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**COMPUDOSE,  
ITS EFFECTS ON HEREFORD X  
FRIESIAN HEIFERS**

A thesis presented in partial fulfilment  
of the requirement for the degree of  
Master of Applied Science (in Animal Science)  
at Massey University.

**Jennifer L Burke**

**1997**

Dedicated to my family and friends.

## **ABSTRACT**

**Burke, J.L. 1997. Compudose, its effects on Hereford x Friesian heifers. M. Appl. Sc. Thesis, Massey University, Palmerston North, New Zealand. 96 pp.**

Compudose is an oestrogenic growth promotant that improves liveweight gain and feed conversion efficiency in steers. In the past it has not been recommended for use in breeding heifers because of the adverse effects of oestrogen treatment on reproductive performance. The effects of Compudose on liveweight gain, skeletal development, lactational performance, carcass characteristics and offspring performance in heifers at pasture are unknown. However, the expected increase in liveweight gain from Compudose treatment may allow target growth rates to be attained in heifers at critical times of the year, without lactational performance being affected.

This study investigated the effect of treating Hereford x Friesian (H x F) once-bred heifers (OBH) with Compudose 400 at 3 months (90 days) of age (Compudose 90) and 7 months (210 days) of age (Compudose 210) compared to non-treated heifers (Control). Compudose 90 heifers (n = 14) gained 0.63 kg/day compared with Control heifers (n = 17) which gained 0.59 kg/day for 385 days from the time of implantation (6.8% increase,  $P < 0.05$ ). Subsequently Compudose 90 heifers achieved greater liveweights than Control ( $P > 0.05$ ) and Compudose 210 (n = 17) heifers ( $P < 0.05$ ) at pre-mating (by 17.7 kg and 16.9 kg, respectively), pre-calving (by 13.7 kg and 25.3 kg, respectively), weaning (by 13.9 kg and 32.8 kg, respectively) and slaughter (by 23.1 kg and 39.9 kg, respectively). Liveweight gain between Control and Compudose 210 heifers did not differ over the 383 day period of implantation. Carcasses of Compudose 90 heifers were 10.5 kg heavier than carcasses of Control ( $P > 0.05$ ) and 19.2 kg heavier than carcasses of Compudose 210 heifers ( $P < 0.05$ ).

Wither height, girth, hip height, hip width, pelvic height and pelvic area did not differ significantly between treatment groups, but pelvic width of Compudose-treated heifers was significantly smaller than that of Control heifers ( $P < 0.01$ ).

Compudose 90 and Compudose 210 heifers had an average calving date 8 days ( $P < 0.10$ ) and 10 days ( $P < 0.05$ ), respectively, later than the average calving date of Control heifers, and more compact calving spreads.

There were no treatment differences in calf birthweight, however calves born to Control heifers were 15.3 kg and 16.2 kg heavier at weaning than calves born to Compudose 90 and Compudose 210 heifers ( $P < 0.05$ ).

Compudose treatment and associated higher growth rates did not affect milk yield, as determined by the weight-nurse-weigh (WNW) method, at 4 and 8 weeks of lactation; udder volume at 4 weeks, 8 weeks and 12 weeks of lactation (weaning), and at pre-slaughter; or udder weight at slaughter.

There were no significant differences in carcass quality characteristics between treatment groups. However, carcasses of Compudose-treated heifers tended to be shorter ( $P < 0.10$ ) and have a greater rib-eye area. No significant differences in fat content, as determined by the weight of kidney and pelvic fat and fat depth, were detected between treatment groups.

Implanting heifers with Compudose at 3 months of age is more beneficial than implanting heifers with Compudose at 7 months of age, but the small liveweight gain advantage would not be economically advantageous. It is concluded that Compudose is not a practical solution for improving the rearing of beef or dairy heifers, despite lactational performance not being affected.

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## LIST OF ABBREVIATIONS

\$	dollar
%	percent
®	Registered Trademark
°C	degree celcius
μCi	microcurie
~	approximately
c.	about
DES	Diethylstilboestrol
DNA	deoxy ribose nucleic acid
DO%	dressing-out percent
E	oestradiol
H x F	Hereford x Friesian
h	hour (s)
HBI	High breeding index
I	iodine
IU	international unit(s)
kg	kilogram(s)
kg/day	kilogram(s) per day
kg <sup>0.75</sup>	metabolic bodyweight
kgDM	kilogram(s) of dry matter
l	litre
LBI	Low breeding index
LD	<i>Longissimus thoracis</i> muscle
LH	lutensising hormone
mg	milligram(s)
mins	minute(s)
ml	millilitre(s)
mm	millimetre(s)

mM	millimole(s)
MY	Milk yield
n	number of observations
ng/l	nanogram(s) per litre
OBH	Once-bred heifer
r	correlation
RNA	ribose nucleic acid
SE	standard error
TOH	tritiated water
US	United States
UV	Udder volume
VFI	Voluntary feed intake
vs	versus
WNW	weigh-nurse-weigh

#### Levels of significance

\*\*\* P < 0.001

## Chapter 1

### INTRODUCTION

New Zealand beef production systems have suffered a period of reduced profitability through the recent decline in world beef prices. As a result farmers have had to adopt systems that will produce beef more efficiently. An effective way of doing this is the use of growth promotants. Compudose<sup>®1</sup>, an oestrogenic growth promotant, has been shown under pastoral and feedlot conditions to increase the financial returns from farming beef by improving liveweight gains and the efficiency of feed conversion into lean meat, and promoting changes in carcass and meat quality. Generally liveweight gains in steers ranging from 5-30% under pastoral conditions, and feed conversion efficiency responses of 10-16% under both feedlot and pastoral environments have been reported, along with heavier carcasses containing more protein, more moisture and less fat. Therefore, under New Zealand's current carcass weight based schedule Compudose implants have economical advantages for New Zealand beef producers battling the recent downturn.

Currently the majority of those animals treated with Compudose are steers and heifers not destined for breeding purposes. In the past, regulations have prevented use of the oestrogenic growth promotant, Compudose, in breeding heifers owing to the negative consequences on reproductive performance reported from the use of oestrogenic compounds. A decrease in conception rates, delays in puberty, a high incidence of non-ovulatory oestrus, delayed oestrous cycles, abortions and abnormal mammary gland development have been reported following the use of oestrogenic compounds. Despite these effects it may still be viable to use Compudose, a slow release growth promotant, on heifers, particularly to reach target liveweights at critical times of the year.

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<sup>1</sup> Compudose<sup>®</sup>, Oestradiol 17 $\beta$  implant, ELANCO Animal Health (NZ) Ltd.

Dairy and beef farmers have repeatedly had difficulty in reaching target liveweights at puberty, mating, and first calving, therefore the ability of Compudose to improve liveweight gains may have considerable benefits in heifer and cow systems. An example is the 'Once-bred heifer' (OBH) system, a recent development in beef production systems, where heifers are mated at 15 months of age to produce a calf which is then finished at liveweights of 450-500 kg at 30-36 months of age. The success of this system is determined by the ability of dairy-cross heifers to reach critical target liveweights at various times of the year, i.e. a 300-330 kg mating liveweight at 15 months and a 400-450 kg calving liveweight at 24 months.

Dairy farmers may also find Compudose to be advantageous in their systems by allowing target growth rates and liveweights at puberty, mating and first calving to improve the efficiency of their systems. However, the negative effects that the naturally occurring compound in Compudose, oestradiol 17 $\beta$ , has on heifer reproductive and lactational performance may prevent the use of this hormonal growth promotant in breeding cow systems. Given that Compudose improves liveweight gains and feed efficiency, and research carried out by Sejrnsen et al. (1982, 1983) and Little and Kay (1979) which showed that heifers grown at rates of more than 0.8 kg/day around the time of puberty had abnormal mammary gland development and milk production, Compudose could potentially have negative consequences on lactational performance.

The occurrence of hormonal residues in the meat and milk of treated animals may interfere with New Zealand's ability to trade with certain markets. However, meat produced from treated steers and heifers commonly exhibit levels of anabolic steroid residues lower than those occurring naturally in mature bulls and pregnant cows, respectively (McCutcheon, 1989; Roche and Quirke, 1992). It is also worth noting that the levels of steroids which could be ingested by eating meat are several orders of magnitude lower than those produced within the human body (McCutcheon, 1989; Roche and Quirke, 1992). Despite this evidence consumers

are still aware of the occurrence of chemical residues in the food they consume which may influence the use of growth promotants in all classes of animal in the future.

More information is needed about the effects of Compudose on heifer liveweight gain, reproductive performance, offspring performance and lactational performance before Compudose becomes widely used in New Zealand heifer beef or dairy production systems. The objective of this study was to investigate the effects of Compudose on heifer liveweight gain, calf growth and lactational performance of dairy-cross heifers in the OBH system.

## Chapter 2

### **LITERATURE REVIEW**

The purpose of this chapter is to review the literature on the responses achieved with Compudose in steers and heifers. Owing to the lack of information regarding Compudose in heifers, the effects of other oestrogenic growth promotants on heifers are reviewed, as are the effects on milk production and udder development from growing heifers at high growth rates around the time of puberty. Measuring udder development and milk production in beef heifers is not as simple as in dairy heifers, therefore the various techniques that have been used in other studies will be reviewed.

#### **2.1 GROWTH PROMOTANTS**

Growth promoting agents are non-nutritional substances that promote increased growth rates of animals without themselves being used to provide nutrients for growth (McCutcheon, 1989). Exogenous administration of growth promoting agents alter the animals' metabolism so that they lay down more body tissues and grow more rapidly. Changes in conformation, growth rates, mature weight or efficiency of growth frequently result from using growth promoting agents (McCutcheon, 1989). Several types of substances that are classified as growth promoting agents are currently available in New Zealand and overseas. They fall under three categories according to their mode of action and the influence they have on the growth process:

1. Agents that stimulate feed intake;
2. Agents that alter the efficiency of the digestive process by improving the supply and/or balance of nutrients derived per unit of feed consumed (MacColl, 1994);
3. Agents that alter the utilisation and partitioning of absorbed nutrients (McCutcheon, 1989; MacColl, 1994).

### **2.1.1 Agents which stimulate appetite**

Growth promoting agents in this category include exogenous agents that are administered to stimulate animal appetite in order to improve growth rates and the production of saleable meat. Agents such as anti-bacterial agents, oestrogens, androgens and possibly somatotropins have had the effect of improving voluntary feed intake (VFI). However, the increased appetite is not the primary mechanism by which these agents improve growth; rather it is their nutrient partitioning effect (McCutcheon, 1989).

### **2.1.2 Agents which alter digestive processes**

These include antibacterial agents and rumen additives that improve nutrient availability and growth performance by causing beneficial changes to the ruminal or intestinal microflora (Blackman, 1990; MacColl, 1994). Antibacterial agents are mainly used in non-ruminants and act on the bacterial populations to cause an effect on growth and feed conversion efficiency. Rumen additives or rumen modifiers are administered via the feed or intraruminal controlled release devices, and alter the metabolism of the rumen microflora. Examples of rumen modifiers include monensin (Rumensin<sup>®</sup>) and lasaloacid (Bovatec<sup>®</sup>), which are both ionophores. These are beneficial to ruminants because they alter the ratio of volatile fatty acids produced in the rumen by increasing the production of propionate, while acetate and butyrate production declines, and the production of

methane and hydrogen is reduced. Propionate is utilised more efficiently by tissues than acetate or butyrate, therefore advantages in productivity of cattle have been observed with use of these products (McCutcheon, 1989; MacColl, 1994).

### **2.1.3 Agents which alter nutrient utilisation and partitioning**

Agents that have this role are hormones that modify the endocrine system with the effect of altering the partitioning and utilisation of nutrients, thus changing the relative rates at which tissues are synthesised. The technology developed to modify the endocrine system of cattle and alter the partitioning process has resulted in the commercial use of hormonal growth promotants. They are a group of productivity enhancers that have the effect of improving growth rates, feed conversion efficiency and carcass quality when implanted into cattle (MacColl, 1994). These anabolic agents alter the metabolic processes involved in increasing nitrogen retention and protein deposition at the expense of fat deposition. They may also create a negative feedback effect on the production and/or release of naturally occurring sex hormones, reducing the side effects normally associated with secondary sexual characteristics resulting from sex hormones (Blackman, 1990; MacColl, 1994).

Anabolic agents have physiological properties similar to the natural androgenic and oestrogenic sex steroids, testosterone and oestradiol. When implanted into cattle and sheep they supplement the endogenous sex steroids and create a hormonal status more favourable for growth. To obtain maximum growth stimulation, androgens and oestrogens should both be present in concentrations that approximate those normally circulating in entire bulls and cows, respectively (Reynolds, 1980). The principle that dictates which type of hormone is to be used in beef cattle is the need to supplement or replace the particular hormone type that is considered to be deficient in the animal to be treated (Heitzman, 1975; Roche and Quirke, 1986). The best responses to anabolic agents have been observed in steers and veal calves treated with combined preparations of an androgen and an oestrogen, and in heifers treated with an androgen (Reynolds et al., 1980).

The exact mode of action of hormonal growth promotants is not completely understood. It is thought they probably act both directly at the level of the muscle cell through specific steroid receptors, and indirectly via circulating metabolic hormones that alter the metabolism (Reynolds, 1980; Peters, 1985; Sawyer and Barker, 1988).

Hormonal growth promotant products available for use in cattle in New Zealand are in the form of slow release subcutaneous ear implants. The products are either compressed pellets or silastic rubber implants of oestrogen, androgen, progestagen or a combination of these, in their natural or synthetic analogue forms. Examples of these products available in New Zealand include Compudose 200 and 400, Ralgro, Revalor, Synovex-H and Synovex-S (McCutcheon, 1989; MacColl, 1994).

## **2.2 HISTORY OF THE USE OF OESTROGENIC COMPOUNDS**

Synthetic oestrogens were the first growth promoting agents applied in practical beef production with increased liveweight gains occurring. Diethylstilboestrol (DES) was used in heifers from 1948 with improvements in liveweight gain and nitrogen retention resulting from its use (Fitzpatrick, 1986). Oral administration and implantation of this synthetic oestrogen to bulls, steers and lambs had the effect of increasing weight gain and protein deposition (Van der Wal, 1975). DES and Synovex, an implant containing the naturally occurring steroids oestradiol and progesterone, were widely used by the beef industry in the US during the 1960's and 1970's. The benefits from their use was clearly visible to producers. Another anabolic compound, zeranol, was introduced in the late 1960's and marketed as Ralgro. Zeranol is a chemical substance with oestrogenic properties produced by the pasture and grain mould, *Giberalle zeae* (Fitzpatrick, 1986). All these products were marketed in the US from 1969 to 1978 when the use of DES in beef cattle was prohibited owing to public health concerns about safety, and failure of the manufacturers to adequately demonstrate safety. Compudose became available worldwide in 1982.

## 2.3 COMPUDOSE

Compudose is a hormonal growth promotant that contains the active ingredient, oestradiol 17 $\beta$ , a form of oestrogen that occurs naturally in all mammals. Oestradiol 17 $\beta$  is synthesised in the Graafian follicles of the ovaries of all mammalian females. This hormone is responsible for causing the behavioural pattern of oestrus around the time of ovulation (Peters, 1985). Sites of production also include the foetal placenta during the latter stages of gestation and the gonads and/or adrenals in bulls and steers (Velle, 1975).

Oestradiol 17 $\beta$  is incorporated into a silicone rubber matrix which allows the hormone to slowly diffuse out, resulting in a controlled release of hormone over a long period of time. Work carried out by researchers has shown that when steers are treated with oestradiol 17 $\beta$  the concentration of oestradiol 17 $\beta$  in the blood rises in the first few days (to 70 ng/l) and then falls to low levels (5-15 ng/l) within 14 days and remains at these levels throughout the next 100 days (Galbraith and Topps, 1981). The product is available as 200 or 400 day implants with 24 mg supplied in the 200 day implant and 45 mg supplied in the 400 day implant. (Wagner, 1983; Peters, 1985; Sawyer and Barker, 1988).

### 2.3.1 Mode of action

The precise way oestrogen exerts its influence on the growth process is not fully understood, but it appears to influence growth via other metabolic hormones. Oestradiol 17 $\beta$  causes the pituitary, thyroid and adrenal glands to enlarge which stimulates the secretion of growth hormone, insulin, thyroxine and androgens. (Trenkle, 1975; Wagner et al., 1978; Buttery and Sinnott-Smith, 1982; Trenkle, 1983; Buttery, 1985; Peters, 1985; Fitzpatrick, 1986; MacColl, 1994). The increase in plasma concentrations of growth hormone causes blood glucose levels and insulin levels to rise and consequently protein synthesis is stimulated. Oestradiol primarily targets the anterior pituitary gland, but the anabolic response of increasing protein accretion may be affected by a combination of hormones

including growth hormone and insulin (Trenkle, 1975). Growth hormone influences the metabolism of carbohydrates, stimulates lipolysis and inhibits lipogenesis, and alters protein and amino acid metabolism (Davis et al., 1984).

Oestrogenic hormones also increase the quantity of androgens circulating in the blood with the effect of increasing growth (Trenkle, 1975). This hormone is also thought to have a direct effect on muscle cells by binding to specific oestradiol receptors in muscle (Roche and Quirke, 1992). The net effect of these possible modes of action is an increase in protein accumulation inside the muscle cell, and an overall effect of improved rates of daily liveweight gain, feed conversion efficiency and proportion of lean meat in the carcass (Peters, 1985).

