THE UNIVERSITY OF MANITOBA

INJECTABLE COPPER AND ZINC FOR GRAZING AND FINISHING YEARLING BEEF STEERS

BY

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ABSTRACT

An experiment with a grazing trial and a finishing trial was conducted using yearling beef steers to study the effect of injectable preparations of Prolontex-Cu★ and Prolontex-Zn★ upon body weights, and Cu and Zn status.

In the grazing trial, 60 yearling beef steers (37 Herefords, Group I, and 23 Angus-Charolais-North-Devon crosses, Group II) were used in a 113-day grazing trial at the community pasture at Narcisse, Manitoba to determine the effect of injectable Cu and Zn upon body weight and Cu and Zn concentrations in liver and blood serum. Liver tissue was obtained at the start, middle and at end of the grazing trial; blood serum was obtained at the start, and at 28-day intervals. Steers were weighed at the start and at 28-day intervals. Liver tissue and blood serum were analyzed for Cu and Zn concentrations.

Forage samples from the grazed paddocks were collected at 28-day intervals. These were analyzed for Ca, Mg, P, S, Cu, Zn, Mo, Fe, Mn, CP, DM and ADF.

In both Groups I and II, injectable Cu (ICU) treatment did not influence ($P>0.05$) body weight responses of steers. In Group I, injectable Zn (IZN) treatment reduced ($P<0.10$) body weight responses of steers but did not influence ($P>0.05$) body weight responses of similarly treated steers of Group II.

Injectable Cu increased ($P<0.05$) and maintained high levels of liver Cu of Group I steers throughout the grazing trial. In Group II, ICU treated steers had higher ($P<0.05$) liver Cu than ICU untreated steers at sampling day 63 and not thereafter. ICU treated steers of Group I had higher ($P<0.05$) blood serum Cu than ICU untreated steers at sampling day 84 and not thereafter ($P>0.05$). For both Group I and Group II steers, IZN treatment did not influence liver Zn ($P>0.05$) or blood serum Zn ($P>0.05$).

Copper and Zn contents in forage samples were present in lower concentrations relative to published requirements for cattle. Total S and Mo were less than 2.0 g and 1.5 mg/kg DM respectively in all forage samples analyzed.

In the finishing trial, 44 steers (the 37 steers of Group I and the seven heaviest steers of Group II used during the grazing trial) were put on a barley-based finishing ration for a period of 79 days. Steers were weighed on day 0, 35 and 79 of the finishing trial. Liver tissue which was analyzed for Cu and Zn was obtained from all steers at the time of slaughter.

Steers treated with ICU on day 0 of the grazing trial had lower ($P<0.05$) body weights on each sampling day of the finishing trial when compared to day 0 of the finishing trial. IZN treatment on day 0 of the grazing trial did not influence ($P>0.05$) body weight of steers during the finishing

trial. Liver Cu and liver Zn during the finishing trial was not influenced ($P>0.05$) by ICU or IZN treatment at day 0 of the grazing trial.

Under the circumstances of this experiment neither Prolontex-Cu nor Prolontex-Zn was beneficial in increasing body weight responses of steers. Prolontex-Cu did modify the levels of Cu in liver and blood serum while Prolontex-Zn did not have an influence on Zn levels in liver and blood serum.

★ Prolontex-Cu and Prolontex-Zn are trade Marks for injectable Cu and Zn products manufacturd by Roussel Uclaf, Paris, France. These products were obtained for the present study through Hoechst, Canada, Limited.

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INTRODUCTION

Copper (Cu) deficiency in cattle under grazing conditions has been reported in several parts of Canada (Brockman, 1977; Smart et al., 1980; Hidiroglou, 1980; Smart et al., 1981; Steacy et al., 1983; Boila et al., 1984a). In Manitoba, evidence of Cu deficiency was first reported by Cunningham (1950) and Cunningham et al. (1953). Subsequent studies with grazing cattle and sheep in different parts of the Province have since confirmed these earlier findings of Cu deficiency (Smith, 1955; Findlay, 1965; Drysdale, 1979; Smart et al., 1980; Boila et al., 1984a, 1984b).

The metabolic requirement for Cu by cattle is normally derived from the herbage or from body stores. Where the herbage Cu is inadequate a primary Cu deficiency will occur after body stores of the element are exhausted. In secondary Cu deficiency the intake of Cu may be adequate but utilization is impaired by elevated dietary Molybdenum (Mo) and/or sulphur (S). Other elements such as Zn, Fe and cadmium (Cd) may influence the utilization of Cu (Mills, 1980).

Depending on the species, severity and extent, Cu deficiency can lead to anaemia, bone and skeletal disorders, depigmentation of hair and wool, impaired keratinization of hair and wool, diarrhoea, cardiovascular disorders, central nervous disorders, retarded growth and reduced fertility (Underwood, 1977, 1981).

Zinc is a ubiquitous element and a deficiency in cattle under grazing conditions is very rare (Legg and Sears, 1960). In cattle, Zn is required for growth and reproduction (Mills et al., 1967; Underwood and Somers, 1969).

Severe Zn deficiency in cattle is normally manifested by retarded growth, reduced reproductive capacity, skeletal abnormalities, parakeratosis, deterioration of hair and changes in activities of Zn-containing enzymes (Miller and Miller, 1960; Underwood and Somers, 1969; Underwood, 1977, 1981). Like Cu, Zn utilization is also influenced by other dietary components such as Cu, Fe, Cd and phytate.

Several methods (injectable and dietary) are being used to prevent or correct Cu and Zn deficiencies in areas where primary or secondary deficiencies of these minerals are known to occur. Dietary supplementation of Cu and Zn under grazing situations can be accomplished by the addition of Cu and Zn salts to the drinking water, systematic dosing by drenching or offering licks or mineral mixtures containing these salts or by topdressing of pastures with fertilizers containing these minerals. Where dietary supplementation is done there is usually the problem of adequate intake of minerals; also in places where Mo and S contents in the herbage are high, this dietary method of supplementation may not be effective. With injectable preparations there is usually a slow release of mineral from the site of injection over the grazing period. When provided as an injectable preparation

the effects of other dietary components on the utilization of these minerals will be greatly reduced.

The study reported herein was carried out in two trials: (a) Grazing and (b) Finishing trials. The grazing trial was conducted to determine the effect of injectable Prolontex-Cu and Prolontex-Zn supplementation upon body weight responses and Cu and Zn status of grazing yearling beef steers. In the Finishing trial, the effect of Prolontex-Cu and Prolontex-Zn supplementation at the start of the grazing trial upon body weight, liver Cu and liver Zn of steers during and at the end of the finishing trial was investigated.

REVIEW OF LITERATURE

The essentiality and the requirement of copper and zinc.

Essentiality of copper

The essential role of Cu in vertebrates was first demonstrated by Hart et al (1928) and Mann and Keilin (1938). Its role in cattle and sheep nutrition has been studied (Neal et al., 1931; Sjollema, 1933; Bennets and Chapman, 1937; Beck, 1941; Dick, 1952; Cunningham, 1959; Hill et al., 1962; Underwood, 1962, 1971, 1977, and 1981; Ammerman, 1970; Mills et al., 1976; Suttle, 1979).

Mann and Keilin (1938) first isolated a Cu-containing enzyme from bovine erythrocytes and since then several Cu containing enzymes have been isolated and characterized (Frieden 1968, 1971; O'Dell, 1976). The functional changes of these enzymes during deficiency conditions have been reviewed (Adelstein and Valle, 1962; Mills et al., 1976; Prasad, 1978).

The basic dietary requirement of Cu for cattle is 10 mg per kilogram dry matter (mg/kg DM) when the dietary Mo and S are less than 1.0 mg and 1.0 g per kilogram diet respectively (Miltimore et al., 1964). The availability of Cu to cattle is influenced by several dietary substances including Mo

and S (Dick, 1952, 1953, 1956; Alloway, 1973; Dick et al., 1975). Mills et al. (1976) reported that 8.0 mg Cu/kg DM was inadequate for Friesian calves; they suggested 10.0 mg Cu/kg DM as the minimum requirement. Thornton et al., (1972a) reported the occurrence of Cu deficiency in cattle pastures in which the herbage Cu content was 10-14 mg/kg DM. From these findings, they suggested that whenever the Mo levels exceed 1.0 mg/kg DM, the absolute Cu to Mo ratio be considered. Waldern (1971) and Underwood (1977) have suggested a dietary ratio of four parts Cu to one part Mo to be adequate in preventing the inhibitory effect of Mo upon Cu utilization.

Essentiality of Zinc

The essentiality of zinc for mammals was first established by Todd et al (1934) and since then Zinc deficiency has been experimentally produced in cattle (Miller and Miller, 1960, 1962, 1963), goats (Miller et al., 1963), sheep (Ott et al., 1964; Underwood and Somers, 1969), pigs (Tucker and Salmon, 1955; Stevenson and Earle, 1956) and poultry (Blamberg et al., 1960; Zeigler et al., 1961; Sunde, 1972).

The main functions of zinc in animal metabolism appears to be enzymatic; there are over seventy metalloenzymes known to require zinc for their function (Riordan and Vallee, 1976). Zinc metalloenzymes are present throughout all phylla

and participate in a wide variety of metabolic processes including carbohydrate,lipid,protein,and nucleic acid synthesis or degradation (Prasad,1976).

Zinc is present in several dehydrogenases, aldolases, peptidases, and phosphatases and each of the six categories of enzymes designated by the International Union of Biochemistry (I.U.B) commission on enzyme nomenclature contains at least an example of a zinc metalloenzyme (Riordan and Valle, 1976).

In cattle and sheep, zinc improves growth, reproduction and wool production (Mills et al 1967; Underwood and Somers, 1969; Underwood, 1977). The minimum requirement of Zn for beef cattle lies between 20 and 30 mg/kg DM of the diet (NRC, 1978; ARC, 1980). The availability of zinc from the diet is influenced by several organic and inorganic substances; the effect of these substances on zinc utilization will be discussed in later sections.

Body levels of copper and zinc

Body levels of copper

Copper is distributed throughout the body. The concentration of this element in tissues and organs varies with species, age, Cu intake and physiological state of the animal (Dick, 1954; Bush et al., 1955; Herbert and Sternlieb, 1960; Wiener and Field 1969, Underwood, 1977).

The liver, brain, heart and kidneys, in decreasing order contain high concentrations of Cu. Intermediate concentrations are found in the lung, intestine and spleen. Endocrine glands, muscle and bone have the lowest concentrations of Cu (Moule et al., 1959; Cartwright and Wintrobe, 1964a, 1964b; O'Mary et al., 1969; Claypool et al., 1975). The livers of normal animals in most species contain 10-50 mg/kg DM (dry basis) with a high proportion containing 15-30 mgCu/kg DM (Lorenzen and Smith, 1947).

In ruminants, the capacity for storage of Cu in liver tissue is high. The level of this element in liver often closely reflects the dietary level of Cu with or without supplementation (Cunningham, 1931). Table 1 shows values for the concentration of copper in cattle and sheep. In cattle, liver Cu concentrations below 40 and 55 mg/kg DM for adult and newborn respectively, are generally regarded as levels indicative of a Cu deficiency (Dick, 1954, Claypool et al., 1975). Liver copper levels are normally higher in the newborn of most species, except cattle and sheep (Underwood, 1977). In cattle, liver Cu levels change very little from birth to old age, while in sheep, liver Cu concentrations have been shown to rise continuously from birth (Underwood, 1977).

Table 1. Influence of Age, and Cu Intake on the Concentration of Cu in the Liver and Cattle and Sheep.#

Species	Age and Cu Treatment	No. of Animals	mg Cu/kg DM Average	Range
Sheep	Newborn - Normal diet	27	168.0	74 - 430
	Newborn - Cu deficient diet	29	13.0	4 - 34
	Adult - Normal diet	44	599.0	186 - 1374
	Adult - Cu deficient	35	27.0	7 - 106
Cattle	Newborn - Normal diet	41	381.0	143 - 655
	Newborn - Cu - deficient	20	55.0	8 - 109
	Adult- Normal diet	23	200.0	23 - 409
	Adult - Cu deficient	41	11.5	3 - 32

Adapted from **Cunningham** (1946).

Copper in whole blood (Evans, 1973) is present in five separate fractions:

1. Erythrocyte superoxide dismutase,
2. An unidentified Cu complex in erythrocytes,
3. Plasma albumin,
4. Plasma caeruloplasmin and
5. Amino acids.

About 60% of erythrocyte Cu is associated with a low molecular weight protein (M.W 35,000) called superoxide dismutase which is also known as erythrocuprein (Mann and Keilin, 1939; Shields et al., 1961; Cartwright and Wintrobe, 1964a). Superoxide dismutase has two atoms of Cu per molecule (0.34% Cu) in the cupric form. The remainder of erythrocyte Cu is loosely bound to an unidentified protein and is much more labile than erythrocuprein (Shields et al., 1961; Cartwright and Wintrobe, 1964a, and 1964).

Copper is present in several forms in the blood plasma of cattle and sheep. Caeruloplasmin which constitutes about 60-99% plasma Cu is an α-globulin with a molecular weight of 157,000 and contains 8 atoms of Cu per molecule (Holmberg and Laurell, 1947, 1948; Wintrobe et al., 1953; Butler, 1963; Cartwright and Wintrobe, 1964; Starcher and Hill, 1965). As an oxidase enzyme, it catalyses the oxidation of various substances including polyphenols and biological compounds (Martin et al., 1964; Underwood, 1977). Albumin-bound Cu is referred to as direct reacting Cu because of its

reaction with dithizone. It is non dialyzable and is reversibly bound to serum albumin (Bush et al., 1956; Bowland et al., 1961). Plasma also contains Cu enzymes such as lysyl oxidase, cytochrome oxidase, monoamine oxidase, tyrosinase, lacase, ascorbic oxidase, δ-aminolevulenic acid, dehydrase and dopamine-β-hydroxylase (Sass-Kortsak, 1965). A small fraction of plasma Cu is bound to amino acids; this latter fraction is believed to be important for the transport and cellular metabolism of Cu (Sass-Kortsak, 1965).

In healthy animals, the normal range for serum Cu is 0.50 to 1.50 ug/ml with a high proportion of values falling between 0.80 to 1.20 ug/ml (Beck, 1961). For cattle and sheep, serum Cu values below 0.60 ug/ml often indicate a Cu deficiency, with values consistently below 0.50 ug/ml demonstrating a severe Cu deficiency (Claypool et al., 1975; Underwood 1977).

The effect of pregnancy on blood Cu levels in various species has been studied (Fay et al., 1949; Schienberg et al., 1954; Butler, 1963; Halstead et al., 1968). A decline in whole blood, plasma, caeruloplasmin and erythrocyte Cu during pregnancy was observed in housed (Butler, 1963) and grazing ewes (Allcroft and Uvarov 1959; Butler and Barlow, 1963). Howell et al., (1968) reported increases in blood and caeruloplasmin Cu at about one week postpartum. Newborn lambs of these ewes had low blood and caeruloplasmin Cu but these Cu levels were shown to increase to a normal adult

range within one week. In newborn calves, whole blood and plasma Cu levels were significantly higher than in their mothers (Bingley and Dufty, 1969).

Several factors have been shown to influence the distribution of Cu in the body (Lahey et al., 1952; Dick et al., 1975; Mills, 1979). Disease and parasitic infestation cause variations in blood Cu parameters of cattle and sheep (Cancilla, 1968; McCosker, 1968; Patterson et al., 1968; Thomas and George 1968), and other species (Cartwright et al, 1954,1960; Danks et al., 1973). Infestation with internal parasites depresses Cu values (Bremner, 1959; McCosker, 1968).

The concentration of Cu in milk varies with species, age, stage of lactation and Cu nutriture of the animal (Underwood, 1977). The normal Cu level in milk is approximately 0.64 ug/ml for cattle (Cox and Miller, 1937) and a range of 0.20 to 0.60 ug/ml for sheep (Beck, 1941). However, Dunkley et al., (1963) have reported a substantial elevation of milk Cu for at least four weeks following subcutaneous injections of cows with 300 mg Cu as Cu glycinate.

The concentration of Cu in hair and wool often reflects the dietary level of Cu (Underwood, 1977). Van Koetsveld (1958) observed that cattle with hair having less than 8 mg Cu/kg DM showed signs of Cu deficiency. Cunningham and Hogan (1958) did not find any relationship between the Cu concen-

tration of cattle hair and the level of this element in the diet or liver. O'Mary et al., (1969) reported a range of 10 to 31 mg Cu/kg DM for the hair of Hereford cattle. Colour had no effect on Cu concentration, although season and the time of sampling influenced the concentration of Cu in hair.

Body levels of Zinc

Zinc is distributed throughout the body in concentrations that vary from tissue to tissue, but with little variation among species (Macapinlac et al., 1967; Hamilton et al., 1973). Liver, kidneys, bone, retina, prostate and muscle contain higher concentrations of Zn (MacCapinlac et al., 1967; Hamilton et al., 1972, 1973).

The normal concentration range of Zn in the liver of cattle is 25 to 50 mg/kg ~~DM~~ (wet basis) and unlike Cu there is no special storage form of Zn, thus suggesting that a regular supply of dietary Zn is necessary for optimal Zn homeostasis (Sandstead, 1981). Substantial mobilization and redistribution of Zn from skeletal muscle to the liver and other tissues is known to occur especially during rapid growth and in cases of healing tissues (Savlou and Huegin, 1962; Lichti et al 1972).

Zinc is present in plasma erythrocytes, leukocytes, and platelets (Underwood, 1977). The accepted normal range of Zn

in blood serum is 0.70 to 1.40 ug/ml. Foley et al.,(1968) reported the concentration of Zn in serum to be 16% higher than in plasma; they attributed the difference to the liberation of Zn from blood platelets during the process of clotting and invisible hemolysis of red cells.

Zinc in erythrocytes occurs as a component of the enzyme, carbonic anhydrase (Hove et al., 1937, 1938 and 1940). About 60-70% of plasma Zn is found loosely bound to albumin and 30-40% firmly bound to an α_2- macroglobulin, while a small fraction occurs in association with other Zn enzymes, transferrin and amino acids such as histidine, glutamine, threonine, cysteine, and lysine (Prasad and Oberleas, 1970a)

Ott et al., (1965) reported that 1.0 ug Zn/ml plasma was optimal for growth in sheep. Mills et al., (1965) observed satisfactory growth rates in sheep whose plasma Zn levels were below 1.0 ug.ml.

Pregnancy has a profound influence upon plasma Zn in sheep, goats and to a lesser extent in cows (Halstead et al., 1968; Pryor, 1975). Increases in plasma Zn for both pregnant sheep and goats has heen reported. In cows, there is usually a marked reduction in plasma Zn during and immediately after parturition (Halstead et al 1968). In sheep and goats, plasma Zn levels are higher in the fetus than in the mother.

Dietary levels of Zn also have a profound influence on the levels of Zn in serum and plasma. Large doses of Zn have been observed to increase whole blood and plasma Zn concentration in rats, rabbits, cats, pigs, sheep and cattle (Ott et al., 1966; Hoekstra et al 1969). Perry et al., (1968) reported a rise in serum Zn from 1.50 to 2.70 ug/ml when the dietary Zn of cattle was changed from 18 to 189 mg/kg DM . Miller et al., (1966) and Mills et al., (1967) reported a decrease in serum plasma and whole blood Zn in Zn-deficient sheep, calves and goats.

The concentration of Zn in milk varies with species, stage of lactation, and Zn content in the diet. Colostrum has been reported to be three to four times richer in Zn than true milk (Kirchgessner, 1959; Earle and Stevenson, 1965). The normal concentration range of Zn in milk is 2.30 to 5.00 ug/ml, but this concentration is known to decline with advancing lactation (Underwood, 1977).

The normal concentration of Zn in the hair of cattle is 100-150 mg/kg DM. O'Mary et al., (1969) and Hidiroglou and Spurr (1975) reported a range of 115 to 135 mg/kg DM for Hereford cattle; they also noted that Zn concentration in hair is not influenced by colour. However, Hidiroglou and Spurr (1975) observed lower Zn concentration in the winter hair than in the summer hair of cattle. The range of Zn in wool is 70-130 mg/kg DM. Burns et al., (1964) reported a mean of 115 mg/kg DM for 45 samples of wool from various areas. Hair

Zn concentration has also been shown to reflect dietary intakes of the element in cattle (Miller et al., 1965, 1966; Beeson et al., 1977) and goats (Combs et al., 1983).

Metabolism of copper and zinc

Absorption of copper

The absorption and retention of copper depends on several factors which include: 1. The chemical forms in which the element is ingested. 2. Dietary levels of other minerals and organic substances. 3. The age and the copper status of the animal (Underwood, 1977).

The exact mechanism of Cu absorption is not well known. In most animals, absorption takes place from the stomach and all sections of the small intestine, particularly the upper sections of the small intestine (Aspin and Sass-Kortsak, 1981). Grace (1975) reported that the absorption of Cu in sheep takes place in the large intestine, while Prokuchin et al., (1976) reported that absorption of Cu in sheep takes place in the forestomach. Using ligated sections of the gastrointestinal tract of the rat, Van Campen and Mitchell (1965) demonstrated that the rate and extent of Cu absorption was greatest in the stomach and decreases progressively along the digestive tract. In similar experiments with ligated rat intestine, Crampton et al., (1965) reported that

the transport of Cu from the mucosal side to the serosal fluids and passage of Cu originally present in the serosal fluids predominated in the lower small intestine, suggesting that the site of maximum absorptive ability is in the lower region.

The Cu status of the animal and the dietary level of Cu have been reported to affect the rate and extent of absorption. There is an increase in absorption at dietary levels below body need.

The chemical form of the element affects absorption of Cu. Oxides of Cu are less available to ruminants than sulphates, nitrates, and carbonates and other water-soluble forms of copper (Stake, 1977). Chapman et al., (1963) studied the relative absorption and excretion by beef cattle of Cu from various sources; they reported that cupric carbonate had the highest rate of absorption but was poorly retained. They suggested the order of availability of Cu from various sources to cattle as follows:

$CuCO_3 > Cu(NO_2)_3 > CuSO_4 > CuCl_2 > Cu_2O >$

CuO powder>CuO needles>Cu wire.

Underwood, (1977) reported that the movement of Cu through the intestinal mucosa occurred in three phases:

1. As ionic Cu;
2. Cu-binding protein from epithelial cells of the intestinal wall and
3. Cu bound to amino acids.

Mills (1954) suggested that Cu is transported through the intestinal mucosa both in ionic form and in the form of complexes similar to those found in herbages. Where Cu becomes bound to single amino acids, its rate of absorption will depend on the type of amino acid, its configuration and the degree of polymerization (Mills 1954). Two different mechanisms for the transport of Cu from the intestinal lumen to the blood have been proposed (Crampton et al., 1965). The first mechanism involves the transport of Cu from the intestinal lumen into the mucosal cells, while the second involves its transfer from the mucosal cells into the blood system. Studies with isolated hamster intestine (Crampton et a., 1965) revealed that the uptake of Cu from the mucosal side is probably the result of the binding of Cu to sites on the surface or within cells, while transport to the serosal side must involve a special mechanism dependent on metabolic energy.

In vitro studies of human saliva, gastric juice, hepatic and gall bladder bile (Gollan et al., 1971) showed that these secretions contained substances with the ability to bind Cu and form soluble complexes under alkaline conditions. Gall bladder bile had the greatest affinity for Cu. The Cu binding components in saliva and gastric juice were observed to form low molecular weight complexes compared to bile which contained a macromolecular binding component. From these observations, it was concluded that the net ab-

sorption of dietary Cu in man could represent an interplay between the opposing influences of endogeneous low molecular weight ligands and the macromolecular Cu binding moiety in bile.

Starcher (1969) identified a single metal-binding protein of molecular weight 1000 daltons in the chick duodenum and demonstrated that this protein binds Cu as well as Zn and Cd. Evans (1970, 1973) and Evans and Leblanc (1976) have found Cu in association with metal-binding ligands and macromolecules in the intestine of the rat and in the duodenum of the bovine. The properties of this protein or ligand described above were found to be similar to those of metallothionein and other Cu, Zn and Cd binding proteins. The presence of such proteins in intestinal mucosal cells suggested that they were involved in the absorption and transport of Cu. Chan et al., (1979) suggested that metallothionein was responsible for the intestinal transport and storage of Cu and that the component responsible for antagonism between metal ions that inhibited Cu absorption was metallothionein; these researchers proposed that antagonism between metal ions for absorption occurs at the brush border.

An inverse relationship between Cu in its binding to metallothionein and its transfer into blood has been reported by Cohen et al., (1979). The binding to metallothionein was regarded as a stumbling block to the transfer of Cu to the blood rather than a necessary step in transfer. They con-

cluded that the process of absorption involves two steps: (a) the uptake of Cu into mucosal cells and (b) the transfer of Cu from mucosal cells to the serosal side.

Although it has been demonstrated that the efficiency of absorption of Cu is higher during periods of low intake, there is as yet no clear evidence that high intakes of Cu cause an increase in the production of metallothionein which in turn might reduce the efficiency of Cu absorption.

Transport of Copper

Absorbed Cu is transported from the intestine in portal blood mostly in combination with albumin. A small portion is also transported by amino acids. In addition to facilitating the transport of Cu in blood, Cu-amino acid complexes also facilitate the transport of Cu across cell membranes in the liver and kidney (Harris and Sass-Kortsak, 1967) and erythrocytes (Neuman and Silverberg, 1966). Free cupric ion which is in equilibrium with Cu loosely bound to albumin is also present in minute proportions in plasma. It is thought that when Cu first enters the plasma from the gastrointestinal tract it is probably in this form (Evans, 1973; Underwood, 1977).

The pathway followed by absorbed Cu from the intestine has been studied using radioactive Cu. Absorbed Cu was found loosely bound to plasma proteins, including transferrin;

most of the Cu is directed to the liver (Bearn and Kunkel, 1954; Evans and Wiederands, 1967; Evans, 1973). The liver plays a central role in Cu metabolism. Its major functions in Cu metabolism appear to be storage of Cu, synthesis of caeruloplasmin and the excretion of Cu in an unabsorbable form (Owen, 1964). In sheep and cattle, caeruloplasmin Cu constitutes 69-99% of the total plasma Cu (Evans, 1973) and this does not exchange with non caeruloplasmin Cu. Caeruloplasmin does not function in transportation of absorbed Cu through portal blood to the liver (Neuman et al., 1966; Evans, 1973). Schienberg and Sternlieb, (1960), Frieden (1968), and Evans (1973) have suggested that caeruloplasmin is involved in the the transport of Cu from the liver to extrahepatic tissues for incorporation of Cu into enzymes such as cytochrome oxidase, superoxide dismutase and lysyl oxidase.

Owen (1964), Owen and Hazelrig (1966) and Marceau and Aspin (1972) reported that intravenous injections of radioactive Cu did not lead to immediate accumulation of the metal in extrahepatic organs of rats until after the emergence of caeruloplasmin-Cu. This suggested that caeruloplasmin served as a Cu donor to extrahepatic tissues. As the Cu in caeruloplasmin is not readily dissociated, it was thought that the exchange of Cu between caeruloplasmin and extrahepatic tissues probably involved a degradative mechanism taking place either on the cellular membrane or within the cell (Neuman et al., 1966; Evans, 1973).

Smith et al., (1968) and Bremner and Young (1978) reported that the release of Cu from plasma albumin was affected in ruminants receiving diets with high Mo content. Such animals have increased plasma Cu concentration due to a decreased rate of clearance of Cu from the plasma. Mills and Bremner (1980) suggested that thiomolybdates or related compounds formed in the rumen, when subsequently absorbed, can increase the affinity of albumin for Cu thereby causing a decrease in the rate of clearance of Cu from plasma.

About 60% of the total Cu in red cells is associated with the enzyme superoxide dismutase (erythroccuprein), while the remainder, a labile pool is contained within a freely dialyzable compartment (Scheinberg and Sternlieb 1960; Evans 1973). The total Cu content of erythrocytes is not affected by Cu deficiency. Even though Cu in erythrocytes is circulating this Cu is not involved in transporting the metal to and from tissues (Evans, 1973; Underwood, 1977; Aspin and Sass-Kortsak 1981).

Storage of Copper

According to Bremner and Mills (1981), the period over which a reduced intake of an essential element can be tolerated is not only influenced by the extent to which stores of the element have accumulated previously but also the ease with which they can be mobilized. Sheep and cattle can accu-

mulate high levels of Cu in the liver during periods when dietary intake of the metal is high (Underwood ,1977). Copper is stored temporarily in the parenchymal cells of the liver, heart and kidney (Bremner and Mills,1981). This temporarily stored Cu appears to be Cu in combination with liver metallothionein (Evans, 1973; Underwood, 1977; Hansard, 1983).

When radioactive Cu was injected into rats, there were rapid changes in the distribution of Cu in the liver (Marceau and Aspin, 1972). Bremner and Mills (1981) reported that when Cu is taken up by the liver, it was initially associated with metallothionein but was transferred rapidly to other hepatic proteins, including superoxide dismutase. When rats were injected with large quantities (100-300 ug) of non radioactive Cu, the pattern of retention was different (Bremner et al., 1978). A maximum concentration of Cu-metallothionein was measured only after ten hours. This protein persisted for much longer with a half- life of seventeen hours (Bremner et al., 1978). The difference between the two studies was explained as follows: In the tracer studies, the binding of Cu to metallothionein was assumed to have occured by exchange reaction whereas the incorporation of the non-radioactive Cu into the protein appeared to be a consequence of de novo synthesis of the protein. Thus the incorporation of [^{35}S]-Cysteine into metallothionein was stimulated by the administration of Cu (Bremner et al., 1978) and was inhibit-

ed by actinomycin D (Premakumar et al., 1975), a drug known to prevent the transcription of DNA to produce RNA thereby blocking protein synthesis. This suggested that the induction of metallothionein synthesis was under transcriptional control. The synthesis of Cu-metallothionein was also blocked by administration of cycloheximide, but this did not prevent the uptake of Cu by the liver (Bremner et al., 1978; Wiener and Cousins, 1980). Metallothionein is a major Cu-binding protein in the liver of animals receiving Cu in the diet as opposed to parenterally (Bremner and Mills, 1981). Up to 40% of the Cu in pig liver was found present as Cu-metallothionein (Bremner et al., 1978). The concentration of this protein is directly related to the liver content of metallothionein.

Hartmann and Weser (1977) and Riordan and Ricchards (1980) reported that Cu-metallothionein was frequently present in exceptionally high levels in the liver of foetal or neonatal animals. Williams et al., (1978) reported that about half the total body Cu in the developing fetus was usually present in the liver. It was thought that these reserves of Cu usually provided the newborn animal with a readily available reserve at a time when the intake of Cu from the milk was low. McDonald et al., (1979) however reported that much of the hepatic Cu in newly weaned calves was lost by endogeneous secretion. Evans and Reiss (1978), suggested that the accumulation of Cu in the liver of new-

born animals probably was a result of limited capacity for excretion rather than a need to form Cu reserves during foetal development which will be required in the neonatal period.

Excretion of Copper

Studies with different animals have shown that bile is the major pathway for the excretion of Cu from the mammalian body (Evans,1973; Cartwright and Wintrobe 1964). Cartwright and Wintrobe (1964) reported that in man and dogs, about 80% Cu was excreted with bile, 16% passed directly through the intestinal wall and approximately 4% was excreted in the urine. In ruminants, the main site for the excretion of Cu appears to be in the gastrointestinal tract where excess Cu is eliminated in the faeces (Aspin and Sass-Kortsak, 1981). Soli and Rambaek (1978) reported that ruminants excrete Cu in bile at a very low rate that is approximately equal to their rate of Cu excretion in urine.

Sass-Kortsak (1965) suggested that since Cu was poorly absorbed in ruminants, the main route of excretion of this metal from the body was in the faeces. The amount and rate of Cu excretion in the faeces depended on dietary level of the metal, chemical form of Cu in the diet, as well as the presence or absence of other dietary components known to be antagonistic to Cu absorption.

Lassiter and Bell (1960), studied the routes of excretion of Cu from various inorganic sources in sheep. When oral doses of cupric sulphate, cupric chloride, cupric oxide needles and cupric nitrate were administered, cumulative excretions averaged 78 to 87 % of the dose in 96 hours for the sulfate, chloride and nitrate, but only 3% of the dose for copper oxide needles. When intravenous injections of cupric sulfate, nitrate and chloride were provided, the cumulative excretion of the chloride in the urine was greater than that from the sulphate or nitrate forms. The amount of Cu voided in the feces and urine after intravenous injections were not significantly different. When oral doses of cupric oxide powder, cupric oxide needles, cupric carbonate and cuprous oxide were provided, cumulative fecal excretion averaged 7, 80 and 90% respectively for the cupric oxide needles, cupric carbonate and cuprous oxide powder. The greatest cumulative urine excretion was 3.1% of the dose from the cupric carbonate and only 0.19% to 0.23% from the other treatments.

Bremner and Young (1978), suggested that the increase in urinary Cu when diets high in Mo and S were fed to ruminants was due to the presence of Cu-Mo-sulfate complex in plasma which though not available for normal metabolic process is taken up by the kidney for excretion in the urine. Mills (1979, 1980), and Underwood (1977), have suggested that as high dietary Mo and sulfur levels increase plasma Cu, or Cu loosely bound to albumin, this may favour the urinary excretion of Cu, especially during conditions of proteinuria.

Bioavailability of Copper and Factors Affecting Bioavailability.

The chemical form of Cu in the diet influences the availability of this element to ruminants (Lassiter and Bell, 1960; Chapman and Bell, 1963). Copper usually exists in three states:

1. As the free, neutral atom;
2. As the cuprous ion (with one electron removed); and
3. As the cupric ion (with two electrons removed).

One ion is easily converted to the other by the addition or the removal of an electron, which gives Cu great versatility as an electron acceptor or electron donor (Frieden, 1968).

The availability of Cu in forages varies between different growth periods. Hartmans and Bosman, (1970) observed that the availability of Cu increased as herbage matured and was higher in hay than in fresh herbage. The decline in hepatic reserves on turning cattle out to graze short grass was more rapid than when grass at a later stage of growth was consumed. Copper in herbage was present as water-solubles neutral or negatively charged organic compounds (Mills, 1954). These water-soluble complexes of Cu in dried herbage were more effective than copper sulfate in increasing liver Cu in rats (Bremner and Mills, 1981).

Proteins also affect the utilization of Cu. Copper ions reacted with amino acids or proteins more strongly than

other ions did and consequently formed very stable chelates with biologically active subsstances (Evans 1973). Davis et al., (1962) reported that isolated soybean protein affected the utilization of Cu, Zn as well as Mn. Animal protein is more effective than vegetable protein for increasing weight gain, and food efficiency in pigs fed high levels of dietary Cu (Barber et al., 1962). MacPherson and Hemingway (1965) noted that the toxic effects of feeding high levels of Cu to sheep could be significantly reduced by increasing the protein level of the ration. An adequate or more than adequate level of dietary protein inhibited the accumulation of a toxic level of Cu in the liver of rats when large amounts of Cu were ingested (McCall and Davis 1961). These researchers suggested that the possible mechanism by which protein may prevent the accumulation of toxic levels of Cu in the liver is through the formation in the intestinal tract of a Cu-protein chelate which would be physiologically unavailable. Protein intake may make sheep less susceptible to Cu poisoning resulting from high dietary Cu levels (MacPherson and Hemingway 1965).

Absorption of Zinc

Zinc can enter the intestinal cells from either the intestinal lumen or the vascular supply (Smith et al., 1978). Absorption occurs mainly in the small intestine with the

duodenum or the proximal part of the small intestine being more active than the lower sections of the small intestine (Miller and Cragle, 1965, Methfessel et al., 1969; Miller, 1969; Methfessel and Spencer, 1973; Antonson et al., 1980).

Zinc absorption has been reported to occur by a saturable mechanism consistent with an enzyme or carrier-mediated process that occurs via a rapid initial phase followed by a slower phase that appears to involve a different mechanism (Antonson et al., 1980). Bremner and Mills (1980) reported that the slow phase and possibly the rapid phase in Zn absorption was preceded by uptake and accumulation of Zn by the mucosa. The transfer of Zn from mucosal cells to plasma appears to be the rate-limiting step in the slow phase of Zn absorption (Bremner and Mills 1981). Kowarski et al., (1974) suggested that the transfer of Zn across the brush border membrane into intestinal cells required energy and was related to an ATP-dependent system.

A number of amino acids, proteins and other substances have been reported to facilitate the absorption of Zn. One such compound, picolinic acid, a metabolic product of pyridine-dependent tryptophane metabolism facilitates uptake of Zn by intestinal mucosal cells (Evans and Johnson, 1980; Evans, 1980). This acid was present in both human and bovine milk (Evans and Johnson, 1980), in pancreatic secretions and in intestinal mucosa (Evans, 1980).

Zinc has the potential to form complexes with many low molecular weight components found in the intestinal lumen (Han and Evans, 1973). Prasad (1976) reported that the absorptive transfer of Zn and Cu from the small intestine to blood plasma was facilitated by low molecular weight proteins in the intestinal wall. He concluded that these proteins are not highly specific for either Cu or Zn as high intake of either element inhibited the absorption of the other. Histidine, EDTA, ProstaglandinE_2a,8-hydroxyquinoline, acetylsalicylate and penicillamine facilitate the transfer of Zn across the small intestine (Aspin and Sass-Kortsak 1981).

In mucosal cells, Zn may pass through to the serosal side or may remain within these cells where it forms complexes with metallothionein, a protein whose synthesis is induced by Zn itself (Bremner et al., 1976; Smith and Cousins 1980). Sandstead, (1981) and Smith and Cousins (1980) have suggested that a high body content of Zn resulted in the uptake of Zn by intestinal mucosal cells from the circulation followed by synthesis of metallothionein with an apperent suppression of Zn absorption from the lumen. Much of the Zn in the metallothionein complex was lost via desquamation of mucosal epithelium (Underwood, 1977).

Entry of Zn from the mucosal cell membrane into circulation occurs through the formation of complexes with transferrin, which takes up Zn from enterocyte plasma membrane. A

small fraction is also taken up by albumin (Evans, 1976). Smith and Cousins (1980) and Cousins (1979) reported that Zn was bound to albumin as it left the serosal surface of the gut. From the available evidence it would appear that Zn is transported to the liver mostly by transferrin before becoming bound to albumin or being incorporated to α-macroglobulin. In normal plasma, Zn is reported to be about 2/3 loosely bound to albumin and 1/3 tightly bound to α-macroglobulin with traces bound to the amino acids, histidine and cysteine (Kirchgessner 1978; Sandstead 1981).

Liver metabolism of Zinc

The liver is a major organ involved in Zn metabolism (Underwood, 1977). Studies with human subjects and experimental animals have shown an increase in the concentration of Zn in the liver shortly after oral or parenteral administration of Zn (Methfessel and Spencer, 1973; Richards and Cousins, 1976; Chen et al., 1977).

Certain humoral factors are known to stimulate the uptake of Zn by the liver. These include adrenocorticotropic hormone (ACTH), leukocyte endogeneous mediator (LEM), endotoxin and parathyroid hormone (Failla and Cousins 1978; Prasad, 1978). ACTH appeared to stimulate Zn uptake by the liver independent of its effect on the adrenal cortex. Leukocyte endogeneous mediator released from phagocytosing leukocytes

stimulated the liver uptake of Zn and Fe and the synthesis of acute phase proteins including caeruloplasmin (Cousins 1979; Evans, 1980).

Bremner and Davis (1973), and Oh and Whanger (1979) reported that Zn in the liver induced the synthesis of and combined with metallothionein. Zinc could form complexes with proteins which may be incorporated into the structure of other proteins and nucleic acids whose functions require its presence (Sandstead 1981). Synthesis of hepatic metallothionein was stimulated by elevation of dietary protein (Richards and Cousins 1976; Chen et al., 1977), or parenteral administration of Zn. Metallothionein serves as a storage protein for Zn (Chen et al., 1974), before its utilization in essential functions (Bremner and Davis 1973). Induced liver metallothionein has a half-life of 18-20 hours and removal of Zn from this protein occurs by proteolysis. The rate of degradation depends on the Zn status of the individual animal (Cousins, 1979a, 1979b)

Glucocorticoids have also been reported to stimulate the accumulation of Zn by hepatic cells. The accumulated Zn is bound initially in the cytosol fraction as metallothionein thus indicating that de novo metallothionein synthesis can be induced by glucocorticords. This is a humoral link in Zn transport system (Cousins, 1979a, 1979b).

Excretion of Zinc

The gastrointestinal tract is the primary route for the excretion of Zn (Miller et al 1966; Kirchgessner and Weigand 1983). Fecal Zn consists mainly of unabsorbable dietary Zn and a small fraction of endogeneous Zn secreted into the tract. The endogenous fraction originates from the desquamation of the epthelial lining, the discharge of mucins and digestive secretions such as saliva, gastric and intestinal juices, bile and pancreatic fluid, and from the transmucosal flux directed from the vascular bed to the luminal pool (Methfessel and Spencer 1973b; Weigand and Kirchgessner 1980; Evans et al., 1979; Kirchgessner and Weigand 1983). Georgievskii et al., (1982) reported that in the calf about a quarter of the total endogeneous Zn loss was contributed by the pancreas.

The quantity of fecal Zn varies with the dietary intake and the Zn status of the animal (Georgievskii et al., 1982; Kirchgessner and Weigand 1983). Feaster et al., (1954) reported a deminished Zn secretion via bile and pancreatic juice in Zn-depleted calves and goats; they observed that when radioactive Zn was administered orally to steers, 70% and 0.25% of the dose was recovered from the feces and urine respectively. When given intravenously, 20% and 0.25% appeared in the feces and in the urine respectively. The amount of Zn excreted with the urine or with sweat in farm animals is insignificant (Georgieveskii et al., 1982).

Bioavailability of Zinc and Factors Affecting Bioavailability

Underwood (1977) and Kirchgessner and Weigand (1983) have suggested that the the uptake of Zn from the intestinal lumen and subsequent transfer to the serosal side was influenced by the following factors:

1. dietary organic and inorganic constituents;
2. the level of Zn in the diet;
3. the Zn status of the animal and
4. the physiological state of the animal.

