 and 7 post-surgery (Figure 4a). Mean plasma haptoglobin concentrations were low in Control calves at all times, but were elevated (P<0.05) in castrates until day 7 post-surgery (Figure 4b).

DISCUSSION

Although temporary suppression of growth following castration has been reported in a number of studies, the possible mechanisms involved are not well understood. In both experiments in this paper, the growth rate of castrated animals was suppressed to a severe degree in the period immediately after surgery. Three possible causative mechanisms are proposed for the reduction in growth. They are: 1) the sudden removal of testosterone (a potent anabolic steroid) from the circulation; 2) the catabolic effects of chronically-elevated plasma corticosteroid concentration resulting from a stressor; and 3) a reduction in feed intake quality or quantity. It is possible that the two latter mecha-

nisms could be evoked under similar circumstances, thus a painful trauma site could result in a stress response and/or reduced appetite.

Surgical castration of post-pubertal bulls resulted in a rapid and permanent decrease in plasma testosterone concentrations. However, there is evidence that this decrease in testosterone may not be responsible for much of the observed suppression in growth rate. In Experiment 1, the growth rate of castrates recovered to be close to that of intact bulls 3 weeks after surgery, when mean plasma testosterone concentration was less than 1.0 ng/ml. Furthermore, the growth rates of Handled animals showed a slight decrease for 1 week compared with Controls, even though their plasma testosterone concentration was not different for more than a few hours post-surgery (Carragher *et al.*, 1997). In addition, the 5-month-old bulls in Experiment 2 had similar growth checks following castration, even though circulating testosterone concentrations would be low at this age.

In studies with immunocastrated bulls (Jago *et al.*, 1996), only a slight suppression in growth rate was reported following a decline in plasma testosterone concentration. The replacement of the anabolic effects of testosterone by implanting cattle with Ralgro at castration did not prevent castrated animals growing about 70% less during the 28 days following castration compared with intact bulls (ZoBell *et al.*, 1993). These observations suggest that removal of the bulls' major source of testosterone is not likely to be the main cause of the severe growth suppression recorded in the period following castration. In the longer term, the removal of testosterone does cause steers to have a lower growth rate than bulls (Field, 1971), and thus it may be difficult to totally separate the effects of removal of androgen from castration-induced stress in understanding changes in growth following castration.

Data from the studies described in this paper show that plasma cortisol concentrations remain elevated compared with Controls for 1 to 2 weeks post-castration, indicating a chronic stress response. Chronically-elevated plasma corticosteroid concentrations of sufficient magnitude are known to have a catabolic effect upon body reserves (Dickson, 1977). However, there were no significant changes in circulating concentrations of NEFA, BOH or urea following castration in Experiment 1, which suggests there were no catabolic effects on body tissue. It is probable that the duration and magnitude of the elevations in plasma cortisol concentrations in Experiments 1 and 2 were not sufficient to induce a significant catabolic effect.

The cortisol data from Experiment 1 may be confounded by the blood sampling protocol; the animals were mustered and sampled in the same yards in which treatments were administered, and there may have been a short-term stress response when they returned to the yards (Pascoe, 1986). Plasma cortisol concentrations in blood samples collected by venipuncture from animals in the yards were significantly higher than in samples taken from animals at pasture using a remote blood sampling device (DracPac; Ingram *et al.*, 1994). Thus, the usefulness of any cortisol measurements in blood samples collected using restraint and venipuncture may be highly questionable. The same criticism cannot be made of Experiment 2, and these data do show that castrated animals had chronically-elevated plasma cortisol concentrations for several days post-surgery.

The data from Experiment 2 indicate that during the period when growth rate was most affected, plasma cortisol concentration remained somewhat elevated; but the cause of this chronic stress remains unknown. Two possible sources of post-castration stress are firstly, ongoing pain or discomfort at the site of the surgical trauma; and secondly, that the castrated animals rapidly lose social status in the hierarchy and receive a higher level of aggression and less access to resources. In support of the first hypothesis, plasma concentrations of haptoglobin in Experiment 2 were elevated for 7 days post-surgery, suggesting that there was an ongoing inflammation response which may have been painful. However, this is complicated by the ability of inflammation *per se* to induce glucocorticoid secretion. There is also some circumstantial evidence to support the second hypothesis. In unpublished studies, Knight *et al.* found that the growth check of recently-castrated post-pubertal bulls re-mixed with a group of entire bulls was greater (in both magnitude and duration) than when they were mixed with steers or maintained as a separate group.

There is a strong suggestion in Experiment 2 that feed intake is markedly reduced in recently castrated animals, even when available *ad libitum*. Thus, it seems likely that the growth check may be largely a consequence of reduced feed intake during the immediate post-castration period and is not dependent upon acts of inter-individual aggression.

We have proposed several mechanisms which could be responsible for causing the growth check observed after cattle are castrated. Our tentative conclusion is that the growth check appears to be a consequence of reduced feed intake in the post-surgery period, which, in turn, appears to occur in response to the chronic stress of pain arising from the site of tissue injury and subsequent inflammation. Future research should examine this possibility by attempting to separate the effects of the stress of castration from the removal of testosterone and partitioning post-castration stress into inflammatory and pain responses. Clearly, the determination of these mechanisms would allow the development of strategies for minimising the impact of castration on growth in cattle.

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