### **2.3.2 Safety and residues**

To maintain a marketing advantage it is important that New Zealand maintains its “clean green” image, therefore using hormonal growth promotants may be seen by some as not living up to this requirement. All the products licensed for use in New Zealand contain naturally occurring hormones, or analogues of them, that have potent hormonal activity, therefore it is important that they present no risk to the meat consumer. Concerns over human safety arise from the possibility of side effects on human reproductive function and sexual characteristics, and the association of reproductive hormones with cancer (Peters, 1985). Meat produced from treated steers and heifers commonly contains levels of anabolic steroid residues lower than those naturally occurring in mature bulls and pregnant cows. It is also worth noting that the levels of steroids which could be ingested by eating meat are several orders of magnitude lower than those produced within the human body (McCutcheon, 1989; Roche and Quirke, 1992). The increase in hormone concentrations from consuming meat from animals correctly implanted with growth promotants is negligible, and the fact that the anabolic agents have a low oral activity and are rapidly metabolised in the liver and excreted by the enterohepatic system further reinforces the safety of consuming hormone-treated products (Peters, 1985; Fitzpatrick, 1986; Roche and Quirke, 1992). Ruminants

transform oestradiol 17 $\beta$  into oestrone, and then into the compound oestradiol 17 $\alpha$  which has low biological activity. This compound is excreted in free-form in the faeces (60-70%) and the remainder is excreted via the kidney as glucuronide and sulphide derivatives (Rico, 1983; Roche and Quirke, 1992).

Tissues from animals treated with anabolic agents may contain residues of the growth promoting agent plus its metabolites that have low biological activity. At the end of the withdrawal period less than one percent of the administered dose is present in tissues with greater amounts, up to 10 per cent of the initial dose, present at the site of implantation. Therefore, it is important that all implantation is done at the base of the ear, beneath the loose skin overlying the conchal cartilage. This is a non-edible part of the carcass and is discarded at slaughter. By observing withdrawal periods and following proper implantation procedures anabolic agent residues in meat are at a low level, similar to the levels of naturally occurring steroids in untreated animals (Reynolds, 1980; Roche and Quirke, 1992)

All the compounds currently available in New Zealand and overseas have undergone exhaustive toxicological investigations to satisfy worldwide licensing requirements (Reynolds, 1980). The results of all investigations have conclusively shown that the growth promotants we have registered in New Zealand satisfy all health and safety standards worldwide. All the evidence points towards growth promotants used in our beef industry improving growth rates, feed conversion efficiency and profitability.

Nevertheless, there are some markets that perceive growth promotants as undesirable. Therefore, if New Zealand producers become reliant on using growth promotants as a means of improving their beef production systems, New Zealand's access to some markets may be denied.

## 2.4 RESPONSES FROM USING OESTROGENIC HORMONAL GROWTH PROMOTANTS

Research investigating the effects of natural or synthetic oestrogens in beef production systems overseas and in New Zealand has shown variable improvements in growth and feed efficiency in calves, steers, heifers and bulls (Roche and Quirke, 1992). The degree of the response is dependent on the type of anabolic agent used, class, age and sex of the animal, and the environmental conditions they are subjected to, such as nutrition, husbandry practices and disease status.

Age at implantation and level of feeding influences the response likely to be achieved, with a greater and more consistent response occurring in yearling beef cattle than in calves. This is because the efficiency of lean meat deposition decreases with age, hence older animals are capable of better growth responses to anabolic agents than young fast-growing efficient animals (Roche and Quirke, 1992).

The response is also dependent on the management and feeding system in place, with greater benefits resulting from implanting anabolic growth promotants in beef animals already growing at a fast rate (Peters, 1985), or on a high plane of nutrition. Heifers and steers treated with zeranol and on a high plane of nutrition had a greater rate of gain advantage than those on a lower level of nutrition (Staigmiller et al., 1983; Sawyer and Barker, 1988).

Generally oestrogenic growth promotants are more effective in castrated ruminants, particularly steers, than in bulls or entire females (McCutcheon, 1989) as they reverse the decrease in circulating endogenous hormone levels that results from castration (Van der Wal, 1975; Sammons, 1980; O'Lamhna and Roche, 1983). Oestrogenic implants are generally not renowned for exerting a positive influence on growth in growing bulls because they inhibit testicular development and secretion of androgens (McCutcheon, 1989). Inconsistent growth responses have been found from using oestrogenic compounds in bulls. Van der Wal (1975)

reported that young growing bulls treated with DES and zeranol, both synthetic oestrogen implants, and Compudose (Gray et al., 1986), had slightly improved growth rates, feed conversion efficiencies and nitrogen retention, however more noticeable was the fattening effect following the use of oestrogenic implants (Trenkle, 1975). Oestrogen use in bulls increases the rate of lipogenesis (Unruh, 1986) compared to a reduction in the accumulation of lipids in adipose tissue that normally occurs in steers treated with oestrogenic compounds (MacColl, 1994). Under UK and European conditions oestrogenic implants in bulls have produced a 2-3% increase in average daily gain and 3-7% increase in feed conversion efficiency, and an associated increase in fat cover and decrease in eye muscle area (Gray et al., 1986).

Side effects from the use of oestrogenic compounds in growing bulls are sexual retardation, early onset of testicular degeneration and increased behavioural control, particularly in prepubertal bulls (MacColl, 1994). Entire males in the UK and Ireland had reduced mounting and less aggressive behaviour patterns following oestrogen hormone use (O'Lamhna and Roche, 1983). These effects are due to oestrogen suppressing the gonadotrophic output from the anterior pituitary gland (Roche and Quirke, 1992). Therefore, using oestrogenic growth promoting agents in entire bulls may have management advantages by helping to avoid problems by minimising their aggressive behaviour.

## 2.5 RESPONSES OF COMPUDOSE IN STEERS AND HEIFERS

### 2.5.1 Effects in steers

Research investigating the effects of Compudose has mainly focused on the responses occurring in steers because of the current regulations where Compudose is recommended for use in steers of all ages. Treating heifers, particularly those destined for breeding (Sawyer and Barker, 1988), with Compudose is not advocated because of the negative effects it has on the development and function of the reproductive organs (Moran et al., 1990).

#### 2.5.1.1 *Effects on liveweight gain and feed conversion efficiency*

Steers have shown the largest response to oestrogenic implants, with improvements under feedlot and pasture conditions in liveweight gain, feed conversion efficiency and carcass composition (Mathison and Stobbs, 1983; Bass et al., 1989; Arando-Osorio et al., 1996; Burnham et al., 1997). In a New Zealand trial by Arando-Osorio et al. (1996), 14 month Friesian and Angus x (Hereford x Friesian) steers implanted with Compudose 200 gained an additional 21% liveweight compared to untreated steers. This agrees with Burnham et al. (1997) where Compudose 400-treated yearling steers of Angus, Simmental Cross and Hereford x Angus breeds had a 17% increase in liveweight gain after 266 days of implantation over the control group. Both groups were managed similarly and fed high quality ryegrass and white clover pasture over spring, summer and autumn. Other pasture-based trials where oestradiol implants were used reported an advantage in average daily gain of 22%, 10-30% and 5-30% (Mason et al., 1986; Bass et al., 1989; Baker et al., 1992, respectively).

Oestrogenic growth promoting agents like Compudose are generally used to increase the efficiency of conversion of feed into liveweight. Under both pasture and feedlot conditions improvements in feed conversion efficiency have been

recorded. Arando-Osorio et al. (1996) recorded no difference in herbage intake between implanted and non-implanted steers despite the 21% advantage in liveweight gain, hence treated steers were able to convert more pasture consumed into liveweight gain with an estimated 16% greater efficiency.

Under feedlot conditions cattle implanted with Compudose grew 15% faster, ate 7.6% more feed daily and required 6.7% less feed per unit gain than control steers in a 140-day feeding period (Mathison and Stobbs, 1983). This level of response is comparable to that which should be expected with an oestrogenic implant (Owens and Gill, 1980; Perry, 1980; Minnish and Fox, 1982) and has occurred when Ralgro and Compudose were implanted into steers (Keane, 1983). Variable responses in feed conversion efficiency under feedlot conditions have been observed with Carroll et al. (1979), Parrott et al. (1979), Utley et al. (1980) and Turner et al. (1981) not detecting any improvement in feedlot feed utilisation efficiency compared to Wagner et al. (1979) and Mathison and Stobbs (1983) measuring a 7.7% and 6.7% improvement in feed efficiency, respectively. Wagner et al. (1979) also reported a 17.3% increase in daily gain when 240 kg steers were implanted with Compudose. Ralgro and Compudose have been compared and have similar effects in terms of daily gain. The advantage of Compudose over other implants, like Ralgro, is its long acting effectiveness (Keane, 1983).

#### 2.5.1.2 *Effects on carcass and meat quality*

Compudose and other anabolic growth promotants have a positive influence on carcass and meat quality characteristics by partitioning nutrients towards lean meat production rather than fat (McCutcheon, 1989). Oestrogenic compounds are responsible for prolonging the juvenile growth phase during which nutrient intake is directed into bone and muscle growth rather than fat deposition (Fitzpatrick, 1986). Carcasses of animals treated with Compudose generally have decreased fat content and increased lean meat content (Roche and Quirke, 1992; MacColl, 1994). The decrease in fat to protein ratio in carcasses of animals treated with

Compudose or other oestrogenic compounds is evident through the reduced kidney, heart, pelvic and abdominal fat (Heitzman et al., 1981) and subcutaneous fat depths measured three-fourths of the length of the *longissimus thoracis* muscle (LD) between the 12th and 13th rib (Burnham et al., 1997). Increases are observed in carcass weight, dressing out percent and rib-eye area (Mathison and Stobbs, 1983; Burnham et al., 1997). Such characteristics reflected the steroidal stimulation of protein deposition at the expense of fat deposition in steers and heifers. In contrast, Johnston et al. (1984) found bulls treated with oestrogenic compounds had increased levels of fat content.

Generally oestrogenic compounds do not affect physical and chemical parameters of meat quality (MacColl, 1994). Eating quality of meat with regard to flavour, texture, juiciness, cooking losses and palatability have not been affected by hormonal growth promotants (Patterson and Salter, 1985), however inconsistencies do exist, especially with the tenderness attribute. Oestrogen-treated cattle have tended to produce slightly tougher meat (Apple et al., 1991; Gerken et al., 1995), however many studies have shown no effect (Ntunde et al., 1977; Huck et al., 1991). Preston (1975) stated that feeding oestrogen additives to cattle results in higher hydroxyproline, elastin and mucoprotein hexaamine content of the muscle, indicating increased connective tissue content. Meat from hormone-treated steers and heifers has shown a reduction in fat content and small increases in moisture and protein contents. This implies that meat quality differences, particularly tenderness, are solely attributable to decreases in fat content and marbling scores. However, the influence of oestrogens on tenderness has been inconsistent. Oestrogen-implanted bulls reared for beef had increased levels of intramuscular fat, improved tenderness and flavour (Greathouse et al., 1983). Chemical composition, pH and colour of lean meat were not affected by implantation (Fitzpatrick, 1986).

Compudose use in steers under pastoral conditions causes liveweight gain and feed conversion responses and improvements in carcass quality. An increased dressing-out percent (DO%), rib-eye area, and carcass lean meat content, together with a reduction in fat content, contribute to an improved carcass quality.

## 2.5.2 Effects in heifers

### 2.5.1.2 Effects on liveweight gain and feed conversion efficiency

Little information exists on the effect of Compudose in heifers because of past regulations preventing the use of oestrogenic implants in breeding heifers. However, Johnson et al. (1987) and Stobbs et al. (1988) were the first researchers to demonstrate that Compudose can improve growth rates and feed conversion in feedlot-fed heifers. Stobbs et al. (1988) conducted a series of trials throughout Canada using beef-breed heifers. The heifers, with an average initial weight of 245 kg (228-269 kg), were implanted with Compudose 200 (24 mg) and fed a grower diet (corn silage or chopped brome alfalfa hay) for 84-112 days followed by a finishing diet (75% barley or 50% high moisture corn) for the remainder of the trial (63-70 days). An overall improvement in liveweight gain by 6.7% and feed:gain ratio of 4.1% resulted. Implanted heifers tended to consume more feed with an improvement in feed conversion efficiency still resulting. However, it was noted that of the 174 heifers implanted, responses varied from positive responses to negative responses, which further reinforces the variable effects anabolic implants can have in animals (Basarab et al., 1984). These responses are similar to the results of Johnson et al. (1987) where an overall increase in average daily gain of 8.2% resulted. In that trial Angus heifers grazed bermudagrass-fescue pastures during a 56 day growing period and were fed concentrate and sorghum silage *ad libitum* during a 98 day finishing period. No differences in average daily dry matter intake resulted, therefore there was likely to be an improvement in feed conversion efficiency in Compudose-treated heifers.

### 2.5.2.2 *Effect on carcass and meat quality*

Carcass quality characteristics of Compudose-implanted heifers are similar to those which occur in steers. Johnson et al. (1987) and Stobbs et al. (1988) both reported heavier carcasses, no difference in DO%, increases in rib-eye area and a reduction in the fat thickness. Literature on meat quality of heifers implanted with Compudose was not available.

Heifers treated with Compudose also showed increased udder development. Johnson et al. (1987) subjectively scored udders and reported that treated heifers had udders that were similar to those of heifers 3-4 weeks prior to parturition. Therefore implanted heifers were found to have more developed udders at the time of observation.

Heifers treated with Compudose appear to have similar responses to those of steers with improvements in liveweight gain, feed conversion efficiency and carcass quality occurring, but of lesser magnitude. Compudose appears to have an influence on mammary gland development as was observed by subjective evaluation. However, these responses were from heifers in a feedlot-based environment.

## **2.6 RESPONSES TO OTHER OESTROGENIC IMPLANTS IN HEIFERS**

Responses from using other oestrogenic compounds, for example zeranol, in heifers have been investigated and reported. This work has looked at the effect oestrogen has on reproductive traits, skeletal development, growth rates, udder development, calf performance, and carcass and meat characteristics. Owing to the lack of information available about the effects of Compudose in heifers, inferences can be made about the effects and responses that an oestrogenic compound, like Compudose, may have on heifers.

### **2.6.1 Effects on liveweight gain and feed conversion efficiency**

Consistent improvements in liveweight and liveweight gain have been reported from oestrogenic implants in growing and fattening heifers (Perry et al., 1970; Staigmiller et al., 1983; Deutscher et al., 1986; Moran et al., 1991; Hancock et al., 1994). However, the age and physiological state of the female animal may influence the response (Anthony et al., 1981). The literature results are not always consistent; for example, bodyweight and growth rates of pregnant heifers were not influenced by zeranol implants (36 mg) in one study (Anthony et al., 1981), compared to other studies with younger, non-pregnant heifers where growth rate and liveweight responses were reported (Nelson et al., 1972; Staigmiller et al., 1978; Ward et al., 1978). Therefore, Compudose implants in heifers are likely to result in improvements in liveweight gain. However, reports on feed conversion efficiency are scarce.

### 2.6.2 Effects on skeletal development

Brannang (1971) stated that one of the main effects of oestrogens is their accelerating effect on bone ossification whereas through ovariectomy (castration) ossification is delayed. Hansel and McEntee (1970) stated that, by hastening the ossification of the epiphyseal plates in the long bones of the skeleton, oestrogens will limit skeletal growth. This may lead to shorter bones and carcass length in oestradiol-treated cattle.

Research investigating the effects of oestrogenic compounds on skeletal development has shown an increase in pelvic area (Ellington et al., 1978; Anthony et al., 1981; Staigmiller et al., 1978, 1983; Bolze and Corah, 1988; Deutscher et al., 1986; Hancock et al., 1994), and inconsistent hip height responses in heifers (Staigmiller et al., 1983; Deutscher et al., 1986; Carpenter and Sprott, 1991; Moran et al., 1991).

Hip and shoulder height differences between heifers implanted with growth promotants and control heifers have varied with some studies reporting no differences (Staigmiller et al., 1983; Deutscher et al., 1986; Moran et al., 1991), while Hancock et al. (1994) reported that heifers implanted at 6 and 12 months of age with Synovex C (implants of 10 mg oestradiol benzoate and 100 mg of progesterone) were shorter at 22 months of age than control heifers. These results do not agree with the theory that the oestrogenic properties of implants increases long-bone growth, thus increasing hip height (Carpenter and Sprott, 1991). The implants may cause early maturation of the epiphyseal plates of long bones and retard growth. Whittier et al. (1991) reported that heifers implanted at 2 months of age were shorter than non-implanted heifers that were 18 months of age. Zeranol-implanted heifers had lower body weight-adjusted hip heights than control heifers (Staigmiller et al., 1983). This agrees with Burnham et al. (1997)

where weight-corrected heights of Compudose-treated steers at day 266 were lower than those of non-treated steers. This may suggest a slightly more blocky conformation.

### 2.6.3 Effects on reproductive performance

Oestrogenic implants have been reported to have negative consequences for the reproductive development of breeding heifers. Evidence for this includes a decrease in conception rates (Nelson et al., 1972; Staigmiller et al., 1978; Anthony et al., 1981), a delay in attaining puberty (Deutscher et al., 1986; Moran et al., 1990), a high incidence of abortion (Anthony et al., 1981) and a greater incidence of non-ovulatory oestrus (Deutscher et al., 1986). However, zeranol treatment has not appeared to have any effect on the attainment of puberty in other studies (Staigmiller et al., 1983).

Oestradiol  $17\beta$  and luteinising hormone (LH) have an important role in both the onset of puberty and subsequent reproductive function of heifers (Hansel and Convey, 1983; Kinder et al., 1987). One critical event in triggering first ovulation is thought to be the ability of the hypothalamus to overcome the negative feedback effect of oestradiol (Day et al., 1984). This allows the hypothalamus to stimulate hourly releases of LH from the pituitary, which are essential for ovulation (McLeod et al., 1985; Kinder et al., 1987). In addition, LH plays a crucial role in maintaining the corpus luteum, while oestradiol is thought to have a role in luteolysis. Long-term alterations in blood concentrations of LH and oestradiol would therefore be expected to affect both puberty and subsequent reproductive efficiency of heifers. Compounds like zeranol and oestradiol  $17\beta$ , both implants with oestrogenic properties, inhibit LH secretion, which in turn delays puberty and retards growth of the reproductive tract (Moran et al., 1990). Subsequently oestrous cycling is delayed until the implant expires.

Once a treated heifer ovulates, there appear to be few subsequent effects of anabolic agents on the ovulatory cycle - irrespective of the time after implantation. This is illustrated by the fact that there was no effect of treatment on the occurrence of silent ovulations or transient prepubertal increases in progesterone concentrations (Deutscher et al., 1986; Moran et al., 1990).