The content of Zn in the ration affects its absorption. A higher percentage of dietary Zn is absorbed when dietary Zn is low or subnormal (Miller 1969; Miller et al., 1968). As the dietary Zn level increases, the efficiency of absorption decreases (Miller 1969; Miller et al., 1965). Ruminants have a higher relative absorption than monogastric animals (Georgievskii et al., 1982).

The Zn status of the animal affects Zn absorption. Zinc-deficient animals absorbed a higher percentage of administered Zn (Miller, 1969). Miller et al., (1967) reported that Zn deficient calves had a net absorption as high as 80% of an oral Zn dose. Endogeneous fecal Zn was low during Zn deficiency (Miller, 1968, 1969). The absorption and retention of Zn was inversely related to the age of the animal (Weiegand and Kirchgessner 1980). Younger calves absorbed a high-

er percentage of dietary Zn than older calves (Miller et al., 1965 and 1968). Differences in the efficiency of Zn absorption were not observed when lactating cows were compared with two and six months old calves (Stake et al., 1975). Studies with rats showed a marked decline in the intestinal absorptive capacity for Zn after weaning. This decrease was attributed to the termination of the ability of the intestinal mucosa to take up immunoglobulins and other proteins by pinocytosis, a process which is no longer possible after 18 days of age (Kirchgessner and Weigand, 1983).

The requirements for Zn is greatly increased during pregnancy and lactation in most species of farm animals (Kirchgessner and Weingand 1983). During pregnancy, Zn is required for foetal growth. Additional Zn may be deposited in extrauterine tissues of the maternal body because of pronounced anabolism during pregnancy. This increased requirement has been reported to improve the efficiency of intestinal Zn absorption. Studies with swine (Kirchgessner and Weigand, 1983) have shown marked improved apparent absorption and retention of Zn during the last third of pregnancy. Postnatally, Zn is required for milk production.

The chemical form of Zn has little or no effect on the absorption of Zn (Kirchgessner and Weigand 1983). Seale and Heaton (1983) reported that the uptake of Zn as inorganic salts in simple buffered media varied: $ZnSO_4$ > $ZnCl_2$ > $Zn_3(PO_4)_2$. Edward (1959) found no differences in the extent of

absorption of Cu as the oxide, carbonate, sulphate or metal. Zinc in the sulphate form and Franklinite (oxide of Zn, Fe, and Mn) was largely unabsorbable by chicks.

Many organic substances also influence Zn bioavailability. Picolinic acid, secreted into the intestinal lumen by the pancreas facilitates the absorption of many bivalent cations including zinc (Evans 1980). This acid is also present in human milk and at a lower concentration in bovine milk and muscle tissue (Evans 1980).

In monogastric animals, phytate is considered to be the primary factor responsible for the lower availability of Zn from plant proteins and cereal products (O'Dell et al., 1972 Davis and Olpin 1979; Hardie-Muncy and Rasmussen, 1979). Phytate renders Zn unavailable by forming water-soluble complexes with it. The effect of phytate is potentiated by the presence of high levels of calcium in the intestinal lumen (Hoekstra, 1964; Byrd and Matrone, 1965). The effect of soybean protein or sesame meal in reducing Zn absorption is due to the high phytic acid content.

Phytic acid has little effect on Zn absorption in ruminants (Kirchgessner and Weigand 1983) as phytates are readily metabolized by the microflora in the rumen. Zinc absorption was depressed when isolated soybean protein diet was delivered to the abomasum of calves. There was increased absorption when the same diet was allowed to go through the

rumen (Miller 1967). Phytic acid added to a purified diet did not materially affect a Zn deficiency in lambs (Mills et al., 1967).

Amino acids such as histidine and cystine facilitate intestinal uptake and transmucosal transport of Zn when present in a high molar ratio relative to Zn (Evans and LeBlanc 1976). Amino acid complexation with Zn also facilitates uptake of Zn into cells from blood.

Trace elements interactions and their effect on the utilization of copper and zinc

Iron (Fe), cadimum (Cd), manganese (Mn), mercury (Hg), lead (Pb), silver (Ag), nickel (Ni), molybdenum (Mo), and sulphur (S) influence the absorption and metabolism of Cu and Zn within the body (ARC, 1980; Mills, 1979; Hansard, 1983; Davis, 1980; Nielsen, 1980; Suttle, 1974; Ptering, 1980; Underwood, 1977). Copper influences the utilization of Zn and vice versa.

Both Cu and Zn are members of the family of elements known as the first transition series: elements 21 to 30 of the periodic table which comprises scandium, titanium, vanadium, chromium, Mn, Fe, Co, Ni, Cu and Zn (Nielsen, 1980). These elements differ essentially only in the number of electrons in the 3d shell, and have many physical and chemi-

cal properties in common. Because of their chemical similarity these elements would be expected to compete for various binding sites within specific metabolic systems (Mills 1979; Evans, 1973).

Mills (1979), outlined five different types of antagonistic interactions among trace elements: 1. Reactions that decrease the solubility of the element in the gastrointestinal tract thereby preventing its absorption e.g. (Phosphate-Fe, sulphide-Cu, phytate-Zn, and thiomolybdate-Cu). 2. Antagonistic interactions occuring at sites involved in transport, storage, or excretion of the element or preventing its incorporation into functional sites (e.g. Sulphate-Molybdate, Sulphate-Selenite, Tungstate-Molybdate). 3. Reactions of the antagonist with proteins which increase their affinity for the element (e.g. Thiomolybdate- Protein complex-Cu antagonism). 4. Synthesis de novo, induced by high concentration of antagonist, of sulphydryl-rich proteins having relatively non-selective affinity for heavy metals (e.g. Cd-Zn antagonism). 5. Secondary effects arising from an interdependent involvement of one element in metabolism of a second (e.g. secondary Fe deficiency resulting from loss of Cu-dependent ferroxidase I (caeruloplasmin) activity and subsequent failure to mobilize stored ferritin Fe).

The gastrointestinal tract is a major site for the antagonistic interactions between Cu and Zn. Starcher, (1969) identified a single metal binding protein in chick duodenum

that bound Cu as well as Cd and Zn. Van Campen and Scaife (1967), suggested that Zn interacts with Cu at a site in or on the intestinal mucosa. Evans et al., (1970) isolated and purified metallothionein from bovine duodenum and demonstrated that both Zn and Cd could displace Cu from sulphydryl binding sites on the metallothionein protein.

High dietary Zn induced the synthesis of intestinal thionein which bound both Zn and Cu but has a higher affinity for Cu than Zn (Ogiso et al 1974). High dietary Zn reduced Cu absorption in dairy calves, sheep, Japanese quail, and horses (Ivan and Grieve 1976; Bremner et al., 1976; Hamilton et al., 1979).

The protective effect of Zn against Cu toxicosis in sheep was reported by Bremner et al., (1976). Liver Cu concentrations were reduced by up to 40% in the Zn supplemented animals, with a concommitant reduction of liver damage in the early stages of the experiment. Damage was assessed through measurement of plasma aminotransferase.

Iron is transported from the intestinal mucosa by combining with the protein, transferrin. El-Shobaki and Rummel (1979) suggested that Cu was preferentially bound to transferrin in the mucosa such that when Fe and Cu were administered in excess Fe absorption was inhibited because of the affinity of Cu for transferrin. Suttle and Mills (1966b) reported that the addition of 500 mg Zn or 750 mg Fe per kg/DM

to pig rations containing 750 ppm Cu eliminated the toxic effects of Cu and also brought serum Cu and aspartate transaminase values to normal levels. In another experiment, El-Shobaki and Rummel (1979) reported that the addition of 150 mg Zn and 150 mg Fe to rations containing 250 mg Cu/kg DM or more eliminated all signs of the Cu toxicosis that developed when the Zn and Fe supplements were not added. They concluded that the signs of toxicity that developed in pigs given 250 mg Cu/kg DM were due to interference of Cu with Zn and Fe utilization.

Grant-Frost and Underwood (1958) suggested a three way interaction involving Cu, Fe and Zn. High levels of Zn by depressing Cu or causing a functional deficiency of Cu, would result in poor utilization of Fe. Zinc also competes with Fe for binding sites on transferrin (El-Shobaki and Rummel, 1979). High dietary Zn influences Fe metabolism in two ways 1. Zn may affect ferritin (Fe storage protein) such that the incorporation of Fe into or release from ferritin is impaired. 2. Zinc may impair Fe absorption and limit the storage of Fe as ferritin (Davis, 1980). A high level of Zn has been reported to shorten the life span of the red blood cells, resulting in a faster turnover of Fe (Settlemire and Matrone 1967).

The apparent interaction of Cu, Zn and Fe at the intestinal level is influenced by several factors including the presence of organic molecules such as amino acids, picolinic

acid, and phytate. Other elements such as Ca that influence the pH of the digestive tract also influence the availability of Cu and Zn.

Whanger and Weswig (1970) reported that Cd, Ag and Zn antagonize Cu metabolism within the hepatic cell. They suggested that these elements inhibit caeruloplasmin activity by preventing Cu from inducing the apocaeruloplasmin molecule or by being incorporated into caeruloplasmin in place of Cu, or a combination of both mechanisms. Zinc can displace Cu from Cu-dependent enzymes such as metallothionein and caeruloplasmin (Cunnae, 1982).

High concentrations of Zn, Co, Ni and Fe have been reported to inhibit the oxidation of N'N-dimethyl-P-phenylenediamine by caeruloplasmin (Curzor 1960). These elements inhibit the oxidization of ferrous Fe by caeruloplasmin (Starches 1969). Zinc, Ni and Co inhibit ferroxidase activity as a result of competition with either Cu or substrate for distinct metal binding sites on the native protein (Hubert and Frieden 1970).

Evans et al., (1979) reported that both Cd and Zn competed with Cu for sulphydryl binding sites on metallothionein from bovine liver. They suggested that the interaction between Cu and other chemically similar transition elements results in part from competition for common binding sites on the hepatic storage protein, metallothionein.

Matrone (1974) suggested that the basis for the competitive interaction of certain essential elements arose from the similarities in electron distribution in the outer orbitals of the ions. Each of Cu, Cd and Zn has a 4s and three 4p orbitals vacant for the formation of coordinate bonds in a tetrahedral (sp^3) array. Thus it has been fully established that a competitive trace element antagonism may be exhibited when cations or anions have similar electron distribution in their outer orbitals and when ionic radii are not greatly dissimilar. For example cuprous and cupric ions have a preferred coordination number of four and form tetrahedral and square coplanar complexes respectively. Nickel can possess inner orbital bonding in which the preferred cordination number is four and form either tetrahedral or square complexes. It would appear that when Ni is present in biological systems with a coordination of four, Ni and Cu would have similar chemical parameters leading to a competitive nickel-copper interaction. Copper prevented blindness caused by excessive nickel in sheep (Roshjakou et al., 1972). Dietary Ni supplementation decreased lung and spleen Cu (Schroeder et al., 1974) and alleviated growth retardation and depressive hematocrit and hemoglobin due to a Cu deficiency (Spears et al., 1977).

In ruminants, both organic and inorganic sulphur have a synergistic effect on the antagonistic action of Mo upon the utilization of dietary Cu (Mills 1979; Bremner and Young

1978; Smith et al 1968; Mills and Bremner 1980). Molybdenum in the presence of inorganic sulphur modifies Cu retention by reducing its absorption and increasing its excretion (Marcilese et al., 1969). Dowdy and Matrone (1968a, 1968) suggested that Mo alters Cu metabolism through the formation of a Cu-Mo complex that renders Cu unavailable for cellular metabolism. The effect of Mo and S in reducing or increasing the Cu status of the animal depends on the intake of these elements relative to that of Cu. Chronic Cu poisoning has been reported in sheep consuming diets with moderate Cu and very low levels of molybdenum and sulphur (Underwood, 1977; Dick et al., 1975).

Studies with rats (Kline et al., 1973) have shown that sulphur can aggravate or ameliorate the toxic effect of Mo upon Cu depending on the Cu status of the animal. High dietary intakes of Mo and S by pigs did not show significant reductions in tissue Cu levels even at levels of 1500 mgMo/kg DM which substantially depressed growth and reduced plasma clearance of injected Cu.

Molybdenum exerts a marked effect upon Cu utilization (Fig.1) in situations which favour the formation of oxythiomolylbdate $[MoO_{4-n}S_n]^{2-}$ or tetrathiomolybdates $[MoS_4]^{2-}$ within the digestive tract.(Dick et al., 1975; Mills et al., 1978). The formation of these complexes in the digestive tract arises as a result of the reaction of molybdate (Fig.1) with free sulphur, either generated in the rumen by

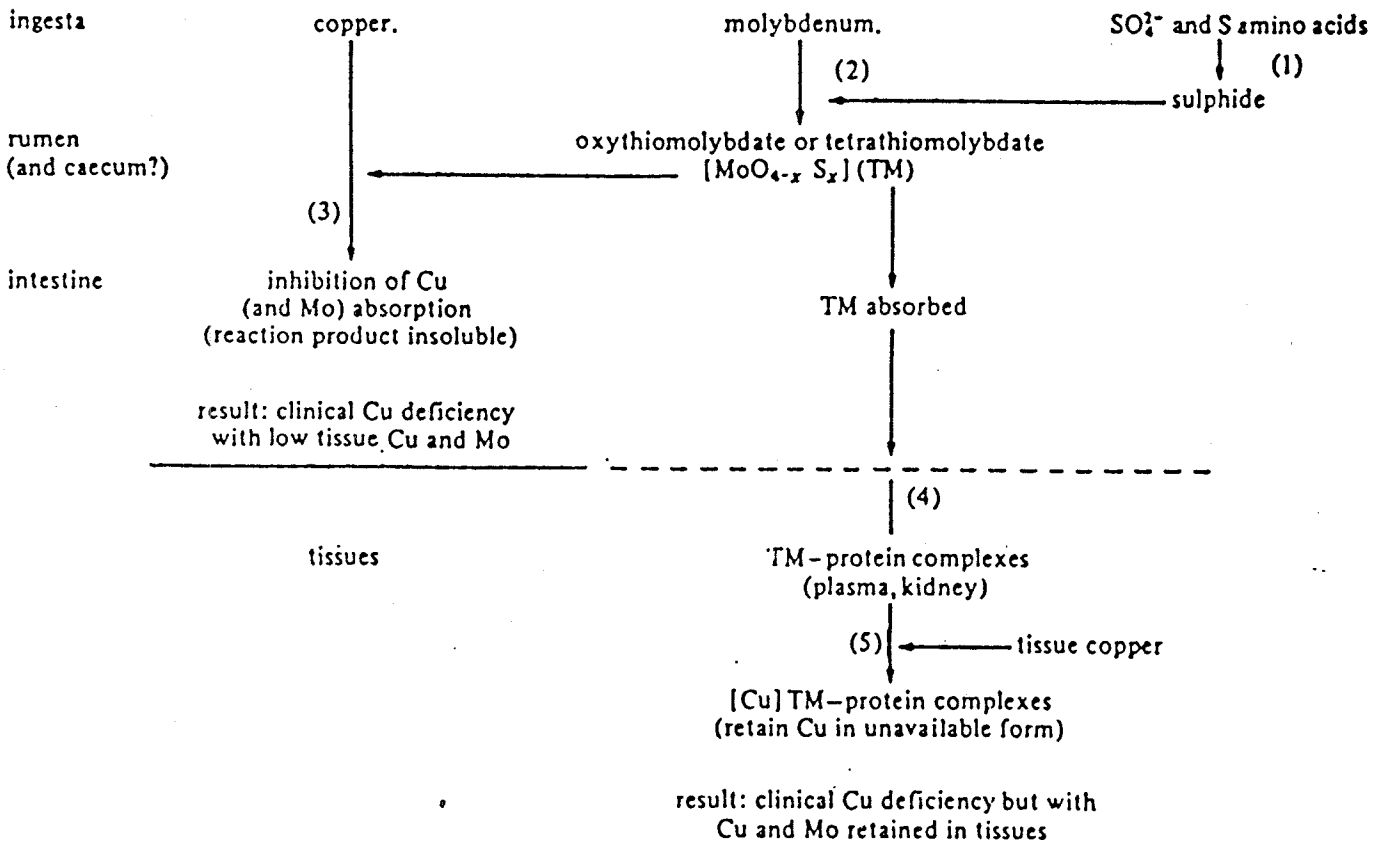


FIGURE 1 Mechanisms involved in the inhibitory action of dietary molybdenum and S upon Cu utilization by ruminants. Sulphide generation (1) in the rumen and the subsequent reaction of this with molybdenum (2) yields oxythiomolybdate or tetrathiomolybdate (TM). Reaction of TM with Cu in the gastrointestinal tract (3) prevents Cu (and Mo) absorption. Alternatively, if TM is present in excess it is absorbed (4) and in association with specific proteins (not yet characterized) reacts with Cu to induce a systemic deficiency of Cu.

Adapted from Mills (1979)

reduction of dietary sulphate or by degradation of S amino acids (Mills 1979). Within the digestive tract, dietary Cu will react with thiomolybdates to form its Cu derivative thereby preventing the absorption of both Cu and Mo (Mills 1979). On the other hand, excess thiomolybdate may be absorbed into the circulation where it reacts with plasma albumin or protein of similar molecular mass or possibly with other tissue proteins to form a stable complex (Mills 1979).

Huisingh et al., (1973) proposed two additional routes by which Cu may become unavailable. Copper may interact with molybdate to form a biologically unavailable Cu-Mo complex called cupric molybdate which is absorbed, transported and excreted as a unit making both Cu and Mo less available. Insoluble cupric sulfide may be formed in the rumen, intestine, or tissues.

Mills et al., (1976) reported that the presence of a Mo-containing fraction in the blood stream caused a rise in blood Cu even though biochemical and clinical indications of a Cu deficiency eventually developed in the animal. The signs of Cu deficiency were prevented by parenteral injection of Cu. This parenteral administration did not inhibit the absorption of thiomolybdate nor the increase in tissue Mo and blood Cu content that resulted from its presence. Mills (1979), Bremner and Young (1978), Mills et al., (1978), Smith et al., (1968), Mills and Bremner (1980) , Marcilese et al., (1969) and Dowdy and Matrone (1968) have

confirmed that the interference of Mo on Cu metabolism was not limited to that found in the gastrointestinal tract.

Insoluble cupric sulphide has been reported to accumulate in molybdenotic animals as a consequence of the depressed sulfide oxidase activity (Halverson et al., 1960; Spais et al., 1968). Copper is needed for the activity of sulfide oxidase, which is inhibited by Mo. When dietary Cu becomes unavailable, the inhibitory effect of Mo on sulfide oxidase leads to the accumulation of insoluble cupric sulfide and Mo in tissues (Underwood, 1977).

Copper Deficiency in Ruminants

Copper deficiency in ruminants can either be a primary (absolute) or a secondary (conditioned) deficiency (Underwood, 1977; Mills, 1979; Hansard, 1983). Primary Cu deficiency results from low or inadequate intake of the metal relative to the animal requirement. In a conditioned Cu deficiency, the dietary intake of the metal may be adequate but its absorption and utilization are impaired by elevated dietary levels of Mo and /or sulphur (Underwood, 1977). Copper deficiency due to Mo has been reported from such varied areas as Australia, Britain, Canada, New Zealand, Tanzania and the U.S.A (Cunningham, 1950; Cunningham et al., 1953; Underwood, 1962; Givens and Hopkins, 1978; Merry et al.,

1983). The severity and extent of Cu deficiency varies with the species, age, and the length of time the animal is exposed to the deficiency situation (Mills et al., 1967; Underwood, 1977; Mills, 1979; Smart et al., 1981).

Depending on species, Cu deficiency can lead to anemia, bone and skeletal disorders, depigmentation of hair and wool, impaired keratinization of hair and wool, diarrhoea (scouring) of cattle, cardiovascular disorders, central nervous disorders and infertility (Underwood, 1977).

Anemia is a common expression of Cu deficieny in all species of farm animals affected by severe or prolonged Cu deficiency (Underwood, 1977). The anaemia in young cattle is microcytic normochromic (Smart et al., 1981). In ewes and cattle it is hypochromic and microcytic. Copper is essential for the production and maintenance of the integrity of erythrocytes in the circulation. Normal haematopoiesis cannot occur in mammals when the blood Cu levels fall from 0.80-1.20 to 0.10-0.12 ug Cu/ml (Underwood, 1977).

Caeruloplasmin, a Cu-containing metalloenzyme is essential for the synthesis of hemoglobin and other Fe-containing proteins (Frieden, 1968). Caeruloplasmin is essential for the synthesis of Fe(III) transferrin, the transport vehicle for Fe. It is the Fe in Fe(III) transferrin that is donated directly to the developing reticulocyte in the bone marrow (Freiden, 1968).

In Cu deficiency, Fe absorption is unaffected but release of Fe into plasma is impaired. Release from liver parenchymal cells is also reduced and mobilization of Fe from the reticulo-endothelial system into plasma is also lowered in Cu deficiency.

Cattle and sheep grazing Cu-deficient pastures developed bone defects characterized by spontaneous fractures (Bull, 1980). Similar changes have been reported for Cu-deficient foals, chicks, pigs and dogs (Marcilese et al., 1969; Suttle et al., 1972; Bull, 1980; Smith et al., 1975). Copper is essential for the activity of amine oxidase or lysyl oxidase, a Cu-containing metalloprotein involved in collagen synthesis (Suttle and Angus, 1976). The structural integrity of collagen depends on cross-linking between collagen precursors which is believed to require the action of lysyl oxidase to convert lysyl residues into the ϵ-aldehyde products thought to form the cross-linkages (Rucker et al., 1969). In Cu-deficient tissues this conversion is impaired, cross-linking is prevented and the result is fragility and loss of strength in bone collagen (Irwin et al., 1974; Smith et al., 1975; Mills et al., 1976). Impaired collagen formation results in defective bone formation.

Depigmentation of hair and wool is a common clinical symptom of Cu-deficiency in most farm animals except pigs (Underwood, 1977). The condition is also described as achromotrichia (the absence or loss of melanin granules).

The condition is characterized by a failure in pigment production in black-wooled sheep and greying of the hair around the eyes in cattle. Phenyl oxidase, a Cu-containing enzyme is needed to catalyze the synthesis of melanin from L-tyrosinase at the wool or hair follicle. When Cu is deficient the pigment for black is not incorporated into the follicle. This then results in a whitening of hair or wool (Bull, 1981; Underwood, 1981). The color of hair produced due to failure in the synthesis of melanin depends on the original color of the animal's hair; white hair usually turns rusty yellow, while black hair becomes reddish-brown (Underwood, 1981).

Impaired keratinization of hair and wool due to a Cu-deficiency has also been reported in sheep, cattle and other species (Bull, 1980). Copper-deficient sheep failed to impart crimp in wool fibres. This results in almost straight hair-like fibre called 'stringy' or "steely" wool. The tensile strength of steely wool is reduced, its elastic properties are abnormal and it tends to set permanently when stretched (Underwood,1981). Similar hair changes have been observed in humans suffering from Menke's Kinky Hair Syndrome which is an abnormal error of Cu metabolism inherited as an X-linked recessive trait (Dekaban and Steusing, 1974). The physical properties of wool and hair are dependent on the presence of disulfide groups which provide cross-links or bonding of keratin and on the alignment or orientation of

the long chain fibrillae (Underwood, 1981). Cu is required for the formation or incorporation of disulphide groups during keratin synthesis (Gallagher, 1979).

Diarrhea (scouring) has been reported in cattle receiving high dietary Mo relative to Cu (Underwood, 1981) and in Cu deficient cattle (Mills et al.,1976; Suttle, 1975). The condition is usually associated with low blood Cu (Alcroft and Parker, 1949; Leigh, 1975). Fell et al., (1975) and Mills et al., (1976) reported reduced cytochrome activity and extensive mitochondrial damage in the small intestine of Cu deficient cattle. There was also mucosal atrophy, elongation of crypts and hyperplasia of goblet cells. The condition was reversed when Cu was added to the diet.

Cardiovascular disorders resulting from a Cu deficiency was first reported in Western Australia where the disease was described as "falling" disease (Underwood, 1977; Prasad, 1980; Hurley, 1981). The lesions included acute atrophy of the myocardium with replacement fibrosis (Underwood, 1977). Cytochrome oxidase, a Cu-containing metalloenzyme is required by the heart to maintain its oxidative activity. The level of this enzyme is low in the myocardium of Cu-deficient animals. Cytochrome oxidase is the principal terminal oxidase in all animals and is involved in the oxidation of intermediate energy carriers. All energy in animals is derived from oxidative reactions; hence, a deficiency of the terminal oxidase will constitute a defect in energy metabolism (Mills et al., 1976, Fell et al., 1975).

Internal haemorrhage due to rupture of the heart, aorta and other large vessels has been reported in cattle (Leigh,1975), rats (O'Dell,1961), and guinea pigs (Leigh, 1975). Biosynthesis of elastin and collagen is decreased in Cu deficient animals and this sometimes leads to rupture of major blood vessels and replacement of myocardium tissue with fibrous tissue. Copper is present in the enzyme, lysyl oxidase, an enzyme that makes the cross-linkages between polypeptide chains in collagen and elastin. Animals deficient in Cu develop defective collagen molecules lacking cross-links with the result that the collagen and the elastin in the walls of major arteries become weakened and arteries tend to rupture (Underwood, 1977; Everson et al., 1967).

Central nervous disorders due to severe Cu deficiency have been reported in lambs, goats, pigs, guinea pigs and rats (Evans, 1973; Mills, 1979; Hurley, 1981). This disease has not been reported in cattle. In lambs the disease is commonly referred to as swayback or neonatal ataxia. Swayback in lambs is characterized by uncoordinated movement of the hind limbs, a stiff and staggering gait and swaying of the hind quarters. Lambs are often born dead or die shortly after birth. Those lambs that survive usually become paralyzed within a few days after birth (Underwood, 1981). The major effect of a Cu deficiency in the brain of ataxic lambs appears to be abnormal myelination which includes myelin aplasia, which is a faulty development of myelin rather than

excessive myelin degeneration. Frieden, (1968) suggested that swayback disease results from the degeneration of the sheath around the spinal cord. Lack of Cu impairs the animal's ability to synthesize the phospholipids that form the outer covering of the nerves. There was also a reduction in the activity of cytochrome oxidase in the large motor neurons of the red nucleus in brain. This low level of cytochrome oxidase activity may lead to tissue anorexia and insufficient production of ATP needed for synthesis of phospholipids which form the major components of myelin (Prasad, 1978; Hurley, 1981).

Reduced fertility and high incidence of fetal resorption and still births resulting from Cu deficiency has been reported in grazing cattle (Donaldson, 1964; Underwood, 1981). Copper deficiency causes anaemia and haemorrhages in the developing fetuses resulting in mortality due to lack of adequate synthesis of elastin (and collagen) for normal embryonic development (Bull, 1980).

Zinc Deficiency in Ruminants

Zinc deficiency has been experimentally produced in cattle (Miller and Miller, 1960), sheep (Ott et al., 1965b; Mills et al., 1967) and goats (Miller et al., 1964) using semisynthetic diets. Evidence of Zn deficiency in ruminants

under grazing conditions has been reported by Legg and Sears (1960), Haaranen and Hyppola (1961), Haaranen (1963), Pierson (1966) and Papasteriadis (1973).

Severe Zn deficiency in ruminants results in retardation of growth, reduced reproductive capacity, skeletal abnormalities, delayed wound healing, parakeratosis, deterioration of hair and wool texture followed by loss of hair and changes in activities of Zn-containing enzymes (Miller and Miller, 1960; Miller et al., 1964; Ott et al., 1965; Mills et al., 1967; Blackmon et al., 1967; Underwood and Somers, 1969; Smart et al., 1981; Hansard, 1983; Lamand, 1980, 1984).

Growth retardation and reduced weight gains are common expressions of Zn deficiency in most species, including man (Perry et al., 1968; Mills et al., 1969; Underwood and Somers, 1969; Hill and Miller, 1983). Abrupt cessation in weight gain and arrested growth within two weeks occurred in lambs and calves receiving diets containing 1.2 mg Cu/kg Dm (Underwood and Somers 1969). The effect of Zn deficiency on growth is thought to be partly due to reduced food consumption and partly due to impaired food utilization (Miller et al., 1968). Zinc is present in the bacterial cell wall where it contributes to stabilization of the interactions between the various components of other cells and possibly to binding of bacterial cells to particles or to other cells. Zinc is thought to be essential for the adherence of cellulolytic

rumen bacteria to feed fibre in the rumen (Zeherebtsov et al., 1972). Zinc increased in vitro and in vivo digestibility of fibre and microbial protein synthesis (Zeherebtsov et al., 1972).

Zinc is required for spermatogenesis and the development of the primary and secondary sex glands in male animals. It is essential for all phases of the reproductive processes in the female from estrous to parturition and lactation (Underwood and Somers, 1969; Underwood, 1977). Zinc deficiency caused decreased fertility and resulted in abnormal estrous behaviour in cows, and retarded testicular growth in bulls, goats and lambs (Miller et al., 1964, Pitts et al., 1966; Underwood and Somers, 1969; Miller, 1970). The exact role of Zn in reproduction is not well established. Impaired development of the secondary sex glands may be secondary to the inanition of Zn deficiency which could result in a reduced gonadotropin output and consequent fall in androgen production (Miller et al., 1966).

Abnormal skeletal development resulting from a Zn deficiency has been reported in sheep, cattle, and goats (Miller and Miller, 1960; Miller et al., 1964; Demertzis and Mills, 1973; Demertzis et al., 1978; Brown et al., 1978). Stiff gait, bowed legs, and soft swelling of the hocks, knees and fetlocks were due to an accumulation of fluids. Cows with severe Zn deficiency developed inflammatory lesions of the skin and bones of the hind limbs. The edematous swelling of

the coronets of the rear legs, infectious pododermatitis in cattle and foot rot disease in cattle and sheep have been reported to respond to Zn treatment (Demertzis and Mills 1973; Demertzis et al., 1978; Hidiroglou, 1980). In goats cracks in the skin of the coronary bands around the hooves was prevented by Zn treatment; male kids from Zn-deficient goats suffered from dwarfism (Miller et al., 1964; Hidiroglou, 1980). Gross abnormalities and other deformities including agenesis of the limbs, dorsal curvature of the spine, shortened and fused vertebrae, decreased osteoblastic activity, and reduced chondrogenesis associated with matrix has been reported in Zn deficient pigs and chickens (O'Dell et al., 1958; Blamberg et al., 1960).

Parakeratosis of the muzzle, scrotum, neck, ears and back of the hind limbs, bowing of the joints of the hind limbs, stiffness of the joints and swelling of the hocks often develop in calves during severe Zn deficiency (Miller and Miller, 1962). In sheep the quantity and the quality of the wool are adversely affected (Underwood, 1977). In horned lambs the normal structure of the horns often disappear from new horns and are ultimately shed and replaced by spongy out growths that continually haemorrhage (Miller and Miller, 1962).

Zinc deficiency has also been implicated in delayed wound healing (Pories and Strain, 1966). Zinc accelerated the process of wound healing in calves, man, and rats (Stanstead

and Rinaldi, 1968; Lavy, 1972; McClain et al., 1973; Rahmat et al., 1974). McClain et al., (1973) suggested the role of Zn in wound healing to be related to a heightened metabolic demand for this metal for collagen synthesis in the process of tissue repair. The migration, proliferation and maturation of the epidermis are partly controlled by peripatetic Zn. The failure of epithelial proliferation associated with Zn deficiency may be evidence of lack of catalysing Zn-containing enzymes consequent inability to synthesize RNA and DNA (Pories and Strain, 1966)

Changes in the blood and Zn enzyme activities in tissues have been reported in severe Zn deficiency (Paris and Vallee, 1970). The level of serum alkaline phosphatase and serum or plasma Zn are greatly reduced during Zn deficiency (Miller et al., 1965, 1968). Low plasma vitamin A values in the presence of adequate vitamin A in the diet have also been observed in Zn-deficient rats, pigs, goats, and lambs (Smith et al., 1976).

Supplementation with Copper

Supplementation of cattle with Cu can be achieved by one of several methods:

1. Top dressing of pastures with fertilizers containing Cu salts.
2. Incorporation of Cu salts in concentrate mixtures

complete mixed diets.

3. systematic dosing by drenching or offering licks or mineral mixtures containing Cu.
4. Addition of Cu salts to the drinking water, and
5. Administering injectable Cu preparations at specific intervals.

Dosing or drenching cattle at intervals with Cu provided temporary remedy in severely Cu deficient areas (Cunningham, 1950) Underwood, 1962; and Dewey, 1977). Molybdenum scours in cattle was controlled by a weekly dosage of 3.5 grams copper sulphate. In pregnant ewes, 1.5 grams of copper sulphate administered seven weeks prepartum was effective in preventing swayback in lambs (Cunningham, 1950). Delayed and subsequent development of ataxia in lambs was prevented by doses of 35 mg copper sulphate twice weekly from birth. Fergusson et al., (1938) reported that Mo induced scours in cattle was controlled by daily doses of 1 or 2 grams copper sulphate. Under conditions in Manitoba it was recommended that Cu deficient cattle should be drenched with 2 to 4 grams copper sulphate daily until complete recovery and there after animals should be drenched with 1 to 2 grams of the compound as long the animals remain exposed to Cu deficiency (Cunningham, 1950).

Top dressing or application of Cu-containing compounds to pastures not only raises the Cu content of the herbage to adequate levels, for cattle, but also increases herbage

yield (Cunningham and Perrin 1946; Underwood 1962). Cunningham and Perrin (1946) reported an increase in herbage Cu content from 4 to 6 mg/kg DM to approximately 10 mg/kg DM following application of 4.5 kg $CuSO_4$ per hectare of pasture land. In Australia, the application of 4.5 to 6.3 kg $CuSO_4$ or its equivalent in Cu ores per hectare of land increased herbage Cu content to adequate levels for cattle for several years (Underwood, 1962). Aerial application of Cu salts, to rough or semi-forested pasture also increased the Cu content in the herbage (Anderson and Cunningham, 1946). Top dressing method of supplementation is not very feasible in calcareous soils where high pH reduces Cu solubility and a reduction in Cu uptake by plants nor is it feasible when high levels of supplementation are required to counteract a molybdenum problem.

Under range conditions, animals can be provided with Cu in the form of salt licks containing 0.5 to 1.0% $CuSO_4$ or its equivalent in other Cu salts (Cunningham, 1950; Underwood, 1977). Where salt licks are provided, great care must be taken to ensure adequate intake of Cu and at the same time prevent Cu poisoning. Calves under the age of five to six months are known to consume less mineral supplement.

The biological availability of Cu from Cu-containing compounds should be considered before supplementation. Copper from cupric nitrate, cupric sulphate and cupric chloride was absorbed to similar extent by cattle; cupric carbonate was

poorly retained . Of these compounds, cupric sulphate was the most suitable source of Cu for cattle (Chapman and Bell, 1963).

Injectable Cu complexes and preparations are widely used in preventing and treating Cu deficiency in sheep and cattle. They are particularly effective under range conditions. The method has several advantages over other supplemental methods. The exact dosage received by each animal is known, the frequency and handling of animals is reduced and most important is the fact that the utilization of these injected compounds is not impaired by dietary constituents known to reduce absorption of Cu in the digestive tract. Thus, the biological availability of Cu in injected Cu supplements appears to be superior to that of oral supplements.

Several injectable Cu complexes are currently available and these include: (a) copper glycinate, (b) copper-calcium edetate (CuCaEDTA), (c) copper methionate, (d) $CuSO_4$ and (e) copper as diethylamine oxyquinoline sulphonate. Many of these products differ in the local tissue reactions which they induce, in the extent to which the are taken up and retained by hepatic and extrahepatic tissues and their acute toxicity (Suttle, 1981). The choice of injectable Cu supplement should ideally be made by balancing effectiveness of disease control against risks of local and general toxicity.

Injectable Cu complexes are administered by one of three routes: (a) Subcutaneously: in front of the brisket, that is into the loose fold of the dewlap, (b) Intramuscularly: in the hind quarter into either the gluteal or the biceps femoris and semitendinosus muscle; into the fore quarter into the triceps and supraspintous muscle; or in the neck into the trapezius muscle. (c) Intravenously: into the jugular vein (Alcroft and Uvarov, 1959). A subcutaneous route of administration is preferable in beef cattle in order to avoid abscesses following intramuscular injections. Alcroft and Uvarov (1959) reported greater increases in hepatic Cu concentration after intravenous injections of $CuSO_4$ and observed that the rate of fall or depletion of liver Cu was more rapid after intravenous injection than after subcutaneous or intramuscular injections. Uptake of Cu from the site of injection was slowest when intramuscular injections were administered.

Responses of sheep and cattle to injectable Cu supplementation has been investigated (MacPherson et. al, 1979, Alexander et. al, 1967, Steacy et al 1983, Mahmoud and Ford, 1981; Miltimore et al., 1964; Boila et al., 1984a). MacPherson et al., (1979) reported significant increases in mean live weight gains of Cu treated calves ranging from 19.9 to 34.3 kg per head relative to untreated animals. Alcroft and Uvarov, (1959) reported that parenteral administration of Cu glycinate to the dam within few months of calving increased the Cu-status of the newborn calves.

Injectable Cu supplements are administered three to six months intervals and at levels of 120 and 240 mg active Cu for calves and adult cattle respectively, and at 30 to 40 mg for adult sheep. Calves under 140 kg body weight should be treated with 60 mg Cu and very young calves with 30 mg (Allcroft and Uvarov, 1959; Underwood, 1962; Smith et al., 1975).

Where an induced Cu deficiency is severe, cows should be injected with Cu supplements one month prepartum and calves from such cows should be injected with Cu supplements at three months of age and there after at four to six months intervals. In sheep, administration of 40 to 50 mg Cu in mid pregnancy or immediately prior to mating was effective in preventing swayback in offspring lambs (Alcroft and Uvarov, 1959; Habel, 1974; Hemingway et. al., 1970; Suttle, 1974).

Boila et al., (1984a) evaluated Cu methionate, Cu calcium edetate, and two forms of Cu glycinate (one from the U.S.A and the other from New Zealand) as injectable sources of Cu for grazing cattle. All four preparations were effective in raising serum Cu. Weight gains were not influenced by administration of any of the supplements. Copper methionate produced the most severe reaction at the site of injection. This was followed by the two forms of Cu glycinate with Cu-CaEDTA producing the least severe reaction at the site of injection. From the available information it appears that careful use of Cu supplements could serve as a means of en-

suring adequate intake of Cu by animals under range situations.

Supplementation with Zinc

Limited information is currently available on the supplementation of Zn for grazing cattle; this lack of much information may be attributed to the absence in many areas of clearly defined symptoms of Zn deficiency in cattle under grazing conditions. Supplementation with Zn in grazing ruminants has been shown to produce variable responses in body weights and reproduction (Legg and Sears, 1960; Haaranen and Hyppola, 1961; Dynna and Harve, 1963; Ott et al., 1966; Lamand, 1980; Mayland et al 1980; Pond, 1983).

Zinc in the metal form as well as Zn-containing compounds such as the oxide, carbonate, sulphate and chloride have been successfully used as sources of Zn for cattle (Legg and Sears, 1960; Miller, 1970; Mayland et al., 1980; Lamand, 1980).

The basic procedures applied to Cu supplementation may also be applied for Zn. The type of supplementation used will however, depend upon such factors as number of animals involved, severity of the Zn deficiency, levels of other nutrients in the forage known to influence Zn utilization and other environmental factors.

MATERIALS AND METHOD

A grazing trial was carried out at the Narcisse community pasture from June 13 to October 5, 1983. The Narcisse Community pasture is administered by the Prairie Farm Farm Rehabilitation Administration (P.F.R.A).

Description of the Study Area

The Narcisse Community Pasture, located about 121 kilometers North of Winnipeg, was establihed in 1968 by the Faculty of Agriculture, University of Manitoba, P.F.R.A and the Manitoba Department of Agriculture (Clark and Ingalls, 1970).

The soil type of this area is Inwood-Meleb, which covers 428,700 hectares in the southern portion of the Interlake district. The characteristic features of this soil type include an extensive area of low ridges and swale topography, being very stony and subject to water-logging during wet seasons. Virgin areas of this soil type are covered by a dense growth of small aspen. The soils are also known to become calcareous at the surface when cultivated and are therefore unsuitable for grain crop production (Clark and Ingalls, 1970).

Eight paddocks (2.4 hectares per paddock) covering an area of 19 hectares were allocated to this study. In 1968, these paddocks were seeded with mixtures of grasses and legumes based on plot studies and green house studies carried out on the soil type by Clark (1968). The seeded mixtures and the actual amounts seeded are shown on Table 2. However, over the years, different species have become established on the paddocks. The botanical composition of the eight paddocks as determined in August 1983 is listed on Table 3.

Animals and Management

Sixty-eight yearling beef steers (40 Herefords and 28 Angus-Charolais -North Devon Crosses) were available for this grazing trial. Liver tissue and blood samples were obtained from all animals on May 10 1983. Blood serum was obtained from whole blood, placed in a plastic vials, capped and stored in a freezer for analysis of Cu and Zn. Liver tissues were analyzed for Cu and Zn.

Sixty steers (37 Herefords, Group I and 23 Angus-Charolais-North- Devon crosses Group II) from the original 68 were selected for the grazing trial. During their one-month pretreatment stay at the Glenlea Research station, the steers were fed alfalfa silage followed by grass hay (Table 4).

Table 2: Grasses and Legumes used in the seeding mixtures and the paddock to which they were sown.

Mixture #[1]	Kg/Acre	Paddock
#1		
Brome grass	2.0	3 & 7
Crested wheat grass	1.5	
Alfalfa	1.2	
#2		
Russian wild rye grass	2.4	4 & 8
Slender wheat grass	1.3	
Alfalfa	1.0	
#3		
Brome grass	1.7	2 & 6
Meadow fescue	1.0	
Creeping red fescue	1.0	
Alfalfa	1.0	
#4		
Reed canary grass	0.9	1 & 5
Timothy	0.5	
Birds Foot Trefoil	0.9	

SOURCE: CLARK AND INGALLS 1970.

Table 3: Botanical composition of the 8 paddocks+ as estimated August 16, 1983.

	FORAGE SPECIES#											
Paddock	AC	ALF	AS	BF	BFT	BP	BR	BT	CRF	CWG	RGG	SE.WHT	SW.CL
1					54.7			6.0	26.7		12.7		
5	20		2.7		42.6			3.4	42.6		6.6		
2	6.2	24.1		0.7			14.5		53.8				0.7
6		44.1		0.6			22.4		32.9				
3		36.8				2.1	9.7		44.4	4.9			
7		49.6					35.7		14.0	0.7			
4		54.1					4.5		41.4				
8	9.2	10.8				1.6	0.8		70.0				7.5

+ Expressed as percent of each species in each paddock.

ABBREVIATIONS

AC - White clover
ALF - Alfalfa
AS - Asike
BF - Meadow fescue
BFT - Birds foot trefoil
BP - Blue grass
BR - Brome
BT - Timothy
CRF - Creeping red fescue
CWG - Crested wheat grass
RCG - Reed Canary grass
SE.WHT - Slender wheat grass
SW.CL - Sweet clover.

Table 4: Nutrient composition of the alfalfa silage and grass hay fed to the steers during their stay at the Glenlea Research Station before the grazing trial.

Nutrient	Alfalfa Silage	Grass Hay
Dry matter (%)+	95.6	97.2
ADF (%)+	30.0	38.9
CP (%)+	23.5	9.3
Calcium #	12.0	4.8
Magnesium #	1.5	1.7
Phosphorus #	2.6	2.4
Sulphur #	1.2	1.8
Copper *	6.5	4.0
Zinc *	21.4	15.3
Molybdenum *	1.5	1.0
Iron *	179.4	46.8
Manganese *	40.3	65.4

+ Dry matter, ADF and CP are expressed as percent (All are expressed on dry basis)

Ca, Mg, P and S are expressed as g/kg dry matter

* Cu, Zn, Mo, Fe and Mn are expressed as mg/kg dry matter

The steers were assigned to the four treatment groups (Table 5) on the basis of liver Cu and body weights measured on May 10 1983.

Liver tissue and blood samples were obtained from all steers on June 10. Prolontex-Cu and Prolontex-Zn were administered on this date. One steer in treatment C, Group II became lame on that day and an untreated steer was brought in as a control animal. Thus there were 16,15,14, and 15 steers in treatments A, B, C and D respectively (Table 5). The steers were transported to the Narcisse pasture on June 13, 1983. Henceforth, June 13 is referred to as day 0 of the grazing trial.

The steers were pastured as one group of 60 animals in the eight paddocks with three paddocks opened every two weeks. Loose cobalt-iodized salt was the only supplement provided. Drinking water was available throughout the grazing trial. A combination squeeze chute and scale was stationed at the pasture for the duration of the grazing trial.

Injectable Preparations

The Prolontex-Cu and Prolontex-Zn used in this study were obtained from Roussel, Uclaf, Paris, France, through Hoechst, Canada limited. Prolontex, Cu and Zn are injectable preparations which are offered as metal or oxides in an oil suspension.

Table 5: The four treatment groups together with the number of steers from each of Group I and Group II and the total number of steers in each treatment.

Treatment	Number of steers From Each Group		Total Number of steers
	Group I	Group II	
A Control	9	7	16
B Prolontex–Cu	9	6	15
C Prolontex–Zn	10	4	14
D Prolontex–Cu and Prolontex–Zn	9	6	15

The trace elements are insoluble compounds in aqueous solution, not ionizable, and are slowly released after intramuscular injection, by a moderately inflammatory process, without external signs of reaction. Prolontex preparations are available in volumes of 10 ml in a 20 ml bottle, in order to obtain adequate mix of trace elements after vigorous shaking.

The Prolontex-Cu used in this study was cupric oxide of average granulometry of one to three microns with 12.5 mg active Cu per milliter in the suspension. The Prolontex-Zn, was zinc metal of average granulometry of one to two microns with a concentration of 60 mg active Zn per milliter in suspension. A washed and purified plant-oil is used as the suspension medium for both Prolontex-Cu and Prolontex-Zn.

Manufacturers recommendation (Table 6) were strictly followed. Both preparations were administered intramuscularly into the middle third of the neck. Copper was administered on the right side of the neck and Zn on the left. Sites of injections were examined for signs of swelling as a measure of a possible inflammatory reaction at the site of injection. Both Prolontex-Cu and Prolontex-Zn were given in 10 ml doses.

Sampling Procedure

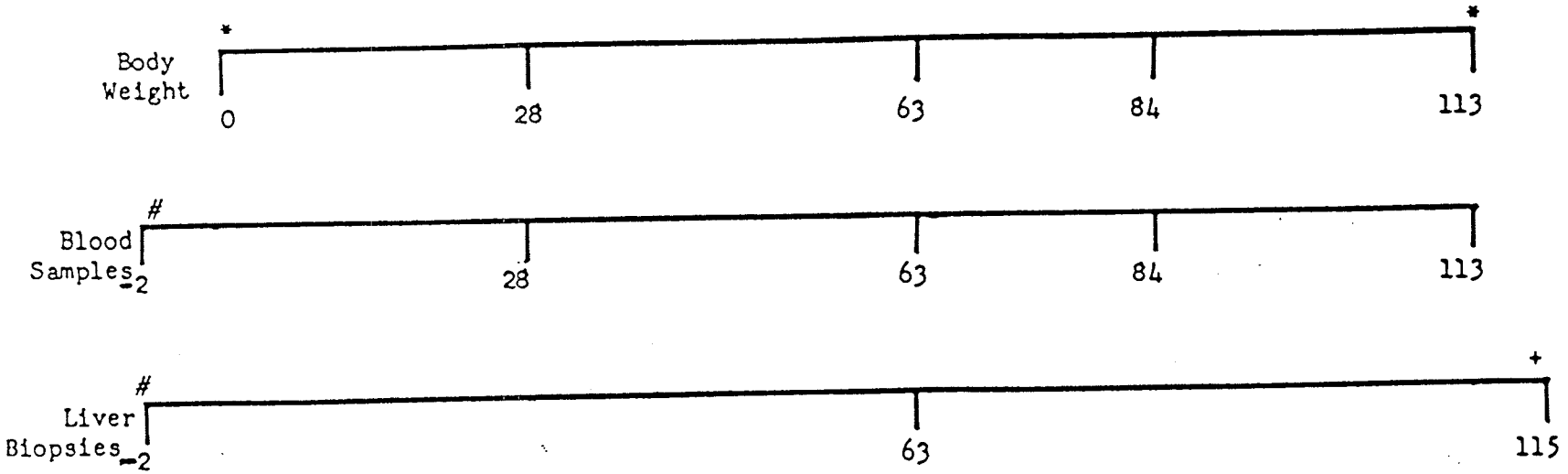
Body weights, liver tissue, and blood samples were obtained from all animals during the grazing trial (Fig.2).

Table 6: Recommended Doses of Prolontex-Cu and Prolontex-Zn for cattle at different stages of growth.

Preparation	Stage of Growth	Recommended Dose (ml)	Milligrams of Element in dose
Prolontex-Cu[1]	Adults	10	125
	Calves(100-250 kg)	5	62.5
	Calves(100kg)	3	37.5
Prolontex-Zn	Adults	10	600
	Calves (100-250)	5	300
	Calves (100kg)	3	180

1 Prolontex-Cu and Prolontex-Zn are trade marks for injectable Cu and Zn products manufactured by Roussel Uclaf, Paris, France.

Fig.2: Experimental design and sampling days for body weights, blood samples and liver tissues.



* Day 0 is June 13 1983, Day 113 is October 4 1983,

Liver tissue and blood samples were obtained 2 days before the start of grazing trial

+ The last sample of liver tissue was obtained two days after termination of grazing trial.

Body weights were measured on days 0, 28, 63, 84 and 113 of the grazing trial. Blood samples for serum Cu and Zn analysis were obtained from the jugular vein two days before the start of the grazing trial and on days 28,63,84 and 113 of the grazing trial. Blood serum was separated from whole blood within 12 hours after collection, placed in plastic vials, capped and stored in a freezer for later analysis.

Liver tissue was obtained two days before the start of the grazing trial and on days 63 and 115. Day 115 was two days after the termination of the grazing trial. This last liver tissue was taken at the Glenlea Research station. Liver tissue was taken by the method described by Chapman et al., (1963) and samples were placed in labelled plastic vials and stored for analysis of Cu and Zn.

Forage samples of alfalfa, grasses and birdsfoot trefoil were obtained from the grazed paddocks at intervals of 28 days beginning on day 15 of the grazing trial. Forage samples from each paddock were treated separately. All samples were dried to constant weight in an oven set at $60^{0}C$. Dried samples were ground using a Wiley hammer mill equipped with 1mm stainless steel sieve. Ground samples of each forage category, legume or grass from each paddock of each collection were analyzed for DM, ADF, CP, S, Ca, S, Mg, P, Cu, Zn, Mo, Fe and Mn.

Chemical analysis

Liver Tissue and Blood Serum

Liver and blood serum were prepared for analysis using a modification of the Thompson and Blanchflower, (1970) wet ashing procedure.

For liver tissues, 0.25g sample was placed in a clean dry weighed tarred vial. The vial was placed in an oven set at 35^{0}C and left to dry for 24 hours. The final dry weight of the sample was equal to the difference in weight between the weight of the dry sample and vial less the weight of the empty vial. Concentrations of both Cu and Zn in liver tissue were calculated on both wet and dry basis. The next step in the preparation of the samples was the addition of 3ml of nitric- perchloric acid mixture (4:1 by volume) and a small glass bead was dropped into the vial. Appropriate blanks were also included. The vials were placed on a drilled Aluminum heating block in a fume hood and left to stand over night for predigestion at room temperature. Complete digestion was attained the following day by heating the vials on the aluminum block until nitric oxide and nitrogen dioxide fumes were no longer produced, leaving a clear solution which was then evaporated by steadily raising the temperature of the heating block. The wet ashing procedure was complete when no visible liquid was in the vial. Vials were

then removed from the heating block and allowed to cool before adding 5 ml of 5% (vol.%) hydrochloric acid solution to each vial to dissolve the residue. The solution was aspirated directly into an Instrumentation Laboratory Model 551 atomic absorption spectrophotometer for determination of Cu and Zn.

Blood serum samples were also prepared by the wet ashing procedure. Two milliters of blood serum were measured into a vial and 3ml of nitric-perchloric acid mixture (4:1 by volume) was added. The period of predigestion at room temperature and the subsequent digestion on the hot plate was as described for liver tissue.

The concentrations of Cu or Zn were reported as mg/kg dry matter (DM) for liver tissue or as ug/ml for blood serum. Liver Cu and Zn were also calculated as mg/kg wet basis.

Forage samples

Forage samples were prepared by the modified wet ashing procedure described by Thompson and Blanchflower (1970). About 0.25g of ground forage sample was digested in 5ml of nitric-perchloric acid (4:1 by volume). After the completion of the wet ashing procedure 5ml of 5% (volume/volume %) hydrochloric acid was added to each vial to dissolve the residue. This solution was aspirated directly into the atomic absorption spectrophotometer for the determination of Cu,

Zn, Fe, and Mn. For the determination of Mg and Ca, 0.25g of the 5 ml solution was diluted to 10 ml using 2 ml of 5% (vollue by volume) lanthanum solution and 7.7 ml deionized distilled water. The final 10 ml solution has a concentration of 1% lanthanum. The lanthanum solution eliminates the interference of other elements in the atomic absorption determination of Mg and Ca. Phosphorus was determined colorimetrically (AOAC, 1980) using a Bausch and Lomb Spectronic 20 colorometer. Total sulphur was determined by the procedure described by Boila et al., (1984b).

Crude protein, acid detergent fibre and dry matter in forage samples were determined using standard laboratory methods (AOAC 1980).

FINISHING TRIAL

Following the termination of the grazing trial on October 4, 1983, the steers were transported to the Glenlea Research station on October 5, and liver tissues were obtained from all steers on October 6 (Day 115). The steers were then allowed to adjust to their new environment for two weeks before they were placed on a barley based finishing ration on October 18, 1983 (Day 0 of the finishing trial).

Animals and Management

The eleven heaviest steers of each treatment group (A, B, C and D) were selected for the finihing trial. The 44 steers were divided into eleven groups of four each. The first

pen consisted of the heaviest animal from each treatment group, and the second pen the next heaviest animal from each treatment group and so on until the eleven pens were filled. There was no Group I vs Group II separation. Two steers (one from treatment A and another another from treatment B died during the finishing trial. Statistical analysis was done only on data for 42 steers.

A barley-based diet (Tables 7a and 7b) was fed ad lib throughout the finishing trial. The steers were placed on the finishing ration on October 18,1983 and were fed this ration until January 5,1984, for a period of 79 days. Straw was provided as bedding.

Liver tissue was taken form all steers at the abattoir at the time of slaughter. These were wet ashed by the Thompson and Blanchflower (1970) method and analyzed for Cu and Zn.

Statistical analysis

For the grazing trial, there were 60 steers from two breed groups and four treatment groups. Animals were assigned to treatment groups on the basis of liver Cu (dry basis) and body weight measures obtained on May 10, 1983.

Treatment means and standard errors of means were calculated for body weight, serum Cu, serum Zn, liver Cu and liver Zn for each day samples were taken. Data were subjected to analysis of variance using split plot design with repeated

Table 7a. Chemical composition of the barley grain used in the finishing trial.

Nutrient	Concentration
DM (%)	97.9
ADF (%)	12.9
CP (%)	12.6
Ca#	0.62
Mg#	3.7
P#	1.3
S#	1.3
Cu+	4.3
Zn+	26.4
Mo+	1.5
Mn+	203.8
Fe+	29.0

Ca, Mg, P, and S are expressed in g/Kg DM

+ Cu, Zn, Mo, Mn and Fe are expressed in mg/Kg DM

ADF and CP are expressed on dry matter basis.

Table 7b. Composition of the finishing

Rolled barley	492.0 Kg
Cobalt-iodized salt	2.5 Kg
Limestone ($CaCo_3$)	3.5 Kg
*Wheat Middlings with Vitamin A	2.0 Kg

*Four grams of vitamin A palmitate were mixed with wheat middlings before being incorporated into the finish mix. One gram vitamin A palmitate contained 325,000 USP Units Vitamin A.

measures over time as described by Snedecor and Cochran, (1980).

The effect of Group on the response of steers to injectable-Cu and injectable Zn treatments was investigated. Data for both Group I and Group II steers were analyzed separately. Linear contrast analysis was performed on all analysis of variance whenever ICU or IZN treatment were significant or there was a significant interaction ($P<0.05$, $P<0.01$) between ICU and sampling day or IZN and sampling day for any of the parameters under study. Linear contrast analysis was reported significant at $P<0.10$, $P<0.05$ or $P<0.01$. Carcass weight data at the end of the finishing trial were analyzed using a 2 x 2 factorial design (Snedecor and Cochran, 1980).

Means and standard errors (SE) of means for Ca, Mg, P, S Cu, Zn, Mo, Fe, Mn, CP, ADF and DM in forages were calculated. Forages were divided into two categories viz, grasses and legumes, the latter being represented by alfalfa and birdsfoot trefoil.

RESULTS

The means for body weights, serum Cu, serum Zn, liver Cu and liver Zn for Group I and Group II steers in the four treatment groups measured on May 10, 1983 are listed in Appendix Table A1. Raw data for live weight, serum Cu, liver Cu, serum Zn and liver Zn for individual steers measured on May 10, during the grazing and finishing trials are listed in Appendix Table A2.

Raw means(± SE) and range for body weight, serum Cu, liver Cu, serum Zn and liver Zn for Group I and Group II during the grazing trial are reported in Appendix Tables A3 and A4 respectively. Means (± SE) for body weight, liver Cu, liver Zn and carcass weight for the finishing trial are reported in Appendix Table A5. Statistical analyses were performed on the raw data obtained during the grazing and finishing trials. For the data obtained during the grazing trial, statistical analysis was first done on a treatment basis across breed groups. As the two groups of steers used in this study belonged to two different breeds and also came from two different farms, the data were sorted out into two breed groups for analysis on a group basis. This was done in order to identify any responses to treatment that could be attributed to breed of steer.

The data for forage are reported in Appendix Table A6. Means (±SE) and range for Ca, Mg, P, S, Cu, Zn, Mo, Mn, Fe,

CP, ADF and DM are listed in Appendix Table A6. Means (±SE) for Cu, Zn, S and Mo in grasses and forages for the entire grazing trial are reported in Table 8.

Body Weights of Steers During the Grazing Trial

Body weights of all steers

Least square means for body weight of all steers are illustrated in Figs 3 and 4 which show that steers in all treatment groups gained weight throughout the grazing trial.

Body weights differed ($P<0.01$) between Group I and Group II steers (Appendix Table B1). There were no main effects ($P>0.05$) for either ICU (Fig.3) or IZN (Fig.4) on body weight of steers (Appendix Table B1). There was no interaction ($P>0.05$) between ICU and IZN for body weight.

Subplot analysis showed a significant effect ($P<0.01$) of sampling day upon body weight. There were significant interactions ($P<0.05$) between Group and sampling day and between IZN and sampling day (Fig.4) for body weight. There was no interaction between ICU and sampling day for body weight ($P>0.05$).

Linear contrast analysis of each sampling day relative to Day 0 was done to determine the effect of IZN on body weight of steers at each sampling day. At sampling days 84 and 113,

Table 8: Mean[+] Concentrations of copper, zinc, molybdenum and total sulphur in grasses, alfalfa and birds foot trefoil consumed by steers during the grazing trial.

Forage	Number of samples	NUTRIENT			
		Copper*	Zinc	Molybdenum	Sulphur[#]
Grasses	31	4.4 (0.2)	15.7 (0.5)	1.2 (0.1)	1.8 (0.1)
Alfalfa	17	6.9 (0.2)	16.5 (1.5)	1.2 (0.1)	2.0 (0.2)
Birds foot trefoil	7	5.0 (0.8)	15.0 (3.4)	1.1 (0.2)	1.4 (0.3)

[+]Mean followed by standard errors in parenthesis.

[*]Copper, zinc and molybdenum are expressed in mg/kg dry matter forage.

[#]Total sulphur is expressed in g/kg dry matter forage.

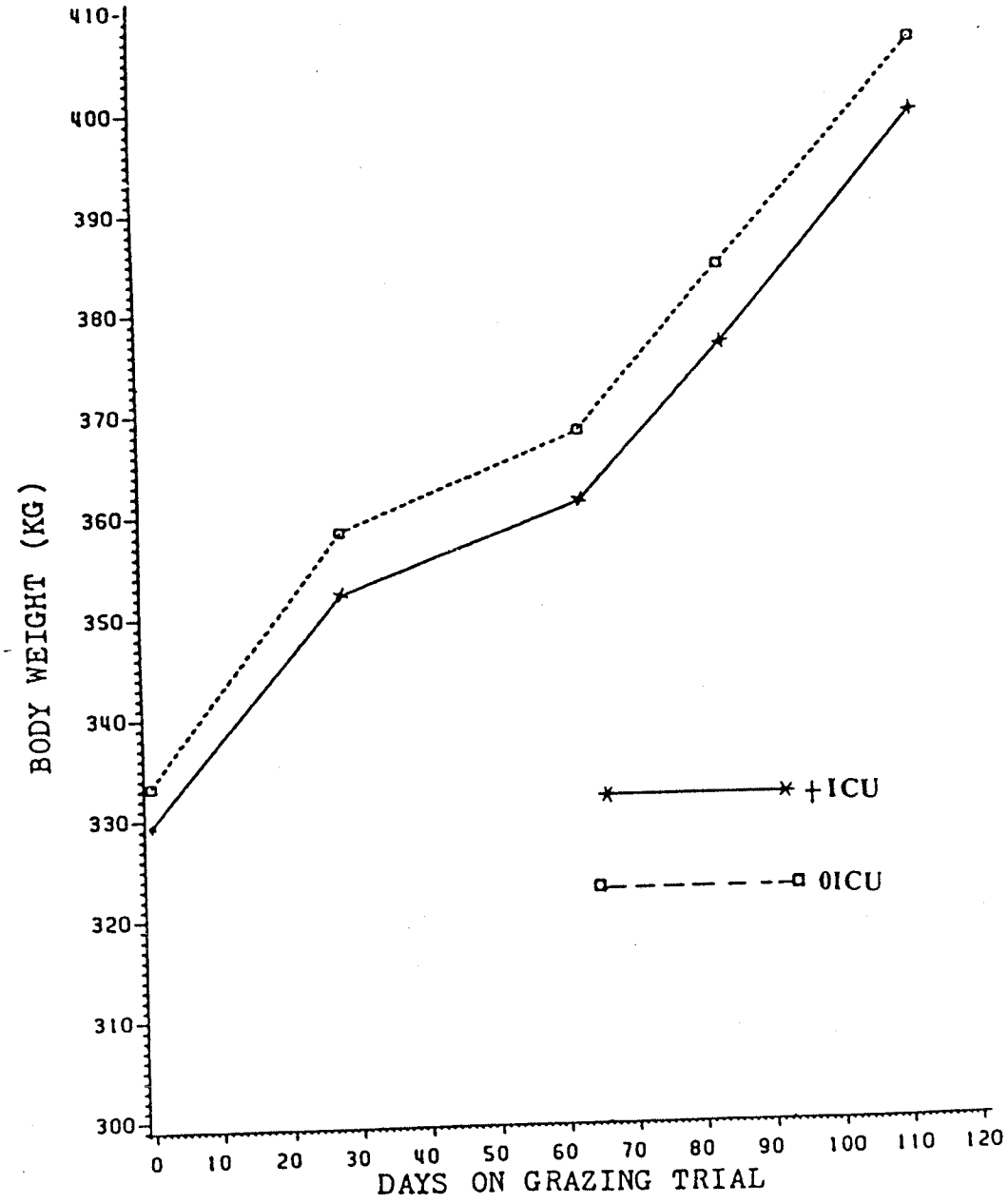


Fig.3: Least square means for body weight of all steers treated with ICU (+ICU)or not treated with ICU (OICU)• Treated steers received a dose of Prolontex-Cu at the start of the grazing trial.

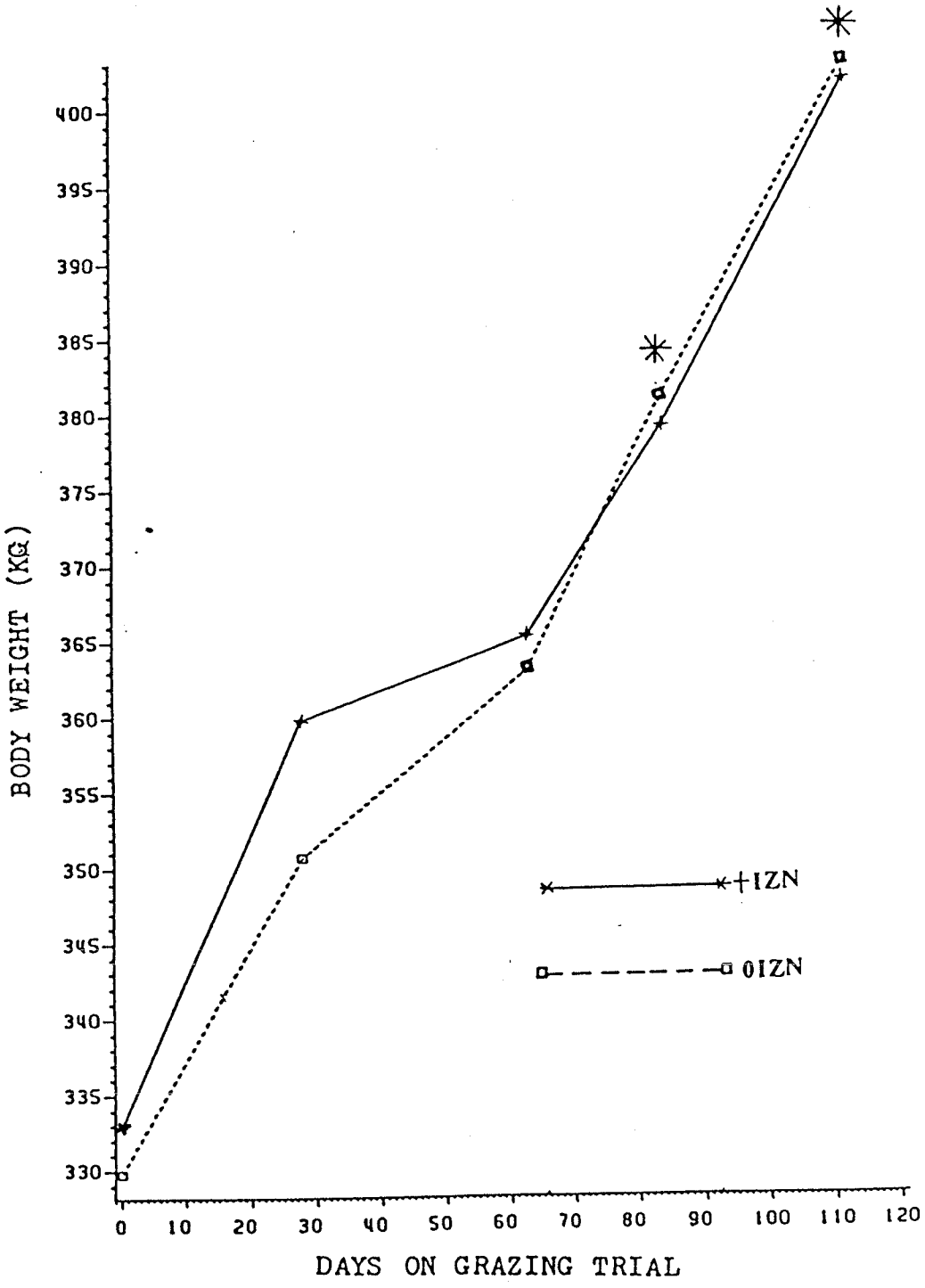


Fig.4: Least square means for body weight of all steers treated with IZN (+IZN) or not treated with IZN (0IZN). Treated steers received a dose of Prolontex-Zn at the start of the grazing trial. Linear contrast of each sampling day relative to day 0 (Appendix Table B2) were significant ($P<0.10$) if noted with an asterisk (*)

IZN treatment had an effect ($P<0.10$) upon body weight (Fig.4). Based on linear contrast analysis, at sampling Day 84, steers treated with IZN weighed 6.8 kg less ($P<0.10$) than steers not treated with IZN (Appendix Table B2); on sampling Day 113, IZN treated steers weighed 6.5 Kg less ($P<0.10$) than steers not treated with IZN (Appendix Table B2).

Body weight of Group I steers

Least square means for body weight of Group I steers during the grazing trial are illustrated in Figs 5 and 6. Steers in all treatment groups grew throughout the grazing trial.

Treatment with ICU (Fig.5) and IZN (Fig.6) did not influence ($P>0.05$) body weight of Group I steers (Appendix Table B3). There was no interaction between ICU and IZN for body weight ($P>0.05$).

Subplot analysis revealed a significant effect ($P<0.01$) of sampling day upon body weight. There was a significant ($P<0.05$) interaction between IZN and sampling day for body weight (Fig.6). Other subplot interactions were not significant ($P>0.05$).

The linear contrast analysis reported in Appendix Table B4 showed that mean body weight of IZN treated steers on

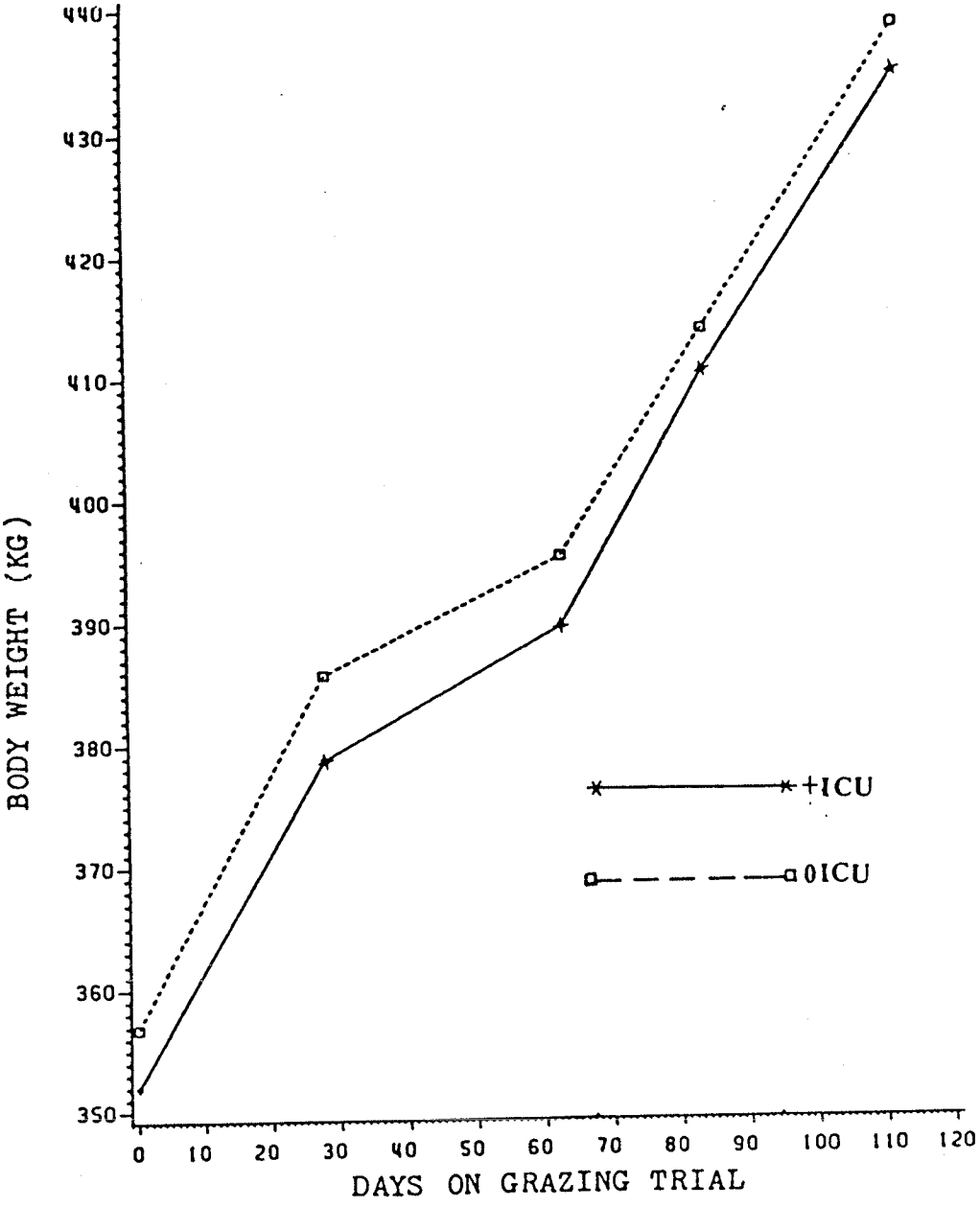


Fig.5: Least square means for body weight of Group I steers treated with ICU (+ICU) or not treated with ICU (OICU). Treated steers received a dose of ICU at the start of the grazing trial.

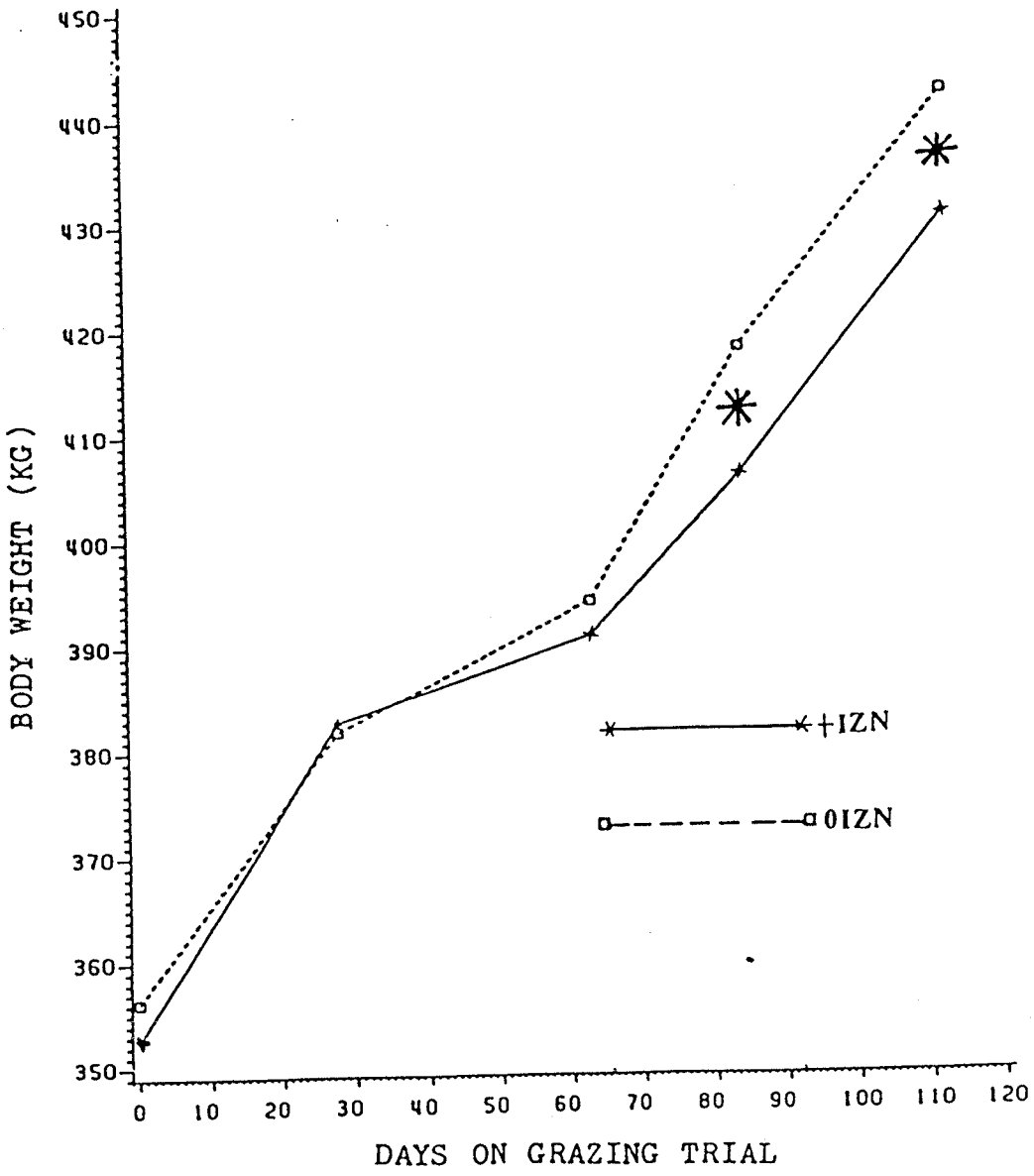


Fig.6: Least square means for body weight of Group I steers treated with IZN (+IZN) or not treated with IZN (OIZN). Treated steers received a dose of Prolontex-Zn at the start of the grazing trial. Linear contrast of each sampling day relative to day 0 (Appendix Table B4) were significant ($P<0.10$) if noted with an asterisk(*)

sampling days 84 and 113 were different ($P<0.10$) from that of IZN untreated steers (Fig.5). The estimated differences in mean body weight of IZN treated steers and untreated steers on sampling days 28, 63, 84 and 113 are listed in Appendix Table B4. On sampling Day 84 IZN treated steers weighed 8.3 kg less ($P<0.10$) than steers not treated with IZN, while on sampling day 113, IZN treated steers weighed 8.0 kg less ($P<0.10$) than steers not treated with IZN. There were no differences in body weight between IZN treated and steers not treated with IZN at sampling days 28 and 63 (Fig. 6; Appendix Table B4).

Body weights of Group II steers

Least square means for body weight of Group II steers are illustrated in Fig.7 and Fig.8 on the basis of ICU and IZN treatments, respectively.

Neither ICU (Fig.7) nor IZN treatment (Fig.8) had an influence ($P>0.05$) on body weight of Group II steers (Appendix Table B5). There was no interaction ($P>0.05$) between ICU and IZN for body weight. Subplot analysis showed a significant effect ($P<0.01$) of sampling day upon body weight (Appendix Table B5). All subplot interactions were not significant ($P>0.05$).

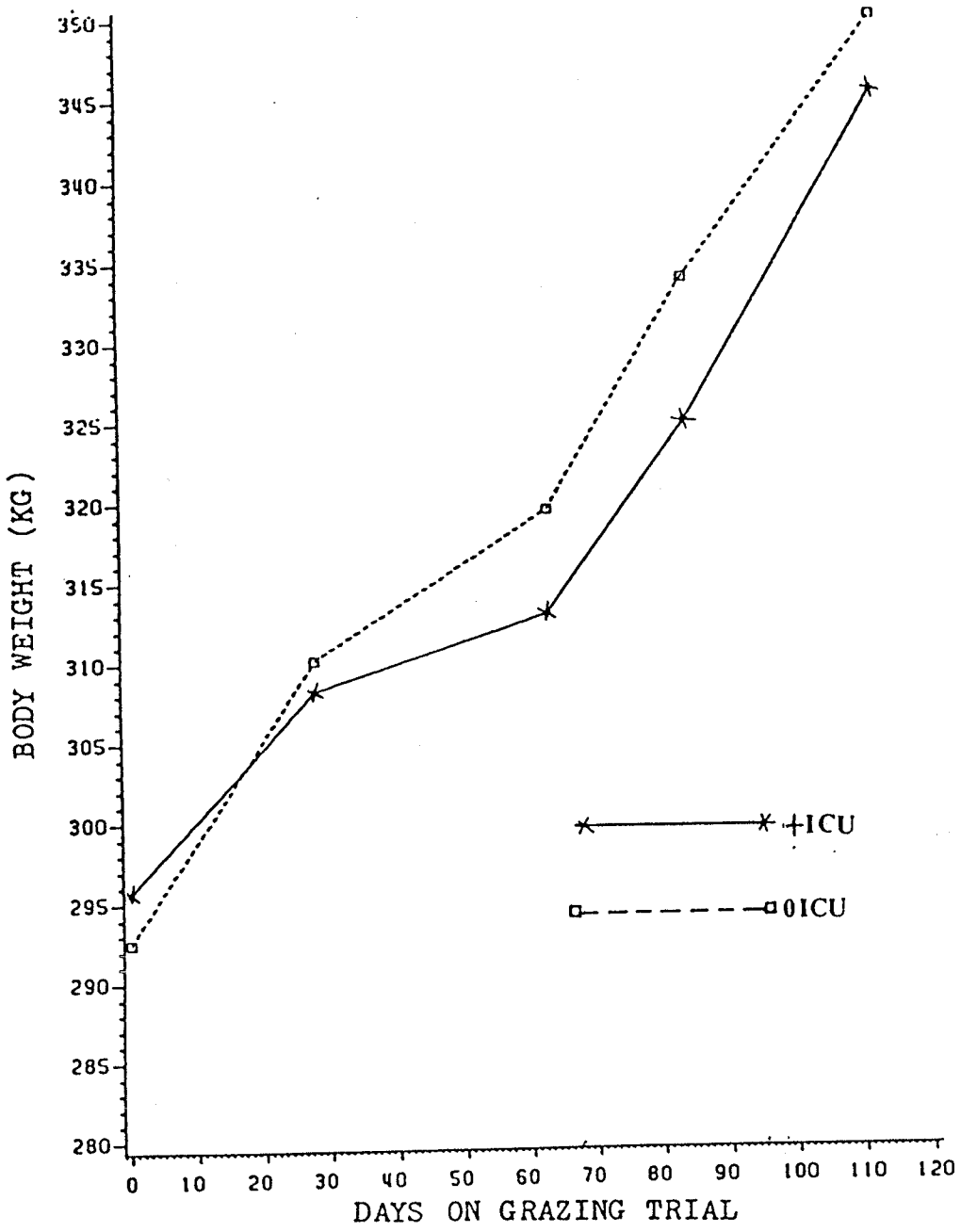


Fig.7: Least square means for body weight of Group II steers treated with ICU (+ICU) or not treated with ICU (0ICU). Treated steers received a dose of Prolontex-Cu at the start of the grazing trial.

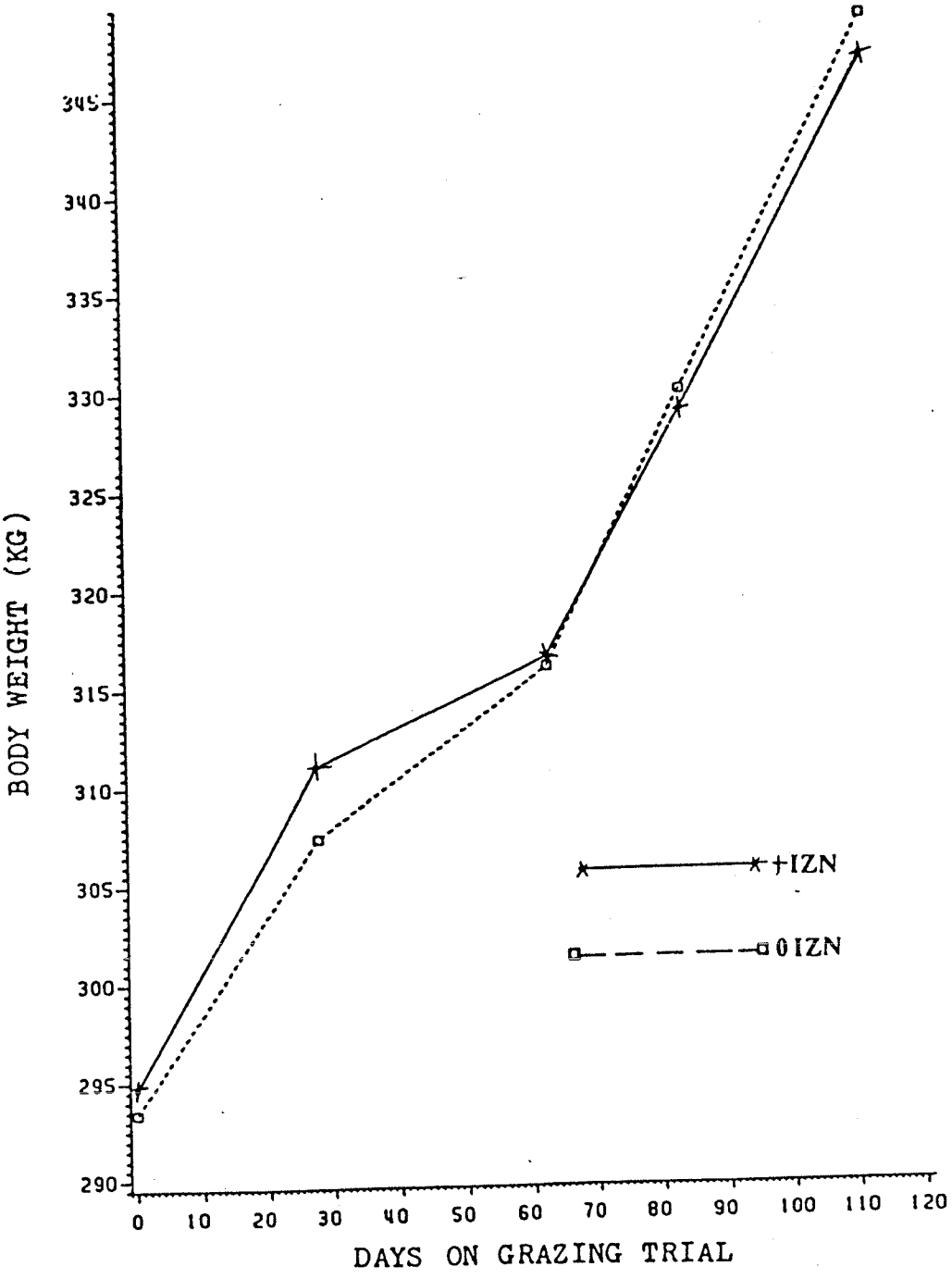


Fig.8: Least square means for body weight of Group II steers treated with IZN (+IZN) or not treated with IZN (OIZN). Treated steers received a dose of Prolontex-Zn at the start of the grazing trial.