A high incidence of abortions and reabsorption of foetuses was reported to occur when heifers were implanted with zeranol at 100 days of gestation (Anthony et al., 1981). The ability of zeranol to bind to uterine oestrogen receptors in rats (Katzenellenbogen et al., 1979) and calves (Anthony et al., 1981) is thought to cause this effect.

Oestrogens have a tendency to increase vulva length which illustrates that the growth of these tissues is probably dependent upon oestrogens in normal circumstances. The presence of oestrogenic compounds circulating in the blood, such as oestradiol and zeranol, accelerates growth of the vulva (Moran et al., 1990). Cystic corpora lutea or a luteinized follicle were evident in the ovaries of some non-pregnant heifers that had been treated with zeranol (Deutscher et al., 1986). This abnormality may occur in about 2% of the cow population with the cause of this condition uncertain, but it is associated with anoestrus (Salisbury et al., 1978).

Oestrogenic growth promotants, like Compudose, will expose the heifer to more than the acceptable amounts of oestrogen, resulting in an interaction with oestrogen receptors which evokes many of the same biological and biochemical responses that occur naturally (Katzenellenbogen et al., 1979). Therefore, long term implantation may stimulate a negative feedback to the pituitary gland, disrupt cyclical activity, retard development and compromise normal fertility (Reynolds, 1980). Work carried out using Ralgro, which is a weakly oestrogenic compound (between  $1/300$  and  $1/800$  times that of oestradiol  $17\beta$ ) has affected the reproductive performance and fertility of heifers (Nelson et al., 1972; Katzenellenbogen et al., 1979; Staigmiller et al., 1983; Deutscher et al., 1984; Moller, 1984). Noticeable adverse effects are decreases in conception rates (Nelson et al., 1972; Staigmiller

et al., 1983; Moller, 1984), a delay in puberty (Staigmiller et al., 1983), and delayed oestrous cycling (Deutscher et al., 1984). Generally oestrogenic growth promotants, like Compudose, may impair normal reproductive development and function in heifers.

#### **2.6.4 Effects on calving difficulty and calf performance**

Pelvic size is one factor influencing dystocia levels in heifers, therefore an increase in pelvic area may lower the dystocia levels recorded in first calving heifers (Anthony et al., 1981; Basarab et al., 1993). The increase in pelvic area is based on the theory that the oestrogenic properties of zeranol stimulate flat (pelvic) bone growth (Staigmiller et al., 1983). The differences in pelvic area have been reported to be through differences observed in the vertical and horizontal measurements of the pelvis (Staigmiller et al., 1983). In studies by Anthony et al. (1981) and Deutscher et al. (1986), vertical pelvic measurements were significantly greater than horizontal measurements which was reflected in the observed difference in pelvic area between zeranol-treated and non-treated heifers. This indicates more growth in the length of the illea shafts than in the width between them. Adjusting pelvic size measurements for body weight indicates that skeletal pelvic size grows more rapidly than soft tissue when females are implanted with zeranol (Deutscher et al., 1986; Staigmiller et al., 1983). Zeranol is capable of increasing pelvic area dimensions in nursing heifer calves (Ellington et al., 1978) and in heifers post-weaning (Staigmiller et al., 1978). However, the differences in pelvic development observed between treated and non-treated heifers are temporary. In a study by Anthony et al. (1981) heifers treated with zeranol (36 mg) implants had larger pelvic areas during the first, third and fourth months after treatment compared to untreated heifers. No differences were observed between control and treated animals during the fifth month after treatment. This was thought to be due to the expiration of the implant (Anthony et al., 1981).

Oestrogenic implants like zeranol appear to have very little influence on calving performance with calf birth weight, weaning weight and calving difficulty not being affected in treated dams compared to untreated dams (Anthony et al., 1981; Deutscher et al., 1986). Deutscher et al. (1986) reported that heifers implanted once only at 1 month of age, or implanted at 1 month and again at 9 months of age, had slightly lower calf birth weights than control heifers, but this may have been due to nutritional differences. In the same trial, heifers implanted at 6 months of age and 9 months of age had no differences in calf birthweight compared to non-implanted heifers. Anthony et al. (1981) reported that heifers treated with zeranol at 100 days of gestation tended to produce calves lighter at birth in one trial, but in a second trial there were no differences in calf birth weight or weaning weight between treated and untreated heifers. Calving difficulty scores have been reported to be improved following zeranol treatment (Anthony et al., 1981), but other evidence suggests no differences in dystocia between treated and non-treated heifers. (Deutscher et al., 1986). This may reflect a combination of lighter calf birthweights and/or an increase in pelvic area.

No evidence of adverse effects of zeranol on cow rebreeding has been observed (Deutscher et al., 1986). Moran et al. (1990) reported that once a treated heifer ovulates there appears to be few subsequent effects of anabolic agents on the ovulatory cycle - irrespective of time after implantation.

The increased growth rates that Compudose causes in dams do not appear to exist in the offspring, instead the occurrence of lighter calves is more likely. This in combination with larger pelvic size would be expected to reduce the levels of dystocia. This may be particularly important in the OBH system where dairy-cross heifers mated to large beef breed sires experience calving difficulty. However, management practices appear to have alleviated this problem.

### 2.6.5 Effects on mammary development and milk production

Both ovarian and pituitary gland hormones stimulate mammary development. The primary stimulators of mammary growth are oestrogen and progesterone, which act synergistically with prolactin, growth hormone and adrenocorticotropin (Cowie et al., 1966; Emery, 1969). The development of secretory tissue (mammogenesis), the onset of secretory activity in the individual secretory cells (lactogenesis) and the cow's ability to maintain an already established yield (galactopoiesis) are processes that influence the cow's milk yield and are under hormonal control. Therefore, alteration of the normal endogenous hormonal status of heifers may have severe implications for mammary gland development and milk yield (Sejrsen, 1978). A major part of the development of mammary glands takes place between birth and first calving. Consequently the way in which heifers are reared has a profound effect on mammogenesis.

Mammary glands begin to form at the embryonic stage and continue to develop and grow throughout the foetal stage. The growth of the mammary gland is isometric in the foetus (same relative growth rate as the body) (Cowie and Tindal, 1971). Oestrogen is mainly responsible for the ductal development in mammary glands (Cowie and Tindal, 1971). Oestrogen administration during gestation results in foetal mammary malformations in rats, the severity of which depends on the dose of oestrogen, ranging from mere hypertrophy of the nipple to complete disappearance of the mammary gland (Cowie and Tindal, 1971).

From birth to puberty, mammary gland growth is minimal. Studies by Sinha and Tucker (1969) illustrated the pattern of mammary gland growth in heifers from birth to 1 year of age based on DNA, RNA, collagen and lipid concentrations. From birth to 2 months of age mammary DNA content increases 1.6 times faster than body weight, which is followed by a phase of greatly accelerated mammary growth until about 6-9 months of age when the DNA increases 3.5 to 5.5 times faster than body weight (Tucker, 1969). This phase of positive allometric growth (when the udder is growing at a relatively higher rate than the body) coincides with the onset of oestrogen secretion from the ovary in the rat (Cowie, 1949) and

the heifer (Wallace, 1953). This continues until about 2-3 months after first oestrus in the heifer (Sinha and Tucker, 1969; Pritchard et al., 1972). There is a rapid increase in the size of the fat pad and the ducts grow into the fat pad. Therefore the fat pad predetermines the maximal outgrowth of the ducts (Sejrsen, 1984).

After the period of allometric growth, the mammary growth becomes isometric again until the heifer becomes pregnant. The DNA studies by Sinha and Tucker (1969) found that the growth of the mammary gland were 1-1.5 times greater than body weight during this period. Sinha and Tucker (1969) found that 9 month old heifers having showed oestrus at 7-months had the same amount of secretory tissue per 100 kg liveweight as 16 month old heifers. The growth consists mainly of stromal tissue with some branching of collecting ducts. The amount of mammary gland tissue formed in a non-pregnant heifer is negligible relative to the growth during pregnancy. Furthermore, the amount of mammary gland tissue present at puberty is poorly correlated with subsequent milk yield (Tucker et al., 1973). Nevertheless, events which occur at the time of puberty and before mating can have a profound influence on subsequent milk production. (Holmes and Wilson, 1987).

Most of the heifer's mammary gland growth occurs during pregnancy at an exponential rate so that at the end of each month there is 25-35 percent more tissue present than there was at the end of the previous month (Swanson and Poffenbarger, 1979). This is mainly the period of lobulo-alveolar development (Sejrsen, 1984). During the first 3-4 months of pregnancy relatively little mammary growth takes place, whereas during the last two to three months of pregnancy most tissue is laid down. During the first half of pregnancy the tissue increase is mainly an extension of the duct system through the fatty pad and proliferation of the supporting tissues. At the fifth to sixth month of pregnancy a small amount of serous secretion is present. This coincides with the appearance of alveoli, and during the remainder of pregnancy these increase in number. Most of the growth of the udder during the last part of pregnancy is an accumulation of

secretions, rather than from cell division (Cowie and Tindal, 1971). However, it has been shown that cell division takes place throughout pregnancy and that the amount of secretory tissue does, in fact, increase (Munford, 1964).

Mammary gland growth is essentially complete at the time of parturition (Cowie et al., 1980) with the fully developed mammary epithelium consisting of branching ducts, and lobes and lobules of alveoli (Cowie and Tindal, 1971). Experiments with goats (Anderson et al., 1981) and ewes (Davis et al., 1980) suggest that some growth can occur very early in lactation, particularly if extra milking stimulus is given, for example cows suckling several calves or when lactation is induced. Following peak lactation the number of secretory cells decreases, through either autolysis or reabsorption, there is a decline in the activity of the secretory cells and a decrease in the supply of nutrients to the tissue (Holmes and Wilson, 1987).

Mammary gland development in heifers is significantly influenced by the plane of nutrition before puberty. Several experiments have reported reduced milk production in heifers fed high planes of nutrition during the allometric phase of mammary growth (pre-puberty) when heifers are between 200 and 350 kg liveweight (Sejrsen, 1978; Sejrsen et al., 1982). Excessively high rates of liveweight gain, greater than 0.8 kg/day, especially during prepubertal growth, have a negative effect on milk production (Little and Kay, 1979). However, when high planes of nutrition are applied post-puberty and during pregnancy or lactation the opposite effect on milk yield occurs (Johnsson, 1988). There appears to be a critical upper limit for average daily gain of 0.7-0.8 kg/day in Holstein heifers beyond which milk yield progressively declines (Amir and Kali 1974, 1975). This agrees with the study of Swanson (1967) where Jersey heifers had an optimum growth rate of 0.5 to 0.6 kg/day from birth to conception. The high level of energy intake between 200 and 350 kg liveweight which equates to high daily liveweight gain is of great importance for later milk yield. Heifers growing at more than 1 kg/day from 3 to 9 months of age had a much lower milk yield than heifers not putting on more than 0.65 kg/day. From this evidence the already mentioned critical limits were derived (Sejrsen, 1978).

A similar effect of high growth rates from a high plane of nutrition and an energy-dense diet between 2 and 8 months of age was evident in Hereford heifers. Hereford heifers fed a high (H), moderate (M) or low (L) level of a grain and hay ration between 2 and 8 months of age grew at 0.91, 0.67 and 0.55 kg/day, respectively, during this period. Between 8 and 14 months of age, intake was restricted in half of the H heifers (HL), *ad libitum* in half of the low group (LH) and was moderate in all other heifers (HM, MM and LM, respectively). Despite weighing significantly less at first calving, LM heifers produced 50% more milk and weaned heavier calves (+41 kg) in the first lactation than either the MM or HM heifers. In two subsequent lactations, LM heifers continued to produce 30-50% more milk and wean heavier calves than HM heifers. Of particular importance was that the severe restriction of the growth of rapidly-reared heifers after 8 months of age (HL) only marginally overcame the detrimental effects of the initial rapid growth, while *ad libitum* feeding of restricted heifers after 8 months of age (LH), so that these heifers achieved the same liveweight at 14 months as HM heifers, did not result in a similar depression in milk yield (Johnsson and Obst, 1984).

It has been suggested that the reduced milk production is due to two different factors, excessive fat deposition in the developing udder and reduced mammary development due to high energy intakes lowering circulating levels of the hormones which promote udder development (Sejrsen et al., 1983). Amir et al. (1968) investigated the effect of feed energy level on udder development and found that the amount of fat in the udder increased with the level of energy. On the other hand, the amount of secretory tissue decreased in inverse proportion to the increase in energy level. Therefore, the impaired milk production in heifers on a high plane of nutrition may be from impaired development of mammary secretory tissue (Swanson, 1960). Swanson (1960) slaughtered cows reared at different feeding intensities and found that mammary development appeared to be impaired by high planes of nutrition during rearing. Similar conclusions were drawn by Little and Kay (1979) based on visual appraisals. Examination of cross-sections of udders of normally grown and fattened heifers has shown that normally reared heifers were essentially normal in gross appearance and structure compared

to fattened heifers which had abnormal internal structures. In fattened heifers, the outer areas of what should have been milk secreting tissue were filled with cysts or cavities. These spaces were connected to the ducts of the normal part of the mammary gland, indicating that they were probably part of the gland which failed to develop properly (Swanson, 1967). Udders of well-fed heifers had incompletely-developed parenchyma on the periphery of glands excised from fattened twins with low milk yields at the end of their second lactation. The duct and framework of the gland appeared to be normal but alveolar-secreting tissue had not filled the spaces provided (Swanson, 1960).

Swanson (1960, 1975) has suggested that abnormal mammary gland development may be caused by storage adipose tissue in the inguinal pad being perhaps more dense and less vascular, or consisting of a different type of fat, than in a lean heifer. Overfeeding calves results in the loose connective tissue that the duct system normally branches into being replaced by fat deposits. This causes poor or abnormal growth of the secretory tissue into the fat-packed udder area. Dissection of the mammary parenchymal tissue of heifers reared on different levels of food intake relative to udder development and subsequent milk yield was carried out by Amir et al. (1968). They reported that at four different ages between 6 and 16 months, udder weight and parenchymal mass were directly related to liveweight and to plane of nutrition. No evidence was found that the greater fat deposition in rapidly-reared heifers had restricted parenchymal development as Swanson (1960) hypothesised.

Mammary growth is a component of the reproductive system of the animal and is closely related to general body growth and under similar hormonal control. Feeding high energy diets before puberty might affect the secretion or biological action of certain mammogenic hormones at a critical period when the gland is undergoing a major phase of primary duct extension. It has been shown experimentally that abnormal hormone treatment can produce abnormal mammary glands which are deficient in lactation, so it is quite possible that the true cause of deficient development of fat mammary glands is associated in some with hormone action (Swanson, 1967; Sejrsen et al., 1983; Sejrsen et al., 1986). Before

puberty, overfed heifers have lower circulating growth hormone levels (Sejrsen et al., 1983) and recent evidence suggests that the administration of exogenous growth hormone can increase development in prepubertal heifers (Sejrsen et al., 1986).

Sejrsen et al. (1982) found that the plane of nutrition did affect mammary development in heifers during the allometric period of mammary growth with less mammary gland secretory tissue and more adipose tissue occurring in overfed heifers compared to normally fed animals. Heifers were grown at 0.6 kg/day (restricted feeding) or 1.2 kg/day (*ad libitum* feeding) from either 7 months of age (180 kg) (pre-puberty) to a common final liveweight of 320 kg or from 300 kg to 440 kg (post-puberty). *Ad libitum*-fed pre-pubertal heifers had heavier mammary glands than those on restricted feed. The increase in weight of the mammary gland, however, was from an increase in the amount of adipose tissue because restricted-fed heifers had 30% more mammary parenchyma and 47% more total parenchymal DNA than rapidly-reared heifers at 320 kg liveweight. In contrast, there was no difference in growth of mammary secretory tissue between feeding levels in postpubertal heifers fed restricted or *ad libitum* diets. Composition of the mammary parenchyma was not affected by plane of nutrition in prepubertal or postpubertal heifers. Harrison et al. (1983) fed heifers to grow at 0.57 (L), 0.76 (M) or 1.18 (H) kg/day between 3 and 12 months of age to encompass the greater portion of the allometric growth period. When compared at the same age, the weight of the dissected mammary parenchyma was 71% greater in L heifers than in H heifers. An analysis of the individual parenchymal weights indicated a quadratic relationship with liveweight gain during rearing similar to that reported previously for milk yield (Little and Harrison, 1981).

The implications of Compudose treatment for mammary development in breeding heifers are relatively unknown. Johnson et al. (1987) reported that heifers treated with Compudose generally had udders that had developed similarly to those of untreated heifers 3-4 weeks prior to parturition. Other oestrogenic compounds were reported to increase teat and udder development (Deutscher et al., 1986; Moran et al., 1991) because of the stimulatory effect oestrogen has on mammary

gland development (Sejrsen, 1984). However, zeranol implants have been reported not to have adverse effects on milk production as reflected by calf weaning weights (Deutscher et al., 1986).

Growing heifers more rapidly by feeding high planes of nutrition or altering the hormone balance around the time of puberty influences mammary development and subsequent milk yield. Implanting heifers with Compudose to increase growth rates may cause hormonal imbalances which have negative side effects on mammary gland development and milk production. However, the mode of action of oestradiol  $17\beta$  needs to be considered. If oestradiol  $17\beta$  influences growth hormone secretion, the effect of greater liveweight gains from more protein deposition may cause mammary gland development to improve. Furthermore, Compudose affects the partitioning of nutrients from fat to protein, therefore maybe excessive fat deposition will not occur in the developing udder and consequently mammary gland development will not be impaired.