Serum copper

Serum Copper of all Steers

Least square means of serum Cu for all steers treated with or not treated with ICU during the grazing trial are illustrated in Fig.9. Serum Cu in both treated and untreated steers fluctuated throughout the grazing trial. At the end of the grazing trial, mean concentrations of serum Cu for both treated and untreated steers were approximately 80% of values measured on sampling day 0.

There were no main effects ($P>0.05$) for either ICU or IZN treatment on serum Cu (Appendix Table B1). There was no interaction between ICU and IZN for serum Cu ($P>0.05$). There was a significant interaction ($P<0.05$) between ICU, IZN and Group for serum Cu.

Subplot analysis by split plot procedure showed a significant effect ($P<0.01$) of sampling day upon serum Cu (Fig.9). There were also significant interactions between breed group and sampling day ($P<0.05$) and between ICU and sampling day (Fig.9). Other subplot interactions were not significant ($p>0.05$).

The linear contrast analysis listed in Appendix Table B6 showed that serum Cu of ICU treated steers was significantly different ($P>0.01$) from that of untreated steers at sampling day 84 of the grazing trial (Fig.9). At sampling day

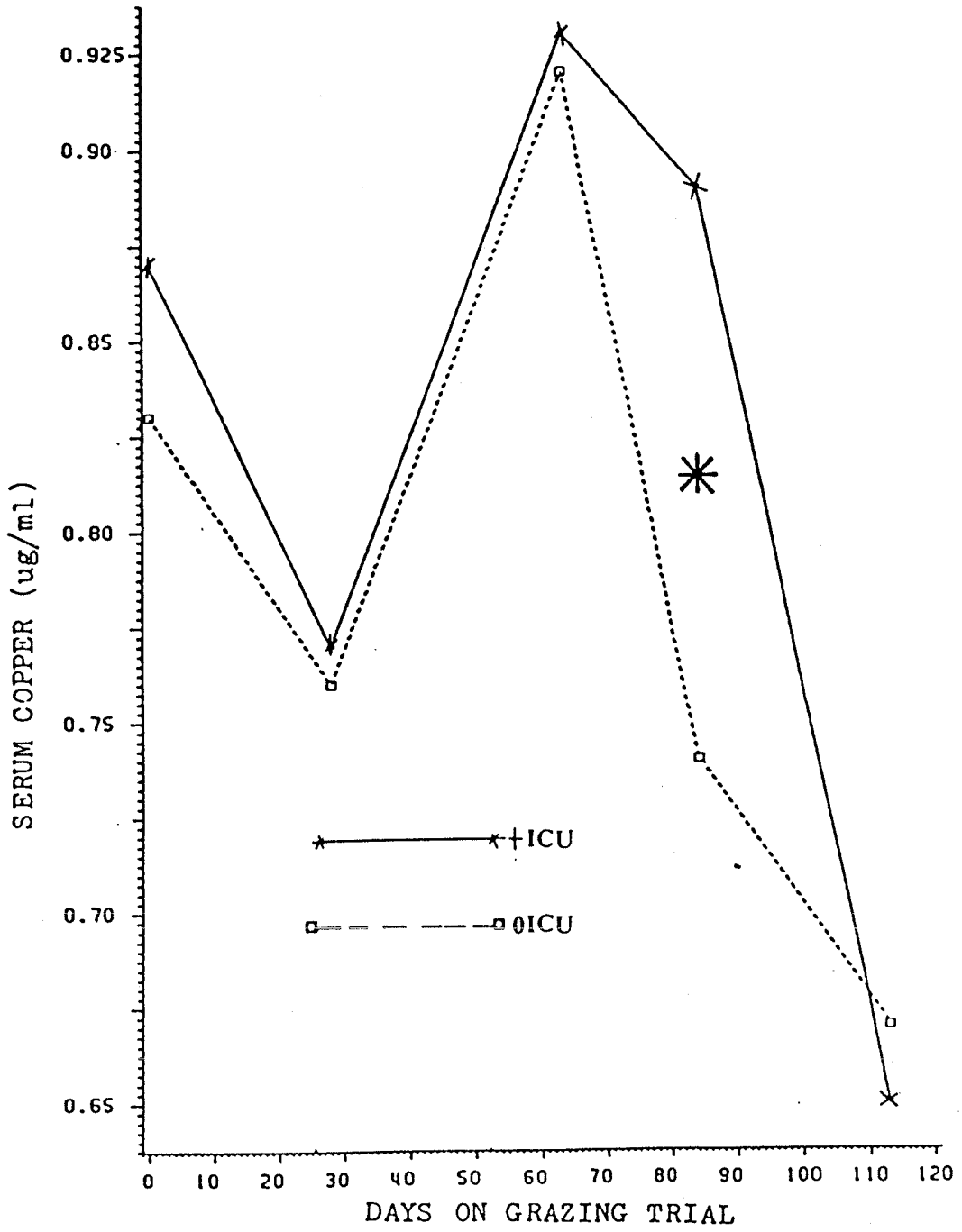


Fig.9: Least square mean concentration of serum copper of all steers treated with ICU (+ICU) or not treated with ICU (OICU). Treated steers received a dose of Prolontex-Cu at the start of the grazing trial. Linear contrast of each sampling day relative to day 0 (Appendix Table B7) were significant ($P<0.05$) if noted with an asterisk (*).

84 serum Cu was 0.89 and 0.69 ug/ml for ICU treated and untreated steers respectively; at sampling day 113, there were no significant differences (P>0.05) in serum Cu between ICU treated and untreated steers respectively (Fig.9).

Serum Copper for Group I steers

The mean concentrations of serum Cu of Group I steers during the grazing trial are illustrated in Fig.10. On sampling day 0, of the grazing trial, the range of serum Cu in both ICU treated and untreated steers was 0.82 and 0.95 ug/ml, but at sampling day 113, the serum Cu range for both ICU treated and untreated had declined to 0.59 to 0.67 ug/ml; thus final serum Cu range was approximately 75% of day 0 values.

Split plot analysis of variance for serum Cu data of Group I steers is reported in Appendix Table B3. There were no significant main effects (P>0.05) for either ICU or IZN treatment upon serum Cu. There was no interaction (P>0.05) between ICU and IZN for serum Cu. Sub plot analysis showed a significant effect (P<0.05) of sampling day (Fig.10) upon serum Cu. There was also a significant interaction (P>0.05) between ICU treatment and sampling day (Fig.10) for serum Cu.

From the linear contrast analysis (Appendix Table B7), steers treated with ICU had higher (P<0.05) serum Cu at sampling day 84 (fig.10) but at sampling day 113, there were

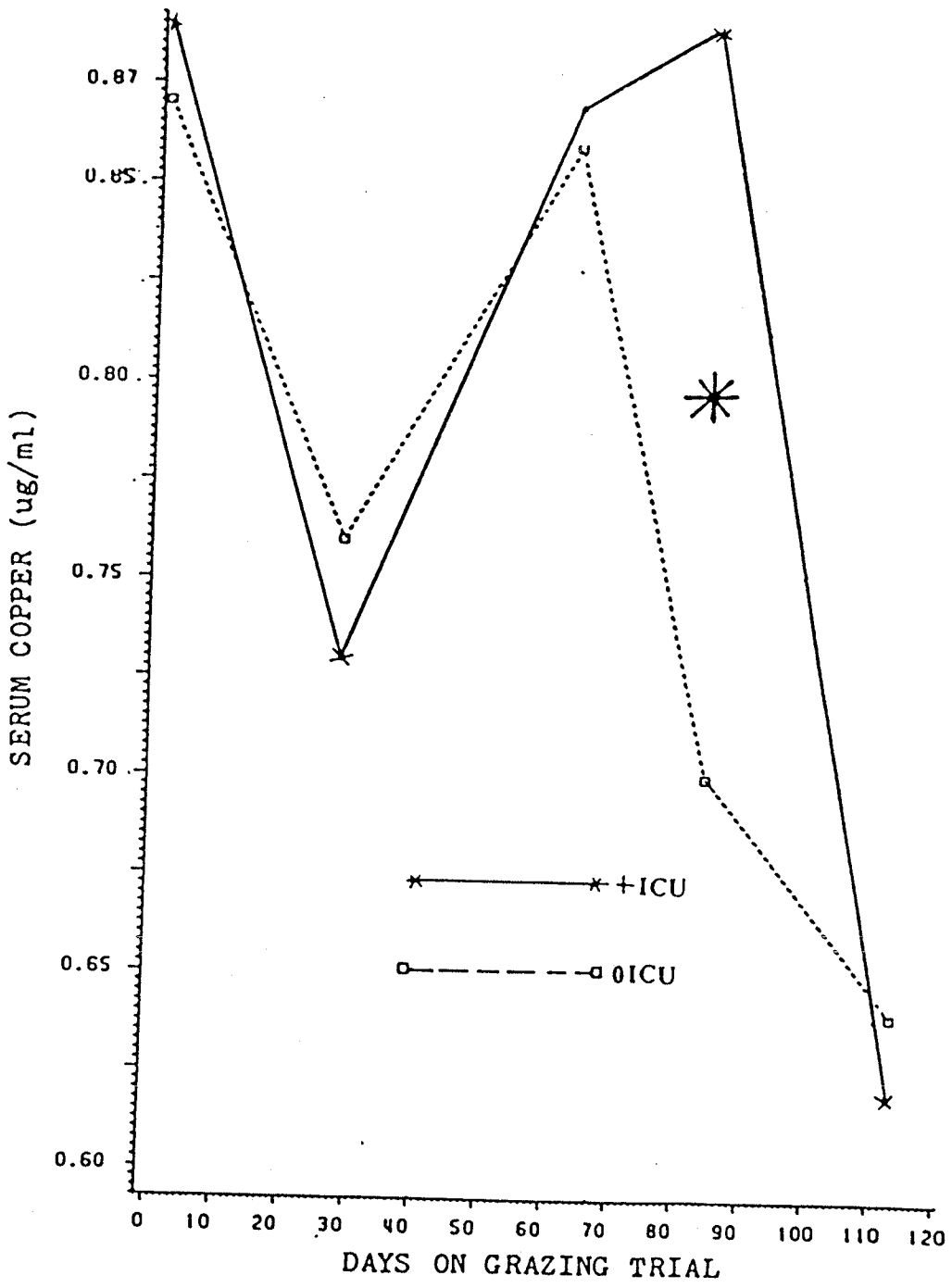


Fig.10: Least square mean concentration of serum copper for Group I steers treated with ICU (+ICU) or not treated with ICU (OICU) Treated steers received a dose of Prolontex-Cu at the start of the grazing trial. Linear contrast of each sampling day relative to day 0 (Appendix Table B8) were significant ($P<0.05$) if noted with an asterisk (*).

no significant differences in serum Cu ($P>0.05$) between ICU treated and untreated steers.

Serum Copper for Group II steers

For both ICU treated and ICU untreated steers, serum Cu fluctuated throughtout the grazing trial (Fig.11). Mean serum Cu measured on day 0 for all 23 steers of Group II was in the range of 0.72-0.87 ug/ml. At sampling day 113, the range had decreased to 0.64-0.70 ug/ml. Mean concentrations of serum Cu on day 0 was 0.86 and 0.76 ug/ml for ICU treated and ICU untreated steers respectively. At sampling day 113, serum Cu was 0.68 and 0.69 ug/ml for ICU treated and untreatd steers respectively (Fig.11).

Serum copper of Group II steers was not influenced ($P>0.05$) by either ICU or IZN treatment (Appendix Table B8). There was no interaction ($P>0.05$) between ICU and IZN for serum Cu of Group II steers. Subplot analysis showed a highly significant effect ($P<0.01$) of sampling day upon serum Cu (Fig.11). All subplot interactions were not significant ($P>0.05$).

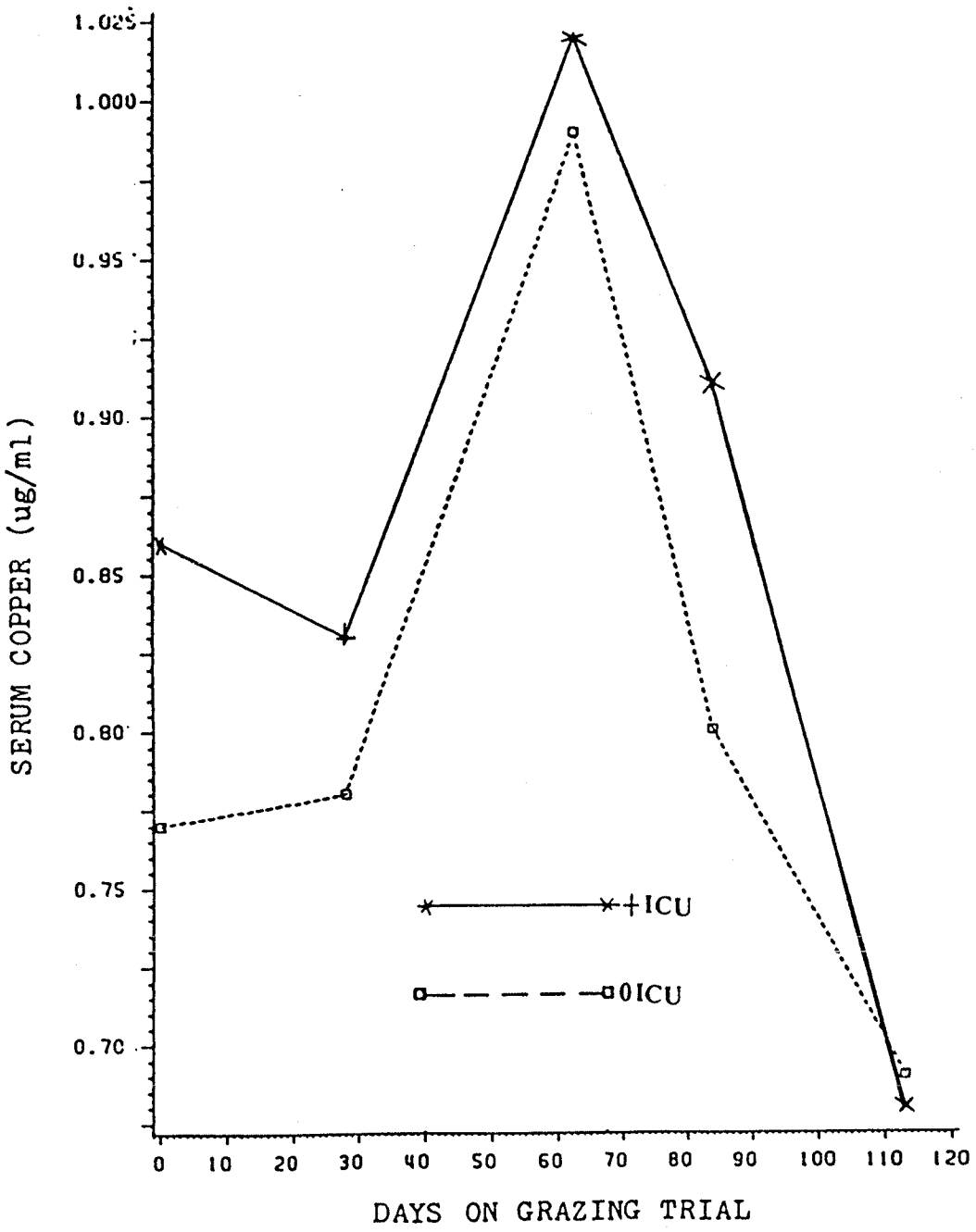


Fig. 11: Least square mean concentration of serum copper for Group II steers treated with ICU (+ICU) or not treated with ICU (OICU) Treated steers received a dose of Prolontex-Cu at the start of the grazing trial.

Serum zinc

Serum Zinc for all Steers

Least square mean concentrations of serum Zn of all steers during the grazing trial are illustrated in Fig.12. Serum Zn fluctuated throughout the grazing trial (Fig.12). At the end of the grazing trial, mean serum Zn for both IZN treated and untreated steers was approximately 75% of values measured on day 0.

Neither ICU nor IZN treatment had an effect ($P>0.05$) upon serum Zn (Appendix Table B1). There was no Group, Group x IZN or Group x ICU effects ($P>0.05$) on serum Zn.

Sub plot analysis showed a significant effect ($P<0.05$) of sampling day (Fig.12) on serum Zn. There were no significant ($P>0.05$) subplot interactions.

Serum Zinc of Group I steers

Serum Zn for Group I steers fluctuated throughout the grazing trial (Fig.13). At day 0 the range of serum Zn for both IZN treated and untreated steers was 1.11-1.15 ug/ml; at sampling day 113, the range for serum Zn had declined to 0.80-0.91 ug/ml. Final serum Zn values were approximately 75% of values measured on day 0.

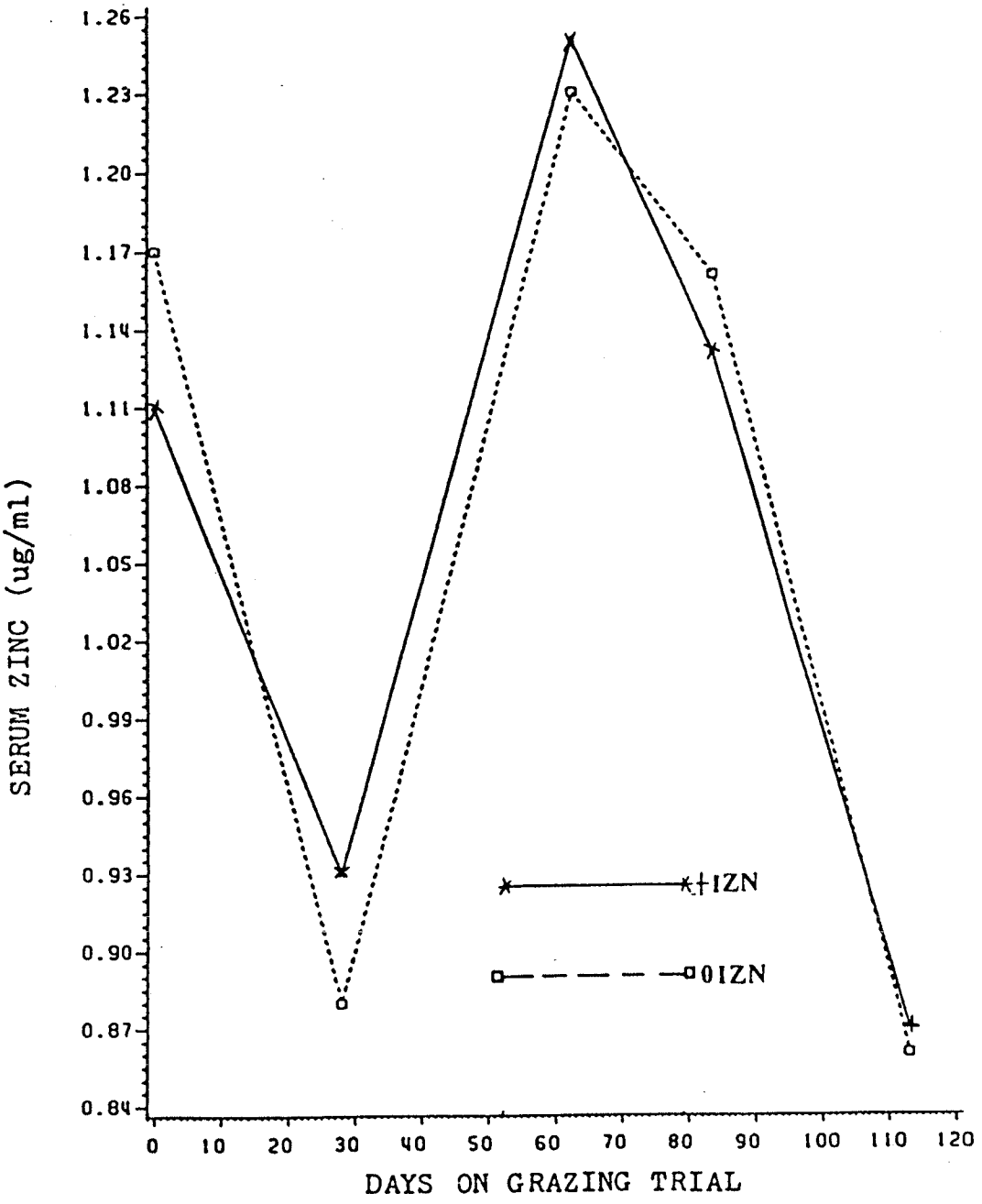


Fig. 12: Least square mean concentration of serum zinc of all steers treated with IZN (+IZN) or not treated with IZN (OIZN). Treated steers received a dose of Prolontex-Zn at the start of the grazing trial.

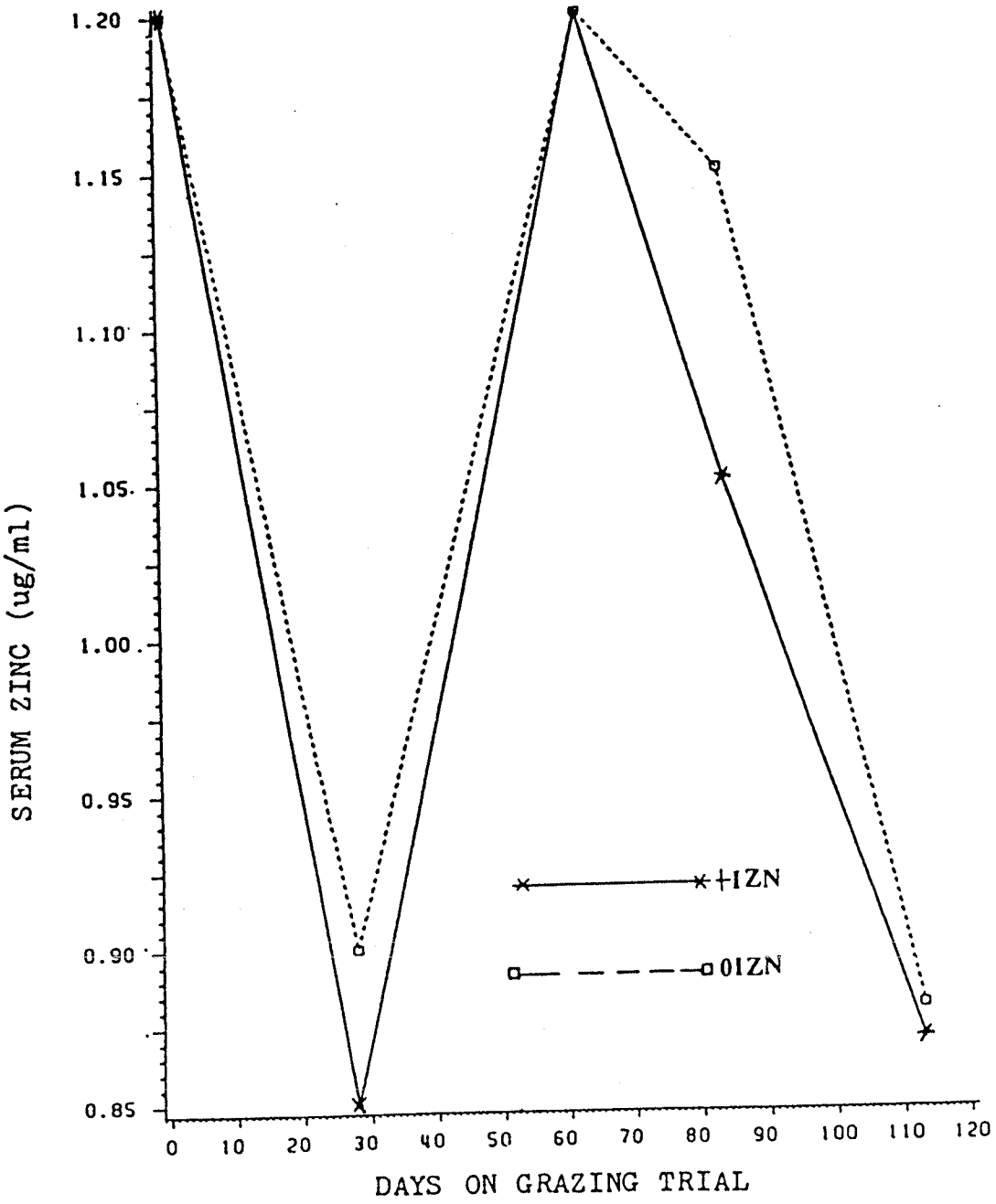


Fig. 13: Least square mean concentration of serum zinc of Group I steers treated with IZN (+IZN) or not treated with IZN (OIZN). Treated steers received a dose of Prolontex-Zn at the start of the grazing trial.

Neither IZN nor ICU treatment had an influence ($P>0.05$) upon serum Zn of Group I steers (Appendix Table B3); there was no interaction ($P>0.05$) between ICU and IZN treatment for serum Zn.

Subplot analysis showed a significant effect ($P<0.01$) of sampling day (Fig.13) on serum Zn (Appendix Table B3). There were no significant ($P>0.05$) subplot interactions for serum Zn.

Serum Zinc for Group II steers

Least square mean concentrations of serum Zn for Group II steers are illustrated in Fig.14. Serum Zn of Group II steers also fluctuated throughout the grazing trial, with means on sampling day 113 lower than those measured at day 0 of the grazing trial.

Neither IZN nor ICU treatment had an influence ($P>0.05$) upon serum Zn (Appendix Table B8). There was no interaction ($P>0.05$) between IZNxICU for serum Zn. Subplot analysis showed a significant effect ($P<0.01$) of sampling day upon serum Zn (Fig.14). All subplot interactions were non significant ($P>0.05$).

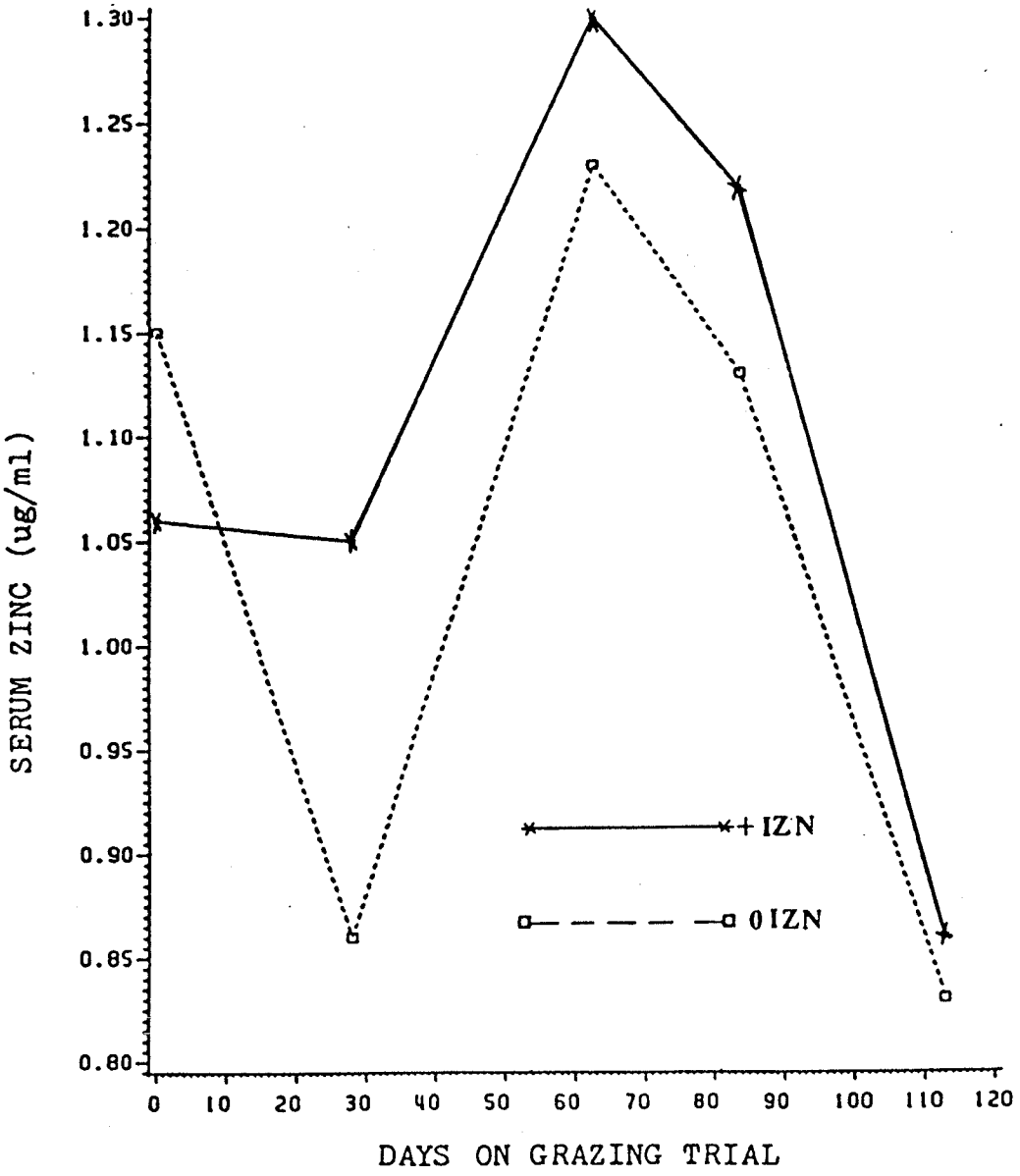


Fig. 14: Least square mean concentration of serum zinc for Group II steers treated with IZN (+IZN) or not treated with IZN (OIZN) Treated steers received a dose of Prolontex-Zn at the start of the grazing trial.

Liver copper

Liver Copper for all Steers

Least square means for liver Cu for all steers during the grazing trial are illustrated in Fig.15. For both ICU treated and untreated steers there was an increase in liver Cu to approximately twice the values measured on day 0.

Main plot analysis showed no significant effects ($P>0.05$) of ICU, IZN, or ICU x IZN for liver Cu (Appendix Table B9). All main plot interactions were not significant ($P>0.05$).

Subplot analysis revealed a significant effect ($P<0.01$) of sampling day upon liver Cu (Fig.15). There were also significant interactions ($P<0.01$) between ICU and Group (Appendix Table B9) and between ICU and sampling day (Fig.15) for liver Cu. Other subplot interactions were not significant ($P>0.05$).

Based on linear contrast analysis (Appendix Table B9), mean liver Cu on sampling day 63 was 32.4 mg/kg dry matter higher ($P<0.01$) than that of ICU untreated steers (Fig.15). On sampling day 113 mean liver Cu of ICU treated steers was 22.8 mg/kg dry matter higher ($P<0.05$) than that of ICU untreated steers (Fig.15).

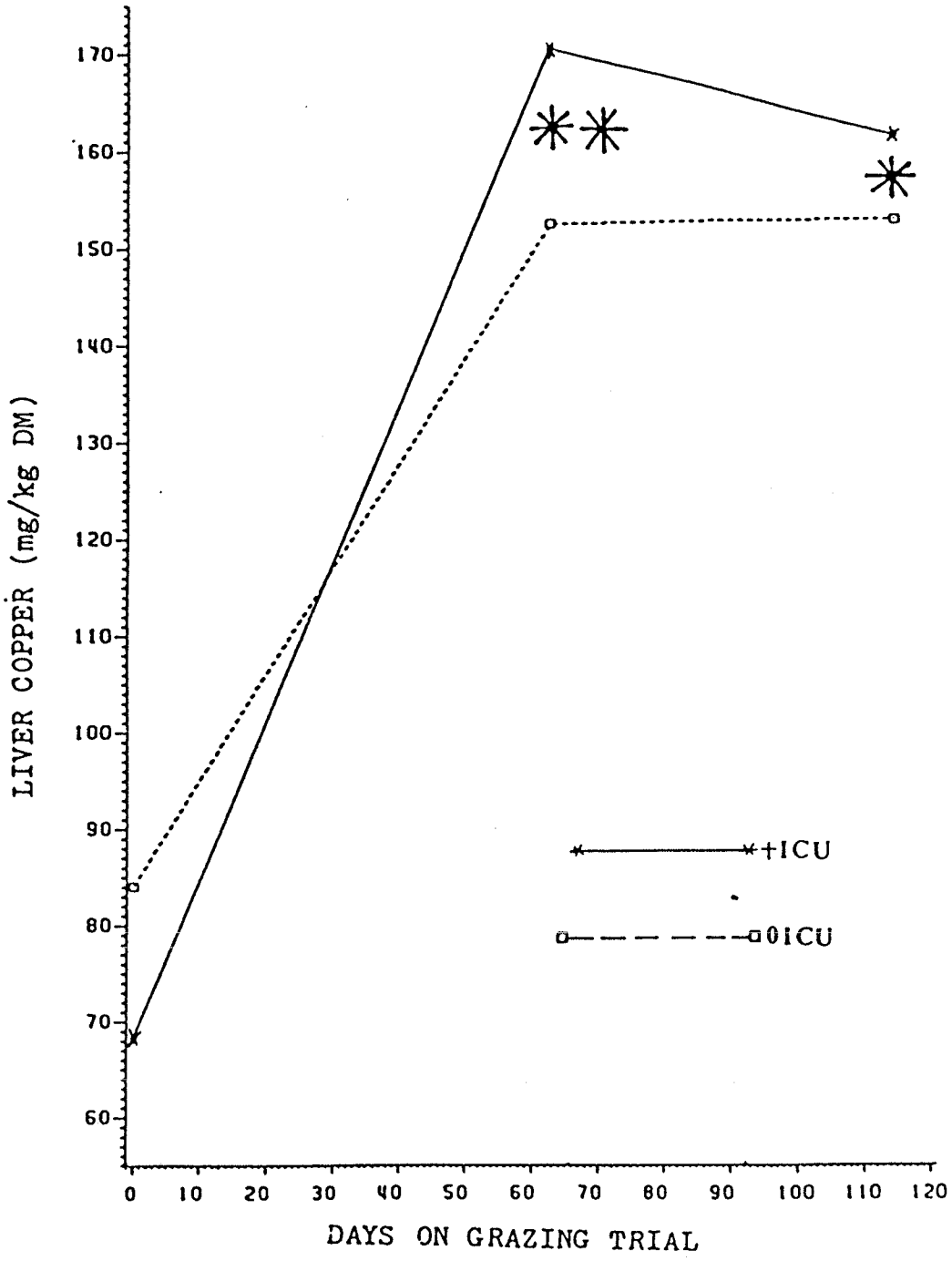


Fig. 15: Least square mean concentration of liver copper of all steers treated with ICU (+ICU) or not treated with ICU (OICU). Treated steers received a dose of Prolontex-Cu at the start of the grazing trial. Linear contrast of each sampling day relative to day 0 (Appendix Table B9) were significant if noted with asterisks (* $P<0.05$, ** $P<0.01$).

Liver Copper for Group I Steers

For both ICU treated and untreated steers, liver Cu increased to approximately twice the values measured on day 0 (Fig.16).

Main plot analysis showed no significant effects ($P>0.05$) of ICU, IZN, or ICU x IZN for liver Cu (Appendix Table B10). Subplot analysis revealed a significant effect ($P<0.01$) of sampling day on liver Cu (Fig.16). There was also a significant interaction ($P<0.01$) between sampling day and ICU treatment (Fig.16) for liver Cu (Appendix Table B10). Other subplot interactions were not significant($P>0.05$).

Linear contrast analysis (Appendix Table B10) showed liver Cu of ICU treated steers on sampling days 63 and 115, was significantly different ($P<0.05$) from that of ICU untreated steers (Fig.16). On sampling day 63, mean liver Cu of ICU treated steers was 27.3 mg/kg DM higher ($P<0.05$) than that of ICU untreated steers (Appendix Table B10). On sampling day 115 liver Cu of ICU treated steers was 31.5 mg/kg DM higher ($P<0.05$) than that of ICU untreated steers (Appendix TAble B10).

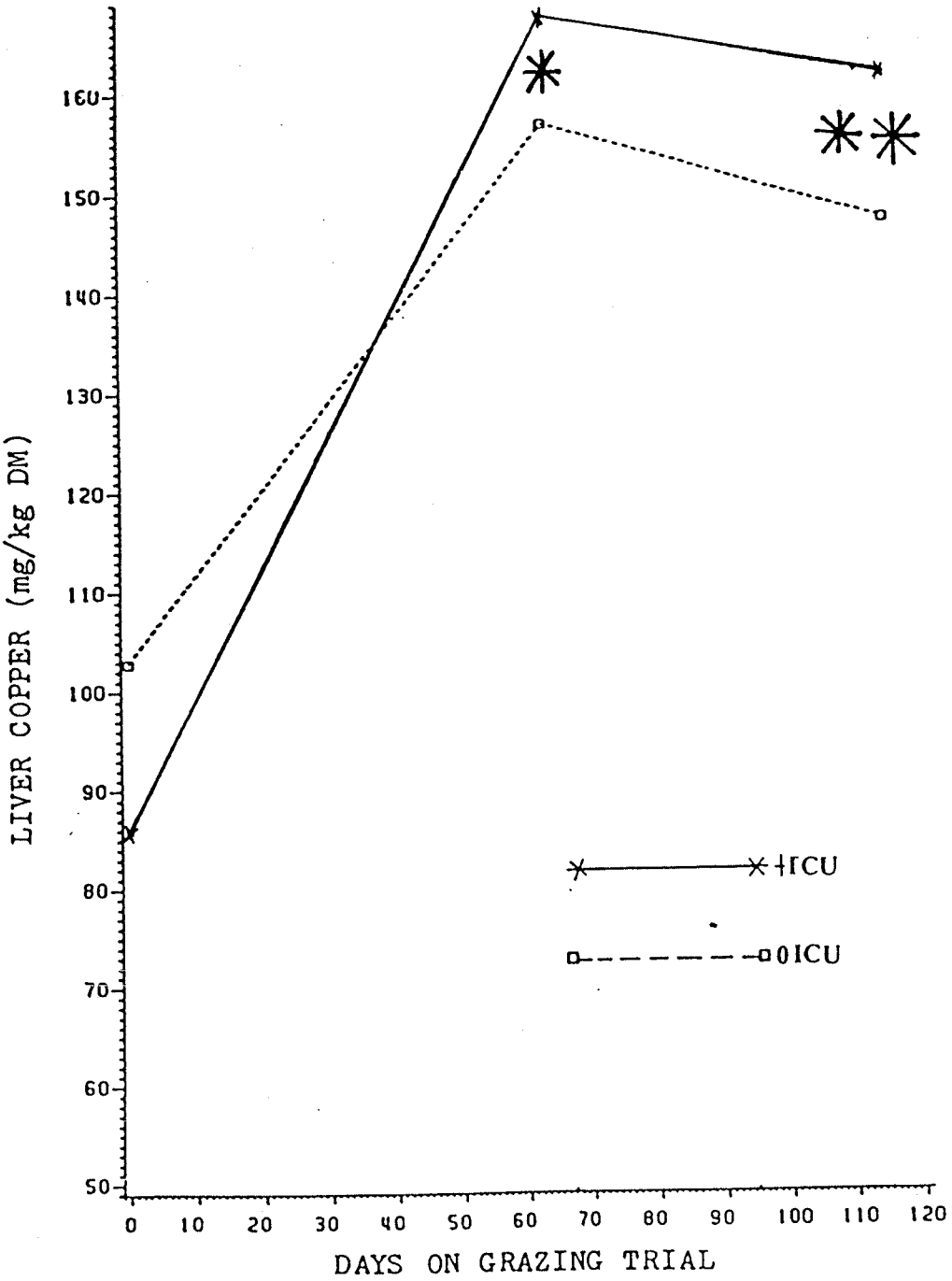


Fig. 16: Least square mean concentration of liver copper for Group I steers treated with ICU (+ICU) or not treated with ICU (OICU). Treated steers received a dose of Prolontex-Cu at the start of the grazing trial. Linear contrast of each sampling day relative to day 0 (Appendix Table B10) were significant if noted with asterisks (* $P<0.05$, ** $P<0.01$).

Liver Copper for Group II Steers

Least square means for liver Cu of Group II steers treated with ICU or not treated with ICU are illustrated in Fig.17. For both ICU treated and untreated steers, mean liver Cu on sampling day 63 was more than twice the values measured on day 0; the values remained at these high levels at sampling day 115.

Split plot analysis of variance for liver Cu showed no significant main effects ($P>0.05$) of ICU, IZN or ICU x IZN on liver Cu (Appendix Table B11). Subplot analysis showed a significant effect ($P<0.01$) of sampling day on liver Cu (Fig.17). There was also a significant interaction ($P<0.05$) between ICU and sampling day for liver Cu (Fig. 17). Other subplot interactions were not significant ($P>0.05$).