## **2.7 DETERMINING MILK PRODUCTION IN BEEF COWS AND HEIFERS**

A dam's milk production is the most important factor influencing the weaning weight and growth rates of calves pre-weaning (Neville, 1962). Therefore several investigations have been undertaken to determine the milk production of single-suckled cows (Barton, 1970). Determining the milk producing ability and lactation pattern in dairy cows is easier than in beef cows because of the regular milking routine they are subjected to, the use of milking sheds and the herd testing facilities available. Estimating the lactation characteristics of beef cows is much more difficult with each method having its own advantages, disadvantages and purpose. Weaning weight and growth rate from birth to weaning have been used to estimate the level of milk production (Barton, 1970). High positive correlations between milk production of the cow and growth of her offspring have been reported to range from 0.30 to 0.82, with the size of the correlation dependent on

the stage of lactation (Barton, 1970). As lactation proceeds the size of the correlation between milk production and calf growth rate tends to decrease reflecting the diminishing dependence of the older calf on milk for its growth (Barton, 1970). In a study by Rutledge et al. (1971) approximately 60% of the variation in 205-day weight could be attributed to the direct influence of the dam's milk yield. Other methods have been employed to provide a more accurate picture of milk production. These methods include the following, alone or in combination:

- Weigh-nurse-weigh method
- Oxytocin technique
- Isotope dilution technique
- Water dilution technique

### **2.7.1 Weigh-nurse-weigh**

The weigh-nurse-weigh (WNW) technique involves separating the calf from its dam for c. 16 hours, weighing, allowing it to suckle and reweighing again. The difference between the two weights is assumed to equal the weight of milk consumed (Barton, 1970; Huw and Morgan, 1991). This technique is based on the assumption that the milk produced is equal to that consumed by its calf; hence it is limited by the ability of the calf to consume milk and therefore depends on the calf's appetite at the time, rather than the cow's capacity to produce milk. Barton (1970) points out that errors may arise due to an interaction between the cow and calf, which may depend on such factors as sex of the calf, its birthweight and subsequent weight changes, its vigour, breed, age, and its ability to stimulate the cow's let-down process. The WNW method suffers from the major disadvantage of having to determine a small increase in liveweight, due to milk consumption in a relatively large animal. Furthermore, extreme care is required to avoid cross-suckling between cow-calf pairs, or calf urination and defaecation between

suckling and weighing (Nicol and Irvine, 1973). This method also interferes with the normal behaviour of grazing animals (Coombe et al., 1960) and provides intermittent samples.

Also known as the plunketing system (Walker and Pos, 1963) or weigh-suckle-weigh method (Williams et al., 1979a), it is significantly dependent on the time of separation of the calf from its dam (Williams et al., 1979a, 1979b). The time of separation has varied from 4 hours (Dawson et al., 1960) to 16 to 19 hours (Kress and Anderson, 1974 cited by Williams et al., 1979a). Williams et al. (1979a) investigated the effect of the separation interval on the WNW milk yield estimates and found that the 8 hour separation interval gave the best estimate of milk production during early lactation. This is because it: had less measurement error and a higher correlation with calf average daily gain than the 4 hour interval ( $r = 0.46$  compared to  $0.27$ ); produced less observable irritation and discomfort to the cows; and was closer to the natural interval than the 16 hour interval (Williams et al. 1979a). The shorter separation interval of 4 hours between nursing yielded more milk per 24 hours, suggesting that the additional milk was due to frequent oxytocin release (Williams et al. 1979a). This agrees with the dairy industry reports where milking 3 times a day increased milk production (Knight et al., 1988). After 16 hours of separation, the udder was subjected to increased pressure compared to the 4 hour and 8 hour separation interval where there was a decrease in udder pressure, which may be a factor causing reduced production for the 16 hour interval. Least square means for the 24 hour milk production following 4 hour, 8 hour and 16 hour separation were  $9.2 \pm 0.21$ ,  $7.6 \pm 0.21$  and  $5.9 \pm 0.21$  kg, respectively (Williams et al., 1979a).

Williams et al. (1979b) reported repeatabilities between consecutive measurements (7-21 days and 28-56 days of lactation, respectively) for 4, 8 and 16 hour milk production estimates of 0.55, 0.61 and 0.79, respectively. The higher repeatabilities for the 8 hour and 16 hour milk production estimates compared to the 4 hour milk production estimates possibly occurred because the 4 hour estimates involved more measurement error, or because the 8 hour and especially the 16 hour estimates were measuring udder capacity rather than actual milk

production potential. The decreased coefficient of variation as separation time increased also suggests that some other factor was reducing the natural variation in milk production and that measurement error was less for the longer intervals (Williams et al. 1979b).

### **2.7.2 Oxytocin Technique**

To overcome the problems of the WNW method, the oxytocin technique was developed initially by McCance (1959) for use in sheep, and has since been adapted for use in beef cows (Lamond et al., 1969). Oxytocin is a hormone released from the posterior pituitary gland following stimulation of the sensory nerve endings in the teat and base of the udder. The oxytocin reacts with the receptors on the myoepithelial cells in the mammary gland and causes them to contract. Contraction of the myoepithelial cells increases the pressure in the alveoli resulting in milk ejection from the mammary gland (Whittemore, 1980; Holmes and Wilson, 1987).

Milk production of the cow can be measured by total evacuation of the udder with oxytocin (10 to 20 IU) before and after a 6 hour separation period of the cow and calf (Nicol and Irvine, 1973). The udders can be emptied by machines or by hand milking. The milk obtained at the beginning of the separation period is discarded, while the milk collected at the end of the separation period is weighed to provide an estimate of milk yield (Nicol and Irvine, 1973; Le Du et al., 1979). The milk production measured by this technique will only equal milk intake if the rate attained during the measurement period is the same as the rate obtained by the calf or lamb before and after the measurement period (Moore, 1967).

Huw and Morgan (1991) compared the monthly milk production estimates from the WNW method, following a 17 hour separation, to those obtained by the oxytocin method. Results showed that both the WNW and oxytocin method were of similar precision in predicting calf liveweight gain. This agrees with Le Du et al. (1979) who found no significant difference between the two techniques in

estimating milk yield. The excellent agreement of the two techniques may not be as good in the very early stages of lactation owing to the consumption of milk by the calf and the dams milk production not reaching equilibrium (Le Du et al., 1979). The oxytocin method is more convenient and less time consuming than the WNW technique (Moore, 1967; Le Du et al., 1979). However, the main criticism with the oxytocin method is the influence that high doses of oxytocin may have on the subsequent rate of milk secretion (Sprain et al., 1954; Donker et al., 1954).

Machine milking beef cows without the use of exogenous oxytocin is not a satisfactory method of measuring milk production when compared with the WNW technique (Somerville and Lowman, 1980). Cows milked by machines showed trends of drying off earlier and milk yields that were more variable than those determined by the calf suckling technique (Somerville and Lowman, 1980; Totusek et al., 1973). The limited appetite of the calf for milk in early lactation noted by Drewry et al. (1959) and Le Neindre and Petit (1975) may have reduced the potential yield of individual heifers and hence reduced the variability of yield estimates. Another reason for the greater variation in machine milked cows may be the failure of the pre-milking stimulus to elicit a satisfactory milk-ejection reflex. In this context, stress before and during milking may have been a contributory factor (Somerville and Lowman, 1980).

### 2.7.3 Isotope Dilution Technique

This was developed to eliminate the errors from the WNW method and to avoid possibilities of biased results using oxytocin. Basically the technique is an enhancement of the WNW method and estimates calf milk consumption following a 6-hour separation period. By measuring the ratio of two isotopes in the calf's blood, one administered as a known quantity to the calf and the other obtained by the calf suckling an unknown quantity of milk of known radioactivity per ml, milk intake can be calculated. The two isotopes,  $^{131}\text{I}$  and  $^{125}\text{I}$  are given as sodium iodide since iodide is secreted in milk and readily absorbed from the gut (Miller

and Swanson, 1963). This method overcomes the disadvantages of using oxytocin, however considerable animal handling is still necessary, as well as separation of the calf from the cow for six hours.

This technique involves removing cows and calves from pasture and separating them for 2 hours. A known volume of milk is removed from the cow and then the calf is allowed to suckle in order to empty the udder. The calf is then removed and each cow receives an intravenous injection in the mammary vein of  $^{131}\text{I}$  as iodide in sterile water. Cows and calves are isolated for 6 hours with cows having access to pasture and water. This period allows the build up of the products of 6 hours' milk secretion and the equilibration of the milk with the  $^{131}\text{I}$  iodide. The milk sample taken is incubated for 6 hours at  $37^\circ\text{C}$  with  $^{125}\text{I}$  to stimulate the conditions applying to the  $^{131}\text{I}$ . After the 6 hours, but before suckling, the cow's milk is sampled for  $^{131}\text{I}/\text{ml}$ , and the  $^{125}\text{I}$  ml sample that was incubated for 6 hours is forced up the teat canal. Suckling is then permitted. One to 4 hours after suckling, blood samples are taken and the  $^{125}\text{I}$  to  $^{131}\text{I}$  ratio measured. Using the formula below the six hour milk consumption can be calculated:

$$\text{6-hour milk yield (l)} = \frac{\text{Total } ^{125}\text{I administered}}{^{131}\text{I in 1 litre milk}} \frac{^{131}\text{I in calf blood}}{^{125}\text{I in calf blood}}$$

This technique is more demanding than the WNW technique in equipment and facilities, but does not require separating the calves for periods longer than 6 hours to allow sufficient milk build-up, and avoids weighing the calves which involves several inaccuracies. Confidence in using this technique to measure calf milk intake was shown by Nicol and Irvine (1973) where the calculated milk intake from the  $^{131}\text{I}/^{125}\text{I}$  ratios in Friesian calves agreed with the actual intake of a known volume of  $^{131}\text{I}$ -labelled milk and known dose of  $^{125}\text{I}$ . The consistency of the plasma isotope ratio in calves 30 to 320 minutes after suckling, and the fact that the calves consumed most of the  $^{125}\text{I}$  dose that was injected into the teat, further reinforces the validity of this method. Using radioactive isotopes to measure milk production is not recommended because of safety problems and regulations regarding their use.

## 2.7.4 Water Dilution Technique

This technique was developed in an attempt to measure calf milk consumption over longer periods of time in young ruminants (Macfarlane et al., 1969; Yates et al., 1971; Wright et al., 1974) and is a reasonably accurate method of predicting milk intake. It is based on the principle that lambs or calves obtain water entirely from milk in the first weeks of life and by measuring the rate at which an injection of tritiated water (TOH) is diluted over periods of days or weeks, an assessment of milk intake can be made. Milk is more than 80 percent water and the oxidation of hydrogen in milksolids yields a volume of water near to that of the solids themselves, so that measurement of water turnover in the young provides a close estimate of milk intake (Macfarlane et al., 1969). The use of tritiated water in the young allows estimation of the body solids content as well as an integrated measure of milk intake, while mother and off-spring live undisturbed (apart from capture at 7-10 day intervals) in their normal environment.

This method involves animals being brought in from pasture, separated, weighed and given two hours to equilibrate milk or feed in their stomach. Animals are injected intramuscularly with 10  $\mu\text{Ci/kg}$  of TOH made up in 150 mM sodium chloride. As calves get older the amount of TOH water injected is increased to about 90  $\mu\text{Ci/kg}$  (Yates et al., 1971). Equilibration of the labelled water requires less than 2 hours in young lambs and calves, 6 hours in adult sheep and 7 to 8 hours in adult cattle, because of the slow mixing of rumen water with the other body fluids. At the end of the equilibration period a blood sample is taken and then the cows and calves are reunited and allowed to graze freely. After a period of 7-15 days the animals are reweighed and another blood sample taken. A second dose is injected to determine the change in body water volume and content of solids during growth. Serum samples are processed either by sublimation or ethanol precipitation of proteins and counted in a liquid scintillation counter. Ethanol precipitation gave similar results to sublimation and enabled samples to be processed more quickly (Wright et al., 1974).

The validity of this technique was shown by Wright et al. (1974) where water turnover rates calculated from the dilution rate of tritiated water in calves and lambs up to 4 weeks of age were only slightly in excess of known intakes. However, poor drinkers and calves older than 4 weeks of age had calculated intakes much greater than known intakes. This is probably because of the higher intake of pasture. In calves up to four weeks of age the intake of water from pasture had apparently little effect on water turnover measurements. The amount of milk that a young lamb or calf can consume may influence the accuracy of the prediction of milk intake and/or milk production from water turnover rates. Comparison of ewe milk production using oxytocin with estimated milk intake from water turnover rates in lambs illustrates the importance of lamb and calf consumption capacity at an early age. Estimated milk intake was considerably less than the ewes milk production in the first week of age; however, by week 2 the calculated intake was very close to the measured milk production (Wright et al., 1974).

Each method has its advantages and disadvantages, but the WNW technique is one that is adopted by most researchers investigating milk production in beef heifers and cows. However, the separation interval is debated often. More recent studies (Huw and Morgan, 1991; Noricumbo-Saenz, 1995) separated the cow and calf pair for c. 16 hours in order to determine the level of milk production, despite some studies (Williams et al., 1979a, 1979b) illustrating the advantages of a shorter separation interval of about 8 hours.

## **2.8 TECHNIQUES FOR DETERMINING UDDER SIZE**

Milk production is a function of the number of secretory cells in the udder and their productivity (Davis et al., 1983, 1985). The productivity of mammary tissue is relatively constant both across and within species at a rate of 1.9 ml/g tissue/day (Linzell, 1960). Indices of secretory cell numbers, such as post-milking udder weight or volume, vary proportionally with milk production (Davis et al., 1983, 1985; Davis and Hughson, 1988). Assessment of secretory cell numbers by

determining post-milking udder volume has been carried out during pregnancy and lactation in goats (Linzell, 1966) and sheep (Davis et al., 1980). A decline in milk production in goats was associated with a decline in cell numbers, a decrease in cell productivity and a decrease in udder size (Davis et al., 1985). Relationships between udder size and milk production in cattle are similar, with high genetic merit Jersey cows having a greater peak milk production and post-milking udder volume compared to unimproved Jersey cows (Davis et al., 1983).

Measurement of udder size usually requires surgical or post-slaughter removal of the udder for the appropriate determinations (DNA content, gross weight, etc.) to be made. However, the morphology of the udder in ruminants allows udder size to be determined as a volume by various techniques. These include the water displacement technique, the use of plaster casts, and more recently measurements of various dimensions on sheep and cattle udders which can be used to calculate udder volume.

### **2.8.1 Water Displacement Technique**

The water displacement technique is one method that has the advantage of being able to take many measurements on the same animal. This technique has been applied to lactation studies in goats (Linzell, 1966) and Scottish women (Hyttén, 1954) and is found to be closely related to udder and mammary gland volume in goats and women, respectively. The udder is completely submerged in a container full of water and the residual water subtracted from the volume of the container to estimate udder volume. When only the volume of tissue is required, the animal can be milked, and milked again after an injection of oxytocin (0.1-0.4 i.u. intravenously) to completely remove all milk before the measurement is made. When using this method it is important to make sure that the tissue is fully submerged (Linzell, 1966). Larger and pendulous udders, especially those found in multiparous animals, make it easier to measure the displacement of water.

### 2.8.2 Plaster Casts

Plaster casts of the mammary gland have also been used to measure udder size. An impression of the glands is made with calcium alginate, and a plaster of Paris cast made from the mould. This technique is basically the same as that used in dentistry. The appearance of the cast once set is compared to that of the tissue to check the accuracy of reproduction of the base of the glands, which is able to be corrected if necessary. The volume of the wet cast is measured by displacement of water (Linzell, 1966).

Udder volume from both these techniques is closely related to udder weight at slaughter. The estimated mean weight of the udder using the water displacement method was 99% of the true weight, but ranged from 15% too low to 31% too high and had a coefficient of variation of 11%. This compares with the plaster technique where weight estimates were 4% too low to 15% too high and the mean estimated weight was 102% of the true weight, with a coefficient of variation of 2% (Linzell, 1966). Comparing the water displacement and plaster cast volumes in the live animal with these estimates of volume after death, high correlation coefficients of 0.9952 for water and 0.9858 for the plaster cast were found (Linzell, 1966).

### 2.8.3 Udder Volume Dimensions

A more recent method of calculating udder volume was developed by Davis et al. (1983) in which post milking udder height, width and length is measured. Udder height is the distance from the rear attachment to the base of the rear teat; width is the average width measured approximately 5-8 cm above the front and rear teats; and length is defined as the distance from the base of the rear teat to the anterior junction where the udder joins with abdomen. Half the product of these measurements estimates udder volume. The accuracy of this method in determining udder volume is dependent on how closely the udder approximates a wedge shape. Thus cows with pendulous or asymmetric udders are not suitable

(Davis et al., 1983). The change in udder volume following removal of a known volume of milk from 10 Jersey cows was similar to the mean milk yield at both morning and afternoon milkings, and post milking udder volume did not differ significantly with time of milking (Davis et al., 1983). Udder volume is a measure of the total amount of mammary tissue in the udder, and as such does not distinguish between secretory and non-secretory tissue (Eichler and McFadden, 1996).

This technique has been used by other researchers (Davis et al., 1985; Davis and Hughson, 1988) to measure udder capacity through monitoring the rate of milk accumulation in the udder (Davis and Hughson, 1988), and to study the pattern of udder development in Jersey cows of known high and low breeding index through late pregnancy, early lactation and at peak lactation (Davis et al., 1983, 1985). Stage of lactation has little effect on udder volume (Davis and Bryant, 1985; Eichler and McFadden, 1996), however nutritional and environmental conditions can decrease udder volume (~ 10%) and milk production (~ 15%) (Eichler and McFadden, 1996). Udder capacity calculated from bulk and residual milk yields is an index of mammary secretory cell number (Carruthers et al., 1993) and is affected by stage of lactation, as is milk yield. Udder volume is not a good measure of functional mammary gland size owing to the lack of changes in udder volume, despite the differences in milk yield and udder capacity during lactation (Eichler and McFadden, 1996).