Linear contrast analysis (Appendix Table B11) showed that at sampling day 63 liver Cu of ICU treated steers was significantly different ($P<0.05$) from that of ICU untreated steers. At sampling day 115,liver Cu for ICU treated steers was not significantly different ($P>0.10$) from that of control steers. On sampling day 63 liver Cu of ICU treated steers was 39.3 mg/kg dry matter higher than that of ICU untreated steers (Appendix Table B11).

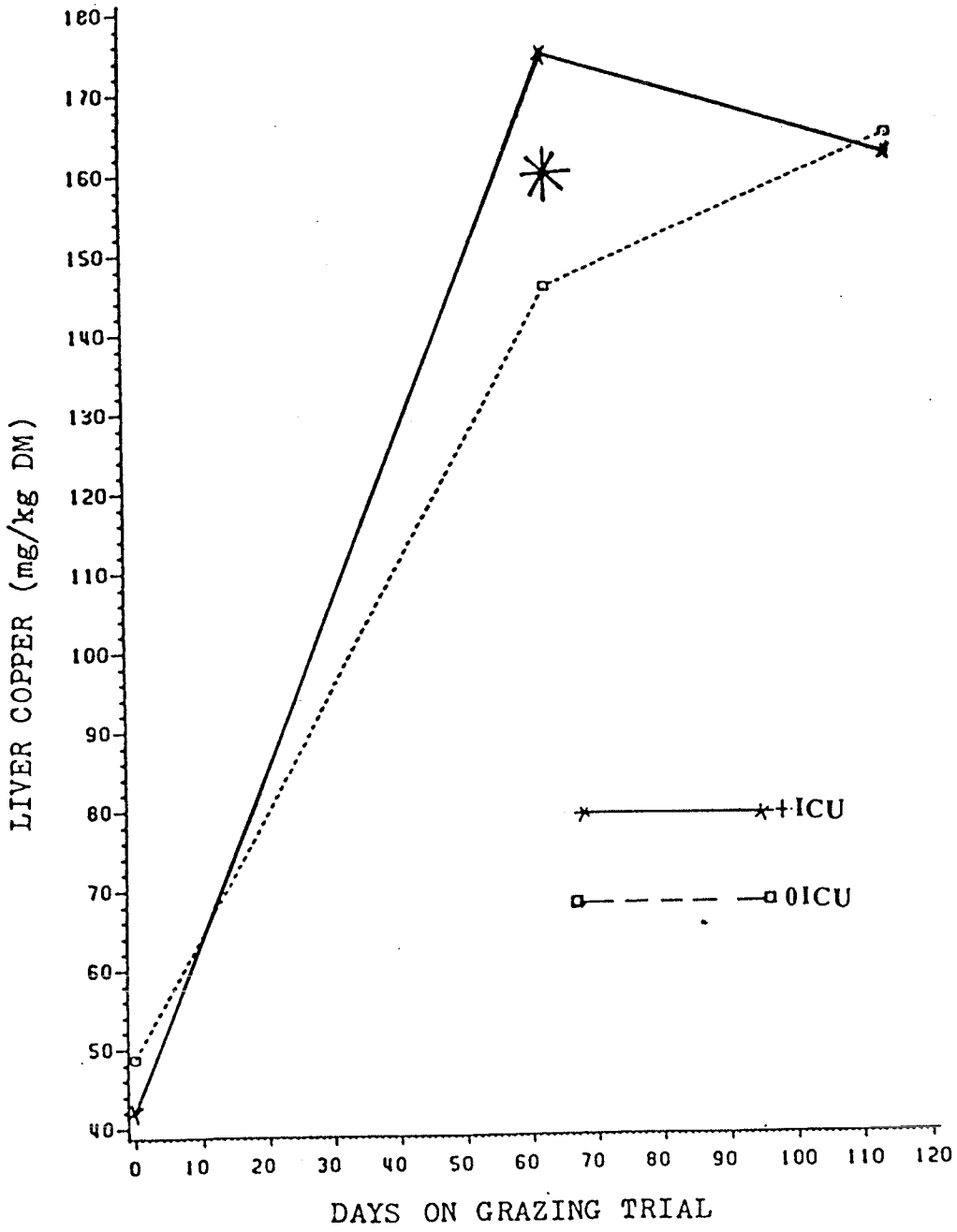


Fig. 17: Least square mean concentration of liver copper of Group II steers treated with ICU (+ICU) or not treated with ICU (OICU). Treated steers received a dose of Prolontex-Cu at the start of grazing trial. Linear contrast of each sampling day relative to day 0 (Appendis Table B11) were significant if noted with an asterisk (*P<0.05).

Liver zinc

Liver Zinc for all steers

There was an overall decrease in liver Zn for both IZN treated and IZN untreated steers during the grazing trial (Fig.18). Liver Zn measured on day 0 was 95.8 and 98.2 mg/kg dry matter for IZN treated and IZN untreated steers respectively; at sampling day 115, mean liver Zn concentrations were 88.8 and 88.3 mg kg dry matter for IZN treated and IZN untreated steers respectively (Fig.18).

Neither ICU nor IZN treatment had an influence ($P>0.05$) upon liver Zn (Appendix Table B12). There was no interaction ($P>0.05$) between ICU and IZN for liver Zn; there were no Group differences ($P>0.05$) on the response of liver Zn to either IZN or ICU treatment. All main plot interactions were not significant ($P>0.05$). Subplot analysis showed a significant effect ($P<0.05$) of sampling day upon liver Zn (Fig.18). Mean liver zinc for both IZN treated and IZN untreated steers were lower at sampling day 115 compared to sampling day 0 (Fig.17). This drop was not relateed to IZN treatment ($P>0.05$).

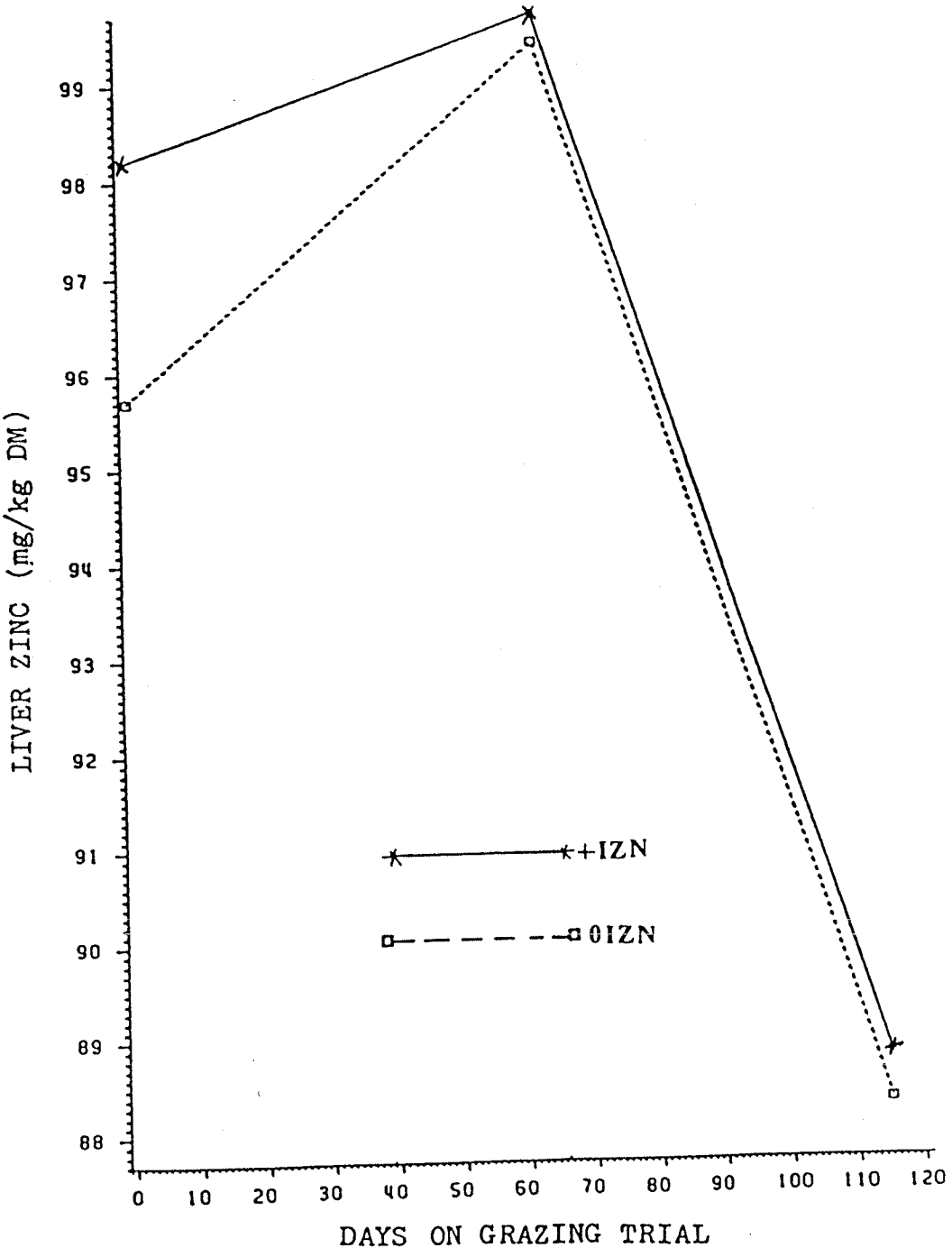


Fig. 18: Least square mean concentration of liver zinc of all steers treated with IZN (+IZN) or not treated with IZN (OIZN). Treated steers received a dose of Prolontex-Zn at the start of the grazing trial.

Liver Zinc for Group I Steers

Least square means for liver Zn of Group I steers are illustrated in Fig.19. There was a decrease in liver Zn for both IZN treated and IZN untreated steers during the grazing trial (Fig.19). Mean initial liver Zn values on day 0 were 102.8 and 97.8 mg/kg dry matter for IZN treated and IZN untreated steers respectively; at sampling day 115, liver Zn concentrations were 88.4 and 86.4 mg/kg dry matter for IZN treated and IZN untreated steers respectively.

Liver Zn was not influenced ($P>0.05$) by either IZN or ICU treatment (Appendix Table B13). There was no interaction ($P>0.05$) between ICU and IZN for liver Zn. Liver Zn however, differed ($P<0.01$) between sampling days (Fig.19). All subplot interactions were not significant ($P>0.05$).

Liver Zinc for Group II Steers

Mean concentrations of liver Zn for Group II steers are illustrated in Fig.20. Liver Zn measured on sampling day 115 was lower than that measured on sampling days 0 and 63 of the grazing trial (Fig.20) but this difference was not significant ($P>0.05$), because the sampling day effect was not significant ($P>0.05$; Appendix Table B14).

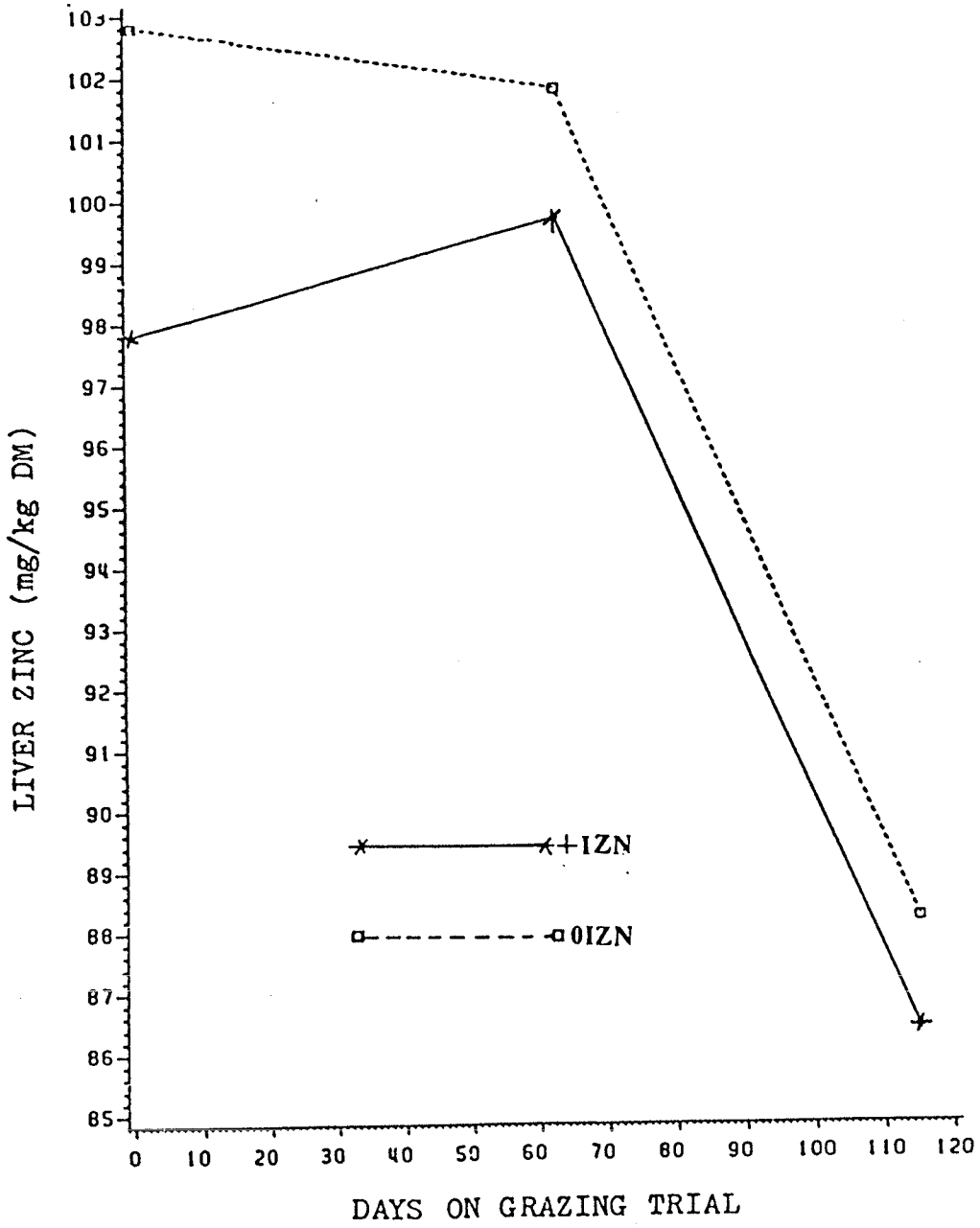


Fig. 19: Least square mean concentration of liver zinc of Group I steers treated with IZN (+IZN) or not treated with IZN (OIZN). Treated steers received a dose of Prolontex- Zn at the start of the grazing trial.

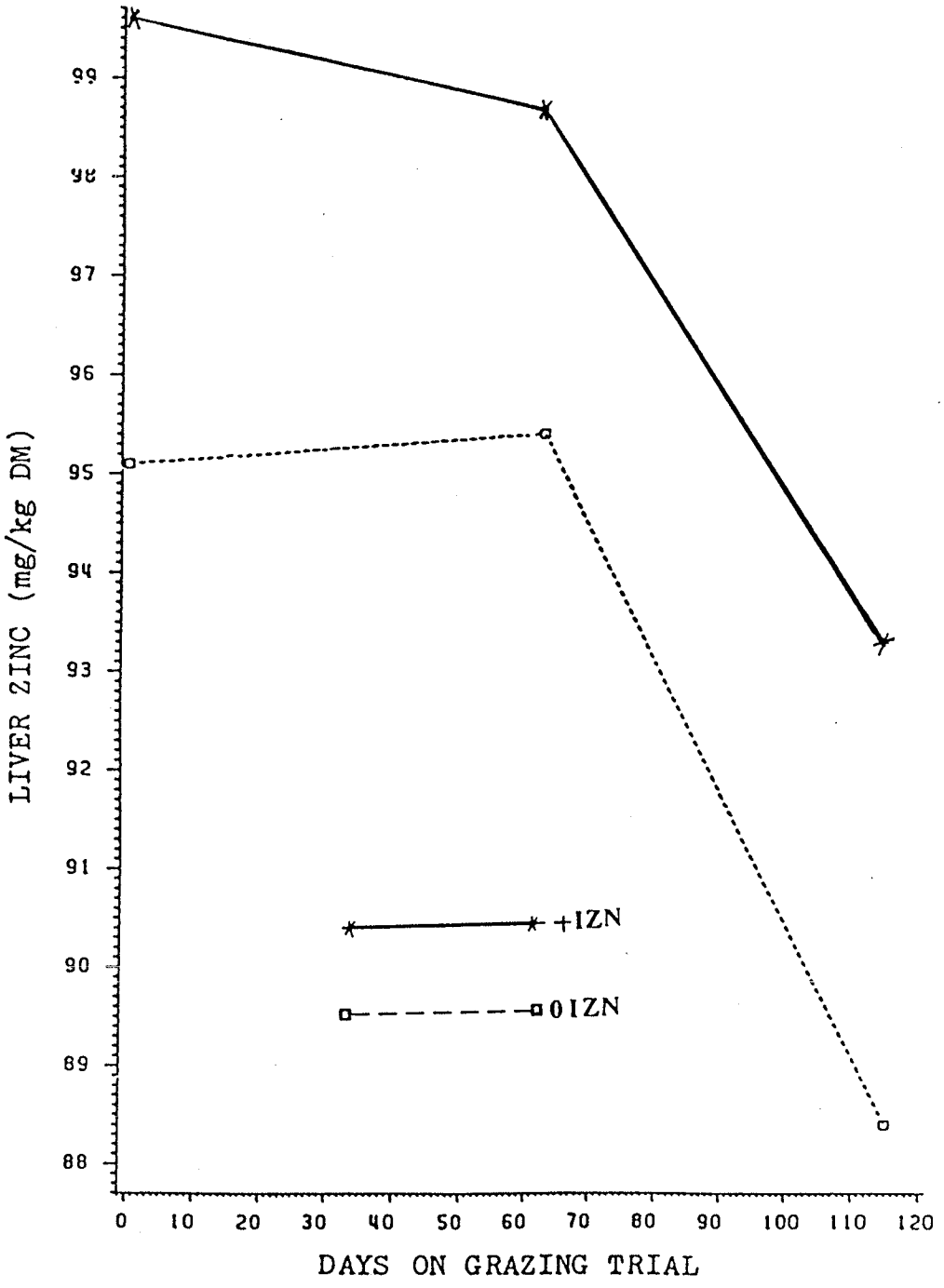


Fig. 20: Least square mean concentration of liver zinc for Group II steers treated with IZN (+IZN) or not treated with IZN (OIZN). Treated steers received a dose of Prolontex-Zn at the start of the grazing trial.

Neither ICU nor IZN treatment had an influence (P>0.05) upon liver Zn (Appendix Table B14). There was no interaction between ICU and IZN for liver Zn. All subplot interactions were not significant (P>0.05).

Forage Mineral concentrations

The data for the nutrient composition of the forage consumed by the steers during the grazing trial are listed in Appendix Tables A6, and Table 8. Copper and Zn were present in low concentrations (relative to requirements for cattle) in all forage samples. Molybdenum and S were less than 1.5 mg and 2.0 g/kg DM respectively. Iron and Mn were normal in all species analyzed; Ca was higher in legumes than in grasses. Phosphorus and Mg were both low in all forage species.

Reactions at the site of Injection

Sites of injection for both ICU and IZN were examined on sampling day 28 of the grazing trial for external signs of swelling. No external signs of swelling were noted at sites of injections for either preparation.

Finishing Trial

Body Weights and Carcass Weights of Steers

Means for body weights and carcass weight of steers measured during the finishing trial are listed in Appendix Table A5. Steers in all treatment groups gained weight throughout the finishing trial whether ICU (Fig.21) or IZN (Fig.22) treatment was considered.

ICU treatment at day 0 of the grazing trial significantly influenced ($P<0.05$) body weight gain of steers during the finishing trial (Appendix table B15). Mean body weights at the start of the finishing trial were 415.7 and 425.4 kg for ICU treated and untreated steers, respectively. At the end of the finishing trial, mean body weights were 510.8 and 533.3 kg for ICU treated and ICU untreated steers, respectively (Fig.21).

IZN treatment did not influence ($P>0.05$) body weight of steers (Fig.22). Subplot analysis showed a significant effect ($P<0.01$) of sampling day upon body weight. There was a significant ($P<0.05$) interaction between ICU and sampling day upon body weight. Other subplot interactions were not significant ($P>0.05$).

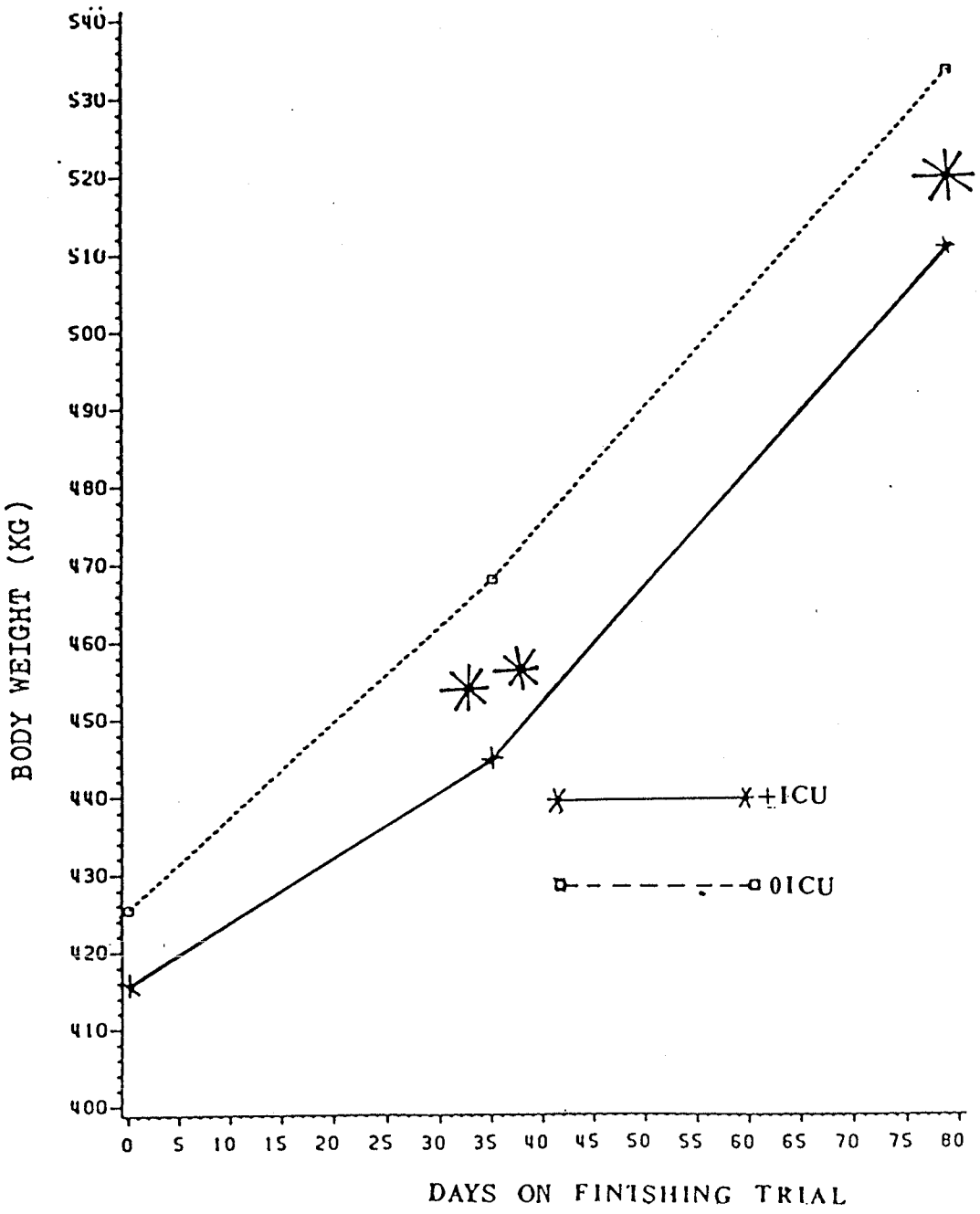


Fig. 21: Least square means for body weight of steers treated with ICU (+ICU) or not treated with ICU (0ICU). Treated steers received a dose of Prolontex-Cu at the start of the grazing trial. Linear contrast analysis of each sampling day relative to day 0 (Appendix Table B15) was significant ($P<0.05$, $P<0.01$) if noted with asterisk (*).

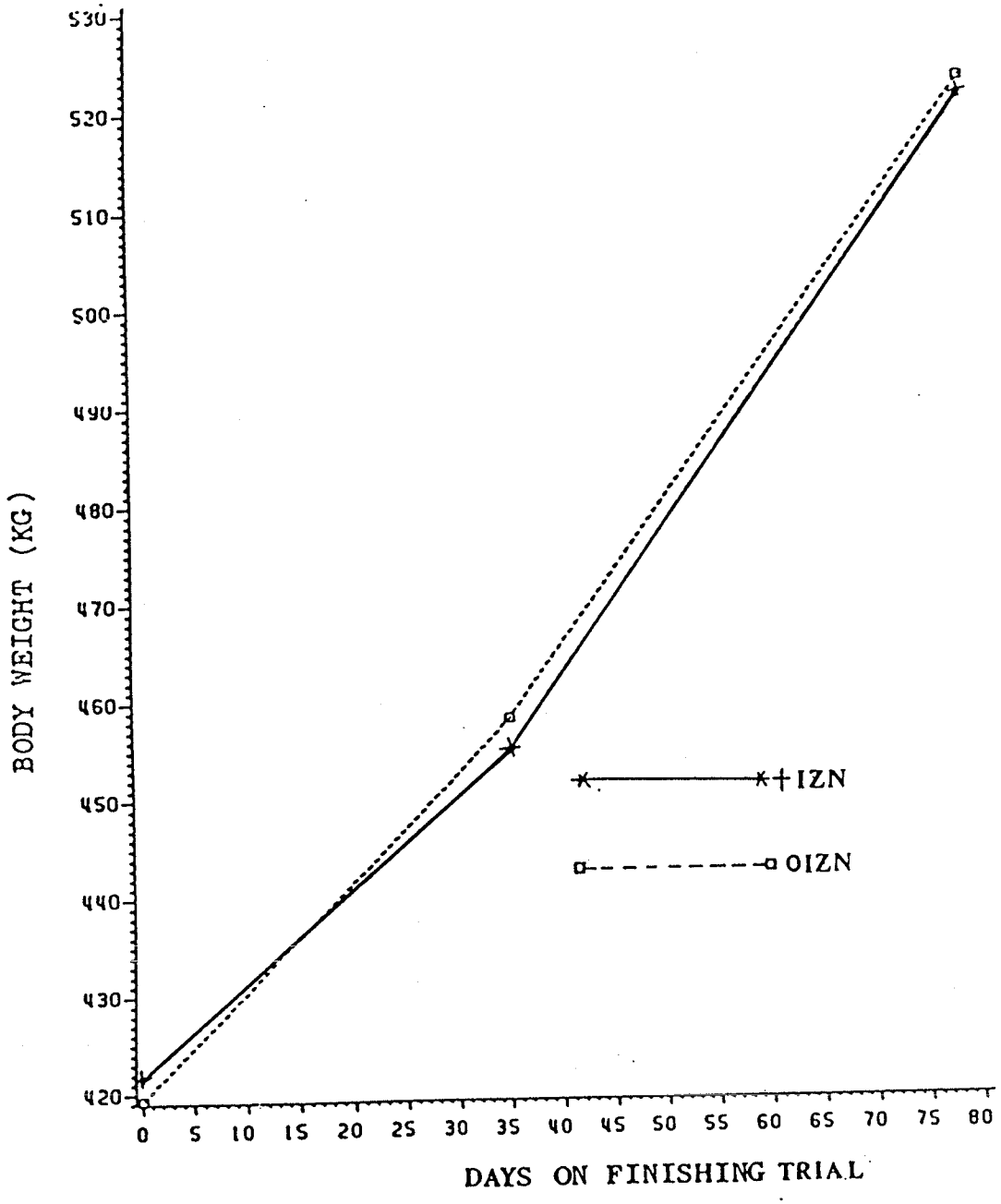


Fig. 22: Least square means for body weight of steers treated with IZN (+IZN) or not treated with IZN (OIZN). Treated steers received a dose of Prolontex-Zn at the start of the grazing trial.

Linear contrast analysis (Appendix Table B15) showed that ICU treated steers had lower body weights at sampling days 35 ($P<0.05$) and 79 ($P<0.10$) of the finishing trial. At sampling day 35, mean body weight (based on linear contrast analysis, Appendix Table B15) of ICU treated steers was 13.7 kg lower ($P<0.05$) than that of untreated steers; at sampling day 79, mean body weight of ICU treated steers was 11.6 kg lower ($P<0.10$) than that of untreated steers (Appendix Table B15). There was no interaction ($P>0.05$) between ICU and IZN for body weights of steers.

Analysis of variance for carcass weight of steers is reported in Appendix Table B16. ICU treatment on day 0 of the grazing trial significantly influenced ($P<0.05$) carcass weight of steers. Mean carcass weights were 279.1 and 291.5 Kg for ICU treated and ICU untreated steers respectively; these means were significantly different ($P<0.05$). IZN treatment did not influence ($P>0.05$) carcass weight. There was no interaction ($P>0.05$) between ICU and IZN for carcass weight. Carcass weight as a percent of body weight was 54.6 for both ICU treated and untreated steers.

Liver copper of steers during the finishing trial

Least square means for liver Cu of steers during the finishing trial are listed in Appendix Table A5. For both ICU

treated and ICU untreated steers, there was a large decrease in liver Cu from the beginning to the end of the finishing trial.

Mean liver Cu at the end of the grazing trial was 159.3 and 145.5 mg/kg dry matter for ICU treated and ICU untreated steers respectively; at the end of the finishing trial, mean liver Cu was 81.2 and 89.8 mg/kg dry matter for ICU treated and ICU untreated steers respectively. These latter means were not significantly different ($P>0.05$).

Analysis of variance for liver Cu during the finishing trial is reported in Appendix Table B17. There was no significant effect ($P>0.05$) for ICU, IZN or ICU x IZN for liver Cu (Appendix Table B17). Subplot analysis revealed a significant effect ($P<0.01$) of sampling day upon liver Cu. There was a significant interaction ($P<0.05$) between sampling day and ICU for liver Cu. All other subplot interactions were not significant ($P>0.05$).

Linear contrast analysis was performed to estimate the difference in mean liver Cu of ICU treated and untreated steers at the end of the grazing trial and at the end of the finishing trial (Appendix Table B17). The contrast analysis showed that mean liver Cu of ICU treated steers at the end of the grazing trial was significantly higher than that of untreated steers ($P<0.05$). At the end of the finishing trial mean concentrations of liver Cu for

both treated and untreated steers were not significantly different ($P>0.05$) from one another. For both ICU treated and untreated steers, mean liver Cu at the end of the finishing trial was significantly different from that measured at the end of the grazing trial. Mean liver Cu of all steers at the end of the finishing trial was significantly lower ($P<0.01$) than that measured at the end of the grazing trial.

Liver zinc of steers during the finishing trial

Means for liver Zn of steers at the end of the finishing trial are listed in Appendix Table A5. Liver Zn concentrations at the end of the grazing trial was 87.6 and 87.7 mg/kg DM for IZN treated and untreated steers respectively. At the end of the finishing trial, mean liver Zn concentrations were 110.2 and 109.5 mg/kg DM for IZN treated and untreated steers respectively.

Neither IZN nor ICU treatment had an effect ($P>0.05$) on liver Zn of steers at the end of the finishing trial (Appendix Table B18). There was no interaction ($P>0.05$) between ICU and IZN for liver Zn. Subplot analysis of variance showed a significant effect ($P<0.05$) of sampling day for liver Zn. All subplot interactions were not significant ($P>0.05$).

DISCUSSION

Clinical conditions, Copper status and Zinc status of steers at the start of the grazing trial.

All the steers were apparently healthy at the start of the grazing trial. There were no visible signs of either a Cu or Zn deficiency.

Initial Copper status

Underwood, (1977) suggested the normal range for liver Cu in cattle to be 100-400 mg/kg dry matter. More recently liver Cu has been defined as deficient, marginal, or adequate with values less than 40, 40-90 and over 90 mg/kg DM respectively (Agriculture Canada, 1981).

Group I steers started the grazing trial with a mean liver copper of 94.2 mg/kg dry matter which is indicative of an adequate Cu status. Group II steers had a mean concentration of 45.4 mg/kg DM; this value was indicative of a marginal Cu deficiency.

Mean serum Cu concentration for Group I steers was 0.90 ug/ml. In Group II mean serum Cu was 0.82 ug/ml. The normal accepted range for serum Cu has been set at 0.80-1.20 ug/ml (Beck, 1961; Smith et al., 1974). In cattle, serum Cu of 0.60 ug/ml or less has been reported as indicative of Cu deficiency (Underwood, 1977). Jamieson and Allcroft, (1950), Thornton et al., (1972a), Bingley and Anderson (1972), and

Thompson and Todd, (1976) have used 0.70 ugCu/ml as the lower limit for normal serum Cu. However, Claypool et al., (1975) reported that low Cu level in blood though indicative of Cu deficiency, does not necessarily represent the adequacy of body stores of Cu; they concluded that only with Cu of less than 0.50 ug/ml plasma was there a linear relationship with liver Cu of less than 40 mg/kg dry matter.

Boila et al., (1984a) have suggested that the serum Cu of cattle may be defined as deficient, marginal or adequate with values of serum Cu of less than 0.60, 0.61-0.70 and more than 0.70 ug/ml blood serum respectively. Based on this definition for serum Cu, steers of both Group I and Group II started the grazing trial with serum Cu levels indicative of an adequate Cu status.

Initial Zinc status

The concentration of Zn in liver and blood serum may be used to identify the Zn status of cattle. Liver Zn and blood serum Zn have both been defined as being deficient, marginal or adequate (Agriculture Canada, 1981). Liver Zn values less than 70, 70-90 and over 90 mg/kg dry matterfor cattle have been indicative of a deficient, marginal and adequate status respectively (Agriculture Canada, 1981). Serum Zn values less than 0.50, 0.50-0.70 and more than 0.70 ug/ml are described for cattle as indicative of a deficient, marginal or adequate status respectively (Agriculture Canada, 1981).

Based on the above definitions for serum Zn and liver Zn, steers of both Group I and Group II started the grazing trial with serum Zn and liver Zn indicative of an adequate Zn status (Appendix Tables A3 and A4).

Effect of ICU treatment on body weight of steers

Steers of both Group I (Fig.5) and Group II (Fig.7) gained weight throughout the grazing trial. Administration of ICU did not influence body weight of either Group I or Group II steers. In both Group I (Fig.5) and Group II (Fig.7) steers, rate of weight gain was slowest between sampling days 28 and 63 of the grazing trial. One possible explanation for this depression in growth rate was the long period of dry weather characterized by high environmental temperatures of between 30 to 40°C which may have resulted in heat stress. Heat stress is known to cause a reduction in feed consumption in many species of animals including cattle. The reduced feed consumption is usually accompained by a depression in body weight gains (Folk, 1974).

Copper supplementation of cattle has not always produced positive responses in body weight even in situations were cattle are known to be deficient in Cu. Studies with beef cattle (Drysdale, 1979) in Norhtwestern Manitoba showed that only four of sixty-four herd groups of cattle showed significant positive responses in body weights, while two herds showed significant negative responses. Bingley and Ander-

son, (1972), Thornton et al., (1972), Mills et al., (1976), MacPherson et al., (1979) and Boila et al., (1984a, 1984b) have reported variable responses of body weight to Cu supplementation. Donaldson et al., (1964) Miltimore et al.,(1964), Alexander et al., (1967), Maro and Kategile, (1980) and Plasto et al., (1983) reported significant increases in body weight of cattle following Cu supplementation. The variable responses obtained from Cu supplementation to cattle do revealed that an understanding of the Cu status of the animal is required before Cu supplementation is undertaken.

Several factors have been shown to influence body weight responses to Cu supplementation. These include the age of the animal, current Cu status, length of time the animal is exposed to Cu supplementation and the presence or absence of dietary components known to affect the utilization of Cu by ruminants (Thornton et al., 1972; Felsman et al., 1972; Maro and Kategile, 1980).

The age of cattle has been shown to influence body weight responses to Cu supplementation. Poole et al., (1974) indicated that the critical period for growing cattle to receive supplementatal Cu was the first five to six months of life. They noted that cattle are more likely to respond to Cu supplementation during this period of life. Chapman and Kidder, (1963), Clawson et al., (1972), Thornton et al., (1972b) and Suttle and Angus, (1976) have reported that cattle on low Cu

intake but not showing clinical symptoms of Cu deficiency may or may not show a response in body weight to Cu supplementation.

Dietary constituents such as Mo, S, Zn, Cd and Fe may affect the utilization of Cu by cattle. Molybdenum is known to interact with Cu in both the digestive tract and at tissue level (Mills et al., 1976). Thornton et al., (1972a) suggested that body weight response in cattle will occur only when Mo is the overriding cause of the Cu deficiency and concluded that the degree of response to Cu supplementation is determined by factors of management and environment. The lack of body weight response to Cu supplementation in present study may not have been due to the effect of other dietary constituents such as Mo, S, Zn and Fe which are known to influence the utilization of Cu. These minerals were all present in low concentrations relative to the requirement of cattle or relative to levels indicative of toxicities in cattle (Table 8; Appendix Table A6).

Effect of IZN treatment on body weight of steers

Body weight was influenced in IZN treated steers of Group I (Fig.5) with no difference for similarly treated steers of Group II (Fig.8). At sampling days 84 and 113, body weight of steers was lower for IZN treated steers of Group I (Fig.6), but there were no significant differences in body weight at sampling days 28 and 63.

There have been a few cases in which Zn supplementation was shown to reduce body weight gains in cattle (Ott et al., 1966a; Beeson et al., 1977) and in all of these cases, the quantity of supplemental Zn provided was much higher than those recommended for cattle (NRC, 1984). Ott et al., (1966b) reported that age, sex and breed influenced the response of cattle to Zn supplementation. They observed that steers were less tolerant to high dietary levels of Zn than heifers but noted that 500 mgZn/kg DM in the diet did not have an effect on body weight of either Group of steers.

The effect of IZN treatment in influencing body weight responses of Group I steers (Fig.6) in the present study was not consistent with the work of Miller and Miller, (1960), Ott et al., (1965), Mills et al., (1967) Perry et al., (1968), Beeson et al., (1977) and Mayland et al., 1980). These researchers reported significant positive increases in body weight following dietary Zn supplementation. Underwood and Somers (1969) reported significant effects of Zn supplementation upon body weight . However most of these researchers were working with Zn deficient cattle wherein the likelihood of response to Zn supplementation was greater.

The differences in response of body weight of Group I and Group II steers to IZN treatment could not have been due to Zn toxicity but could rather be due to breed differences. The two groups of steers were different breeds. Their previous nutritional history may have played a role in the sub-

sequent effect of IZN treatment on body weight; they came from different farms. The biochemical role of IZN in influencing body weight of steers in this study could not be identified but it may be speculated that there may have been an imbalance of some nutrients whose effects were accentuated by IZN treatment. Based on the lack of statistical interactions between ICU and IZN in all parameters, the Zn effect was presumably not due to an interaction with Cu.

Liver copper

In both Group I (Fig.16) and Group II (Fig.17) steers, there was an increase in liver Cu to approximately twice the pretreatment levels at sampling days 63 and 115 of the grazing trial. This increase in liver Cu occurred in both ICU treated and ICU untreated steers. For Group I, ICU treated steers had higher liver Cu than untreated steers at sampling days 63 and 115 of the grazing trial (Fig.16). The greatest increase in liver Cu occurred in Group II steers (Fig.16). For Group II mean liver Cu at sampling day 63 was approximately three times higher than that measured on day 0. This increase occured in both ICU treated and untreated steers. ICU treated steers of Group II had higher liver Cu at sampling day 63 but not at sampling day 115 (Fig.17).

The main difference between Group I and Group II steers in the response of liver Cu to ICU treatment was that in

Group I (Fig.16), ICU treated steers had higher liver Cu than ICU untreated steers at sampling days 63 and 115, while in Group II, liver Cu was higher (Fig.17) for ICU treated steers at sampling day 63. By sampling day 115, liver Cu was no longer higher for ICU treated steers of Group II (Fig.17). This difference in response of liver Cu of the two groups of steers may not be attributed to breed effect alone, since the Cu status of the two breeds was different at the start of the experiment. Group II steers started the grazing trial with a marginal liver Cu level while Group I had adequate liver Cu level. Thus the ability of a single dose of ICU in maintaining high levels of liver Cu in steers throughout the grazing appeared to have been influenced by the initial Cu status of the animal.

The increase in liver Cu of ICU untreated steers of both Group I and Group II during the grazing trial was in complete contrast to the results of Alexander et al., (1967) and Gleed et al., (1983) who reported significant decreases in liver Cu of steers after they have been turned onto pasture. One possible explanation for the increase in liver Cu in the present study might have been due to differences in the availability of Cu from the forages consumed by the steers. The concentrations of Mo and total S in the forage (Table 8) were relatively low and were not expected to have had a significant effect on the availability of herbage Cu to steers in this atudy.

Serum copper

Serum Cu of all steers (Fig.9) whether Group I (Fig.10) or Group II (Fig.11) fluctuated throughout the grazing trial reaching values at the end of the grazing trail lower than those measured on day 0. For both Group I and Group II steers, mean serum Cu did not fall below marginal levels. These patterns of serum Cu occurred in all steers whether ICU treated or untreated.

Serum copper of Group I steers

The fall in serum Cu of both ICU treated and untreated steers (Fig.10) during the first 28 days and subsequent rise by sampling day 63 may have been due to a number of factors. First, the Cu in young herbage is known to be less available compared to that in matured or conserved herbage (Hartmans and Bosman, 1970). The relative increase in serum Cu of ICU untreated steers between sampling days 28 and 63 and the subsequent decline to below adequate levels (<0.70 ug/ml) after sampling day 63 indicated that the Cu in the herbage though available was not sufficient to sustain normal serum Cu levels during a prolonged grazing trial. For steers treated with ICU, the initial decline in serum Cu during the first 28 days may have been also been due a slow release of Cu from the site of injection; the decline in serum Cu in ICU treated steers after day 84 suggested that a single dose

of prolontex-Cu was not sufficient to maintain serum Cu levels for more than four months. A second dose of ICU might not have been necessary as the serum Cu (Fig.10) and liver Cu (Fig.16) were above deficiency levels throughout the grazing trial.