Udder volumes of heifers and cows of poor genetic merit (low breeding index - LBI) are less than those of mature and cows of high genetic merit (high breeding index - HBI), mainly because of liveweight differences and milk yield (Davis et al., 1983). By inference differences in udder volume are a reflection of differences in secretory cell number. Milk yield (MY) and udder volume (UV) have been found to be linearly related (Davis et al., 1983) and highly correlated (Davis et al., 1985) by the following regression equation:

$$MY = 0.93 UV + 7.33; r = 0.66; P < 0.001; n = 55.$$

Another technique for measuring udders in ewes is described by Mellor and Murray (1985) in which three distances (A, B, C) were measured by following the contours of the udder with a piece of thread. A is the mean of three measurements from the posterior margin to the anterior margin of the udder along the midline and parallel to the midline immediately medial to each teat; B is the distance between the left and right lateral edges of the udder immediately anterior to the teats; and C is the circumference of the udder.

The dimensions of the udder (cm) (A, B and C) are linearly related to the full weight (g) by the following regression equations:

$$W = 127 (\pm 7) A - 1313 (\pm 171) \quad r = 0.99$$

$$W = 124 (\pm 24) B - 2662 (\pm 856) \quad r = 0.88$$

$$W = 103 (\pm 30) C - 5441 (\pm 2085) \quad r = 0.78$$

(The bracketed figures are the standard deviations of the coefficients).

This method of determining udder size is dependent on the animal being measured, time constraints, facilities available and what the measurement is going to predict.

The method of Davis et al. (1983) would provide the best predictor of udder development and secretory cell numbers in the mammary gland because of its ease of use and practicality on live heifers. This method and the WNW technique will be used in order to determine whether Compudose has detrimental consequences for the lactational performance of heifers.

## **2.9 PURPOSE OF THE STUDY**

The purpose of this study was to gain more information on the effects of Compudose in breeding heifers under New Zealand pastoral conditions. Collecting information on liveweight gain, calf performance and lactational performance was the main focus of the study. If Compudose could improve heifer growth rates without reducing lactational or reproductive performance, beef and dairy farmers could find Compudose to be an important strategy of improving output and profitability.

## Chapter 3

# **THE EFFECT OF COMPUDOSE IN BEEF HEIFERS**

### 3.1 INTRODUCTION

Compudose<sup>®</sup>, a growth promotant containing the naturally occurring substance oestradiol 17 $\beta$ , has been shown to improve liveweight gain and feed conversion efficiency in steers. Research under pasture- and feedlot-based environments has found improved liveweight gain and feed conversion efficiency responses in steers of 5-30% and 7-16%, respectively (Mathison and Stobbs, 1983; Mason et al., 1986; Bass et al., 1989; Arando-Osorio et al., 1996; Burnham et al., 1997). Very little research has been conducted on the effects of Compudose in heifers. However, trials overseas under feedlot conditions recorded 7-10% increases in growth rates and 4% improvements in feed efficiency in heifers treated with Compudose (Johnson et al., 1987; Stobbs et al., 1988).

In the past, regulations have prevented the use of Compudose in breeding heifers owing to the negative consequences on reproductive performance reported from using other oestrogenic compounds (Anthony et al., 1981; Staigmiller et al., 1983; Deutscher et al., 1986; Moran et al., 1990; Hancock et al., 1994). Oestrogen has a stimulating effect on mammary gland development (Sejrsen, 1984) with increased teat and udder development being reported from treatment with Compudose and other oestrogenic compounds (Deutscher et al., 1986; Johnson et al., 1987; Moran et al., 1990). However, little research has been conducted about the effects of Compudose on heifer growth, reproduction and lactational performance, and offspring performance under New Zealand pastoral conditions.

Use of Compudose may be advantageous in dairy and beef production systems by allowing target growth rates to be attained in heifers at critical times of the year. However, the reported negative effects of oestrogen on reproduction and lactational performance may prevent the use of this growth promotant in breeding cow systems. Given that Compudose improves liveweight gains and feed efficiency, and research carried out by Sejrsen et al. (1982, 1983) and Little and Kay (1979), showed that heifers grown at rates of more than 0.8 kg/day around the time of puberty had abnormal mammary gland development and milk production, Compudose could potentially have negative consequences on lactational performance.

Therefore, the objective of this study was to investigate the effects of Compudose treatment on heifer growth, skeletal development, lactational performance, offspring performance and carcass quality.

### **3.2 METHODS**

Nineteen Hereford x Friesian (H x F) heifers were implanted with Compudose<sup>®</sup> 400 (46 mg of oestradiol 17 $\beta$  impregnated in silicone rubber with an active life of 400 days: Elanco Products, New Zealand) at c. 3 months of age (90 days of age in November, 1994) and another 19 were implanted at c. 7 months of age (210 days of age in April, 1995). A control group of 19 heifers were not implanted. All heifers were mated at 15 months of age to a Charolais sire over two-and-a-half cycles. A pregnancy rate of 86%, occurred with 49 heifers calving out of the 57 that were mated. A further heifer was removed from the trial owing to unusually poor liveweight gain. Fourteen of the heifers that calved were from the group implanted at 90 days of age (Compudose 90), 17 were from the group implanted at 210 days of age (Compudose 210) and 17 were from the non-implanted control group (Control).

Heifers grazed on pastures of predominantly perennial ryegrass (*Lolium perenne*) and white clover (*Trifolium repens*) at Massey University's Animal Research Unit. For five weeks from July, heifers consumed a Chou moellier (*Brassica napus*) (c. 4-5 kgDM/cow/day) and hay (2-3 kgDM/cow/day) diet, after which all heifers were break-fed pasture and fed hay (3-7 kgDM/cow/day) until calving. Calved heifers grazed ryegrass/clover pastures with a sward height of c. 21 cm on a continuous stocking system designed to permit *ad libitum* levels of intake. This was assumed to be 13 kg DM/head/day for growing lactating beef heifers with an average liveweight of 400 kg (Geenty and Rattray, 1987).

### 3.2.1 Liveweight measurements

Unfasted liveweights were taken monthly from the start of the experiment (28 November 1994) until pre-slaughter (28 January 1997). In July 1996 (c. 8 weeks prior to the planned start of calving) girth, wither height, hip height, hip width, pelvic height and pelvic width were measured to obtain information about skeletal development. External pelvic size was calculated from the hip height taken from the ground (H), and hip width between the left and right *tuber coxae* (W) measured at c. 22 months of age (July 1996). The product of  $(0.18H \times 0.36W)$  predicted pelvic area based on the method of Beriao et al. (1986). Internal measurements of the birth canal were measured by a veterinarian using a Rice Pelvimeter as described by Simons (1986) and Price and Wiltbank (1978). The product of the pelvic width (the distance between the two *shafts of ilea*) and pelvic height (the distance between the *symphysis pubis* and *sacral vertebrae*) predicted internal pelvic area.

Calving commenced on the 25 August 1996 and continued until 15 October 1996 (51 days) with a mean calving date of 20 September 1996. All calves were tagged, weighed, and sex recorded at birth, whether they were alive or dead. No calves died at birth. Occurrence of calving difficulties was noted following morning and evening inspections using a scoring system that ranked calving difficulty from 1 to 3 (1 = no assistance; 2 = assistance, but easy calving; 3 = substantial assistance

(veterinarian required)). Liveweight loss over calving was assumed to be the pre-calving liveweight of the heifer minus the weight of the calf at birth and embryonic fluids (calf birth weight + 15 litres of amniotic and allantoic fluids + weight of placenta, calculated as 14% of calf birth weight, Roberts (1986)). Calves suckled their dams for an average of 12 weeks (c.  $85 \pm 11$  days) post parturition when weaning occurred. The average weaning date was the 14 December 1996. All heifers were slaughtered on 29 January 1997.

### **3.2.2 Milk production and udder dimensions**

An estimate of heifer milk production was obtained using the weigh-nurse-weigh technique described by Barton (1970) and Huw and Morgan (1991). Of the 48 heifer's that calved, 42 were used to determine milk production in the three treatment groups (Control, Compudose 90 and Compudose 210). Heifer and calf pairs were divided into four groups of 9 and one group of 6 with equal numbers of heifers of each treatment in each group. Groups were selected according to calving date. Groups 1 to 5 had mean ( $\pm$  range) calving dates of 3 September (9 days), 11 September (6 days), 16 September (6.5 days), 26 September (11 days) and 1 October (11 days), respectively.

At 4 weeks (c. 28 days) and 8 weeks (c. 56 days) after calving the heifer and calf pairs were brought into the yards at 1600 h and separated. At separation all heifer and calf pairs were weighed. Calves and dams were reunited the following day at c. 0900 h and calves were allowed to suckle their mothers until they lost interest or became satiated. The calves were weighed before and after suckling their dams. Any urine or faecal deposits were collected and weighed. The difference in liveweight (adjusted for excretion of wastes) provided an estimate of milk yield. Ideally this was when the calf had suckled all milk that had accumulated overnight (c. 18 hours). The time interval from separation to reuniting, and suckling time, were recorded for each heifer-calf pair. The nursing times of heifers and calf pairs in the Control, Compudose 90 and Compudose 210 treatment groups at 4 weeks of

lactation were 9.6, 10.2 and 9.8 minutes, respectively, and at 8 weeks of lactation were 11.1, 11.8 and 10.7 minutes, respectively. Measurements were repeated the following day to ensure the method was repeatable.

Udder size was determined at 4 weeks, 8 weeks, 12 weeks (weaning) of lactation and at slaughter using the udder dimension method described by Davis et al. (1983) and Davis and Hughson (1988). Udder height, length, average width, and distance between the front and back teats were obtained on two consecutive days by two operators pre- and post-calf suckling at 4 weeks and 8 weeks of lactation. Udder dimensions were measured on a full udder at weaning as calves did not nurse their heifer dams, and prior to slaughter. Udder volume was calculated as half the product of H, L and W of the udder. The dimensions are defined as follows:

udder height (H) - posterior rear surface of rear mammary glands from rear attachment to base of rear teat;

udder length (L)- base of rear teat to anterior junction where the udder joins the abdomen;

average udder width (W) - measured approximately 5 cm above the front and rear teats.

### **3.2.3 Slaughter procedure and carcass measurements**

Heifers were weighed off pasture at c. 1100 h and transported about 20 km to a meat processing plant (Manawatu Beef Packers, Feilding). Heifers were slaughtered and dressed under normal commercial conditions 24 hours after removal from pasture. The number of erupted permanent incisor teeth and the combined weight of the kidney and pelvic fat from each carcass side were recorded. Carcass length for each carcass side was measured from the distal end of the tarsal bones to the midpoint of the cranial edge of the first rib. Meat plant personnel subjectively evaluated the carcass sides according to the muscling classes 1, 2 and 3 using + and - values to give a nine-point scale with +1 being the highest muscling

and -3 being the lowest. For analysis purposes the scores were converted to values on a 1 to 9 scale (1 = -3 and 9 = +1). Udders were removed from the heifers at slaughter and weighed.

Following overnight chilling at 1-3°C, the left carcass sides were quartered between the 12th and 13th ribs and the cross-sectional area of the rib-eye (LD muscle) were traced. The areas of the tracings were assessed using a Placom digital planimeter. On the quartered carcass at a point over the LD muscle three-fourths the distance from its medial to its lateral edges, the subcutaneous fat thickness was measured. At boning the weights of the three major hind-quarter cuts from each carcass side were weighed. These cuts included the knuckle, inside round and outside round.

Subjective evaluation of fat colour using standards ranging from 0 (pure white) through to 7 (creamy yellow), and electronic meat pH was carried out by meat plant personnel at the meat processing plant.

### **3.2.4 Statistical Analysis**

Data were analysed using the general least-squares procedures within the general linear model procedure of SAS (1985). Heifer liveweights, and heifer and calf liveweight gains were analysed to determine the main effect of Compudose treatment on these variables. Repeated measures analysis was used for heifer liveweight traits in recognition of the fact that sequential liveweights represent repeated measurements of each trait on the same animals (Gill and Hafs, 1971). Carcass composition characteristics were adjusted to a constant carcass weight, and girth, wither height, hip height, hip width and pelvic size were adjusted to a constant liveweight, by covariate analysis. Calf liveweights at birth, 4 weeks, 8 weeks and 12 weeks (weaning); milk yield at 4 and 8 weeks; and udder volume at 4 weeks, 8 weeks, 12 weeks (weaning) were adjusted to a constant date of birth using the previously mentioned statistical procedure. Pre-slaughter udder volume was adjusted to a constant period from weaning to slaughter by covariate analysis. Analysis of milk yield and udder volume considered the measurements taken on

consecutive days and the measurements taken on the udder by two operators. The effect of calf sex on calf liveweight or milk yield was not significant, and was therefore removed from the model. Age of calf did not have a significant effect on milk yield at 4 and 8 weeks of lactation, or on udder volume at 4 weeks, 8 weeks, 12 weeks (weaning) of lactation and pre-slaughter, and therefore was not included in the model to determine the effects of Compudose treatment on these variables.

### 3.3 RESULTS

#### 3.3.1 Heifer Liveweights

Liveweight changes from the start of the experiment (28 November, 1994) to final liveweight (29 January, 1997) of the 48 heifers which completed the trial are illustrated in Figure 3.1. Table 3.1 summarises the key liveweight gains for the heifers implanted at 90 days (Compudose 90) and 210 days of age (Compudose 210), and the Control group.

Compudose 90 heifers had consistently greater liveweights ( $P < 0.10$ ) throughout the trial than Compudose 210 heifers and the Control group owing to slightly greater liveweight gains. The Compudose 210 heifers did not differ significantly from the Control group in liveweight performance throughout the trial, although the trend was for this group to be lighter than the Control and Compudose 90 group of heifers.

The 400 day Compudose implant allowed the Compudose 90 heifers to achieve growth rates of 0.63 kg/day for a period of 385 days from the time of implantation (28 November, 1994). This was a 6.8% growth rate advantage over the Control group ( $P < 0.05$ ) resulting in the Compudose 90 group having a liveweight advantage of 5.5% over the Control group after 385 days of implantation ( $P < 0.10$ ). Heifers implanted with Compudose at 210 days of age (6 April, 1995) and Control heifers did not differ in liveweight gain over the 383 day period of implantation ( $0.59 \pm 0.01$  kg/day *vs*  $0.58 \pm 0.01$  kg/day), or liveweight at day 383 ( $322.2 \pm 6.4$  kg *vs*  $320.9 \pm 6.4$  kg).

At pre-mating the heifers implanted at 90 days of age were 6.1% heavier ( $P < 0.05$ ) than the control group ( $308.0 \pm 6.6$  kg *vs*  $290.3 \pm 6.0$  kg) and 5.8% heavier ( $P < 0.10$ ) than the heifers implanted at 210 days of age ( $308.0 \pm 6.6$  kg *vs*  $291.1 \pm 6.0$  kg), while the heifers implanted at 210 days of age did not differ significantly from the Control group of heifers ( $291.1 \pm 6.0$  kg *vs*  $290.3 \pm 6.1$  kg). From the time of

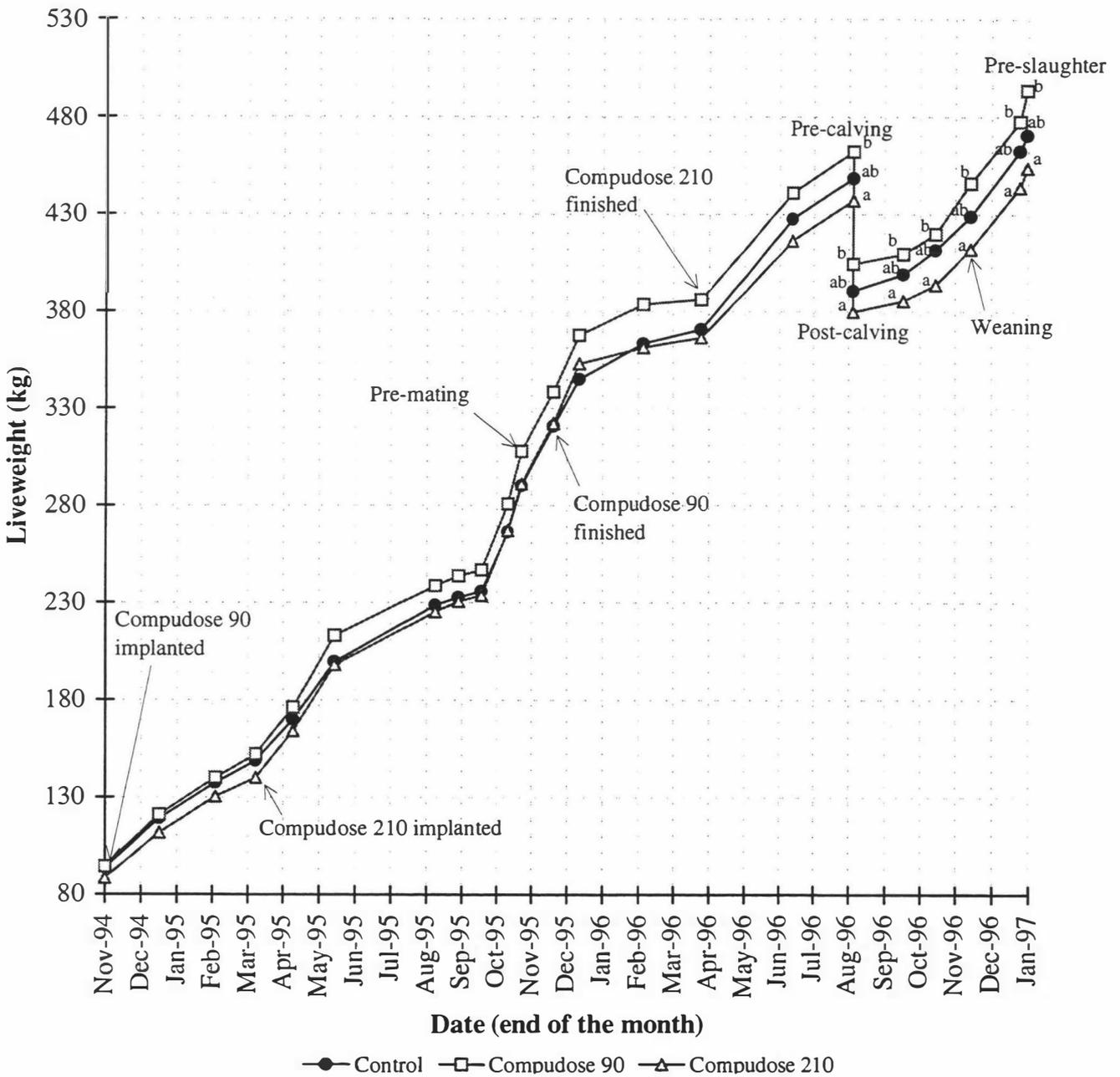
implantation until pre-mating the Compudose 90 and Compudose 210 groups grew 0.05 kg/day and 0.04 kg/day faster than the Control group ( $0.60 \pm 0.01$  kg/day vs  $0.55 \pm 0.01$  kg/day,  $P < 0.01$  and  $0.66 \pm 0.01$  kg/day vs  $0.62 \pm 0.02$  kg/day,  $P < 0.05$ , respectively).

The pre-calving liveweights of Compudose 90 heifers were 3.1% greater than the Control heifers ( $462.2 \pm 8.9$  kg vs  $448.5 \pm 8.0$  kg,  $P > 0.10$ ) and 5.8% greater than the Compudose 210 heifers ( $462.2 \pm 8.9$  kg vs  $436.9 \pm 8.0$  kg,  $P < 0.05$ ). Post-calving liveweight differences between the three treatment groups followed a similar pattern with Compudose 90 heifers being 24.8 kg ( $P < 0.05$ ) and 14.1 kg ( $P > 0.10$ ) heavier than Compudose 210 and Control heifers.

The liveweight advantage of Compudose 90 heifers over non-treated heifers continued through to pre-slaughter, however the 23 kg advantage was not significant ( $P > 0.05$ ). At pre-slaughter, Compudose 210 heifers were 39.9 kg lighter than heifers treated with Compudose 90 heifers ( $P < 0.01$ ), and 16.8 kg lighter than Control heifers ( $P > 0.10$ ). Growth rates from implantation to pre-slaughter in the Compudose 210 group were consistently lower than in the Compudose 90 group, while the Compudose 90 group maintained a non-significant 6.3% growth rate advantage over non-treated heifers from implantation to final liveweight.

**Figure 3.1. Liveweight profile of Hereford x Friesian heifers not implanted with Compudose (Control) and heifers implanted with Compudose at 90 days (Compudose 90) and 210 days of age (Compudose 210).**

(<sup>a,b</sup> = means with different superscripts are significantly different, P < 0.05).



**Table 3.1. Least-square means  $\pm$  standard errors showing the effect of no Compudose implants (Control), implants at 90 days of age (Compudose 90) and implants at 210 days of age (Compudose 210) on liveweight gains from implantation to pre-slaughter.**

	Treatment		
	Control	Compudose 90	Compudose 210
Number of animals <sup>1</sup>	17	14	17
<b>Liveweight gains (kg/day)</b>			
Compudose 90 heifers implanted to when Compudose 210 heifers implanted	0.43 $\pm$ 0.02	0.45 $\pm$ 0.02	0.40 $\pm$ 0.02
Compudose 90 implanted to when implant finished	0.59 $\pm$ 0.01 <sup>a</sup>	0.63 $\pm$ 0.01 <sup>b</sup>	0.61 $\pm$ 0.01 <sup>ab</sup>
Compudose 210 implanted to when implant finished	0.58 $\pm$ 0.01	0.61 $\pm$ 0.01	0.59 $\pm$ 0.01
Compudose 90 implanted to pre-mating	0.55 $\pm$ 0.01 <sup>a</sup>	0.60 $\pm$ 0.01 <sup>b</sup>	0.57 $\pm$ 0.01 <sup>ab</sup>
Compudose 210 implanted to pre-mating	0.62 $\pm$ 0.01 <sup>a</sup>	0.68 $\pm$ 0.02 <sup>b</sup>	0.66 $\pm$ 0.01 <sup>b</sup>
Pre-mating to pre-calving	0.55 $\pm$ 0.02	0.54 $\pm$ 0.02	0.51 $\pm$ 0.02
Compudose 90 implanted to pre-calving	0.55 $\pm$ 0.01	0.57 $\pm$ 0.01	0.54 $\pm$ 0.01
Compudose 210 implanted to pre-calving	0.58 $\pm$ 0.01	0.60 $\pm$ 0.01	0.58 $\pm$ 0.01
Post-calving to weaning	0.47 $\pm$ 0.05	0.53 $\pm$ 0.05	0.41 $\pm$ 0.05
Weaning to final liveweight	0.77 $\pm$ 0.10	0.96 $\pm$ 0.10	0.84 $\pm$ 0.10
Compudose 90 implanted to final liveweight	0.48 $\pm$ 0.01 <sup>ab</sup>	0.51 $\pm$ 0.01 <sup>b</sup>	0.46 $\pm$ 0.01 <sup>a</sup>
Compudose 210 implanted to final liveweight	0.49 $\pm$ 0.01 <sup>ab</sup>	0.52 $\pm$ 0.01 <sup>b</sup>	0.47 $\pm$ 0.01 <sup>a</sup>

<sup>a, b</sup> Means within rows with different superscripts are significantly different ( $P < 0.05$ ).

<sup>1</sup> Numbers of heifers that actually calved.

### 3.3.2 Calf Birthweights

Birthweight of calves born to Compudose 90, Compudose 210 and Control heifers were not significantly different ( $P > 0.10$ ) as illustrated in Table 3.2.

Heifers not implanted with Compudose had an average calving date of 14 September and a calving spread of 51 days. Compudose 90 and Compudose 210 implanted heifers calved 8 days ( $P < 0.10$ ) and 10 days ( $P < 0.05$ ) later than the Control group, and had smaller calving spreads of 34 days and 39 days, respectively. One heifer in the Compudose 210 group was given a dystocia rating of 2 owing to a breech delivery, while the other heifers experienced no calving difficulties.

Calves born to heifers not implanted with Compudose had significantly greater liveweights at 4 weeks, 8 weeks and 12 weeks post-calving than calves born to heifers implanted at 90 days and 210 days of age after adjusting for date of birth (Figure 3.2).

Despite the obvious advantage in liveweight performance of calves born to heifers not implanted with Compudose compared to Compudose 90 and Compudose 210 heifers, differences in growth rates were not significant. Calves born to control heifers had a 4.5% growth rate advantage from birth to weaning ( $P > 0.10$ ).

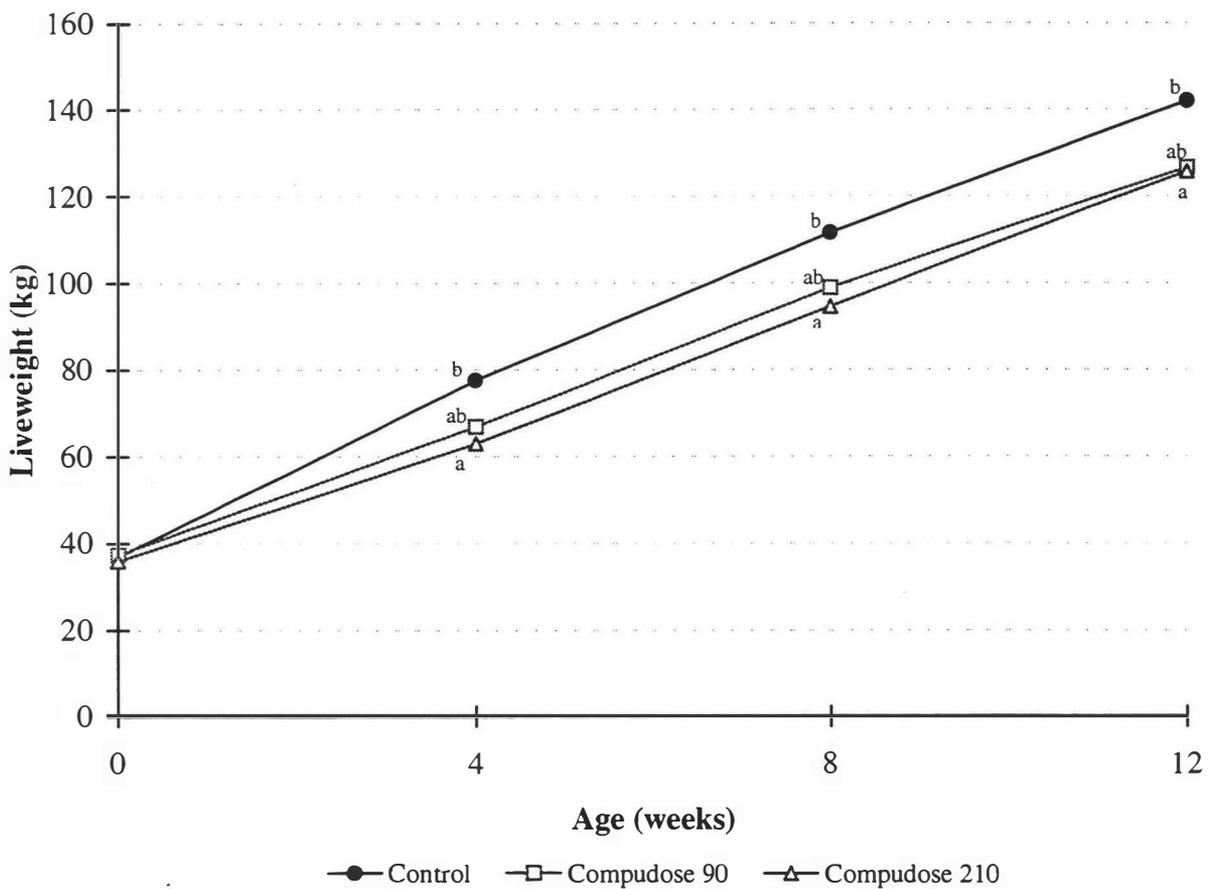
**Table 3.2. Least-square means  $\pm$  standard errors showing the effect of no Compudose implants (Control), implants at 90 days of age (Compudose 90) and implants at 210 days of age (Compudose 210) on date of birth, birthweight, weaning weight (12 weeks of age) and liveweight gain from birth to weaning on Charolais x (Hereford x Friesian) calves.**

	Treatment		
	Control	Compudose 90	Compudose 210
Date of Birth	10 September $\pm$ 3 days <sup>a</sup>	22 September $\pm$ 3 days <sup>b</sup>	24 September $\pm$ 3 days <sup>b</sup>
<b>Liveweight (kg)</b>			
Birthweight	36.9 $\pm$ 0.9	37.4 $\pm$ 1.0	35.8 $\pm$ 0.9
Weaning (12 weeks)	141.9 $\pm$ 3.5 <sup>b</sup>	126.6 $\pm$ 2.4 <sup>a</sup>	125.7 $\pm$ 2.3 <sup>a</sup>
<b>Liveweight gains (kg/day)</b>			
Birth to weaning	1.17 $\pm$ 0.03	1.12 $\pm$ 0.03	1.12 $\pm$ 0.03

<sup>a, b</sup> Means within rows with different superscripts are significantly different ( $P < 0.05$ ).

Figure 3.2. Liveweight profile of Charolais x (Hereford x Friesian) calves born to heifers not implanted with Compudose (Control) and heifers implanted with Compudose at 90 days of age (Compudose 90) and 210 of age (Compudose 210) .

(<sup>a,b</sup> = means with different superscripts are significantly different,  $P < 0.05$ ).



### 3.3.3 Skeletal Development

No significant differences between the Control group of heifers and those implanted with Compudose 90 and Compudose 210 were observed in wither height, girth circumference, hip height and hip width (Table 3.3). Pelvic area measured internally did not differ significantly between treatment groups, however internal pelvic width for the Control group was significantly smaller than for heifers implanted at 90 days and 210 days of age ( $P < 0.01$  in both cases). Subsequently internal pelvic area of the Control group tended to be smaller ( $P > 0.10$ ) than internal pelvic area of Compudose-treated heifers, despite no differences in pelvic height occurring. Calculated external pelvic areas for the three treatment groups at c. 210 days of gestation were larger than those reported by Khadem (1994) in H x F and Simmental x Friesian heifers at 270 days of gestation, and were not highly correlated with internal pelvic area ( $r = 0.24$ ). Therefore, external pelvic area measurements will not be discussed further.

**Table 3.3. Least-square means  $\pm$  standard errors showing the effects of no Compudose implants (Control), implants at 90 days of age (Compudose 90) and implants at 210 days of age (Compudose 210) on liveweight and physical dimensions of Hereford x Friesian heifers at c. 210 days of gestation.**

	Treatment		
	Control	Compudose 90	Compudose 210
Liveweight (kg)	424.7 $\pm$ 7.8 <sup>ab</sup>	437.1 $\pm$ 8.5 <sup>b</sup>	416.3 $\pm$ 8.0 <sup>a</sup>
Wither height (cm)	147.5 $\pm$ 0.7	145.9 $\pm$ 0.8	146.1 $\pm$ 0.7
Girth (cm)	181.1 $\pm$ 1.0	181.2 $\pm$ 1.1	180.9 $\pm$ 1.0
Hip height (H) (cm)	153.1 $\pm$ 0.9	151.4 $\pm$ 0.9	151.3 $\pm$ 0.8
Hip width (W) (cm)	49.7 $\pm$ 0.4	49.4 $\pm$ 0.5	49.1 $\pm$ 0.4
Pelvic height (cm)	13.7 $\pm$ 0.2	13.5 $\pm$ 0.2	13.6 $\pm$ 0.2
Pelvic width (cm)	12.2 $\pm$ 0.2 <sup>a</sup>	12.9 $\pm$ 0.2 <sup>b</sup>	13.0 $\pm$ 0.2 <sup>b</sup>
Internal pelvic area (cm <sup>2</sup> ) <sup>1</sup>	167.3 $\pm$ 4.0	175.42 $\pm$ 4.5	176.4 $\pm$ 4.2
External pelvic area (cm <sup>2</sup> ) <sup>2</sup>	483.4 $\pm$ 5.3	485.2 $\pm$ 6.0	481.1 $\pm$ 5.6

<sup>a, b</sup> Means within rows with different superscripts are significantly different (P < 0.05).

<sup>1</sup> Internal pelvic area calculated as pelvic height x pelvic width.

<sup>2</sup> External pelvic area calculated as (0.18 x H) x (0.36 x W).

### 3.3.4 Milk production and udder volume

No significant differences in milk yield or udder volume at 4 weeks and 8 weeks of lactation were detected between Compudose 90 implanted heifers, Compudose 210 implanted heifers and Control heifers (Table 3.4). On average heifer and calf pairs were separated for 1112 mins (18 h and 32 mins) ranging from 1080 mins to 1140 mins (18 to 19 h). At 8 weeks of lactation the separation interval for the Control group of heifers was significantly less than the separation interval for the heifers and calf pairs in the Compudose 210 implanted group (1084 mins vs 1111 mins).

Milk yield and udder volume increased as lactation progressed within a treatment group, however udder productivity declined with advancing lactation. Heifers implanted at 210 days of age had a significantly lower volume of milk yield per unit volume of udder volume at c. 8 weeks of lactation than heifers implanted at 90 days of age and the Control group ( $P < 0.05$  and  $P < 0.10$ , respectively).

Table 3.5 illustrates the significant correlation and linear relationship between milk yield and udder volume difference following calf nursing for all operators and between operators.

Milk yield measurements taken by the WNW method on consecutive days, and udder dimension measurements taken by two operators and on consecutive days were repeatable with positive correlations of 0.54, 0.53 and 0.50, respectively.

Udder volume at weaning and pre-slaughter did not differ significantly between treatment groups ( $P < 0.05$ , Table 3.6). Compudose-treated heifers and non-treated heifers did not differ in udder weight. Udder volume (UV) and udder weight (UW) had a regression equation and correlation of:

$$UV = 0.67 UW - 0.21 \quad (r = 0.86; P < 0.001; n = 42)$$

**Table 3.4. Least-square means  $\pm$  standard errors showing the effects of no Compudose implants (Control), implants at 90 days of age (Compudose 90) and implants at 210 days of age (Compudose 210) on Hereford x Friesian heifer liveweight, milk yield and udder volume at 4 weeks and 8 weeks of lactation.**

	Treatment		
	Control	Compudose 90	Compudose 210
Number of animals	14	14	14
<b>4 weeks of lactation</b>			
Days post-calving	34 $\pm$ 1 <sup>b</sup>	23 $\pm$ 1 <sup>a</sup>	25 $\pm$ 1 <sup>a</sup>
Cow liveweight (kg)	399.1 $\pm$ 9.5	409.6 $\pm$ 9.5	385.4 $\pm$ 9.5
Milk yield (l/cow/day)	7.53 $\pm$ 0.25	7.41 $\pm$ 0.24	7.49 $\pm$ 0.23
Udder volume difference (l)	3.66 $\pm$ 0.17	3.58 $\pm$ 0.16	3.59 $\pm$ 0.16
Milk yield (ml/kg <sup>0.75</sup> )	85.4 $\pm$ 2.8	81.1 $\pm$ 2.7	86.2 $\pm$ 2.6
Udder volume (ml/kg <sup>0.75</sup> )	41.8 $\pm$ 2.0	39.0 $\pm$ 1.9	41.5 $\pm$ 1.8
Milk yield/udder volume (l/l)	2.21 $\pm$ 0.17	1.97 $\pm$ 0.16	2.33 $\pm$ 0.16
<b>8 weeks of lactation</b>			
Days post calving	62 $\pm$ 1 <sup>b</sup>	51 $\pm$ 1 <sup>a</sup>	53 $\pm$ 1 <sup>a</sup>
Cow liveweight (kg)	411.6 $\pm$ 9.5	419.9 $\pm$ 9.5	393.6 $\pm$ 9.5
Milk yield (l/cow/day)	8.96 $\pm$ 0.36	9.07 $\pm$ 0.35	8.92 $\pm$ 0.34
Udder volume difference (l)	4.33 $\pm$ 0.17	4.30 $\pm$ 0.17	4.60 $\pm$ 0.16
Milk yield (ml/kg <sup>0.75</sup> )	99.6 $\pm$ 4.0	97.2 $\pm$ 3.9	101.0 $\pm$ 3.8
Udder volume (ml/kg <sup>0.75</sup> )	48.1 $\pm$ 1.9	46.2 $\pm$ 1.9	52.1 $\pm$ 1.8
Milk yield/udder volume (l/l)	2.01 $\pm$ 0.07	2.06 $\pm$ 0.07	1.88 $\pm$ 0.07

<sup>a, b</sup> Means within rows with different superscripts are significantly different (P < 0.05).