Serum Copper for Group II steers

Though there was an increase in serum Cu in both ICU treated and untreated steers by sampling day 63 (Fig.11), this increase was not due to ICU treatment. Serum Cu of this group also fluctuated throughout the grazing trial (Fig.11). At the start of the grazing trial, 13% of the steers had serum Cu below 0.60 ug/ml, while at the end of the grazing trial, serum Cu in all steers was between 0.60-0.70 ug/ml.

Todd et al., (1967) reported a lack of response to Cu supplementation in cattle with low blood Cu and no visible symptoms, where the pasture and silage contained normal Cu, Mo and S. Similar results were obtained by Poole and Walshe, (1970) in which many cattle with a low Cu status on pastures of normal Cu and Mo content failed to respond to Cu supplementation. However, Allcroft (1950), Field (1957) and Lewis et al., (1957) have reported positive responses of serum Cu from copper therapy in the form of injections to cattle showing clinical symptoms of either absolute or induced hypocuposis. There is however, limited reports on the effect

of Cu in animals with low blood Cu in the absence of clearly defined subclinical of clinical Cu deficiency symptoms.

The lack of a response in serum Cu of Group I steers (Fig.11) to Cu supplementation in this trail may have been due to several factors. The concentration of Cu in blood serum is known to vary in relation to breed, age and sex (Christenson, 1980). The difference between Group I (Fig.10) and Group II (Fig.11) steers in the response of serum Cu may not have been due to breed alone. The Cu status of the two groups at the start of the grazing trial may have influenced the subsequent response to ICU treatment within Groups.

There appeared to be no direct relationship between serum Cu and liver Cu of steers during the grazing trial. While there was an increase in liver Cu of both Group I (Fig.16) and Group II (Fig.17) there was a gradual decline in serum Cu of both groups (whether ICU treated or untreated) over the grazing trial (Fig.10 and Fig.11) The lack of relationship between serum Cu and liver Cu to some extent suggested that both parameters should be considered when identifying the Cu status of cattle. Claypool et al., (1975) reported that there was a negligible increase in plasma Cu levels when liver Cu was above 40 mg/kg DM; they noted that there was a wide variation in plasma Cu for any given liver Cu levels above 40 mg/kg DM. These researchers suggested that both blood plasma and liver Cu be considered in identifying the Cu status of cattle.

Liver zinc

Administration of IZN did not influence liver Zn of either Group I or Group II steers; however, there was a gradual decrease in liver Zn in all steers throughout the grazing trial (Fig.19 and 20).

The lack of response of liver Zn to IZN treatment was not in agreement with the results of Ott et al., (1966a, 1966b, 1966c, 1966d), Miller et al., (1967), Allen (1968) and Combs et al., (1983). These researchers reported significant increases in liver Zn from normal or above normal dietary Zn supplementation. The absence of a marked decline in liver Zn in all steers whether IZN treated or untreated during the grazing trial (Fig.13 and 14) agreed with the results of Miller and Miller (1962) and Ott et al., (1964) who reported that Zn content of body tissues changes but very little during periods of low Zn intake. This indicates a lack of a specialized storaged form of Zn that could be mobilized during periods of low Zn intake.

Several factors may influence the effect of IZN treatment upon liver Zn. The Zn concentration in the liver of steers at the start of the grazing trial (Fig. 18 and 19) was indicative of an adequate Zn status. A response to IZN treatment was more likely to occur if the steers were Zn deficient. Also there may have been a rapid removal of Zn from the site of injection into the circulatory system, thereby

resulting in a rapid turn over rate and subsequent excretion in faeces and urine.

Studies with calves and goats (Miller et al., 1966b) showed that animals consuming diets containing adequate or high levels of Zn developed symptoms of Zn deficiency about three weeks after Zn was withdrawn from their diets. It appeared that the absence of liver Zn and serum Zn indicative of Zn deficiency in the present study may have been due to high availability of Zn from the herbage consumed by the steers.

Serum zinc

Though there was a decrease in serum Zn in all steers throughout the grazing trial, serum Zn did not fall to levels indicative of of Zn deficiency. The absence of a response of serum Zn to IZN treatment in this study is contrary to the work of Lamand (1978, 1980) who reported significant positive responses in serum Zn of sheep following injectable Zn supplementation.

Mills et al., (1967), Ott et al., (1964) and Perry et al., (1968) have reported significant increases in serum Zn concentration resulting from dietary Zn supplementation. The major difference between the work of these researchers and the work reported herein is in the method of supplementation. Also the steers used in this study were in an adequate

Zn status at the start of the grazing trial. At present there is very limited data on the use of injectable Zn preparations in cattle.

The absence of a marked decrease in serum Zn in all steers whether IZN treated or untreated may have been due to high availability of Zn from the herbage consumed by the steers. Serum Zn is known to reflect the dietary intake of the element and with the possible exception of the pancreas and bone, the Zn content of other tissues changes very little during periods of low Zn intake (Mills et al., 1967).

The concentration of Zn in the forage consumed by the steers in this trial are listed in Table 8 and Appendix Table A6. The concentration of Zn in all forage species was lower than the minimum requirement for cattle (NRC, 1984). A Zn deficiency has been reported in grazing cattle in which the forage Zn content of the herbage was 18.0-24.0 mg/kg DM (Legg and Sears, 1960), 19.0-83.0 mg/kg DM (Dynna and Harve, 1963) and 28-50 mg/kg DM (Haaranen, 1963). In most of these cases of Zn deficiency, the major contributing factors were other dietary components such as Ca, Cu, Cd, and Fe which are known to influence the utilization of Zn.

Haaranen (1963) suggested that the Zn requirement of grazing cows was approximately 45.0 mg/kg DM. Underwood and Somers (1969), Mills et al., (1967) and Miller and Miller (1960) have reported significant improvement in Zn status of

ruminants on rations in which the Zn content was lower than 40.0 mg/kg DM.

Mineral content in the forage consumed by steers during the grazing trial

The concentrations of Cu and Zn in all forage species (Table A5) were below the minimum recommended levels for cattle (Miltimore et al., 1970; ARC, 1980; NRC, 1984). Mo and S, two elements that influence the utilization of Cu were also present in low concentrations (Appendix Tables A5, A6 and Table 8). The Cu to Mo ratios in both grasses and legumes were greater than 4.0. Miltimore and Mason, (1971) and Alloway, (1973) have used Cu:Mo ratios in forages to determine whether a Cu deficiency was due to high levels of dietary Mo. Fisher and Waldern, (1978) suggested that a Cu:Mo ratio of 4:1 was preferred or ideal. This finding was in agreement with the suggestion of Alloway (1973) that a Cu:Mo ratio of 4:1 was the the critical value at above which Mo can no longer exert its adverse effect upon Cu utilization by ruminants. As a result of the increase in liver Cu to approximately twice the pretreatment levels and the absence of blood serum Cu indicative of Cu deficiency in all steers whether ICU treated or untreated, it was assumed that the availability of Cu from the forage was not significantly affected by either Mo or other dietary elements. Both liver

and serum Zn in all steers (Fig.12 and 18) did not fall below adequate levels. Thus a Zn deficiency as a result of the unavailability of Zn from the forage was not apparent.

All other elements in forages (Appendix Tables A6 and A7) were at low concentrations but not necessarily at severely deficient levels present in deficient concentrations (Appendix Tables A6 and A7) relative to the requirements of cattle (NRC. 1984).

Reactions at the site of Injection

No evidence of external swellings were noted for Prolontex-Cu or Prolontex-Zn at the sites of injection. Both preparations had been injected intramuscularly. Injectable preparations of Cu do elicit some reactions to varying degrees at the sites of injection, although the swellings caused by those preparations did disappear over the grazing period (Boila et al., 1984a).

Examination of carcass at the slaughter plant revealed that there were no external evidence of reactions on the carcass of steers injected with either Prolontex-Cu or Prolontex-Zn.

Finishing Trial

After the termination of the grazing trial, the steers were taken to the Glenlea Research station where they were kept for two weeks before being placed on the finishing ration. Of the 44 steers placed in the finishing trial, 37 were from Group I and 7 from Group II. Because of the small number of Group II steers in the finishing trial, no attempt was made to determine any group effects. All steers were treated as either ICU or IZN treated or untreated.

Effect of ICU and IZN treatment upon body weight of steers

All steers grew rapidly during the finishing trial, whether ICU treated (Fig.21) or IZN treated (Fig.22). During the two weeks interval between the end of the grazing trial and the start of the finishing trial, all steers had a reduced growth rate (Fig.23 and 24). Growth rate was greatly improved when they were placed on the finishing ration.

Steers treated with ICU on day 0 of the grazing trial grew less than ICU untreated steers during the finishing trial (Fig.21) There was no interaction between ICU and IZN for body weight of steers. The effect of ICU treatment at the start of the grazing trial in reducing body weight gains during the finishing phase was not identified as ICU treatment did not affect body weights of steers during the grazing trial.

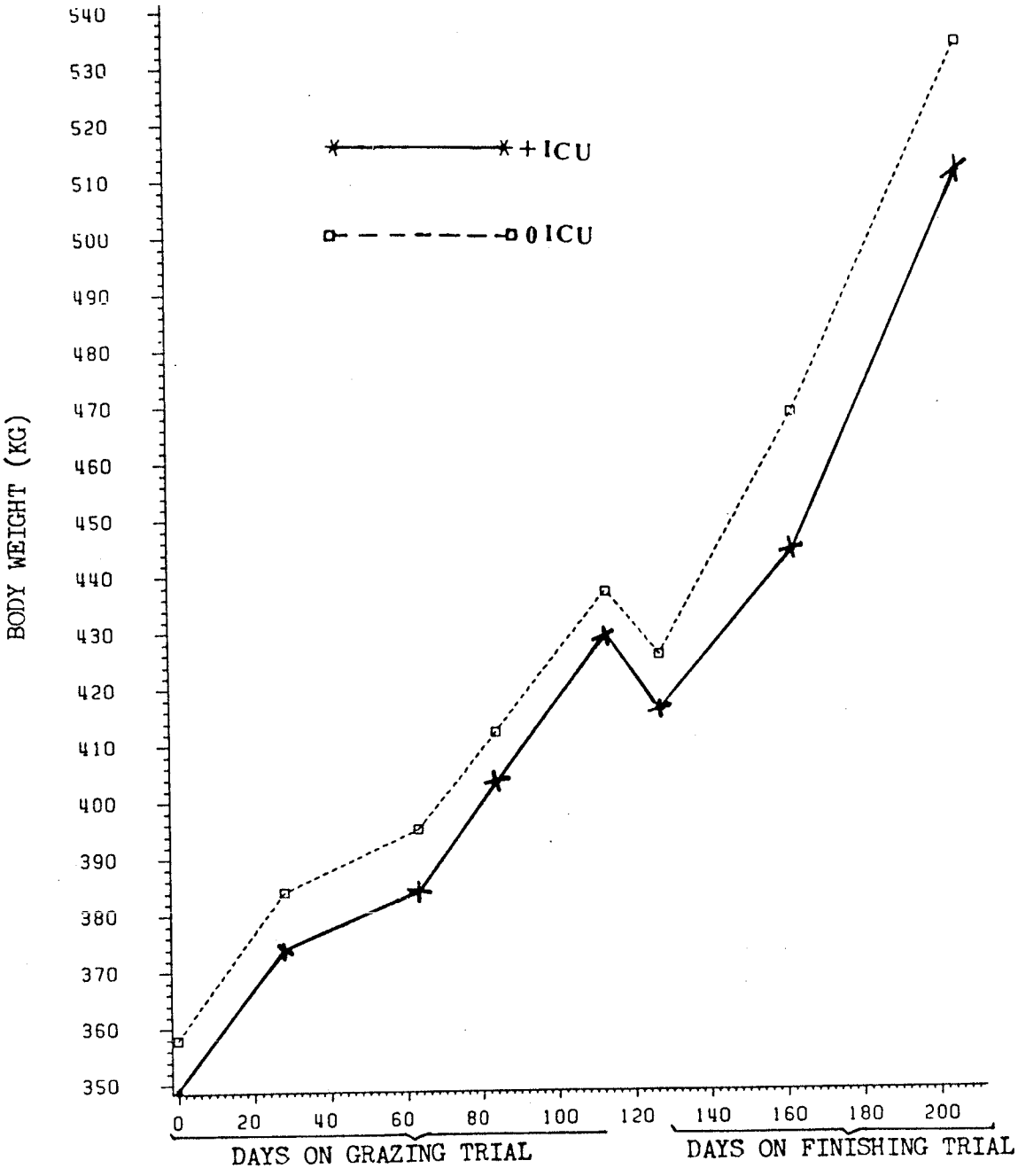


Fig. 23: Least square means for body weight of ICU treated (+ICU) or non treated (OICU) steers during the grazing and finishing trials. Treated steers received a dose of Prolontex-Cu at the start of the grazing trial.

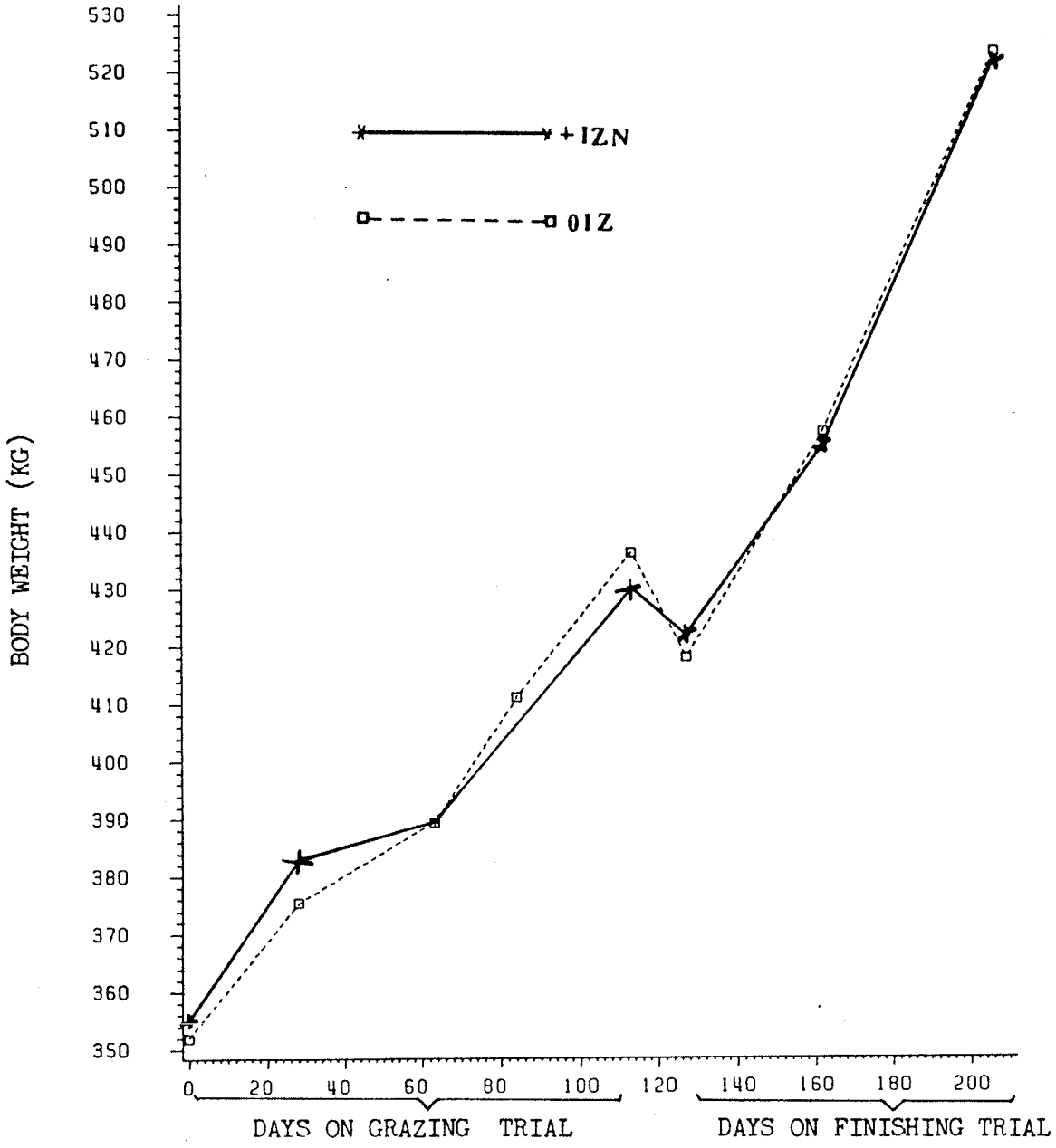


Fig. 24: Least square means for body weight of IZN-treated or non treated steers during the grazing and finishing trials. Treated steers received a dose of Prolontex-Zn at the start of the grazing trial.

Administration of IZN at the start of the grazing trial did not have an influence on body weight of steers during the finishing trial (Fig.22).

Liver copper

There was a marked decrease in liver Cu in both ICU treated and untreated steers during the finishing trial (Fig.25). The decrease in liver Cu may have been due to the low content of Cu in barley fed to the steers (Table 7a) thereby leading to mobilization of Cu from the liver to meet metabolic needs. As the steers were growing rapidly there was the probability of an increased demand for Cu.

The concentration of Cu in the barley used in the finishing ration was 4.3 mg/kg DM (Table 7a). Diets with less than 3 to 4 mg Cu/kg DM have been reported to result in subnormal plasma and liver Cu levels in cattle (Goodrich 1968). In the present study ICU treatment at day 0 of the grazing trial was not effective in maintaining high liver Cu into the finishing trial.

Liver zinc

There was an increase in liver Zinc in all steers whether IZN treated or untreated (Fig.26). IZN treatment at the start of the grazing trial did not have an influence

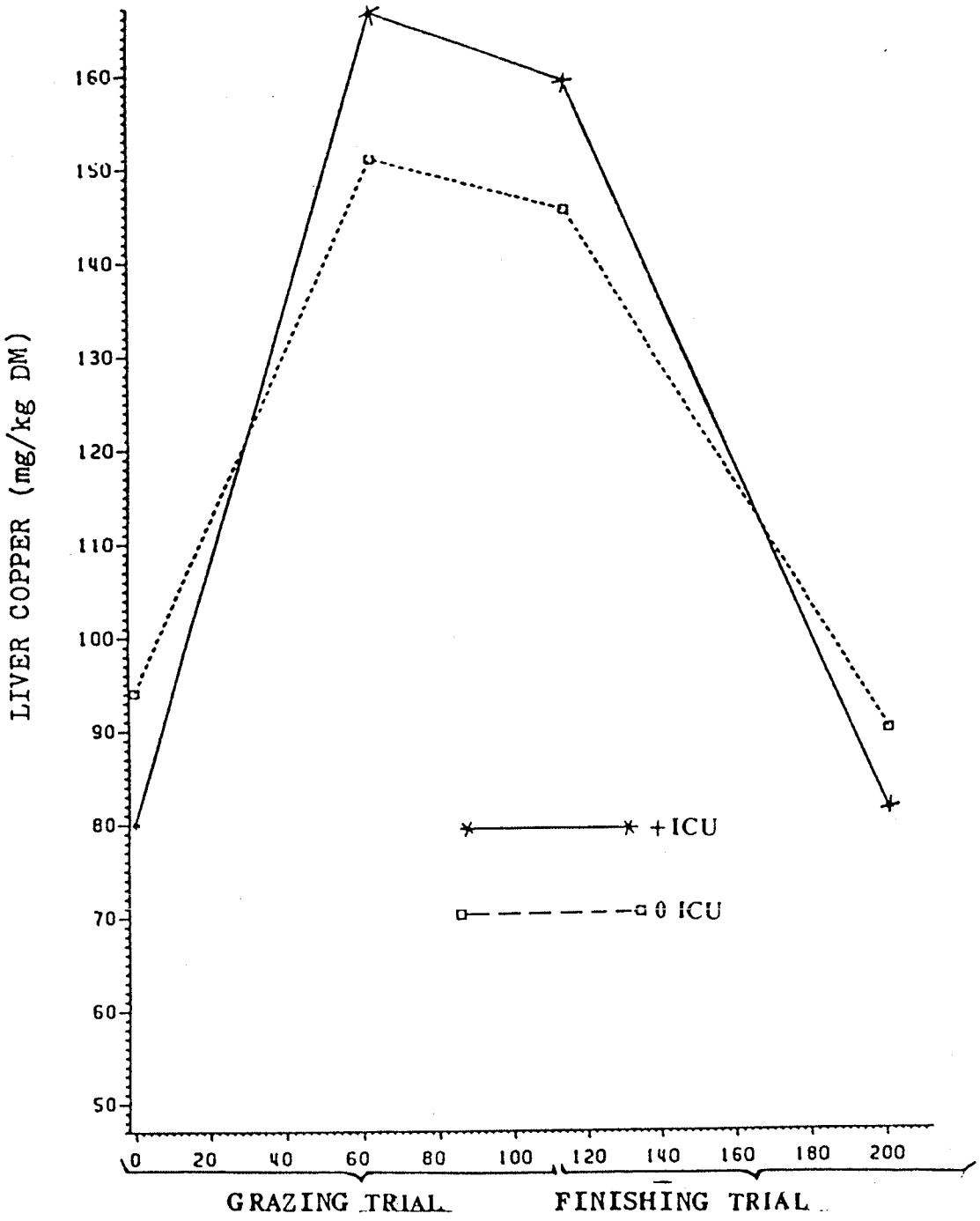


Fig. 25: Changes in concentration of liver copper of steers during the grazing and finishing trials. ICU treated steers (+ICU) received a dose of Prolontex-Cu at the start of the grazing trial.

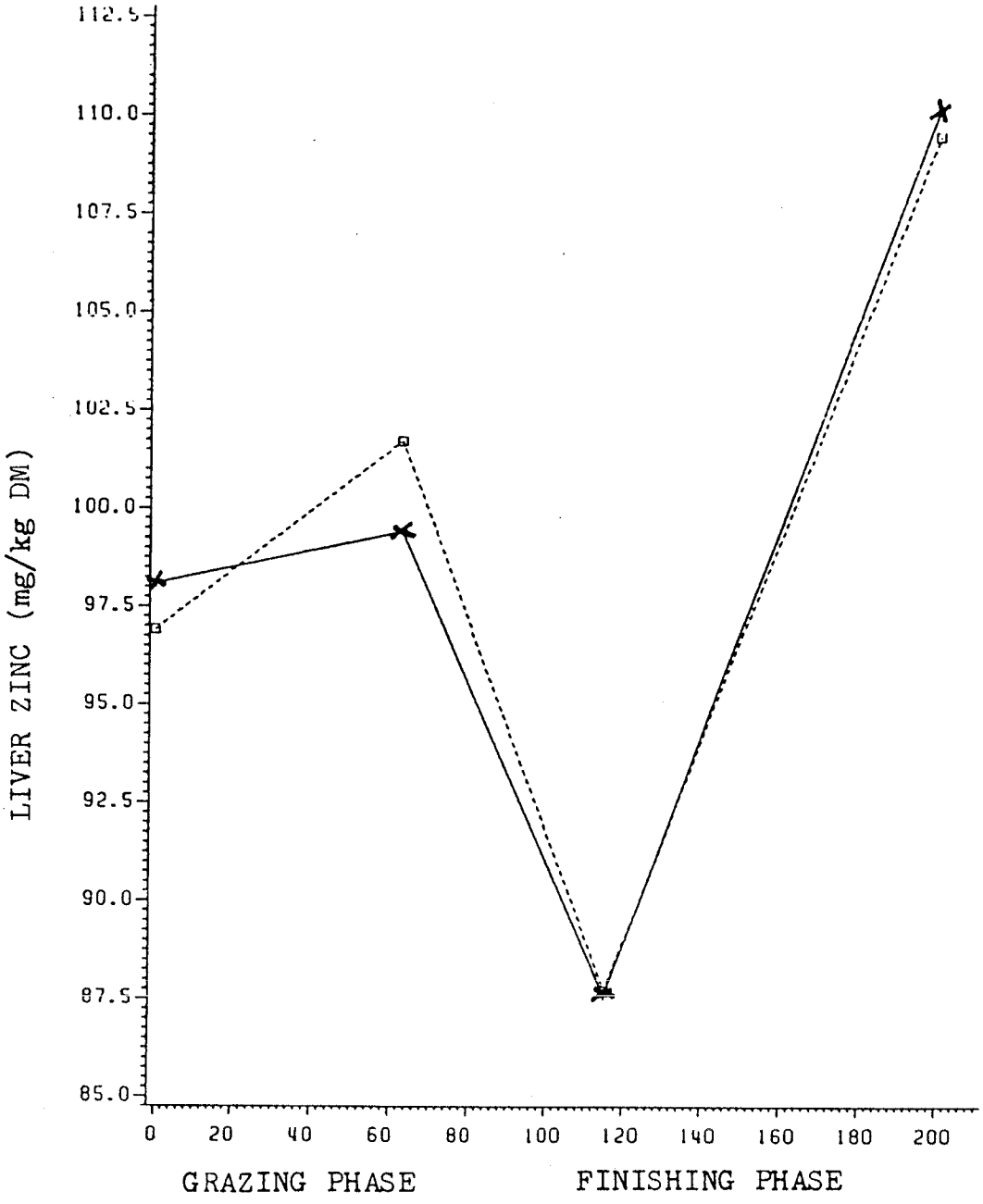


Fig.26 Mean concentrations of liver zinc of IZN treated (IZN) and untreated (OIZN) steers from day 0 of grazing trial to end of finishing trial.

on liver Zn during the finishing trial. The increase in liver Zn during the finishing trial may have been due to high availability of Zn from the finishing ration. The Zn content in the barley was 26.4 mg/kg DM (Table 7a). The Zn content in the finishing ration was much higher than that in the forage consumed by the steers during the grazing trial (Table 8).

SUMMARY AND CONCLUSION

Thirty-seven Herefords (Group I) and twenty-three Angus-Charolais-North-Devon crosses (Group II) yearling beef steers were used in a 113-day grazing trial at the community pasture at Narcisse in the summer of 1983. Steers were divided into four treatment groups: Control, ICU, IZN and ICU + IZN.

Body Weights Response to Treatment

1. Steers of both Group I and Group II grew throughout the the grazing trial.

2. ICU treatment did not influence ($P>0.05$) body weights of either Group I or Group II steers ($P>0.05$).

3. There was a significant ($P<0.05$) IZN x sampling day interaction. IZN treatment significantly reduced body weight gains of Group I steers at sampling days 84 and 115 of the grazing trial, but did not influence ($P>0.10$) body weights of similarly treated steers of Group II.

4. There was no interaction ($P>0.05$) between ICU and IZN for body weights of either Groups of steers.

Serum Copper Responses to Treatment

1. In Group I, ICU treated steers had higher ($P<0.05$) serum Cu at sampling day 84 of the grazing trial but at sampling day 113, there were no significant ($P>0.05$) differences in serum Cu of ICU treated and ICU untreated steers ($P>0.05$).

2. In Group II, ICU treatment did not influence ($P>0.05$) serum Cu. Serum Cu fluctuated throughout the grazing trial.

Liver Copper Responses to Treatment

1. In Group I, ICU treated steers had higher ($P<0.05$) liver Cu than ICU untreated steers at sampling days 63 and 115 of the grazing trial.

2. In Group II, ICU treated steers had higher ($P<0.05$) liver Cu than ICU untreated steers at sampling day 63 but not at sampling day 115 ($P>0.05$).

3 For both Group I and Group II steers, there was no interaction ($P>0.05$) between ICU and IZN treatment for liver Cu.

Liver and Serum Zn

1 For both Group I and Group II, IZN treatment did not have an influence on either liver or serum Zn of steers ($P>0.05$).

Forage Analysis

1. Forage analysis showed low concentrations of Cu and Zn in both grasses and legumes. The concentrations of these minerals (Cu and Zn) were marginal relative to requirements of cattle. Molybdenum and S were less than 1.5 mg and 2.0 g/kg DM in grasses and legumes repectively. Iron and Mn were normal in all species; Ca was higher in legumes than in grasses. Phosphorus was low in both grasses and legumes. Magnesium was marginal in grasses as well as in legumes.

Finishing Trial

1 ICU treatment at day 0 of the grazing trial significantly reduced body weight responses of steers during the finishing trial.

2 IZN treatment at day 0 of the grazing trial did not influence ($P>0.05$) body weight responses of steers during the finishing trial.

3. Carcass weight as a percent of body weight was 56.4% in both ICU treated and IZN treated.

4. ICU treatment at day 0 of the grazing trial did not influence ($P>0.05$) liver Cu of steers. But liver Cu decreased in all steers whether ICU treated or ICU untreated.

5. IZN treatment at day 0 of the grazing trial did not influence ($P>0.05$) liver Zn of steers during the finishing trial. There was an increase in liver Zn of both IZN treated and IZN untreated steers at the end of the finishing trial.

Efficacy of Prolontex-Cu

Prolontex-Cu did not modify body weight responses during grazing trial. Body weight responses were lower for these Prolontex-Cu treated steers during the finishing trial. Previous nutrition of these cattle during the grazing trial with respect to Cu treatment did modify growth during the finishing trial.

During the grazing trial, liver Cu of prolontex-Cu treated steers had increased to levels above that of untreated steers. The effect of Prolontex-Cu lasted into the finishing

trial, with no difference in level of Cu between Prolontex-Cu treated and untreated steers at the end of the finishing trial. Serum Cu levels during the grazing trial were indicative of a response to injectable Cu treatment, but this response was not as clearly indicated as was demonstrated with the concentration of Cu in liver tissue.

Efficacy of Prolontex-Zn

The provision of Prolontex-Zn to steers at the start of the grazing trial, had reduced the growth rate of steers by the end of the grazing trial. This effect of Prolontex-Zn could have been due to metabolic interactions with other minerals or dietary constituents.

Since no statistical ICU x IZN interaction was noted for the parameters studied, the provision of Prolontex-Zn did not modify the Cu metabolism of cattle. The reduction in growth due to Prolontex-Zn did not carry over to the finishing trial.

The concentrations of Zn in liver and serum were not modified by the provision of Prolontex-Zn. Based on these analyses for Zn, cattle were not Zn deficient, and appeared not to require supplemental Zn, either during the grazing trial or during the finishing trial.

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A P P E N D I X

Appendix Table A1: Means for body weight, serum copper, liver copper, serum zinc and liver zinc for group I and group II on May 10, 1983.

Treatment	Variable	Number of steers	Mean	SE. of Mean
		Group I		
A				
Control	WT	9	332.8	6.6
ICU=0	SC	9	0.81	0.04
	LCD	9	141.72	7.36
IZN=0	SZ	9	0.64	0.04
	LZD	9	94.31	5.65
B				
ICU=1	WT	9	333.10	5.4
IZN=0	SC	9	0.81	0.02
	LD	9	126.57	7.64
	SZ	9	0.63	0.03
	LZD	9	104.28	6.00
C				
ICU=0	WT	10	335.1	5.8
IZN=1	SC	10	0.80	0.04
	LCD	10	126.72	11.52
	SZ	10	0.61	0.03
	LZD	10	85.31	7.52
D				
ICU=1	WT	9	337.4	6.6
IZN=1	SC	9	0.77	0.03
	LCD	9	134.54	7.29
	SZ	9	0.64	0.03
	LZD	9	92.28	6.18
		Group II		
A				
Control	WT	7	264.6	16.5
ICU=0	SC	7	0.64	0.03
	LCD	7	52.94	9.52
IZN=0	SZ	7	0.73	0.04
	LZD	7	87.32	3.53

Appendix Table A1: (Cont'd).

Treatment	Variable	Number of steers	Mean	SE. of Mean
B				
ICU=1	WT	6	275.0	19.0
IZN=0	SC	6	0.70	0.03
	LD	6	59.05	16.18
	SZ	6	0.68	0.02
	LZD	6	92.24	7.03
C				
ICU=0	WT	4	277.7	31.6
IZN=1	SC	4	0.65	0.04
	LCD	4	62.92	33.0
	SZ	4	0.71	0.05
	LZD	4	93.26	10.18
D				
ICU=1	WT	6	273.4	20.9
IZN=1	SC	6	0.69	0.03
	LCD	6	52.54	10.19
	SZ	6	0.76	0.03
	LZD	6	91.72	16.29

WT - Weight in Kilograms

SC - Serum Copper (ug/ml)

LCD - Liver copper (mg/kg Dry matter basis)

SZ - Serum zinc (ug/ml)

LZD - Liver zinc (mg/kg Dry matter basis)

Appendix Table A2
Raw Data for Individual Steers Measured
on May 10, During the Grazing and Finishing Trials

Explanation of Abbreviations:

Period refers to Sampling day

Period 1 - May 10, 1983

2 - June 10/13, 1983

3 - July 12, 1983

4 - August 16, 1983

5 - September 6, 1983

6 - October 4/6, 1983

7 - October 18, 1983

8 - November 22, 1983

9 - January 5, 1984

Periods 2, 3, 4, 5 and 6 comprise the grazing trial.

Periods 7, 8 and 9 comprise the finishing phase.

Group A (Also referred to as Group I) - HEREFORDS

Group G (Also referred to as Group II) - ANGUS - CHAROLAIS-
North-Devon Crosses.

ICU - Injectable Copper; OICU - Without Injectable Copper
IZN - Injectable Zinc; OIZN - Without Injectable Zinc
WT. - Weight (lb.), Kg = lb (2.2025)
LCW - Liver Copper (Wet basis) mg/kg wet basis
LCD - Liver Copper (Dry basis) mg/kg dry matter basis
LZW - Liver Zinc (Wet basis) mg/kg wet basis
LZd - Liver Zinc (Dry basis) mg/kg dry matter basis
SC - Serum Zinc ug/ml
CARWT - Carcass weight (lb) Kg = lb (2.2025)

OBS	PERIOD	GROUP	ANIM	ICU	IZN	WT (lb)	LCW	LCD	LZW	LZD	SC	SZ	CARWT
1	1	G	81	0	0	778.8	14.84	51.50	21.47	74.52	0.599	0.737	.
2	1	G	126	0	0	611.6	11.48	38.90	29.42	99.59	0.665	0.599	.
3	1	G	21	0	0	585.2	11.04	37.70	27.23	92.84	0.671	0.875	.
4	1	G	129	0	0	558.8	19.39	66.90	24.42	84.23	0.602	0.750	.
5	1	G	131	0	0	497.2	7.62	22.60	32.72	96.79	0.593	0.704	.
6	1	G	57	0	0	497.2	27.40	100.44	23.04	84.50	0.771	0.794	.
7	1	G	88	0	0	550.0	14.87	52.57	22.29	78.80	0.585	0.630	.
8	1	A	160	0	0	717.2	28.60	102.97	33.33	120.04	0.949	0.680	.
9	1	A	123	0	0	682.0	32.80	122.33	18.27	68.18	0.721	0.544	.
10	1	A	303	0	0	739.2	38.11	131.05	28.23	97.08	0.816	0.696	.
11	1	A	149	0	0	814.0	34.56	131.34	19.16	72.83	0.784	0.784	.
12	1	A	301	0	0	699.6	42.43	144.31	26.73	90.92	0.854	0.557	.
13	1	A	99	0	0	745.8	40.66	149.01	31.32	114.78	0.664	0.811	.
14	1	A	115	0	0	690.8	42.52	157.27	26.64	99.51	0.720	0.636	.
15	1	A	134	0	0	726.0	46.43	163.41	25.35	89.22	0.796	0.535	.
16	1	A	188	0	0	783.2	48.51	173.79	26.86	96.21	1.006	0.544	.
17	1	G	135	1	0	787.6	4.97	17.60	24.02	84.81	0.628	0.664	.
18	1	G	66	1	0	620.4	11.28	39.50	20.53	71.94	0.652	0.641	.
19	1	G	90	1	0	572.0	17.78	66.20	21.38	79.60	0.787	0.787	.
20	1	G	41	1	0	572.0	20.07	73.30	25.29	92.39	0.680	0.637	.
21	1	G	63	1	0	475.2	9.72	30.30	32.62	117.27	0.778	0.656	.
22	1	G	130	1	0	607.2	29.52	127.42	24.89	107.42	0.667	0.722	.
23	1	A	97	1	0	690.8	27.47	89.83	32.05	104.81	0.762	0.577	.
24	1	A	140	1	0	726.0	30.93	102.90	32.14	106.90	0.701	0.712	.
25	1	A	182	1	0	704.0	31.14	107.31	28.48	98.13	0.916	0.672	.
26	1	A	183	1	0	770.0	36.81	132.06	28.22	94.34	0.799	0.537	.
27	1	A	173	1	0	743.6	36.54	123.25	27.15	91.58	0.769	0.513	.
28	1	A	174	1	0	792.0	35.94	132.22	31.72	116.69	0.891	0.777	.
29	1	A	120	1	0	756.8	39.15	143.33	25.36	92.87	0.848	0.557	.
30	1	A	156	1	0	730.4	49.30	149.23	48.08	145.54	0.816	0.673	.
31	1	A	166	1	0	688.6	43.53	158.96	24.65	87.65	0.818	0.641	.
32	1	G	47	0	1	818.4	14.23	47.59	23.92	79.97	0.656	0.701	.
33	1	G	22	0	1	563.2	5.72	20.02	.	.	0.608	0.772	.
34	1	G	127	0	1	545.6	7.06	23.93	33.43	113.26	0.539	0.796	.
35	1	G	78	0	1	519.2	44.78	160.14	24.20	86.55	0.736	0.555	.
36	1	A	177	0	1	712.8	13.84	49.65	6.98	25.04	0.689	0.452	.
37	1	A	304	0	1	693.0	29.43	94.90	30.35	97.86	0.869	0.637	.
38	1	A	118	0	1	792.0	34.53	112.87	27.71	90.59	0.844	0.724	.
39	1	A	153	0	1	712.8	32.93	121.62	31.49	116.33	1.115	0.655	.
40	1	A	145	0	1	730.4	39.60	129.63	25.60	83.80	0.685	0.696	.
41	1	A	101	0	1	822.8	38.30	134.69	25.59	89.98	0.728	0.501	.
42	1	A	161	0	1	748.0	35.51	135.15	24.00	91.35	0.714	0.749	.
43	1	A	199	0	1	712.8	42.11	147.37	22.67	79.34	0.726	0.542	.
44	1	A	105	0	1	717.2	54.43	157.24	27.66	79.91	0.737	0.580	.
45	1	A	194	0	1	739.2	54.17	184.09	29.11	98.91	0.906	0.572	.
46	1	G	34	1	1	814.0	11.80	47.45	20.99	84.40	0.805	0.839	.
47	1	G	132	1	1	633.6	11.43	42.53	21.75	80.93	0.743	0.779	.

OBS	PERIOD	GROUP	ANIM	ICU	IZN	WT (LB)	LCW	LCD	LZW	LZD	SC	SZ	CARWT
48	1	G	9	1	1	572.0	12.72	46.39	46.47	169.52	0.638	0.752	.
49	1	G	60	1	1	545.6	12.51	55.56	13.76	61.11	0.716	0.716	.
50	1	G	12	1	1	501.6	6.80	24.43	17.31	63.36	0.619	0.664	.
51	1	G	71	1	1	545.6	27.10	98.90	24.95	91.01	0.618	0.817	.
52	1	A	119	1	1	756.8	24.18	93.61	16.24	62.91	0.652	0.640	.
53	1	A	147	1	1	726.0	35.21	118.70	25.81	87.00	0.838	0.535	.
54	1	A	136	1	1	827.2	34.41	127.07	23.38	83.61	0.871	0.734	.
55	1	A	162	1	1	708.4	40.94	126.25	28.49	87.88	0.691	0.584	.
56	1	A	155	1	1	704.0	39.90	134.07	33.17	111.55	0.807	0.646	.
57	1	A	104	1	1	787.6	38.91	136.90	29.53	103.90	0.871	0.680	.
58	1	A	148	1	1	708.4	38.83	154.12	31.54	122.02	0.760	0.760	.
59	1	A	175	1	1	708.4	44.64	153.51	20.90	73.73	0.821	0.512	.
60	1	A	108	1	1	761.2	48.40	166.60	28.45	97.92	0.631	0.633	.
61	2	G	81	0	0	853.6	21.59	41.71	40.23	77.72	0.590	1.050	.
62	2	G	126	0	0	673.2	24.49	33.82	65.29	90.14	0.500	0.860	.
63	2	G	21	0	0	598.4	12.26	31.10	37.20	94.34	1.290	1.590	.
64	2	G	129	0	0	620.4	19.39	37.61	48.05	93.18	0.940	1.610	.
65	2	G	131	0	0	558.8	18.76	23.61	108.84	136.98	0.750	1.360	.
66	2	G	57	0	0	541.2	67.04	95.75	79.41	113.41	0.860	1.200	.
67	2	G	88	0	0	598.4	.	.	.	.	.	.	.
68	2	A	160	0	0	730.4	42.63	89.66	44.75	94.14	0.670	0.940	.
69	2	A	123	0	0	743.6	63.57	93.14	62.02	90.86	1.080	1.640	.
70	2	A	303	0	0	765.6	31.52	79.75	37.54	94.97	0.760	0.960	.
71	2	A	149	0	0	875.6	34.68	104.12	33.48	100.51	0.740	0.960	.
72	2	A	301	0	0	761.2	77.36	87.23	82.83	93.40	0.940	1.260	.
73	2	A	99	0	0	783.2	61.35	98.30	100.50	161.04	0.810	1.050	.
74	2	A	115	0	0	748.0	30.35	90.45	30.91	92.13	0.920	1.270	.
75	2	A	134	0	0	792.0	61.88	128.91	44.40	92.49	0.850	1.000	.
76	2	A	188	0	0	858.0	90.85	134.87	57.42	85.23	0.960	0.990	.
77	2	G	135	1	0	840.4	7.89	17.54	41.33	91.93	0.880	1.190	.
78	2	G	66	1	0	671.0	15.90	22.91	51.51	74.23	0.720	0.780	.
79	2	G	90	1	0	640.2	26.20	53.83	39.54	81.25	0.750	1.030	.
80	2	G	41	1	0	596.2	35.23	62.98	58.32	104.24	1.100	1.300	.
81	2	G	63	1	0	521.4	9.78	19.65	46.48	93.43	0.760	0.870	.
82	2	G	130	1	0	675.4	25.14	58.73	38.61	90.20	0.870	0.950	.
83	2	A	97	1	0	752.4	51.26	70.47	76.40	105.03	0.960	1.400	.
84	2	A	140	1	0	770.0	35.31	70.36	56.23	112.05	0.590	1.060	.
85	2	A	182	1	0	726.0	67.41	75.31	82.01	91.62	0.740	0.810	.
86	2	A	183	1	0	842.6	39.10	88.58	54.24	122.88	0.910	1.240	.
87	2	A	173	1	0	759.0	42.42	68.51	71.67	115.75	1.000	1.220	.
88	2	A	174	1	0	860.2	36.43	77.19	46.66	98.86	1.110	1.560	.
89	2	A	120	1	0	789.8	33.32	62.77	53.68	101.14	0.810	0.930	.
90	2	A	156	1	0	765.6	105.56	115.99	83.91	92.20	1.210	1.570	.
91	2	A	166	1	0	796.4	70.99	124.16	60.23	105.33	1.240	1.510	.
92	2	G	47	0	1	886.6	16.70	48.85	34.55	101.05	0.990	1.340	.
93	2	G	22	0	1	618.2	14.08	16.30	105.61	122.31	0.730	0.990	.
94	2	G	127	0	1	563.2	23.98	29.94	82.93	103.56	0.470	0.870	.