**Table 3.5. Regressions of milk yield on udder volume across 4 and 8 weeks of lactation, and correlation between milk yield and udder volume for all operators and between operator 1 and operator 2 across 4 and 8 weeks of lactation (n = 84).**

	Slope ( $\pm$ SE)	Intercept ( $\pm$ SE) (Milk yield, l)	Correlation of milk yield and udder volume (r)	Significance
All operators	0.68 $\pm$ 0.07	3.08 $\pm$ 0.27	0.63	***
Operator 1	0.55 $\pm$ 0.06	3.66 $\pm$ 0.26	0.55	***
Operator 2	0.51 $\pm$ 0.06	3.71 $\pm$ 0.26	0.55	***

\*\*\* P < 0.001

**Table 3.6. Least-square means  $\pm$  standard errors showing the effects of no Compudose implants (Control), implants at 90 days of age (Compudose 90) and implants at 210 days of age (Compudose 210) on Hereford x Friesian heifer udder volume at weaning (12 weeks of lactation) and slaughter.**

	Treatment		
	Control	Compudose 90	Compudose 210
Number of animals	14	14	14
Weaning udder volume (l) <sup>1</sup>	9.18 $\pm$ 0.36	9.01 $\pm$ 0.34	9.91 $\pm$ 0.33
Slaughter udder volume (l)	2.85 $\pm$ 0.20	3.29 $\pm$ 0.20	3.15 $\pm$ 0.20
Udder weight (kg)	4.7 $\pm$ 0.3	5.4 $\pm$ 0.4	5.0 $\pm$ 0.6

<sup>a,b</sup> Means within rows with different subscripts are significantly different (P < 0.05).

<sup>1</sup> Full udder volume.

### 3.3.4 Carcass quality characteristics

Carcass weight differences of Compudose-treated and non-treated heifers followed a similar trend to liveweight differences with heifers treated with Compudose at 210 days of age recording a lighter carcass weight than heifers implanted at 90 days of age ( $P < 0.001$ ), and non-treated heifers ( $P > 0.10$ ). No differences in dressing-out percent (DO%) adjusted to a common carcass weight between treatment groups occurred (Table 3.7).

Differences in carcass length (carcass weight-adjusted) between treatment groups did not exist ( $P > 0.05$ ), but the Control heifers had greater average carcass lengths than carcasses of Compudose 90 heifers ( $P < 0.01$ ). Kidney and pelvic fat weights, and fat depths between the 12th and 13th rib, of the three treatment groups did not differ significantly ( $P > 0.10$ ). However, carcasses of Compudose 210 heifers had lighter kidney and pelvic fat weights and smaller fat depths than carcasses of Compudose 90 and Control heifers, despite the non-significant difference. Carcasses of Control heifers had the smallest average rib-eye area compared to Compudose 210 carcasses ( $P < 0.05$ ) and Compudose 90 carcasses ( $P > 0.10$ ).

No treatment differences in the combined weight of the three hind-quarter cuts occurred, despite the outside round muscle of non-treated heifers being significantly lighter than that of Compudose 210 heifers ( $P < 0.01$ ).

Meat pH recorded electronically at the meat processing plant was lower in Compudose 90 and Compudose 210 heifers compared to control heifers ( $P < 0.10$  in both cases). Compudose 210 heifers generally had greater fat colour scores than Control heifers and Compudose 90 heifers (Figure 3.3).

All carcasses were graded heifer due to none having more than six permanent incisor teeth erupted (Figure 3.4). Subjective assessment of muscling illustrated in Figure 3.5 showed that heifers treated with Compudose 90 were better muscled than Compudose 210 heifers. A greater proportion of the carcasses of Compudose

210 heifers were graded in the L class (1-3 mm backfat thickness) compared to Control and Compudose 90 carcasses, which were generally graded in the P class (4-7 mm backfat thickness) (Figure 3.6).

**Table 3.7. Least-square means  $\pm$  standard errors showing the effects of no Compudose implants (Control), implants at 90 days of age (Compudose 90) and implants at 210 days of age (Compudose 210) on Hereford x Friesian heifer carcass characteristics.**

	Treatment		
	Control	Compudose 90	Compudose 210
Number of animals	17	14	17
<b>Values not adjusted for carcass weight:</b>			
Liveweight <sup>1</sup> (kg)	415.4 $\pm$ 7.8 <sup>ab</sup>	436.4 $\pm$ 8.8 <sup>b</sup>	401.4 $\pm$ 8.0 <sup>a</sup>
Carcass weight (kg)	214.3 $\pm$ 4.6 <sup>ab</sup>	224.8 $\pm$ 5.2 <sup>b</sup>	205.6 $\pm$ 4.7 <sup>a</sup>
<b>Values adjusted for carcass weight:</b>			
Dressing-out %	51.6 $\pm$ 0.2	51.1 $\pm$ 0.3	51.5 $\pm$ 0.31
Carcass length (mm)	2044 $\pm$ 11	2016 $\pm$ 13	2038 $\pm$ 11
Kidney and pelvic fat (kg)	4.81 $\pm$ 0.31	4.75 $\pm$ 0.36	4.55 $\pm$ 0.32
Fat depth (mm)	4.0 $\pm$ 0.5	4.3 $\pm$ 0.6	3.8 $\pm$ 0.5
Rib-eye area (cm <sup>2</sup> )	58.7 $\pm$ 1.2 <sup>a</sup>	60.1 $\pm$ 1.5 <sup>ab</sup>	62.5 $\pm$ 1.3 <sup>b</sup>
Inside cut (kg) <sup>1</sup>	12.8 $\pm$ 0.2	13.2 $\pm$ 0.2	13.2 $\pm$ 0.2
Knuckle cut (kg) <sup>1</sup>	7.9 $\pm$ 0.1	7.9 $\pm$ 0.1	7.9 $\pm$ 0.1
Outside cut (kg) <sup>1</sup>	11.2 $\pm$ 0.1 <sup>a</sup>	11.4 $\pm$ 0.1 <sup>ab</sup>	11.5 $\pm$ 0.1 <sup>b</sup>
3-cut weight (kg) <sup>2</sup>	31.6 $\pm$ 0.3	32.4 $\pm$ 0.3	32.6 $\pm$ 0.3
pH <sup>3</sup>	6.11 $\pm$ 0.05	5.98 $\pm$ 0.05	5.98 $\pm$ 0.05

<sup>a, b</sup> Means within rows with different superscripts are significantly different ( $P < 0.05$ ).

<sup>1</sup> Weight of inside round, knuckle or outside round muscle from both carcass sides.

<sup>2</sup> The sum of the weights of the inside round, knuckle and outside round muscles.

<sup>3</sup> Electronic reading of the muscle recorded at the meat processing plant.

Figure 3.3. Fat colour scores of carcasses of heifers not treated with Compudose (Control) and, heifers implanted with Compudose at 90 days of age (Compudose 90) and 210 days of age (Compudose 210).

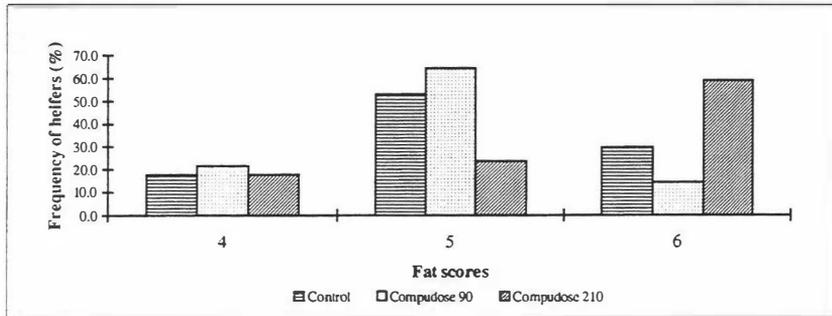


Figure 3.4. Number of teeth in carcasses of heifers not implanted with Compudose (Control) and, heifers implanted with Compudose at 90 days of age (Compudose 90) and 210 days of age (Compudose 210).

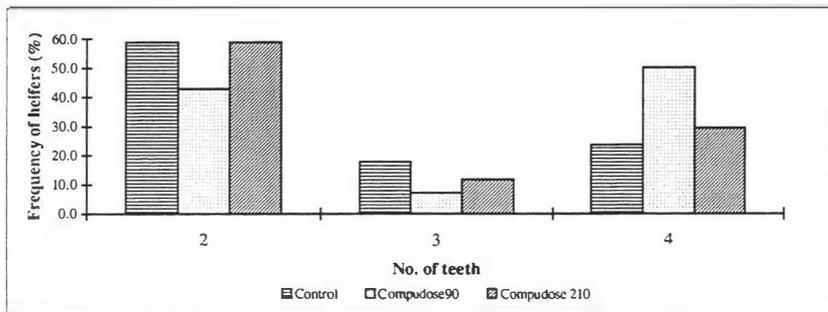


Figure 3.5. Muscling scores of carcasses of heifers not implanted with Compudose (Control) and, heifers implanted with Compudose at 90 days of age (Compudose 90) and 210 days of age (Compudose 210).

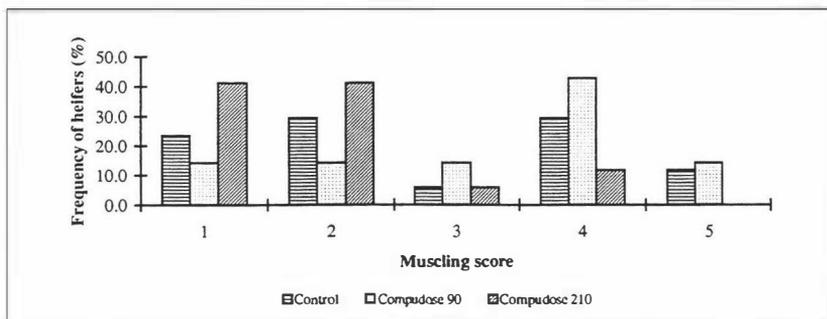
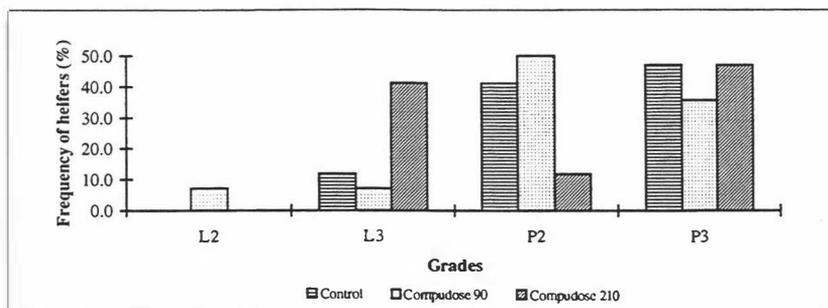


Figure 3.6. Grades assigned to carcasses of heifers not implanted with Compudose (Control), and, heifers implanted with Compudose at 90 days of age (Compudose 90) and 210 days of age (Compudose 210).



## 3.4 DISCUSSION

### 3.4.1 Heifer performance

Various oestrogens (natural and analogues) have been used in studies with heifers, but it does not necessary follow that they will all have the same effects. Therefore, care is needed when comparing results of studies in which different compounds have been used.

The liveweight gains achieved by the H x F heifer's throughout the period of this study are comparable to liveweight gains observed in other studies investigating the performance of once-bred heifers under New Zealand pastoral conditions (Khadem et al., 1993, 1995a, 1995b, 1996; Burke et al., 1997).

The 6.8% growth rate advantage that occurred in pregnant heifers implanted with Compudose 400 (46 mg of oestradiol 17 $\beta$ ) at 90 days of age for 385 days agrees with the 8.2% and 6.7% growth rate advantage that was observed in trials by Johnson et al. (1987) and Stobbs et al. (1988). However, these responses were in older and non-pregnant feedlot heifers that were treated with Compudose implants containing 24 mg of oestradiol 17 $\beta$  that lasted 168 (Stobbs et al., 1988) and 154 (Johnson et al., 1987) days, respectively. H x F heifers implanted at three 84 day intervals from 84 days of age with zeranol (36 mg), and 2 long-acting biodegradable implants of 19 mg oestradiol (2E), exhibited a 7.4% and 6.8% liveweight gain response over 368 days (Moran et al., 1991), respectively. The effects of the oestrogenic growth promotants, zeranol and oestradiol, on breeding heifers have been extensively researched overseas. Moran et al. (1991) reported that the regular release of oestradiol 17 $\beta$  from repeated implants of zeranol (36 mg) over 352 days, and implants of long-acting biodegradable oestradiol (38 mg) provide a regular release of oestradiol over the entire growth phase which is comparable to that of Compudose 365. The 5.5% liveweight advantage that the Compudose 90 heifers achieved in this study over the Control heifers by day 385 compares favourably with the 5.7% and 5.5% significant ( $P < 0.05$ ) liveweight

advantage that zeranol and 2E treated heifers achieved on day 368, respectively (Moran et al., 1991). As was expected, the response observed in the current study with entire heifers was less than that reported in trials investigating the effects of Compudose on steers under New Zealand pastoral conditions (Bass et al., 1989; Arando-Osorio et al., 1996; Burnham et al., 1997).

Previous reports have highlighted the greater response that occurs in older animals as a result of the greater efficiency of deposition of lean meat as age decreases (Deutscher et al., 1986; Roche and Quirke, 1986, 1992). This study contradicts these reports owing to the lack of growth response that occurred over 382 days in heifers implanted with Compudose 400 at 210 days of age. However, Compudose did cause a 6.5% liveweight gain response from implantation at 7 months of age to pre-mating which is in agreement with other reports (Staigmiller et al., 1983; Deutscher et al., 1986; Cohen et al., 1987). Staigmiller et al. (1983) and Deutscher et al. (1986) reported liveweight gain responses of 10.4 and 11.1% ( $P < 0.05$  and  $P < 0.01$ , respectively), and 14.6% ( $P < 0.05$ ) in crossbred beef heifers that were implanted with 36 mg of zeranol at 8 months and 11 months of age, and 6 months and 9 months of age, respectively, and managed under feedlot conditions. The 6.6% ( $P < 0.05$ ) liveweight gain advantage from weaning to breeding in crossbred beef heifers implanted at 7 and 11 months of age with zeranol as reported by Cohen et al. (1987) is more consistent with the present trial, despite these heifers being managed under feedlot conditions. In the current study heifers treated with Compudose at 210 days of age did not achieve greater liveweights at pre-mating than control heifers as was observed in other studies where non significant ( $P < 0.05$ ) liveweight advantages of 5.7% and 6.1% in oestrogenic-treated heifers over non-treated heifers at mating have been recorded.

The lack of overall average daily gain response in Compudose 210 heifers may be due to the effect of pregnancy or age. Heifers treated with zeranol (36 mg) at 100, 170 and 225 days of gestation did not achieve a liveweight gain response at 1, 2, 3, 4 or 5 months after implantation (Anthony et al., 1981) which is inconsistent with responses of younger heifers that were not pregnant (Staigmiller et al., 1983; Deutscher et al., 1986; Cohen et al., 1987; Moran et al., 1991). Therefore,

Compudose 210 heifers in this trial would have been subjected to oestradiol 17 $\beta$  hormone for about 5 months of pregnancy, consequently influencing the overall response. Even though the response of anabolic growth promotants is expected to be greater in older animals, heifers subjected to oestrogenic growth promotants during the finishing or fattening phase of growth, and on high levels of nutrition, do not produce positive growth responses which may help explain the poor overall growth response observed in this study.

### 3.4.2 Skeletal development

The lack of an effect of Compudose on weight-corrected hip height, hip width, wither height and girth in this study agrees with other studies where oestradiol and zeranol implants have no effect on skeletal growth in breeding heifers (Staigmiller et al., 1983; Deutscher et al., 1986; Cohen et al., 1987; Moran et al., 1991). However, weight-corrected wither heights in steers administered with Compudose were generally lower ( $P > 0.05$ ), which agrees with a second trial reported by Staigmiller et al. (1983) where weight-corrected hip heights of zeranol-treated heifers at 8 and 11 months of age were significantly less than those of non-treated heifers ( $P < 0.01$ ). If any effect on skeletal growth had occurred from treating heifers with Compudose at 90 or 210 days of age, the effects would probably have disappeared by the day of measurement (9 months and 4 months, respectively, after the Compudose implant had expired). Oestrogenic growth promotants cause temporary changes as was reported by Anthony et al. (1981) where significantly larger pelvic areas in heifers implanted with zeranol at 100 days of gestation had disappeared by approximately 1 month prior to calving. A further study by Hancock et al. (1994) reported that the increase in pelvic area following implantation of Synovex C (10 mg of oestradiol benzoate and 100 mg of progesterone) at 2 months of age had disappeared by 22 months of age.

Heifers treated with zeranol implants at various stages of the growth phase (1 to 11 months of age) and during pregnancy generally had increased pelvic areas (Ellington et al., 1978; Anthony et al., 1981; Staigmiller et al., 1983; Deutscher et al., 1986; Cohen et al., 1987). This agrees with this study where Compudose-treated heifers tended to have larger pelvic areas than non-treated heifers ( $P > 0.10$ ). Differences in pelvic area in other studies were due to greater pelvic height measurements (Anthony et al., 1981) which contradicts the findings in this study where no differences in pelvic height occurred between treatment groups. However, Compudose-treated heifers had significantly greater internal pelvic width than control heifers. Therefore, these studies confirm that growth promotants with oestrogenic properties may stimulate pelvic girdle or flat (pelvic) bone growth compared to other parts of the skeleton.

Studies that reported larger pelvic areas from treating breeding heifers with zeranol suggested that calving difficulty may have been reduced, however not significantly (Deutscher et al., 1986). The fact that no calving difficulty was experienced in this study agrees with other studies where no differences in dystocia levels were observed in one trial reported by Anthony et al. (1981) while it disagrees with a second trial by Anthony et al. (1981) where heifers treated with zeranol at 100 days of gestation experienced more calving difficulty. Anthony et al. (1981) suggested that this difference was due to other factors such as nutrition, weather and/or genetics.

### **3.4.3 Reproductive performance**

This study demonstrated a later mean calving date and reduced calving spread in Compudose-treated heifers compared to non-treated heifers which may have been caused by a delay in the attainment of puberty and consequently a delay in oestrous cycling, or a greater incidence of non-ovulatory oestrus, in Compudose-treated heifers. This result compares to other studies where attainment of puberty was delayed and the occurrence of non-ovulatory oestrus was greater following zeranol treatment (Staigmiller et al., 1983; Deutscher et al., 1986; Moran et al., 1990;

Hancock et al., 1994). In New Zealand farming systems delayed oestrus and subsequently delayed calving dates are unprofitable. However, the smaller calving spread of the Compudose-treated groups compared to the non-treated group meant that the calving pattern was more compact, and any disadvantages from a delayed calving date are not likely to have adverse consequences in the following year.

This study was not able to conclude that Compudose would adversely affect heifer conception rates owing to the small number of heifers in each treatment group. However, five of the eight empty heifers were from the Compudose 90 group. Other studies have reported decreases in conception rate following zeranol treatment (Nelson et al., 1972; Anthony et al., 1981; Deutscher et al., 1986; Cohen et al., 1987; Moran et al., 1990; Hancock et al., 1994). However, these results have varied and are dependent on the age and time of treatment. Heifers administered with zeranol at birth, and reimplanted at various ages until breeding, experience greater reductions in conception rate (Cohen et al., 1987), than heifers implanted at 7-8 months of age and reimplanted at 9-11 months of age when no reductions in conception rates have been observed (Staigmiller et al., 1983; Cohen et al., 1987). No decrease in conception rates has been reported when heifers were implanted with 36 mg of zeranol at 2-5 months of age (Cohen et al., 1987).

Owing to the small number of heifers in each treatment group this study was not designed to determine the effects of Compudose on reproductive performance. However, there is evidence that Compudose-treatment in breeding heifers tends to delay calving date and decrease the conception rate. More research needs to be conducted on this aspect before a more accurate conclusion can be made.

#### **3.4.4 Calf performance**

The lack of differences in calf birthweight between Compudose-implanted and control heifers in this study is consistent with studies investigating the effects of zeranol (Anthony et al., 1981; Deutscher et al., 1986). Heifers implanted with oestrogenic compounds tended to give birth to lighter calves (but differences were non-significant) (Anthony et al., 1981; Deutscher et al., 1986). In this study it appears that the oestradiol 17 $\beta$  released from Compudose does not transfer from the heifer into the calf and affect calf birthweight. This has advantages in heifer replacement systems in order to avoid high levels of dystocia that can occur from large calves. The significantly lower weaning weight of calves from heifers treated with Compudose 90 and Compudose 210 compared to Control heifers in this study contradicts Anthony et al. (1981) and Deutscher et al. (1986) where no differences in weaning weight were observed between zeranol-treated and non-treated heifers. The smaller, but non significant, calf growth rate from birth to weaning of calves born to Compudose-treated heifers is difficult to explain because no differences in milk production were evident in this study and the three treatment groups were managed similarly.

#### **3.4.5 Milk production and udder volume**

The effect of Compudose or other oestrogenic growth promotants (zeranol and oestradiol) on milk production and udder development during lactation has not been extensively investigated. However, Deutscher et al. (1986) did report that weaning weights of calves from zeranol-treated heifers were not significantly different to those of calves from non-treated heifers. This suggests that milk production was not adversely affected, despite the abnormal early mammary gland development. This result compares with the current study where Compudose-treated heifers did not differ in milk production or udder volume at 4 and 8 weeks of lactation compared to non-implanted heifers. Milk yield results determined by

the weigh-nurse-weigh method in treated and control heifers in this study are of similar magnitude to milk yields determined by the same method by Noricumbo-Saenz (1995).

Udder volume is a measure of the total amount of mammary tissue in the udder, and as such does not distinguish between secretory and non-secretory tissue (Eichler and MacFadden, 1996). However, by inference differences in udder volume reflect differences in secretory cell number (Davis et al., 1983). The lack of difference in udder volume between treatment groups during lactation further implies that Compudose did not adversely alter the composition of the udder.

The high correlations between milk yield and udder volume of 0.63 provides evidence that Compudose has no adverse effects on milk production and udder development, despite calf weaning weights being lower in Compudose-treated heifers. Therefore, lower calf weaning weights must be due to some other factor, rather than milk yield. The correlation and regression equation between milk yield and udder volume in this study compares favourably with Davis et al. (1983) where milk yield and udder volume were linearly related by the regression equation of:  $MY = 0.93UV + 7.33$ ;  $r = 0.66$ ;  $P < 0.001$ ;  $n = 55$ .

Most studies investigating the effects of oestrogenic growth promotants have observed abnormal mammary gland development during the early stages of the heifers' life cycle (Deutscher et al., 1986; Johnson et al., 1987; Moran et al., 1991; Hancock et al., 1994). Oestrogens are responsible for normal mammary gland development (Sejrsen, 1984). Therefore growth promotants with oestrogenic properties are likely to stimulate mammary growth (Deutscher et al., 1986; Johnson et al., 1987; Moran et al., 1991; Hancock et al., 1994). Increased average teat length following zeranol and 2E administration (Deutscher et al., 1986; Moran et al., 1991), and increased subjective udder development scores following Compudose (Johnson et al., 1987) and Synovex C (10 mg oestradiol benzoate and 100 mg of progesterone; Hancock et al., 1994) treatment, have been reported. The increase in udder development scores indicated that heifers treated with growth promotants had extreme udder and teat development (Hancock et al., 1994), or

udder development similar to that of heifers 3-4 weeks prior to parturition (Johnson et al., 1987). In this study udder development observations were not carried out on Compudose-treated heifers in the early stages of growth. However, if udder development was affected by Compudose in the early stages of growth, there were no subsequent adverse effects on udder development or milk production.

Plane of nutrition and growth rates greater than 0.7-0.8 kg/day around the time of puberty can have adverse effects on mammary gland development by decreasing the proportion of secretory tissue in the developing udder and reducing subsequent milk production (Sejrsen, 1978; Little and Kay, 1979; Sejrsen et al., 1982). In this study Control heifers and Compudose 90 heifers grew at 0.61 kg/day and 0.72 kg/day, respectively, from 6 to 9 months of age. From 7 to 9 months of age Compudose 210 heifers grew at 0.87 kg/day, Control heifers grew at 0.76 kg/day and Compudose 90 heifers grew at 0.91 kg/day. These growth rates of Compudose-treated heifers around puberty are close to the critical upper limits derived by Sejrsen (1978), but they did not adversely affect udder development and subsequent milk production.

#### **3.4.6 Carcass quality characteristics**

Differences in carcass quality characteristics between Compudose-treated and non-treated heifers in this study were not as marked as the differences reported in other studies investigating the effects of Compudose on steer and heifer carcass quality characteristics. However, the direction of the treatment effect is the same.

The lack of treatment effect on DO% in this study agrees with other studies investigating the effects of Compudose in heifers (Johnson et al., 1987; Stobbs et al., 1988), while other studies investigating the effects of Compudose in steers reported increases in DO% (Mathison and Stobbs, 1983; Burnham et al., 1997). Therefore in this study, the lack of treatment effects on DO%, in combination with the 4.9% and 8.8% pre-slaughter liveweight advantage of heifers treated with Compudose 90 over non-treated heifers and heifers treated with Compudose 210,

respectively, resulted in similar carcass weight differences. Carcasses of Compudose 90 heifers were 4.9% and 9.3% heavier than carcasses of control and Compudose 210 heifers, respectively. The results of this study compare with the study by Stobbs et al. (1988) where Compudose-treated heifers had a 4.3% carcass weight advantage over non-treated heifers. Therefore, this carcass weight advantage may provide small financial benefits for the New Zealand beef producer owing to New Zealand's current carcass weight-based payment system.

Compudose and other anabolic growth promotants have repeatedly been reported to promote muscle deposition at the expense of fat. Evidence of this effect has been observed in steers and heifers where kidney and pelvic fat weights, and fat depths have been reduced following treatment with oestrogenic implants (Moran et al., 1991), while at the same time rib-eye area has been increased (Johnson et al., 1987; Stobbs et al., 1988). In this study, carcasses of heifers treated with Compudose 210 had significantly greater rib-eye areas than carcasses of non-treated heifers and carcasses of heifers treated with Compudose 90, but differences in fat depth and kidney and pelvic fat weight were not significant. This agrees with a study by Mathison and Stobbs (1983) where Compudose-treated steer carcasses had greater rib-eye areas than non-treated steer carcasses, while at the same time fat depth did not alter. Despite the lack of significant treatment differences in kidney and pelvic fat weight, and fat depths, in this study, Compudose-treated heifers tended to have lighter kidney and pelvic fat weights than Control heifers, and carcasses of Compudose 210 heifers had lower fat depths than carcasses of non-treated heifers. The greater proportion of Compudose 210 carcasses graded in the L class further reinforces the fact that Compudose influences fat content. The greater weight of the outside round muscle in carcasses of Compudose 210 heifers compared to non-treated carcasses illustrates the increase in muscle deposition from Compudose treatment. Compudose does appear to influence the muscle and fat content of carcasses, but the extent of these differences is small. The increase in liveweight of heifers treated with Compudose 90 cannot conclusively be explained by an increase in muscle deposition (determined by weights of the three hind-quarter cuts and rib-eye area) and a decrease in fat content (determined by fat depth and kidney and pelvic fat weight) as has been observed in other studies (Sharp and Dyer, 1971;

Cohen et al., 1987). It is also worth noting that heifers treated with Compudose 210 were lighter and grew more slowly than Control heifers and Compudose 90 heifers, but differences in muscle and fat content did not occur.

Even though carcass weight-adjusted carcass length did not differ between treatment groups, Compudose does appear to have an effect on skeletal development. Carcass length of Compudose 90 heifers was significantly less than that of non-treated heifers, and carcasses of Compudose 210 heifers tended to be shorter than those of Control heifers, but not significantly. These effects of Compudose on carcass length are similar to those of Burnham et al. (1997) where Compudose-treated steers had shorter carcasses than non-treated steers. The direction of the difference in carcass length between treatment groups is the same as that for the other skeletal parameters. Heifers implanted with Compudose 90 were exposed to oestradiol  $17\beta$  for a greater part of the growing and development period than heifers implanted with Compudose 210. Hence this may explain why there was a greater difference in carcass length between Control heifers and Compudose 90 heifers than between Control heifers and Compudose 210 heifers.

The lower meat pH values of Compudose-treated heifers observed in this study compared to non-treated heifers agrees with Burnham et al. (1997) where ultimate pH of Compudose-treated steers was significantly lower than non-treated steers.

### **3.4.7 Implications of using Compudose in beef breeding systems and dairy heifer rearing systems**

The results of this study indicate that Compudose has the greatest effect when implanted into heifers at 90 days of age, rather than at 210 days of age. The 6.8% liveweight gain response that occurs in Compudose 90 heifers over the 382 day period of implantation subsequently results in small, but non-significant liveweight advantages at mating, pre-calving, weaning and slaughter. These advantages will not be economically beneficial for the farmer. Carcass weights of Compudose 90 heifers were 10.5 kg greater, but not significantly, than those of Control heifers which equates to a gross return of \$17.85 per heifer at a schedule price of \$1.70 per kg of carcass weight. When accounting for the cost of the Compudose implant, which currently retails at \$8.17 per implant, a net profit of \$9.68 per heifer could be expected. However, in the OBH system the effect of lower calf weaning weights and possibly lower conception rates make the use of Compudose in breeding heifers an unfeasible option for farmers.

Using Compudose as a means of achieving target liveweights at critical times of a heifer's lifecycle is not a viable option for either the beef or dairy industries. Despite small, but non-significant, advantages occurring at mating and pre-calving by using Compudose, manipulating current management practices would be more successful in improving heifer performance in the beef and dairy industries than using Compudose.

It has been difficult in the OBH system to grow heifers from calving to slaughter at sufficiently high growth rates to achieve high liveweights at slaughter. Implanting heifers with Compudose at 90 and 210 days of age will still not allow heifers to obtain satisfactory liveweights between 30-36 months of age. In order to finish OBH's at greater liveweights Compudose 200 might be implanted after calving. However, the consequences of this practice are unknown.

If Compudose became an acceptable product for use in the dairy industry several ethical and consumer safety issues would arise that have not been confronted by the dairy industry in the past, but have been by the beef industry. The collection, processing and marketing of milk and milk products from animals treated with growth promotants is an issue that the dairy industry does not have strategies in place to deal with. Based on the results of this study Compudose will not become a viable option for achieving target liveweights, therefore the dairy industry will not have to deal with these issues.

Compudose may have been useful in steer production systems with several positive growth, feed conversion efficiency and economic responses being reported, but this is not the case in breeding heifer systems. Small liveweight gain responses, and the unknown effect of Compudose treatment in breeding heifers fed pasture-based diets, mean that Compudose would not be an economically viable option for use in either the beef or dairy industries.

#### **3.4.8 Future Research**

This study did not detect milk production differences, however it must be noted that the WNW method used to determine the effects of Compudose on milk production has several sources of variation. Therefore it may not provide an accurate representation of the effects Compudose has on milk production. The WNW method provides an estimate of milk production based on the ability of the calf to consume milk, and therefore depends on the calf's appetite at that time, rather than the cow's capacity to produce milk. Despite the errors of the method, it has been widely used in beef production systems and is the most practical, least expensive and easiest method to implement. The lack of a treatment difference in milk production and udder volume is backed up by the high repeatabilities of using the WNW method over consecutive days, and the udder dimension measurements taken by two operators on consecutive days. The high correlation between MY and UV also provides confident evidence that there are no milk production differences.

The use of milking machines and/or oxytocin may provide a more informative picture. Further studies into the effects of Compudose on milk production and composition are needed.

Compudose in steers has been extensively investigated in the past, but very little research has been conducted regarding the effects of Compudose in entire breeding heifers, under both pastoral and feedlot conditions. This is a reflection of the current regulations that preclude use of Compudose in breeding heifers because of the adverse consequences for reproductive performance. This is a possible issue that warrants further investigation. This study indicated that Compudose treatment in young heifers (90 days of age) may depress conception rates, possibly reflecting a delay in puberty and a delay in oestrous cycling, but the small numbers of animals used in this study did not allow definitive conclusions regarding Compudose effects on normal heifer fertility. A more in-depth study is needed to investigate this issue. However, owing to the lack of liveweight response and economic benefits from using Compudose in either beef or dairy heifer systems, Compudose will still not be widely adopted for use in breeding heifers, and therefore the effects on the reproductive performance are of minor importance.

A study overseas under feedlot conditions (Stobbs et al., 1988) has found a small (4%) improvement in feed conversion efficiency in heifers treated with Compudose that were older than those in this study and were not pregnant. This agrees with the studies investigating the effects of Compudose in steers where feed conversion efficiency responses of 7-16% have been reported under both pastoral and feedlot conditions. Therefore, it would be worth investigating the effect of Compudose on the feed conversion efficiency of breeding heifers under a pastoral environment.

The poor liveweight gains of calves born to Compudose-treated heifers in this study do not agree with other studies investigating the effect of oestrogenic substances on offspring performance. Therefore, it is an issue that requires further investigation. The depressed calf liveweight from birth to weaning in offspring born to Compudose-treated heifers is an important contributor to the success of both beef and dairy heifer systems and the reasons for this poor performance are unknown

from this study. Milk production, as determined by the WNW method, was not a contributing factor to the lower performance, therefore other factors as a result of Compudose treatment may be affecting calf performance. These other factors may be related to milk composition differences, milk production differences that were not detected in this study because of the inaccuracies of the WNW method, or the transfer of Compudose metabolites from the heifer dam to the calf that have negative effects on calf growth rate from birth to weaning.

There are several issues surrounding the use of Compudose in breeding heifers that require further investigation before the widespread use of Compudose in beef and dairy heifer rearing systems is recommended. However, the poor liveweight response and lack of economic benefits from using Compudose provides enough initial evidence that its use in the beef and dairy industries is not worth recommending.

### 3.5 CONCLUSION

In summary, liveweight gain responses were found to be greater when younger heifers (90 days of age) were treated with Compudose 400 than older heifers (210 days of age). The 6.8% growth rate advantage of heifers implanted with Compudose at 90 days of age compared to non-implanted heifers continued throughout the heifers' lifecycle with liveweights at mating, pre-calving, weaning and slaughter being greater than in heifers not treated, or treated at 7 months of age. Heifers implanted with Compudose at 210 days of age did not respond favourably to Compudose treatment, exhibiting no liveweight advantages over the non-treated heifers at critical times of the lifecycle. Compudose treatment in heifers of 90 days and 210 days of age did not have significant effects on skeletal development, carcass quality characteristics, udder development or milk production. Despite growth rates around the time of puberty being close to the reported critical upper limits of 0.7-0.8 kg/day, when mammary gland development and milk production are adversely affected, this study did not find that high growth rates from Compudose treatment, in combination with hormone administration, adversely affected heifer lactational performance. There appears to be a trend that Compudose treatment in young heifers will adversely affect reproductive performance with later calving dates, more compact calving spreads and a reduction in conception rate, but this aspect of Compudose treatment requires more research. Calves born to Compudose-treated heifers also grew more slowly than non-treated heifers, despite there being no apparent milk production differences and the effect of the implant expiring by calving.

The practical implication of these results is that implanting heifers with Compudose at 3 months of age will be more beneficial than implanting heifers with Compudose at 7 months of age. However, the liveweight advantage of treating 3 month old heifers with Compudose in this study was not great enough to warrant the use of Compudose in beef breeding heifers when the effect of the lower calf weaning

weights and decreased fertility are taken into account. Even in dairy heifer rearing systems, Compudose use may be limited until the effects on heifer fertility are known.

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