OBS	PERIOD	GROUP	ANIM	ICU	IZN	WT (lb)	LCW	LCD	LZW	LZD	SC	SZ	CARWT
95	2	G	78	0	1	545.6	71.58	119.75	57.82	96.74	0.670	0.570	.
96	2	A	177	0	1	765.6	40.45	85.43	41.62	87.90	0.670	0.850	.
97	2	A	304	0	1	743.6	57.95	65.88	81.67	92.84	0.850	1.210	.
98	2	A	118	0	1	875.6	79.52	87.87	89.48	98.88	1.080	1.410	.
99	2	A	153	0	1	765.6	36.02	98.25	35.71	97.42	0.820	0.820	.
100	2	A	145	0	1	765.6	41.96	92.56	47.28	104.29	0.810	0.930	.
101	2	A	101	0	1	875.6	45.19	83.33	55.14	101.67	0.990	1.510	.
102	2	A	161	0	1	785.4	79.00	159.16	59.22	119.30	0.750	1.040	.
103	2	A	199	0	1	761.2	54.38	115.82	44.26	94.28	0.670	0.850	.
104	2	A	105	0	1	761.2	96.98	117.60	80.47	97.65	0.790	1.050	.
105	2	A	194	0	1	774.4	44.43	142.54	34.96	112.15	1.240	1.420	.
106	2	G	34	1	1	858.0	17.19	36.97	40.23	86.49	0.850	1.170	.
107	2	G	132	1	1	688.6	33.53	38.08	97.18	110.38	0.880	1.270	.
108	2	G	9	1	1	611.6	22.64	45.03	51.04	101.52	0.730	1.050	.
109	2	G	60	1	1	583.0	22.77	45.58	41.26	82.59	0.890	1.130	.
110	2	G	12	1	1	534.6	15.04	24.75	58.11	95.66	1.100	1.320	.
111	2	G	71	1	1	591.8	36.77	80.29	38.09	83.18	0.790	1.150	.
112	2	A	119	1	1	798.6	38.84	55.12	66.97	95.05	0.600	0.880	.
113	2	A	147	1	1	767.8	34.23	79.52	36.84	85.57	0.560	0.710	.
114	2	A	136	1	1	853.6	36.27	79.25	40.16	87.75	0.790	0.990	.
115	2	A	162	1	1	708.4	41.60	98.38	36.28	85.79	0.640	1.010	.
116	2	A	155	1	1	737.0	66.31	97.36	77.01	113.08	1.260	1.570	.
117	2	A	104	1	1	822.8	29.77	80.92	38.03	103.38	0.910	1.210	.
118	2	A	148	1	1	712.8	36.41	72.07	43.68	86.47	0.980	1.600	.
119	2	A	175	1	1	723.8	92.14	113.69	68.48	84.50	0.690	0.720	.
120	2	A	108	1	1	765.6	40.69	112.88	40.51	112.39	0.910	1.640	.
121	3	G	81	0	0	875.0	.	.	.	.	0.650	0.868	.
122	3	G	126	0	0	715.0	.	.	.	.	0.839	0.973	.
123	3	G	21	0	0	620.0	.	.	.	.	0.845	0.779	.
124	3	G	129	0	0	680.0	.	.	.	.	0.641	0.877	.
125	3	G	131	0	0	620.0	.	.	.	.	0.675	0.929	.
126	3	G	57	0	0	595.0	.	.	.	.	0.840	1.089	.
127	3	G	88	0	0	635.0	.	.	.	.	0.651	0.659	.
128	3	A	160	0	0	820.0	.	.	.	.	0.890	1.220	.
129	3	A	123	0	0	795.0	.	.	.	.	0.546	0.962	.
130	3	A	303	0	0	810.0	.	.	.	.	0.733	1.018	.
131	3	A	149	0	0	945.0	.	.	.	.	0.851	0.878	.
132	3	A	301	0	0	800.0	.	.	.	.	0.843	0.789	.
133	3	A	99	0	0	870.0	.	.	.	.	0.551	0.848	.
134	3	A	115	0	0	800.0	.	.	.	.	0.659	0.903	.
135	3	A	134	0	0	850.0	.	.	.	.	0.691	0.795	.
136	3	A	188	0	0	945.0	.	.	.	.	0.805	0.895	.
137	3	G	135	1	0	865.0	.	.	.	.	0.586	0.713	.
138	3	G	66	1	0	675.0	.	.	.	.	0.626	0.659	.
139	3	G	90	1	0	680.0	.	.	.	.	0.685	0.718	.
140	3	G	41	1	0	620.0	.	.	.	.	1.043	1.335	.

OBS	PERIOD	GROUP	ANIM	ICU	IZN	WT (lb)	LCW	LCD	LZW	LZD	SC	SZ	CARWT
141	3	G	63	1	0	535.0	.	.	.	.	1.030	0.807	.
142	3	G	130	1	0	685.0	.	.	.	.	0.700	0.742	.
143	3	A	97	1	0	800.0	.	.	.	.	0.674	0.804	.
144	3	A	140	1	0	800.0	.	.	.	.	0.601	0.858	.
145	3	A	182	1	0	775.0	.	.	.	.	0.669	0.807	.
146	3	A	183	1	0	825.0	.	.	.	.	0.679	0.819	.
147	3	A	173	1	0	820.0	.	.	.	.	0.710	0.760	.
148	3	A	174	1	0	955.0	.	.	.	.	0.998	1.340	.
149	3	A	120	1	0	820.0	.	.	.	.	0.679	0.725	.
150	3	A	156	1	0	815.0	.	.	.	.	0.598	0.680	.
151	3	A	166	1	0	890.0	.	.	.	.	0.761	0.913	.
152	3	G	47	0	1	910.0	.	.	.	.	0.939	1.245	.
153	3	G	22	0	1	635.0	.	.	.	.	0.971	1.338	.
154	3	G	127	0	1	640.0	.	.	.	.	0.575	1.053	.
155	3	G	78	0	1	570.0	.	.	.	.	0.808	0.708	.
156	3	A	177	0	1	815.0	.	.	.	.	1.035	1.135	.
157	3	A	304	0	1	790.0	.	.	.	.	0.738	0.738	.
158	3	A	118	0	1	960.0	.	.	.	.	0.765	0.643	.
159	3	A	153	0	1	830.0	.	.	.	.	0.514	0.642	.
160	3	A	145	0	1	800.0	.	.	.	.	1.001	0.752	.
161	3	A	101	0	1	890.0	.	.	.	.	0.705	0.890	.
162	3	A	161	0	1	880.0	.	.	.	.	0.606	0.902	.
163	3	A	199	0	1	855.0	.	.	.	.	0.984	0.905	.
164	3	A	105	0	1	820.0	.	.	.	.	0.613	0.730	.
165	3	A	194	0	1	865.0	.	.	.	.	0.851	0.968	.
166	3	G	34	1	1	845.0	.	.	.	.	1.023	1.216	.
167	3	G	132	1	1	770.0	.	.	.	.	0.786	0.985	.
168	3	G	9	1	1	625.0	.	.	.	.	0.708	0.850	.
169	3	G	60	1	1	.	.	.	.	.	0.893	1.113	.
170	3	G	12	1	1	550	.	.	.	.	1.315	1.187	.
171	3	G	71	1	1	615	.	.	.	.	0.519	0.713	.
172	3	A	119	1	1	885	.	.	.	.	0.514	0.642	.
173	3	A	147	1	1	825	.	.	.	.	0.628	0.627	.
174	3	A	136	1	1	900	.	.	.	.	0.861	1.279	.
175	3	A	162	1	1	760	.	.	.	.	0.600	0.662	.
176	3	A	155	1	1	785	.	.	.	.	0.979	0.878	.
177	3	A	104	1	1	875	.	.	.	.	0.950	1.158	.
178	3	A	148	1	1	800	.	.	.	.	0.781	1.262	.
179	3	A	175	1	1	810	.	.	.	.	0.845	0.933	.
180	3	A	108	1	1	875	.	.	.	.	0.521	0.822	.
181	4	G	81	0	0	910	42.89	140.76	34.83	114.29	1.460	1.861	.
182	4	G	126	0	0	755	28.56	100.39	25.55	89.81	1.252	1.161	.
183	4	G	21	0	0	610	40.53	159.78	27.52	108.47	1.420	1.520	.
184	4	G	129	0	0	685	34.40	102.65	40.40	120.54	1.183	1.331	.
185	4	G	131	0	0	610	36.55	106.32	31.60	91.92	0.707	1.331	.
186	4	G	57	0	0	685	68.82	207.96	28.49	86.10	0.997	1.634	.

OBS	PERIOD	GROUP	ANIM	ICU	IZN	WT	LCW	LCD	LZW	LZD	SC	SZ	CARWT
187	4	G	88	0	0	650	54.34	179.50	26.73	86.28	0.673	0.888	.
188	4	A	160	0	0	845	45.56	127.49	34.26	95.87	0.981	1.185	.
189	4	A	123	0	0	815	45.83	174.86	31.08	118.58	0.780	1.019	.
190	4	A	303	0	0	840	52.35	155.72	34.24	101.85	1.316	1.748	.
191	4	A	149	0	0	920	50.49	149.70	30.68	90.97	0.952	1.043	.
192	4	A	301	0	0	865	76.01	176.64	40.74	94.67	1.011	1.407	.
193	4	A	99	0	0	860	64.10	174.44	38.88	105.80	0.716	1.371	.
194	4	A	115	0	0	835	53.83	161.48	30.02	90.05	0.562	1.261	.
195	4	A	134	0	0	865	53.80	173.61	27.42	88.50	0.651	1.086	.
196	4	A	177	0	1	875	38.44	133.64	25.78	89.63	0.998	1.333	.
197	4	A	188	0	0	1000	37.37	118.74	25.14	79.89	0.808	0.910	.
198	4	G	135	1	0	845	37.46	108.84	37.09	107.77	0.935	1.055	.
199	4	G	66	1	0	.	43.48	142.23	24.04	78.62	0.877	0.944	.
200	4	G	90	1	0	725	55.61	193.62	29.59	103.03	1.291	1.664	.
201	4	G	41	1	0	625	78.98	273.71	24.17	83.76	0.678	0.841	.
202	4	G	63	1	0	535	24.61	88.46	25.92	93.15	0.718	0.890	.
203	4	G	130	1	0	725	46.79	162.66	23.33	81.09	0.983	0.890	.
204	4	A	97	1	0	790	94.77	161.78	62.62	106.89	0.653	0.988	.
205	4	A	140	1	0	855	50.01	161.18	40.39	130.17	1.068	1.429	.
206	4	A	182	1	0	835	57.47	144.05	38.10	95.48	1.122	1.446	.
207	4	A	183	1	0	925	41.81	139.12	47.76	158.93	1.198	0.481	.
208	4	A	173	1	0	875	47.23	155.67	27.27	89.88	0.912	1.056	.
209	4	A	174	1	0	970	66.69	171.39	35.62	91.54	0.822	1.119	.
210	4	A	120	1	0	830	46.05	145.08	31.15	98.15	1.339	1.640	.
211	4	A	156	1	0	820	52.27	179.11	28.91	99.06	0.966	1.595	.
212	4	A	166	1	0	875	67.99	225.65	29.17	96.82	0.999	1.294	.
213	4	G	47	0	1	925	38.72	128.40	28.16	93.37	1.026	1.394	.
214	4	G	22	0	1	670	40.59	126.44	29.43	91.67	0.542	0.874	.
215	4	G	127	0	1	660	43.76	143.88	30.74	101.07	0.815	1.426	.
216	4	G	78	0	1	570	54.50	195.04	24.76	88.62	1.138	0.966	.
217	4	A	304	0	1	810	38.82	124.32	29.74	95.29	0.712	1.318	.
218	4	A	118	0	1	940	52.72	168.86	31.15	99.78	1.066	0.921	.
219	4	A	153	0	1	875	51.64	162.77	26.20	82.57	0.635	0.775	.
220	4	A	145	0	1	850	72.54	115.19	54.70	86.85	0.766	1.073	.
221	4	A	101	0	1	925	34.11	105.16	30.31	93.44	0.878	1.463	.
222	4	A	161	0	1	875	105.38	178.97	71.06	120.68	0.678	1.455	.
223	4	A	199	0	1	830	39.08	132.89	38.98	132.55	1.506	1.210	.
224	4	A	105	0	1	850	57.59	227.43	26.75	105.63	0.463	1.318	.
225	4	A	194	0	1	860	63.62	218.71	28.73	98.78	0.936	1.197	.
226	4	G	34	1	1	840	38.87	143.58	26.82	99.06	1.620	1.598	.
227	4	G	132	1	1	750	46.36	141.17	32.57	99.19	0.780	1.363	.
228	4	G	9	1	1	660	71.42	187.49	47.60	124.96	0.856	1.025	.
229	4	G	60	1	1	655	58.72	175.89	26.86	80.45	1.287	1.625	.
230	4	G	12	1	1	550	76.92	186.28	37.13	89.93	1.048	1.003	.
231	4	G	71	1	1	670	60.02	296.96	26.04	128.85	1.226	1.963	.
232	4	A	119	1	1	915	39.36	118.60	28.57	86.09	0.556	1.583	.
233	4	A	147	1	1	830	56.88	179.74	27.53	86.98	1.033	1.284	.

OBS	PERIOD	GROUP	ANIM	ICU	IZN	WT(lb)	LCW	LCD	LZW	LZD	SC	SZ	CARWT
234	4	A	136	1	1	925	54.94	152.93	33.25	92.55	0.548	0.916	.
235	4	A	162	1	1	775	49.75	138.23	37.12	103.14	0.978	1.262	.
236	4	A	155	1	1	800	80.46	210.28	53.34	139.42	1.042	1.049	.
237	4	A	104	1	1	875	56.92	174.37	31.56	96.69	0.677	1.026	.
238	4	A	148	1	1	805	48.54	149.32	32.09	98.71	0.638	1.346	.
239	4	A	175	1	1	835	92.86	190.94	41.65	86.20	0.537	0.881	.
240	4	A	108	1	1	905	80.09	221.19	35.84	98.99	0.589	1.469	.
241	5	G	81	0	0	930	.	.	.	.	0.898	1.059	.
242	5	G	126	0	0	785	.	.	.	.	0.808	1.248	.
243	5	G	21	0	0	655	.	.	.	.	1.035	1.128	.
244	5	G	129	0	0	745	.	.	.	.	0.952	1.785	.
245	5	G	131	0	0	655	.	.	.	.	0.593	0.996	.
246	5	G	57	0	0	615	.	.	.	.	0.780	0.884	.
247	5	G	88	0	0	675	.	.	.	.	0.750	0.928	.
248	5	A	160	0	0	885	.	.	.	.	0.838	1.258	.
249	5	A	123	0	0	875	.	.	.	.	0.435	0.885	.
250	5	A	303	0	0	860	.	.	.	.	1.097	1.391	.
251	5	A	149	0	0	1005	.	.	.	.	0.682	0.875	.
252	5	A	301	0	0	915	.	.	.	.	0.827	1.141	.
253	5	A	99	0	0	925	.	.	.	.	0.653	1.294	.
254	5	A	115	0	0	860	.	.	.	.	0.673	1.324	.
255	5	A	134	0	0	905	.	.	.	.	0.634	0.911	.
256	5	A	188	0	0	1085	.	.	.	.	0.750	1.095	.
257	5	G	135	1	0	925	.	.	.	.	0.557	0.599	.
258	5	G	66	1	0	705	.	.	.	.	0.863	1.096	.
259	5	G	90	1	0	750	.	.	.	.	0.879	1.396	.
260	5	G	41	1	0	670	.	.	.	.	1.040	1.219	.
261	5	G	63	1	0	590	.	.	.	.	1.254	1.736	.
262	5	G	130	1	0	740	.	.	.	.	0.672	0.688	.
263	5	A	97	1	0	845	.	.	.	.	0.580	0.970	.
264	5	A	140	1	0	905	.	.	.	.	1.078	1.219	.
265	5	A	182	1	0	840	.	.	.	.	1.333	1.211	.
266	5	A	183	1	0	1040	.	.	.	.	0.824	0.981	.
267	5	A	173	1	0	920	.	.	.	.	0.764	1.158	.
268	5	A	174	1	0	1060	.	.	.	.	0.960	1.493	.
269	5	A	120	1	0	885	.	.	.	.	0.852	1.219	.
270	5	A	156	1	0	855	.	.	.	.	0.842	1.179	.
271	5	A	166	1	0	900	.	.	.	.	1.059	1.485	.
272	5	G	47	0	1	965	.	.	.	.	0.785	1.119	.
273	5	G	22	0	1	700	.	.	.	.	0.774	1.179	.
274	5	G	127	0	1	720	.	.	.	.	0.780	1.555	.
275	5	G	78	0	1	605	.	.	.	.	0.717	0.760	.
276	5	A	177	0	1	890	.	.	.	.	0.565	0.866	.
277	5	A	304	0	1	820	.	.	.	.	0.594	1.001	.
278	5	A	118	0	1	950	.	.	.	.	0.573	0.636	.
279	5	A	153	0	1	905	.	.	.	.	0.679	0.846	.
280	5	A	145	0	1	865	.	.	.	.	0.883	1.069	.

OBS	PERIOD	GROUP	ANIM	ICU	IZN	WT	LCW(lb)	LCD	LZW	LZD	SC	SZ	CARWT
281	5	A	101	0	1	970	.	.	.	.	0.859	1.079	.
282	5	A	161	0	1	880	.	.	.	.	0.655	0.968	.
283	5	A	199	0	1	910	.	.	.	.	0.462	0.800	.
284	5	A	105	0	1	880	.	.	.	.	0.727	1.170	.
285	5	A	194	0	1	910	.	.	.	.	0.732	0.971	.
286	5	G	34	1	1	860	.	.	.	.	1.098	1.200	.
287	5	G	132	1	1	765	.	.	.	.	0.829	1.193	.
288	5	G	9	1	1	690	.	.	.	.	0.852	1.463	.
289	5	G	60	1	1	650	.	.	.	.	1.057	1.269	.
290	5	G	12	1	1	580	.	.	.	.	0.835	1.090	.
291	5	G	71	1	1	660	.	.	.	.	0.954	1.460	.
292	5	A	119	1	1	940	.	.	.	.	0.793	1.184	.
293	5	A	147	1	1	870	.	.	.	.	0.697	0.843	.
294	5	A	136	1	1	985	.	.	.	.	0.984	1.315	.
295	5	A	162	1	1	810	.	.	.	.	0.675	1.005	.
296	5	A	155	1	1	840	.	.	.	.	1.212	1.460	.
297	5	A	104	1	1	905	.	.	.	.	0.910	1.378	.
298	5	A	148	1	1	855	.	.	.	.	0.824	1.474	.
299	5	A	175	1	1	860	.	.	.	.	0.849	1.104	.
300	5	A	108	1	1	945	.	.	.	.	0.693	1.343	.
301	6	G	81	0	0	1000	52.85	171.06	27.02	87.45	0.823	0.977	.
302	6	G	126	0	0	840	34.24	96.18	31.06	87.25	0.862	1.188	.
303	6	G	21	0	0	670	51.12	156.03	31.42	95.91	0.717	0.806	.
304	6	G	129	0	0	805	41.34	150.92	24.80	90.55	0.661	0.833	.
305	6	G	131	0	0	680	40.99	122.72	32.55	97.46	0.572	0.986	.
306	6	G	57	0	0	655	61.95	234.32	19.53	73.86	0.584	0.691	.
307	6	G	88	0	0	730	58.59	197.72	24.81	83.72	0.521	0.527	.
308	6	A	160	0	0	920	43.51	139.88	30.59	98.35	0.638	0.913	.
309	6	A	123	0	0	925	38.95	123.23	25.64	81.13	0.534	0.694	.
310	6	A	303	0	0	925	47.40	138.02	34.48	100.42	0.657	0.868	.
311	6	A	149	0	0	1095	47.67	135.01	30.35	85.94	0.576	0.769	.
312	6	A	301	0	0	945	68.88	193.08	30.89	86.59	0.784	0.848	.
313	6	A	99	0	0	975	57.04	163.13	35.86	102.57	0.614	1.085	.
314	6	A	115	0	0	905	44.19	134.31	26.24	79.74	0.497	0.860	.
315	6	A	134	0	0	940	54.24	169.77	24.79	77.90	0.628	0.673	.
316	6	A	188	0	0	1135	49.43	162.95	25.56	95.78	0.596	0.888	.
317	6	G	135	1	0	950	32.12	113.67	26.05	92.18	0.639	0.760	.
318	6	G	66	1	0	755	37.45	122.21	22.65	74.12	0.592	0.648	.
319	6	G	90	1	0	810	48.01	169.68	25.55	90.31	0.546	0.810	.
320	6	G	41	1	0	700	67.56	221.76	24.96	81.94	0.779	0.930	.
321	6	G	63	1	0	605	38.02	133.74	32.31	113.66	0.726	0.871	.
322	6	G	130	1	0	780	50.90	165.07	24.57	79.70	0.597	0.764	.
323	6	A	97	1	0	895	40.24	122.68	28.64	87.32	0.567	0.684	.
324	6	A	140	1	0	945	42.92	124.90	30.75	89.48	0.534	0.865	.
325	6	A	182	1	0	905	49.02	162.57	24.57	80.78	0.593	0.861	.
326	6	A	183	1	0	1090	50.56	156.64	31.50	97.60	0.751	0.890	.
327	6	A	173	1	0	995	41.88	136.89	25.56	83.57	0.798	1.254	.

OBS	PERIOD	GROUP	ANIM	ICU	IZN	WT(lb)	LCW	LCD	LZW	LZD	SC	SZ	CARWT
328	6	A	174	1	0	1120	47.36	160.61	25.76	87.38	0.648	1.061	.
329	6	A	120	1	0	945	61.29	155.09	32.29	81.72	0.562	0.884	.
330	6	A	156	1	0	920	59.82	196.94	22.48	74.01	0.656	0.899	.
331	6	A	166	1	0	950	39.69	152.70	26.10	100.40	0.712	0.791	.
332	6	G	47	0	1	1000	46.45	159.60	28.05	96.40	0.703	0.755	.
333	6	G	22	0	1	705	45.29	152.64	34.59	116.58	0.783	0.901	.
334	6	G	127	0	1	755	42.90	130.79	29.44	89.77	0.638	1.108	.
335	6	G	78	0	1	630	66.14	226.18	24.57	84.03	0.646	0.566	.
336	6	A	177	0	1	950	59.08	165.89	28.97	81.36	0.602	0.866	.
337	6	A	304	0	1	890	35.61	106.24	26.21	78.21	0.667	1.024	.
338	6	A	118	0	1	995	42.88	125.12	34.08	99.43	0.739	0.766	.
339	6	A	153	0	1	940	58.23	188.96	25.06	81.32	0.783	0.908	.
340	6	A	145	0	1	915	49.88	138.07	30.36	84.04	0.739	0.904	.
341	6	A	101	0	1	1085	39.31	125.97	27.12	86.90	0.541	0.786	.
342	6	A	161	0	1	940	60.16	167.32	35.90	99.84	0.624	0.820	.
343	6	A	199	0	1	940	49.69	165.53	27.28	90.88	0.652	0.851	.
344	6	A	105	0	1	935	42.55	131.30	27.99	86.37	0.599	0.886	.
345	6	A	194	0	1	975	49.93	116.60	44.47	103.81	0.773	0.855	.
346	6	G	34	1	1	900	43.98	148.46	26.99	91.13	0.791	0.946	.
347	6	G	132	1	1	905	40.64	126.10	30.50	94.64	0.672	1.003	.
348	6	G	9	1	1	730	74.85	253.77	32.60	110.52	0.701	0.844	.
349	6	G	60	1	1	680	34.65	104.73	26.43	79.91	0.644	0.834	.
350	6	G	12	1	1	620	49.29	189.64	21.55	82.93	0.684	0.700	.
351	6	G	71	1	1	690	53.68	193.29	22.16	79.79	0.738	1.026	.
352	6	A	119	1	1	985	38.01	101.42	31.37	83.72	0.454	0.800	.
353	6	A	147	1	1	925	47.04	152.00	23.03	74.41	0.649	0.736	.
354	6	A	136	1	1	1000	60.36	167.32	29.33	81.29	0.597	0.771	.
355	6	A	162	1	1	850	54.06	184.14	28.41	96.75	0.521	0.795	.
356	6	A	155	1	1	925	80.45	279.69	24.59	85.49	0.723	1.020	.
357	6	A	104	1	1	1005	47.50	146.34	27.79	85.61	0.636	1.168	.
358	6	A	148	1	1	900	46.77	139.84	28.53	85.31	0.616	0.824	.
359	6	A	175	1	1	930	71.79	155.84	31.35	68.06	0.662	0.715	.
360	6	A	108	1	1	940	67.61	210.36	29.55	91.92	0.488	0.890	.
361	7	G	81	0	0	950	.	.	.	.	.	.	.
362	7	G	126	0	0	840	.	.	.	.	.	.	.
363	7	A	160	0	0	884	.	.	.	.	.	.	.
364	7	A	123	0	0	880	.	.	.	.	.	.	.
365	7	A	303	0	0	884	.	.	.	.	.	.	.
366	7	A	149	0	0	997	.	.	.	.	.	.	.
367	7	A	301	0	0	926	.	.	.	.	.	.	.
368	7	A	115	0	0	882	.	.	.	.	.	.	.
369	7	A	134	0	0	902	.	.	.	.	.	.	.
370	7	A	188	0	0	1074	.	.	.	.	.	.	.
371	7	G	135	1	0	990	.	.	.	.	.	.	.
372	7	G	90	1	0	774	.	.	.	.	.	.	.
373	7	A	97	1	0	871	.	.	.	.	.	.	.
374	7	A	140	1	0	924	.	.	.	.	.	.	.

OBS	PERIOD	GROUP	ANIM	ICU	IZN	WT(lb)	LCW	LCD	LZW	LZD	SC	SZ	CARWT	TRT
375	7	A	182	1	0	887	.	.	.	.	.	.	.	.
376	7	A	183	1	0	992	.	.	.	.	.	.	.	.
377	7	A	173	1	0	988	.	.	.	.	.	.	.	.
378	7	A	174	1	0	1016	.	.	.	.	.	.	.	.
379	7	A	120	1	0	924	.	.	.	.	.	.	.	.
380	7	A	156	1	0	887	.	.	.	.	.	.	.	.
381	7	A	166	1	0	928	.	.	.	.	.	.	.	.
382	7	G	47	0	1	1012	.	.	.	.	.	.	.	.
383	7	A	177	0	1	924	.	.	.	.	.	.	.	.
384	7	A	304	0	1	867	.	.	.	.	.	.	.	.
385	7	A	118	0	1	1012	.	.	.	.	.	.	.	.
386	7	A	153	0	1	948	.	.	.	.	.	.	.	.
387	7	A	145	0	1	933	.	.	.	.	.	.	.	.
388	7	A	101	0	1	1065	.	.	.	.	.	.	.	.
389	7	A	161	0	1	891	.	.	.	.	.	.	.	.
390	7	A	199	0	1	924	.	.	.	.	.	.	.	.
391	7	A	105	0	1	920	.	.	.	.	.	.	.	.
392	7	A	194	0	1	975	.	.	.	.	.	.	.	.
393	7	G	34	1	1	891	.	.	.	.	.	.	.	4
394	7	G	132	1	1	807	.	.	.	.	.	.	.	4
395	7	A	119	1	1	931	.	.	.	.	.	.	.	4
396	7	A	147	1	1	882	.	.	.	.	.	.	.	4
397	7	A	136	1	1	1030	.	.	.	.	.	.	.	4
398	7	A	162	1	1	854	.	.	.	.	.	.	.	4
399	7	A	155	1	1	882	.	.	.	.	.	.	.	4
400	7	A	104	1	1	1021	.	.	.	.	.	.	.	4
401	7	A	148	1	1	893	.	.	.	.	.	.	.	4
402	7	A	175	1	1	871	.	.	.	.	.	.	.	4
403	7	A	108	1	1	898	.	.	.	.	.	.	.	4
404	8	G	81	0	0	1047	.	.	.	.	.	.	.	1
405	8	G	126	0	0	946	.	.	.	.	.	.	.	1
406	8	A	160	0	0	972	.	.	.	.	.	.	.	1
407	8	A	123	0	0	1021	.	.	.	.	.	.	.	1
408	8	A	303	0	0	1047	.	.	.	.	.	.	.	1
409	8	A	149	0	0	1131	.	.	.	.	.	.	.	1
410	8	A	301	0	0	1027	.	.	.	.	.	.	.	1
411	8	A	115	0	0	988	.	.	.	.	.	.	.	1
412	8	A	134	0	0	977	.	.	.	.	.	.	.	1
413	8	A	188	0	0	1104	.	.	.	.	.	.	.	1
414	8	G	135	1	0	1034	.	.	.	.	.	.	.	2
415	8	G	90	1	0	933	.	.	.	.	.	.	.	2
416	8	A	97	1	0	959	.	.	.	.	.	.	.	2
417	8	A	140	1	0	968	.	.	.	.	.	.	.	2
418	8	A	182	1	0	937	.	.	.	.	.	.	.	2
419	8	A	183	1	0	1012	.	.	.	.	.	.	.	2
420	8	A	173	1	0	957	.	.	.	.	.	.	.	2
421	8	A	174	1	0	1052	.	.	.	.	.	.	.	2

OBS	PERIOD	GROUP	ANIM	ICU	IZN	WT(lb)	LCW	LCD	LZW	LZD	SC	SZ	CARWT	
422	8	A	120	1	0	975	.	.	.	.	.	.	.	2
423	8	A	156	1	0	992	.	.	.	.	.	.	.	2
424	8	A	166	1	0	1091	.	.	.	.	.	.	.	2
425	8	G	47	0	1	1096	.	.	.	.	.	.	.	3
426	8	A	177	0	1	1003	.	.	.	.	.	.	.	3
427	8	A	304	0	1	924	.	.	.	.	.	.	.	3
428	8	A	118	0	1	1113	.	.	.	.	.	.	.	3
429	8	A	153	0	1	988	.	.	.	.	.	.	.	3
430	8	A	145	0	1	1058	.	.	.	.	.	.	.	3
431	8	A	101	0	1	1131	.	.	.	.	.	.	.	3
432	8	A	161	0	1	1025	.	.	.	.	.	.	.	3
433	8	A	199	0	1	986	.	.	.	.	.	.	.	3
434	8	A	105	0	1	1027	.	.	.	.	.	.	.	3
435	8	A	194	0	1	1047	.	.	.	.	.	.	.	4
436	8	G	34	1	1	1030	.	.	.	.	.	.	.	4
437	8	G	132	1	1	818	.	.	.	.	.	.	.	4
438	8	A	119	1	1	1016	.	.	.	.	.	.	.	4
439	8	A	147	1	1	1008	.	.	.	.	.	.	.	4
440	8	A	136	1	1	1034	.	.	.	.	.	.	.	4
441	8	A	162	1	1	915	.	.	.	.	.	.	.	4
442	8	A	155	1	1	1003	.	.	.	.	.	.	.	4
443	8	A	104	1	1	1049	.	.	.	.	.	.	.	4
444	8	A	148	1	1	920	.	.	.	.	.	.	.	4
445	8	A	175	1	1	920	.	.	.	.	.	.	.	4
446	8	A	108	1	1	928	.	.	.	.	.	.	.	4
447	9	G	81	0	0	1166	16.22	100.77	24.81	88.86	.	.	626	1
448	9	G	126	0	0	1074	22.43	75.27	31.77	106.63	.	.	594	1
449	9	A	160	0	0	1157	23.45	81.83	33.85	118.12	.	.	628	
450	9	A	123	0	0	1144	20.09	67.98	24.67	83.46	.	.	620	
451	9	A	303	0	0	1228	56.11	131.94	46.05	108.27	.	.	650	
452	9	A	149	0	0	1219	27.49	97.16	32.85	116.13	.	.	682	
453	9	A	301	0	0	1168	34.82	119.41	32.50	111.47	.	.	658	
454	9	A	115	0	0	1104	20.04	67.72	28.05	94.79	.	.	610	
455	9	A	134	0	0	1135	28.71	100.14	32.38	112.93	.	.	610	
456	9	A	188	0	0	1258	29.97	103.21	31.46	98.79	.	.	726	
457	9	G	90	1	0	1115	23.11	81.10	33.14	116.28	.	.	604	
458	9	A	97	1	0	1091	18.57	64.39	32.74	113.52	.	.	608	
459	9	A	140	1	0	1087	14.38	50.40	30.36	106.43	.	.	610	
460	9	A	182	1	0	1120	22.14	75.56	33.35	114.93	.	.	624	
461	9	A	183	1	0	1170	21.06	72.59	32.86	113.29	.	.	654	
462	9	A	173	1	0	1122	20.44	70.26	30.95	106.39	.	.	620	
463	9	A	174	1	0	1219	22.73	75.63	35.16	117.01	.	.	670	
464	9	A	120	1	0	1126	24.97	82.54	40.17	132.80	.	.	608	
465	9	A	156	1	0	1144	17.33	99.71	30.46	102.15	.	.	610	
466	9	A	166	1	0	1188	34.68	122.33	36.27	127.91	.	.	650	
467	9	G	47	0	1	1254	28.55	100.25	30.22	106.12	.	.	668	

OBS	PERIOD	GROUP	ANIM	ICU	IZN	WT(lb)	LCW	LCD	LZW	LZD	SC	SZ	
468	9	A	177	0	1	1137	17.06	57.96	25.37	86.19	.	.	626
469	9	A	304	0	1	1054	17.84	63.10	25.46	90.13	.	.	580
470	9	A	118	0	1	1245	16.38	55.72	35.64	121.22	.	.	682
471	9	A	153	0	1	1080	33.48	117.91	23.83	83.95	.	.	600
472	9	A	145	0	1	1210	25.99	91.35	32.16	113.02	.	.	628
473	9	A	101	0	1	1291	19.16	70.90	35.10	129.86	.	.	702
474	9	A	161	0	1	1173	20.28	71.49	36.32	128.05	.	.	636
475	9	A	199	0	1	1206	24.11	79.90	31.94	105.85	.	.	636
476	9	A	105	0	1	1157	31.25	106.17	26.97	91.61	.	.	642
477	9	A	194	0	1	1219	33.74	120.65	46.27	165.47	.	.	678
478	9	G	34	1	1	1188	24.18	87.84	30.07	109.26	.	.	626
479	9	G	132	1	1	964	14.71	67.20	30.26	107.36	.	.	524
480	9	A	119	1	1	1144	13.33	46.80	27.94	98.20	.	.	616
481	9	A	147	1	1	1151	20.52	72.60	28.65	101.38	.	.	622
482	9	A	136	1	1	1144	13.99	48.33	30.27	104.52	.	.	646
483	9	A	162	1	1	1065	22.26	78.72	27.90	98.64	.	.	558
484	9	A	155	1	1	1155	31.24	114.71	26.46	97.15	.	.	620
485	9	A	104	1	1	1166	29.87	101.14	37.15	125.77	.	.	644
486	9	A	148	1	1	1089	27.52	93.23	33.68	114.10	.	.	590
487	9	A	175	1	1	1096	19.23	65.43	26.96	91.69	.	.	602
488	9	A	108	1	1	1069	37.74	136.74	42.56	154.19	.	.	594

Appendix Table A3: Means for body weights, serum copper, liver copper, serum zinc and liver zinc of Group I steers during the grazing trial.

NOTE: Body weight is expressed in Kg.

Serum copper and serum zinc are expressed in ug/ml.

Liver copper and liver zinc are both expressed in mg/Kg wet basis (wb) and in mg/Kg DM (Db).

APPENDIX TABLE A3:

Treatment	Variable	Number of Steers	Sampling date	Mean	SE. of Mean
A	Weight	9	13/6/83	356.0	7.7
"	Serum copper	9	10/6/83	0.90	0.00
"	Liver copper (Wb)	9	"	54.90	7.1
"	Liver copper (Db)	9	"	100.7	6.3
"	Serum zinc	9	"	1.10	0.10
"	Liver zinc (Wb)	9	"	54.90	7.9
B	Weight	9	13/6/83	356.3	6.5
B	Serum copper	9	10/6/83	1.00	0.10
"	Liver copper (Wb)	9	"	53.5	8.0
"	Liver copper (Db)	9	"	83.7	7.3
"	Serum zinc	9	"	1.30	0.10
"	Liver zinc (Wb)	9	"	65.0	4.6
"	Liver zinc (Db)	9	"	105.0	3.5
C	Weight	10	13/6/83	357.5	6.9
"	Serum copper	10	10/6/83	0.90	0.10
"	Liver copper (Wb)		"	57.6	6.5
"	Liver copper (Db)	10	"	104.8	9.1
"	Serum zinc	10	"	1.10	0.10
"	Liver zinc (Wb)	10	"	57.0	6.4
C	Liver zinc (Db)	10	"	100.6	3.0
D	Weight	9	13/6/83	347.6	7.7
"	Serum copper	9	10/6/83	0.80	0.10
"	Liver copper (Wb)	9	"	46.3	6.7
D	Liver copper (Db)	9	"	87.7	6.5

APPENDIX TABLE A3 (Continued)

Treatment	Variable	Number of Steers	Sampling Date	Mean	SE. of Mean
D	Serum zinc	9	10/6/83	1.20	0.10
"	Liver zinc (Wb)	9	"	49.8	5.4
"	Liver zinc (Db)	9	"	94.9	3.9
A	Weight	9	12/7/83	385.2	9.1
A	Serum copper	9	"	0.70	0.00
A	Serum zinc	9	"	0.90	0.00
B	Weight	9	12/7/83	378.3	8.2
B	Serum Copper	9	"	0.70	0.00
B	Serum zinc	9	"	0.90	0.10
C	Weight	10	12/7/83	386.2	7.4
C	Serum copper	10	"	0.80	0.10
C	Serum zinc	10	"	0.80	0.10
D	Weight	9	12/7/83	379.1	7.6
D	Serum copper	9	"	0.70	0.01
D	Serum zinc	9	"	0.90	0.01
A	Weight	9	16/8/83	395.8	8.5
"	Serum copper	9	"	0.90	0.10
"	Liver copper (Wb)	9	"	53.3	3.7
"	Liver copper (Db)	9	"	157.0	7.2
"	Serum zinc	9	"	1.20	0.10
"	Liver zinc (Wb)	9	"	32.5	1.7
"	Liver zinc (Db)	9	"	96.2	3.8

APPENDIX TABLE A3 (Continued)

Treatment	Variable	Number of Steers	Sampling date	Mean	SE. of Mean
B	Weight	9	16/8/83	392.2	8.4
B	Serum copper	9	"	1.00	0.10
B	Liver copper (Wb)	9	"	58.3	5.5
B	Liver copper (Db)	9	"	164.8	8.5
B	Serum zinc	9	"	1.20	0.10
B	Liver zinc (Wb)	9	"	37.9	3.8
B	Liver zinc (Db)	9	"	107.4	7.6
C	Weight	10	16/8/83	394.6	5.7
C	Serum copper	10	"	0.90	0.10
C	Liver copper (Wb)	10	"	55.4	6.8
C	Liver copper (Db)	10	"	156.8	13.4
C	Serum zinc	10	"	1.20	0.10
C	Liver zinc (Wb)	10	"	36.3	4.7
C	Liver zinc (Db)	10	"	100.5	4.9
D	Weight	9	16/8/83	386.7	8.4
D	Serum copper	9	"	0.70	0.10
D	Liver copper (Wb)	9	"	62.2	6.0
D	Liver copper (Db)	9	"	170.6	11.3
D	Serum zinc	9	"	1.20	0.10
D	Liver zinc (Wb)	9	"	35.7	2.6
D	Liver zinc (Db)	9	"	98.8	5.5
A	Weight	9	6/9/83	419.5	11.4
A	Serum copper	9	6/9/83	0.70	0.10
A	Serum zinc	9	6/9/83	1.10	0.10

APPENDIX TABLE A3 (Continued)

Treatment	Variable	Number of Steers	Sampling Date	Mean	S.E. of Mean
B	Weight	9	6/9/83	416.2	12.2
B	Serum copper	9	"	0.90	0.10
B	Serum zinc	9	"	1.20	0.10
C	Weight	10	6/9/83	407.7	6.1
C	Serum copper	10	"	0.80	0.00
C	Serum zinc	10	"	0.90	0.10
D	Weight	9	6/9/83	404.1	8.7
D	Serum copper	9	"	0.80	0.10
D	Serum zinc	9	"	1.20	0.10
A	Weight	9	4/10/83	442.2	12.6
A	Serum copper	9	"	0.61	0.00
A	Liver copper (Wb)	9	6/10/83	50.2	3.0
A	Liver copper (Db)	9	"	151.0	7.5
A	Serum zinc	9	"	0.84	0.00
A	Liver zinc (Wb)	9	"	29.4	1.4
A	Liver Zinz (Db)	9	"	89.5	3.2
B	Weight	9	4/10/83	442.2	12.1
B	Serum copper	9	"	0.65	0.00
B	Liver copper (Wb)	9	6/10/83	48.1	2.7
B	Liver copper (Db)	9	"	152.1	7.5
B	Serum zinc	9	4/10/83	0.91	0.10
B	Liver zinc (Wb)	9	6/10/83	27.5	1.1
B	Liver zinc (Db)	9	"	86.9	2.8

APPENDIX TABLE A3 (Continued)

Treatment	Variable	Number of Steers	Sampling Date	Mean	S.E. of Mean
C	Weight	10	4/10/83	434.3	7.7
C	Serum copper	10	4/10/83	0.67	0.00
C	Liver copper (Wb)	10	6/10/83	48.7	2.7
C	Liver copper (Db)	10	6/10/83	143.1	8.5
C	Serum zinc	10	4/10/83	0.87	0.00
C	Liver zinc (Wb)	10	6/10/83	30.7	1.9
C	Liver zinc (Db)	10	6/10/83	89.2	2.8
D	Weight	9	4/10/83	426.8	7.6
D	Serum copper	9	4/10/83	0.59	0.00
D	Liver copper (Wb)	9	6/10/83	57.1	4.7
D	Liver copper (Db)	9	6/10/83	170.8	16.9
D	Serum zinc	9	4/10/83	0.86	0.10
D	Liver Zinc (Wb)	9	6/10/83	28.2	0.9
D	Liver zinc (Db)	9	6/10/83	83.6	2.8

Appendix Table A4: Means for body weights, serum copper, liver copper, serum zinc, and liver zinc of Group II steers during the grazing trial.

NOTE: Serum copper and serum zinc are expressed in ug/ml.

Liver copper and liver zinc are both expressed in mg/Kg wet basis (wb) and as mg/Kg DM (db).

APPENDIX TABLE A4 (Continued)

Treatment	Variable	Number of Steers	Sampling Date	Mean	SE. of Mean
B	Weight	6	4/10/83	348.1	21.3
"	Serum Copper	6	"	0.65	0.04
"	Liver Copper (wb)	6	6/10/83	45.7	5.2
"	Liver Copper (db)	6	"	154.4	16.3
"	Serum Zinc	6	4/10/83	0.80	0.04
"	Liver Zinc (wb)	6	6/10/83	26.0	1.3
"	Liver Zinc (db)	6	"	88.7	5.7
C	Weight	4	4/10/83	350.7	36.4
C	Serum Copper	4	"	0.69	0.03
"	Liver Copper (wb)	4	6/10/83	50.2	5.4
"	Liver Copper (db)	4	"	167.3	20.6
"	Serum Zinc	4	4/10/83	0.83	0.11
"	Liver Zinc (wb)	4	6/10/83	29.2	2.1
"	Liver Zinc (db)	4	"	96.7	7.1
D	Weight	6	4/10/83	342.4	22.3
"	Serum Copper	6	"	0.71	0.02
"	Liver Copper (wb)	6	6/10/83	49.5	5.2
"	Liver Copper (db)	6	"	169.3	22.1
"	Serum Zinc	6	4/10/83	0.89	0.05
"	Liver Zinc (wb)	6	6/10/83	26.7	1.8
"	Liver Zinc (db)	6	"	89.8	4.8

APPENDIX TABLE A4 (Continued)

Treatment	Variable	Number of Steers	Sampling Date	Mean	SE. of Mean
A	Weight	7	6/9/83	328.2	18.6
"	Serum Copper	7	"	0.83	0.06
"	Serum Zinc	7	"	1.14	0.12
B	Weight	6	6/9/83	331.4	20.7
"	Serum Copper	6	"	0.88	0.10
"	Serum Zinc	6	"	1.12	0.18
C	Weight	4	6/9/83	339.4	34.8
"	Serum Copper	4	"	0.76	0.02
"	Serum Zinc	4	"	1.5	0.16
D	Weight	4	6/9/83	318.2	18.3
D	Serum Copper	4	"	0.94	0.05
D	Serum Zinc	4	"	1.28	0.06
A	Weight	7	4/10/83	349.0	21.2
"	Serum Copper	7	"	0.68	0.10
"	Liver Copper (wb)	7	6/10/83	43.7	3.8
"	Liver Copper (db)	7	6/10/83	161.3	17.3
"	Serum Zinc	7	4/10/83	0.86	0.10
"	Liver Zinc (wb)	7	6/10/83	27.3	1.8
"	Liver Zinc (db)	7	"	88.0	3.0

APPENDIX TABLE A4 (Continued)

Treatment	Variable	Number of Steers	Sampling Date	Mean	SE. of Mean
A	Serum Zinc	7	16/8/83	1.40	0.10
"	Liver Zinc (wb)	7	"	30.7	2.0
"	Liver Zinc (db)	7	"	99.6	5.5
B	Weight	6	16/8/83	313.7	23.7
"	Serum Copper	6	"	0.91	0.10
"	Liver Copper (wb)	6	"	47.8	7.5
"	Liver Copper (db)	6	"	161.6	27.1
"	Serum Zinc	6	"	1.05	0.13
"	Liver Zinc (wb)	6	"	27.4	2.2
"	Liver Zinc (db)	6	"	91.2	4.9
C	Weight	4	16/8/83	320.7	34.6
"	Serum Copper	4	"	0.88	0.13
"	Liver Copper (wb)	4	"	44.4	3.5
"	Liver Copper (db)	4	"	148.4	16.0
"	Serum Zinc	4	"	1.17	0.12
"	Liver Zinc (wb)	4	"	28.3	1.3
"	Liver Zinc (db)	4	"	93.7	2.7
D	Weight	6	16/8/83	312.1	18.2
"	Serum Copper	6	"	1.13	0.10
"	Liver Copper (wb)	6	"	58.7	5.9
"	Liver Copper (db)	6	"	188.6	23.2
"	Serum Zinc	6	"	1.43	0.15
"	Liver Zinc (wb)	6	"	32.8	3.4
"	Liver Zinc (db)	6	"	103.7	7.9

APPENDIX TABLE A4 (Continued)

Treatment	Variable	Number of Steers	Sampling Date	Mean	SE. of Mean
D	Liver Copper (db)	6	10/6/83	45.1	7.7
"	Serum Zinc	6	"	1.18	0.04
"	Liver Zinc (wb)	6	"	54.3	9.1
"	Liver Zinc (db)	6	"	93.3	4.6
A	Weight	7	12/7/83	307.4	16.5
A	Serum Copper	7	"	0.73	0.04
A	Serum Zinc	7	"	0.83	0.04
B	Weight	6	"	307.2	20.1
"	Serum Copper	6	"	0.78	0.08
"	Serum Zinc	6	"	0.83	0.10
C	Weight	4	"	312.7	34.3
"	Serum Copper	4	"	0.32	0.09
"	Serum Zinc	4	"	1.09	0.14
D	Weight	5	"	309.2	24.7
"	Serum Copper	6	"	0.87	0.11
"	Serum Zinc	6	"	1.01	0.03
A	Weight	7	16/8/83	318.1	18.0
"	Serum Copper	7	"	1.10	0.10
"	Liver Copper(wb)	7	"	43.7	5.2
"	Liver Copper (db)	7	"	142.5	15.9

APPENDIX TABLE A4:

(13/6/83)

Treatment	Variable	Number of Steers	Sampling Date	Mean	SE. of Mean
A	Weight	7	13/6/83	288.3	18.1
"	Serum Copper	6	10/6/83	0.83	0.12
"	Liver Copper (wb)	6	"	27.3	3.1
"	Liver Copper (db)	6	"	43.9	10.7
"	Serum Zinc	6	"	1.23	0.11
"	Liver Zinc (wb)	6	"	63.2	11.2
"	Liver Zinc (db)	6	"	101.0	3.6
B	Weight	6	13/6/83	298.5	19.7
"	Serum Copper	6	10/6/83	0.85	0.06
"	Liver Copper (wb)	6	"	20.0	4.3
"	Liver Copper (db)	6	"	39.3	3.7
"	Serum Zinc	6	"	1.02	0.08
"	Liver Zinc (wb)	6	"	46.0	3.2
"	Liver Zinc (db)	6	"	89.2	4.2
C	Weight	4	13/6/83	296.7	36.0
"	Serum Copper	4	10/6/83	0.72	0.11
"	Liver Copper (wb)	4	"	31.6	13.5
"	Liver Copper (db)	4	"	53.2	23.0
"	Serum Zinc	4	"	0.94	0.16
"	Liver Zinc (wb)	4	"	70.2	15.4
"	Liver Zinc (db)	4	"	105.9	5.6
D	Weight	6	13/6/83	292.7	21.5
"	Serum Copper	6	10/6/83	0.87	0.05
"	Liver Copper (wb)	6	10/6/83	24.7	3.6

Appendix Table A5: Means for body weights, liver copper and liver zinc of steers during the finishing trial

Note: Body weight is expressed in kilograms.
CARWT- Carcass weight (Also expressed in kilograms).

Liver copper and liver zinc are both expressed as mg/kg wet basis (wb) or mg/kg dry basis (db).

APPENDIX TABLE A5: Raw data of steers measured during the finishing phase

SAMPLING DAY 0

Treatment	Variable	Number of Steers	Mean	SE. of Mean
A	Weight	10	418.6	9.9
B	Weight	11	420.2	9.6
C	Weight	11	432.2	8.0
D	Weight	11	411.1	9.4
		SAMPLING DAY 35		
A	Weight	10	465.8	8.4
B	Weight	11	450.3	6.9
C	Weight	11	470.5	
D	Weight	11	439.2	9.8

APPENDIX TABLE A5 (Continued)

SAMPLING DAY 79

Treatment	Variable	Number of Steers	Mean	SE. Of Mean
A	Weight	10	529.1	8.1
A	Liver Cu (wb)	10	27.9	3.6
A	Liver Cu (db)	10	94.5	6.8
A	Liver Zn (wb)	10	31.8	1.9
A	Liver Zn (db)	10	103.9	3.8
A	CARWT	10	290.8	5.7
B	Weight	10	516.8	6.1
B	Liver Cu (wb)	10	21.9	1.7
B	Liver Cu (db)	10	79.5	6.2
B	Liver Zn (wb)	10	33.5	1.0
B	Liver Zn (db)	10	115.1	3.0
B	CARWT	10	284.1	3.4
C	Weight	11	537.7	10.0
C	Liver Cu (wb)	11	24.3	2.0
C	Liver Cu (db)	11	85.0	7.1
C	Liver Zn (wb)	11	31.7	2.0
C	Liver Zn (db)	11	111.0	7.3
C	CARWT	11	292.1	5.0
D	Weight	11	504.8	8.8
D	Liver Cu (wb)	11	23.1	2.4
D	Liver Cu (db)	11	83.0	8.3

APPENDIX TABLE A5 (continued)

DAY 79(continued)

Treatment	Variable	Number of Steers	Mean	SE. of Mean
D	Liver Zn(wb)	11	31.1	1.5
D	Liver Zn (db)	11	109.3	5.3
D	CARWT	11	274.2	5.0

Appendix Table A6: Mean Concentration of Nutrients in Grasses Alfalfa and Birds Foot Trefoil Consumed by Steers During the Grazing Trial.

Crude protein and Acid detergent fibre are expressed as percent dry matter.

Calcium, Magnesium, Phosphorus and total Sulphur are expressed in g/kg dry matter.

Copper, Zinc, Molybdenum, Iron, and Manganese are expressed in mg/kg dry matter.

NOTE: Means for these nutrients are given for the months of June, July, August and September.

ABBREVIATIONS:

CP - Crude Protein

Ca - Calcium

Mg - Magnesium

P - Phosphorus

S - Sulphur

Cu - Copper

Zn - Zinc

Mo - Molybdenum

Fe - Iron

Mn - Manganese

DM - Dry matter

ADF - Acid detergent fibre

Appendix Table A6: Mean Concentration of Nutrients in forages consumed by steers during the grazing trial

(JUNE 28)

	GRASSES			Alfalfa			Birds Foot Trefoil		
Nutrient	No of Samples	Mean (± SE)	Range	No. of Samples	Mean (± SE)	Range	No. of Samples	Mean (± SE)	Range
Cp+	8	15(1.5)	13.0-18.0	6	25.4(1.5)	23.0-27.6	2	26.3(1.3)	25.0-27.5
Ca	8	3.7(0.4)	3.4-4.6	6	20.0(4.3)	16.6-28.0	1	16.0(0.0)	16.0-16.0
Mg	8	1.8(0.2)	1.6-2.0	6	2.1(0.1)	2.0-2.2	1	2.0(0.0)	2.0-2.0
P	8	1.9(0.6)	1.0-2.6	6	2.1(0.8)	1.4-3.5	1	1.1(0.0)	1.1-1.1
S	8	2.2(0.2)	2.0-2.5	6	2.4(0.2)	2.0-2.5	1	2.1(0.0)	2.1-2.1
Cu	8	5.4(0.9)	4.5(7.0)	6	6.8(0.9)	5.8-8.5	2	7.7(2.0)	5.8-9.7
Zn	8	18.9(1.4)	16.4-21.0	6	22.8(3.1)	18.2-27.4	2	28.8(2.4)	26.4-31.1
Mo	8	1.0(0.3)	0.7-1.5	6	1.4(0.4)	1.0-2.0	1	0.7(0.0)	0.7-0.7
Fe	8	47.9(12.5)	27.1-63.2	6	57.6(4.9)	50.3-64.1	1	70.4(0.0)	70.4-70.4
Mn	8	49.4(19.8)	19.4-84.0	6	44.1(4.4)	40.7-50.4	1	58.9(0.0)	58.9-58.9
DM	8	80.0(11.8)	53.0-88.0	6	70.3(16.9)	51.0-88.0	2	90.5(1.7)	89.3-91.7
ADF	8	32.2(1.0)	30.0-33.0	6	24.3(1.0)	22.0-28.0	2	17.4(0.4)	17.1-17.6

Table A6(Continued) (September 20, 1983)*

Nutrient	Grasses			Birds Foot Trefoil		
	No. of Samples	Mean (± SE)	Range	No. of Samples	Mean (± SE)	Range
CP	8	8.9(0.3)	7.4-10.0	2	7.9(0.3)	7.6-8.2
Ca	8	5.6(0.1)	4.9-6.3	2	12.7(0.7)	12.0-13.4
Mg	8	2.0(0.0)	1.8-2.1	2	2.2(0.0)	2.2-2.2
P	8	1.4(0.1)	1.0-2.2	2	0.8(0.1)	0.7-0.8
S	8	1.2(0.2)	0.5-1.9	2	0.9(0.3)	0.6-1.2
Cu	8	3.7(0.1)	3.2-4.2	2	3.3(0.3)	3.0-3.7
Zn	8	13.8(0.5)	12.1-16.4	2	6.5(0.7)	5.8-7.1
Mo	8	1.4(0.2)	0.7-2.2	2	0.7(0.3)	0.4-0.9
Fe	8	112.7(5.6)	82.8-128.1	2	82.0(15.1)	66.9-97.0
Mn	8	53.8(8.4)	38.8-111.4	2	29.9(0.9)	29.0-30.8
DM	8	91.8(1.0)	90.0-93.5	2	89.8(1.9)	88.4-91.1
ADF	8	43.2(1.5)	40.9-45.0	2	54.1(1.4)	53.1-55.1

* Samples of alfalfa were not obtained on September 20.

Table A6:(Continued)

(JULY 26, 1983)

Nutrient	Grasses No. of Samples	Grasses Mean (± SE)	Grasses Range	Alfalfa No. of Samples	Alfalfa Mean (±)	Alfalfa Range	Birds Foot Trefoil No of Samples	Birds Foot Trefoil Mean (± SE)	Birds Foot Trefoil Range
CP	7	11.5(0.7)	9.5–14.9	5	22.0(0.7)	19.7–23.5	2	21.3(0.4)	20.9–21.6
Ca	7	4.7(0.2)	3.8–5.6	5	22.6(1.5)	18.5–25.8	2	14.6(0.1)	14.5–14.7
Mg	7	1.9(0.0)	1.6–2.2	5	2.0(0.0)	1.9–2.2	2	2.0(0.1)	1.9–2.1
P	7	1.5(0.2)	0.9–2.2	5	1.2(0.1)	1.0–1.5	2	1.3(0.1)	1.2–1.4
S	7	1.8(0.1)	1.6–2.2	5	2.1(0.4)	0.8–2.9	2	2.0(1.5)	1.8–2.1
Cu	7	4.2(0.4)	3.4–6.1	5	6.7(0.3)	5.7–7.6	2	4.8(0.7)	41- 5.4
Zn	7	15.5(0.9)	12.3–19.6	5	16.2(2.2)	8.0–20.9	2	16.1(3.1)	13.0–19.2
Mo	6	1.1(0.1)	0.6–1.3	4	1.1(0.2)	0.8–1.6	2	1.6(0.4)	1.2–2.0
Fe	7	64.8(1.8)	59.6–73.9	5	63.0(3.5)	55.4–75.8	2	58.8(1.7)	57.1–60.5
Mn	7	102.4(26.8)	46.9–227.5	5	51.1(4.6)	35.4–63.3	2	45.2(0.1)	45.1–45.3
DM	7	60.4(7.8)	50.0–71.7	5	51.2(8.2)	37.3–58.3	2	37.6(9.1)	31.2–44.0
ADF	7	38.2(2.1)	34.3–40.1	5	31.2(4.2)	28.4–38.4	2	27.3(0.5)	26.9–27.6
			(August 23, 1983)						
CP	8	9.5(0.3)	8.0–10.9	6	14.1(1.0)	11.6–18.5	2	11.3(0.2)	11.1–11.4
Ca	8	4.5(0.4)	1.4–5.5	6	21.5(3.0)	15.0–34.2	2	13.2(0.8)	12.3–14.0
Mg	8	1.9(0.0)	1.5–2.1	6	2.0(0.0)	2.0–2.1	2	2.0(0.0)	2.0–2.0

Table A6(Continued)

(August 23, 1983)

Nutrients	Grasses No. of Samples	Grasses Mean(±)	Grasses Range	Alfalfa No. of Samples	Alfalfa Mean(± SE)	Alfalfa Range	Birds Foot Trefoil No. of Samples	Birds Foot Trefoil Mean(± SE)	Birds Foot Trefoil Range
P	8	1.4(0.1)	0.8--1.8	6	0.9(0.1)	0.5–1.3	2	0.7(0.2)	0.5–0.3
S	8	1.8(0.1)	1.1--2.4	6	1.4(0.2)	0.9–2.2	2	1.0(0.5)	0.5–1.4
Cu	8	4.2(0.2)	3.5--4.9	6	7.0(0.3)	6.1–8.0	2	4.0(0.5)	3.6–4.5
Zn	8	14.5(0.4)	12.7--15.8	6	10.5(0.5)	8.6–12.2	2	8.5(2.1)	3.0–6.4
Mo	8	1.3(0.2)	1.0--2.2	6	0.9(0.2)	0.3–1.5	2	1.1(0.3)	0.8–1.5
Fe	8	81.4	65.5--117.5	6	62.6(4.7)	47.1–80.4	2	43.5(1.0)	42.5–44.4
Mn	8	70.0(11.5)	22.6--124.5	6	33.2(2.1)	26.6–39.5	2	33.9(5.8)	28.1–39.6
DM	8	90.5(0.5)	89.9--91.2	6	89.2(0.9)	88.2–90.4	2	89.6(1.1)	88.8–90.4
ADF	8	39.3(1.1)	37.9--41.1	6	40.0(7.7)	25.0–46.6	2	42.4(4.0)	39.6–45.2

Appendix Table A7: Concentration of nutrients in forage during the grazing trial.

ABBREVIATIONS:

Period refers to sampling day:

Period 1 was June 28

2 " July 26

3 " August 23

4 " September 20

SPECIES: G for grass

A for Alfalfa

T for birds foot trefoil

DM Dry matter (%)

CP Crude protein (%)

ADF Acid detergent fibre (%)

Ca Calcium g/Kg DM

Mg Magnesium g/Kg DM

P Phosphorus g/Kg DM

S Sulphur g/Kg "

Cu Copper mg/Kg "

Zn Zinc mg/Kg "

Mo Molybdenum mg/Kg DM

Fe Iron mg/Kg DM

Mn Manganese mg/Kg DM

OBS	PERIOD	PADDOCK	SPECIES	DM	CP	ADF	CA	MG	P	S	CU	ZN	MO	FE	MN
1	1	1	G	78.9	17.7	30.0	0.38	0.18	0.13	0.25	4.51	17.8	0.88	52.1	45.2
2	1	2	G	88.0	13.5	32.6	0.34	0.17	0.14	0.23	5.07	19.6	0.77	46.2	67.5
3	1	3	G	87.5	15.2	33.1	0.36	0.17	0.25	0.21	5.60	16.4	1.52	63.2	47.0
4	1	4	G	88.2	16.0	32.9	0.35	0.20	0.26	0.25	5.45	19.1	0.94	27.1	19.4
5	1	5	G	56.6	13.9	32.7	0.37	0.20	0.17	0.21	4.54	19.9	0.85	58.7	44.1
6	1	6	G	83.1	13.2	32.3	0.35	0.18	0.23	0.20	6.38	19.2	1.16	47.5	83.9
7	1	7	G	82.1	14.2	31.6	0.38	0.16	0.10	0.21	5.00	18.5	0.74	55.9	54.7
8	1	8	G	76.1	16.0	32.6	0.46	0.20	0.25	0.21	6.97	21.0	1.39	32.6	33.6
9	1	2	A	51.4	26.2	24.5	1.66	0.21	0.21	0.25	6.21	23.2	1.23	59.4	40.7
10	1	3	A	81.7	27.6	23.7	1.89	0.21	0.35	0.25	8.46	27.4	2.00	64.1	44.6
11	1	4	A	87.5	24.3	22.0	2.05	0.22	0.18	0.24	5.83	18.2	0.99	50.3	50.4
12	1	6	A	87.0	25.8	23.4	1.83	0.20	0.14	0.24	6.85	22.9	1.26	55.2	42.8
13	1	7	A	58.8	23.4	27.8	1.72	0.20	0.23	0.24	6.39	24.1	1.33	61.0	44.2
14	1	8	A	55.2	25.0	24.6	2.83	0.21	0.15	0.20	7.05	20.9	1.82	55.8	41.8
15	1	1	T	91.7	25.0	17.1	.	.	.	.	9.73	31.1	.	.	.
16	1	5	T	89.3	27.5	17.6	1.60	0.20	0.11	0.21	5.75	26.4	0.73	70.4	58.9
17	2	1	G	50.1	12.6	37.7	0.51	0.20	0.20	0.17	6.09	19.6	1.10	61.9	46.9
18	2	2	G	50.6	14.9	34.3	0.56	0.22	0.22	0.20	4.21	15.9	.	73.9	178.5
19	2	3	G	64.2	11.7	37.2	0.46	0.20	0.14	0.22	4.81	16.2	1.34	62.8	52.0
20	2	4	G	59.4	10.4	39.9	0.43	0.17	0.13	0.17	3.36	13.2	0.59	62.9	81.4
21	2	5	G	71.7	9.5	38.3	0.50	0.21	0.09	0.16	3.53	16.7	0.89	67.7	66.8
22	2	6	G	63.4	11.0	40.1	0.38	0.16	0.14	0.17	3.86	14.7	1.24	59.6	227.5
23	2	7	G	63.2	10.5	39.8	0.42	0.20	0.10	0.20	3.48	12.3	1.27	64.6	63.9
24	2	2	A	37.3	23.5	29.3	2.58	0.19	0.14	0.29	5.73	18.0	0.96	61.1	55.3
25	2	3	A	53.9	20.9	38.4	1.95	0.22	0.12	0.20	7.58	15.8	1.20	55.4	35.4
26	2	4	A	51.0	23.5	28.4	2.38	0.19	0.15	0.08	6.70	18.1	0.79	75.8	63.3
27	2	6	A	55.4	19.7	31.4	2.53	0.20	0.10	0.22	6.32	8.0	.	58.7	51.8
28	2	7	A	58.3	22.5	28.4	1.85	0.20	0.10	0.27	7.17	20.9	1.61	64.0	49.9
29	2	1	T	31.2	20.9	27.6	1.45	0.19	0.14	0.21	4.09	13.0	1.17	60.5	45.1
30	2	5	T	44.0	21.6	26.9	1.47	0.21	0.12	0.18	5.42	19.2	2.01	57.1	45.3
31	3	1	G	90.1	9.6	39.6	0.55	0.19	0.11	0.21	4.90	14.9	1.12	65.5	56.5
32	3	2	G	90.3	10.9	38.9	0.53	0.20	0.16	0.11	4.84	12.7	1.36	81.9	93.2
33	3	3	G	89.9	10.0	40.4	0.44	0.19	0.18	0.16	4.30	15.2	1.45	86.8	35.1
34	3	4	G	89.9	9.3	41.1	0.49	0.21	0.17	0.21	4.20	15.8	1.11	117.5	65.0
35	3	5	G	91.2	8.0	37.9	0.45	0.15	0.08	0.24	3.48	14.8	0.98	75.8	124.5
36	3	6	G	91.2	9.9	38.7	0.52	0.21	0.15	0.18	4.28	14.7	0.95	69.9	78.8
37	3	7	G	90.6	9.7	38.2	0.51	0.19	0.09	0.20	4.09	13.4	0.97	78.7	84.0
38	3	8	G	90.9	8.9	39.9	0.14	0.20	0.15	0.16	3.78	14.5	2.20	75.1	22.6
39	3	2	A	88.5	14.7	41.6	2.59	0.20	0.13	0.18	6.99	9.9	1.02	69.4	31.2
40	3	3	A	88.7	14.7	40.9	2.05	0.20	0.10	0.09	6.93	11.0	1.51	57.4	26.6
41	3	4	A	88.2	13.5	41.4	1.82	0.21	0.09	0.16	7.42	8.6	0.25	56.3	39.5
42	3	6	A	90.4	11.6	46.6	1.50	0.20	0.07	0.22	6.81	10.4	0.89	47.1	30.0
43	3	7	A	89.9	11.7	44.5	1.51	0.21	0.05	0.09	6.07	11.1	0.75	80.4	32.1
44	3	8	A	89.5	18.5	25.0	3.42	0.20	0.10	0.12	8.04	12.2	0.87	64.9	39.5
45	3	1	T	88.8	11.4	39.6	1.40	0.20	0.08	0.05	4.49	6.4	0.78	44.4	39.6
46	3	5	T	90.4	11.1	45.2	1.23	0.20	0.05	0.14	3.59	10.6	1.46	42.5	28.1
47	4	1	G	91.7	9.2	44.2	0.55	0.20	0.22	0.16	3.68	12.4	1.76	99.3	44.3

OBS	PERIOD	PADDOCK	SPECIES	DM	CP	ADF	CA	MG	P	S	CU	ZN	MO	FE	MN
48	4	2	G	93.5	9.2	40.9	0.58	0.21	0.15	0.19	3.17	13.7	1.47	106.1	111.4
49	4	3	G	91.8	9.1	41.3	0.56	0.21	0.14	0.13	3.32	13.1	1.43	82.8	65.4
50	4	4	G	91.1	8.2	42.9	0.49	0.18	0.10	0.15	3.55	12.1	0.68	128.1	67.1
51	4	5	G	91.9	8.6	44.6	0.54	0.19	0.12	0.08	4.24	14.7	1.05	125.6	38.8
52	4	6	G	90.0	7.4	43.6	0.50	0.19	0.15	0.08	3.85	14.2	1.19	117.3	55.2
53	4	7	G	92.2	9.3	42.7	0.63	0.19	0.16	0.08	3.80	16.4	1.23	114.6	49.3
54	4	8	G	91.8	10.0	45.0	0.59	0.19	0.11	0.05	3.59	13.5	2.22	127.8	39.0
55	4	1	T	91.1	7.6	53.1	1.34	0.22	0.07	0.12	3.00	5.8	0.38	97.0	29.0
56	4	5	T	88.4	8.2	55.1	1.20	0.22	0.08	0.06	3.66	7.1	0.94	66.9	30.8

Appendix Table B1: Analysis of variance table for body weight, serum copper and serum zinc for all steers during the grazing trial.

	Source	DF	Body Weight Type III MS	Serum Copper Type III MS	Serum Zinc Type III MS
Main Plot	Group	1	1936892.31**	0.1529	0.0072
	ICU	1	6861.39	0.1369	0.0615
	Group xICU	1	37.56	0.0095	0.0928
	IZN	1	2801.04	0.0068	0.0195
	Group xIZN	1	2751.02	0.0654	0.1729
	ICU x IZN	1	5206.51	0.0032	0.3805
	Group x ICU x IZN	1	1055.46	0.2758**	0.1929
	Error A Anim(Group x ICU x IZN	52	33783.40	0.0574	0.0959
Sub Plot	Sampling day	4	177991.37**	0.6360**	1.5665**
	Group x Sampling day	4	7988.38**	0.0707*	0.0327
	ICU x Sampling day	4	223.57	0.0709*	0.0608
	IZN x Sampling day	4	1299.06*	0.0426	0.0314
	ICUxIZNxSampling day	4	234.79	0.0007	0.0361
	Error B	218	490.37	0.0284	0.0466

** $P < 0.01$

* $P < 0.05$

Appendix Table B2: Linear contrast analysis of variance table for body weight of all steers for the effect of injectable zinc treatment upon body weight together with an estimate of the difference in mean body weight between IZN treated and untreated steers.

Period Covered	Source	DF	Mean Square
Day 28 - 0	IZN	1	228.89 NS
63 - 0	IZN	1	0.95 NS
84 - 0	IZN	1	1671.58*
113 - 0	IZN	1	1535.82*
Day 28 - 0	Group	1	6408.26**
63 - 0	Group	1	8849.52**
84 - 0	Group	1	18019.22**
113 - 0	Group	1	27967.97**
Estimate	**Estimate**		**SE. of Estimate**
Day 28 - 0	5.58		3.7
63 - 0	0.16		3.7
84 - 0	0.81		3.6
113 - 0	6.52		3.6

Weight is expressed in kilograms.

NS - not significant $P > 0.10$

* - $P < 0.10$

** - $P < 0.01$

Appendix Table B3: Analysis of variance table for body weight, serum copper and serum zinc of Group I steers.

	Source	DF	Body Weight Type III MS	Serum Copper Type III MS	Serum Zinc Type III MS
Main Plot	ICU	1	5320.90	0.0479	0.1812
	IZN	1	7358.21	0.0789	0.0710
	ICUxIZN	1	1097.91	0.1400	0.0161
	Error A Anim(ICUxIZN)	33	487323.97	1.6850	0.0824
Sub Plot	Sampling day	4	683224.28**	0.3813**	0.9787**
	ICUxSampling day	4	349.63	0.250*	0.0561
	IZNxSampling day	4	5912.26*	0.0385	0.0098
	ICUxIZNxSampling day	4	853.93	0.0248	0.0442
	Error B	132	497.32	0.0266	0.0420

** $P < 0.01$

* $P < 0.05$

Appendix Table B4: Linear contrast analysis of variance table for the effect of IZN treatment upon body weight of group 1 steers during the grazing trial together with an estimate of the difference in body weight between IZN treated and untreated steers at each sampling day relative to day 0.

Sampling day covered	Source	DF	Type III MS
Day 28-0	IZN	1	447.70 NS
68-0	IZN	1	29.60 NS
84-0	IZN	1	1553.35*
113-0	IZN	1	1447.04*
	Error	132	497.32 NS
Estimate	Estimated difference[1]	SE. of estimate	Significance
Sampling day			
28-0	4.5	4.7	NS
63-0	1.2	4.7	NS
84-0	-8.3	4.7	*
113-0	-9.0	4.7	*

* $P < 0.10$

NS - Not significant $P > 0.10$

1 estimate is expressed in kg
Numbers preceeded by negative signs represent the difference by which IZN treated steers weighed less than control steers.

Appendix Table B5: Analysis of variance table for body weight of Group II steers during the grazing trial

	Source	DF	Type III MS
Main Plot	ICU	1	2310.42
	IZN	1	0.07
	ICUxIZN	1	4386.33
	Error A Anim(ICUxIZN)	19	66823.38
Sub Plot	Sampling day	4	44627.98**
	ICUxSampling day	4	552.64
	IZNxSampling day	4	55.13
	ICUxIZNxSampling day	4	435.21
	Error B	74	497.63

** $P<0.01$

Appendix Table B6: Linear contrast analysis of variance table for serum copper of all steers for the effect of ICU treatment upon serum copper together with an estimate of the difference in serum copper between ICU and control steers on sampling days 28, 63, 84 and 113 relative to day 0.

Contrast	Source	DF	Mean Square
Sampling day			
28 - 0	ICU	1	0.0180
63 - 0	ICU	1	0.0097
84 - 0	ICU	1	0.810*
113 - 0	ICU	1	0.0351

Estimate	Estimate	SE. of Estimate	Significance
Sampling day			
28 - 0	-0.0494	0.06	NS
63 - 0	-0.0363	0.06	NS
84 - 0	-0.1048	0.06	*
113 - 0	-0.0689	0.06	NS

* $P < 0.10$

NS - Not significant ($P > 0.10$)

Appendix Table B7: Linear contrast analysis of variance table for the effect of ICU treatment on serum copper of Group I steers together with an estimate of the difference in serum copper of ICU treated and untreated steers on sampling days 28, 63, 84 and 113 of the grazing trial relative to day 0.

Contrast	Source	DF	Type III MS
Sampling day			
28 - 0	ICU	1	0.0123
63 - 0	ICU	1	0.0004
84 - 0	ICU	1	0.1204*
113 - 0	ICU	1	0.0266
	Error	132	0.0087
Estimate	**Estimated difference**	**SE. of Estimate**	**Significance**
Sampling day			
28 - 0	0.0516	0.0758	NS
63 - 0	0.0092	0.0763	NS
84 - 0	0.1615	0.0759	*
113 - 0	0.0434	0.0759	NS

* $P < 0.05$

NS - Not significant ($P > 0.05$)

Appendix Table B8: Analysis of variance table for serum copper, and serum zinc of group II steers during the grazing trial.

	Source	DF	Serum Copper Type III MS	Serum Zinc Type III MS
Main Plot				
	ICU	1	0.0385	0.0026
	IZN	1	0.0126	0.1224
	ICUxIZN	1	0.1417	0.4616
	Error A			
	Anim(ICUxIZN)	19	0.0277	0.1107
Sub Plot	Sampling day	4	0.3064**	0.6128**
	ICUxSampling day	4	0.0140	0.0113
	IZNxSampling day	4	0.0114	0.0450
	ICUxIZNxSampling day	4	0.0410	0.0908
	Error B	75	0.0319	0.0570

** $P<0.01$

Appendix Table B9: Analysis of variance table for liver copper of all steers together with contrast analysis and estimate of the difference in liver copper of ICU treated and untreated steers at each sampling day relative to day 0.

	Source	DF	Type III MS
Main Plot	Group	1	7245.9531
	ICU	1	735.0479
	GroupxICU	1	104.0817
	IZN	1	2085.1353
	GroupxIZN	1	1188.2068
	IZNxICU	1	0.0148
	Error A		
	Anim (GroupxICUxIZN)	52	2565.0673
Sub Plot	Sampling day	2	144853.63**
	GroupxSampling day	2	11866.16**
	ICUxSampling day	2	4053.82**
	IZNxSampling day	2	45.37
	ICUxIZNxSampling day	2	419.64
	Error B	109	577.6873
Contrast			
Sampling day			
Day 63 - 0	ICU	1	7691.81**
115 - 0	ICU	1	3826.73*
		Estimate	SE. of Estimate
Day 63 - 0		32.43	3.65**
115 - 0		22.88	2.57*

** P < 0.01 * P < 0.05

Appendix Table B10: Analysis of variance table for liver copper of group I steers together with contrast analysis and estimate of the difference in liver copper of ICU treated and untreated steers at sampling days 63 and 115 relative to day 0.

	Source	DF	Type III MS
Main Plot	ICU	1	172.3496
	IZN	1	330.6141
	ICU x IZN	1	742.4661
	Error A		
	Anim (ICUxIZN)	33	1586.5829
Sub Plot	Sampling day	2	49881.4000**
	ICUxSampling day	2	2685.9646**
	IZNxSampling day	2	44.5608
	ICUxIZNxSampling day	2	466.5522
	Error B	66	529.8563
Contrast			
Sampling day			
Day 63 - 0		1	3383.2805*
113 - 0		1	4566.4756**

Estimate	Estimate difference	SE. of estimate	Significance
Day 63 - 0	27.3	10.8	*
115 - 0	31.5	10.7	**

** P < 0.01

* P < 0.05

Appendix Table B11: Analysis of variance table for liver copper of Group II steers together with contrast analysis and estimate of the difference in liver copper of ICU treated and untreated steers at sampling days 63 and 115 relative to day 0.

	Source	DF	Type III MS
Main Plot	ICU	1	536.3971
	IZN	1	1838.6834
	ICUxIZN	1	466.977
	Error A		
	Anim (ICUxIZN)		4275.5185
Sub Plot	Sampling day	2	93480.56**
	ICUxSampling day	2	2378.2523*
	IZNxSampling day	2	184.6717
	IZNxICUxSampling day	2	122.8552
	Error B	37	671.6536

Contrast Period covered	Source	DF	Type III MS
Day 0 - 63	ICU	1	4151.9538*
Day 0 - 115	ICU	1	140.9124

Parameter	Estimate	SE. of Estimate	Significance
ICU			
63 - 0	39.3	15.8073	*
115 -0	7.2	15.8073	NS

** $P < 0.01$

* $P < 0.05$

NS - Not significant ($P > 0.05$)

Appendix Table B12: Split plot analysis of variance table for liver zinc of all steers in the four treatment groups measured during the grazing trial.

	Source	DF	Mean Square
Main Plot	Group	1	67.69
	ICU	1	204.23
	GroupxICU	1	228.47
	IZN	1	26.98
	GroupxIZN	1	555.92
	lCUxIZN	1	87.15
	GroupxICUxIZN	1	314.49
	Error A Anim (GroupxICUxIZN)	52	307.16
Sub Plot	Sampling day	2	1845.64**
	GroupxSampling day	2	210.45
	ICUxSampling day	2	252.08
	IZNxSampling day	2	18.90
	ICUxIZNxSampling day	2	36.32
	Error B	109	132.29

** $P < 0.01$

Appendix Table B13: Split plot analysis of variance table for liver zinc of group I steers measured during the grazing trial.

	Source	DF	Type III MS
Main Plot	ICU	1	7.89
	IZN	1	67.84
	ICUxIZN	1	445.89
	Error A Anim (ICUxIZN)	33	194.37
Sub Plot	Sampling day	2	7720.06**
	Sampling dayxICU	2	53.66
	Sampling dayxIZN	2	172.35
	ICUxIZNxSampling day	2	166.98
	Error B	66	146.45

** $P < 0.01$

Appendix Table B14: Split plot analysis of variance table for liver zinc of group II steers during the grazing trial.

	Source	DF	Type III MS
Main Plot	ICU	1	346.205
	IZN	1	329.580
	ICUxIZN	1	33.540
	Error A Anim (ICUxIZN)	19	242.138
Sub Plot	Sampling day	2	282.271
	ICUxSampling day	2	210.078
	IZNxSampling day	2	6.093
	UCUxIZNxSampling day	2	258.926
	Error B	37	155.255

Appendix Table B15: Analysis of variance table for body weight of steers together with contrast analyses to determine the effect of ICU treatment at the start of the grazing trial on body weight of steers during the finishing trial.

	Source	DF	Type III MS
Main Plot	ICU	1	50925.07**
	IZN	1	264.78
	ICUxIZN	1	16159.93
	Error A		
	Anim (ICUxIZN)	39	9916.08
Sub Plot	Sampling day	2	547651.00***
	ICUxSampling day	2	2820.04**
	IZNxSampling day	2	489.93
	ICUxIZNxSampling day	2	218.18
	Error B	77	903.61
Contrast Period covered			
Day 0 - 35	ICU	1	4860.049**
35 - 79	ICU	1	3457.688*

Parameter	Estimate	SE. of Estimate	Significance
ICU			
Day 35 - 0	13.7	5.9	
Day 79 - 0	11.6	5.9	

* $P < 0.10$

** $P < 0.05$

*** $P < 0.01$

Appendix Table B16: Analysis of variance table for carcass weight of steers at the end of the finishing trial.

Source	DF	Type III MS
ICU	1	7704.146*
IZN	1	938.252
ICUxIZN	1	1641.670
Error	38	1207.536

* $P < 0.05$

Appendix Table B17: Analysis of variance table for liver copper of steers during the finishing trial together with linear contrast analysis and estimated difference in mean liver copper of ICU treated and ICU-untreated steers.

	Source	DF	MS
Main Plot	ICU	1	145.0917
	IZN	1	12.4425
	ICUxIZN	1	857.7890
	Error A		
	Anim(ICUxIZN)	38	1204.0915
Sub Plot	Sampling day	1	93901.1102**
	ICUxSampling day	1	2623.0746
	IZNxSampling day	1	296.4134
	ICUxIZNxSampling day	1	0.2896
	Error B	38	415.8494
Linear Contrast			
ICU Day 0 - 79	ICU	1	2623.0746*

Estimate	Estimated Difference	SE. of Estimate	Significance
ICU			
Day 0 - 79	-22.4[1]	8.91	*

* $P < 0.05$

** $P < 0.01$

1 Liver copper expressed in mg/Kg DM

Appendix Table B18: Analysis of variance table for liver zinc of steers during the finishing trial.

	Source	DF	MS
Main Plot	ICU	1	20.6225
	IZN	1	1.8324
	ICUxIZN	1	362.6252
	Error A		
	Anim(ICUxIZN)	38	252.8709
Sub Plot	Sampling day	1	10342.7541**
	ICUxSampling day	1	284.6715
	IZNxSampling day	1	2.8039
	ICUxIZNxSampling day	1	108.5370
	Error B	38	107.6454

** $P<0.01$