

COPPER OXIDE NEEDLES FOR GRAZING
COWS AND CALVES

By

Heather J. Cameron

A Thesis

Submitted to the Faculty of Graduate Studies
In Partial Fulfillment of the Requirements for the Degree of
Master of Science

Department of Animal Science

University of Manitoba

Winnipeg, Manitoba

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ABSTRACT

Copper oxide needles (CuON) provided as a single oral dose at the start of a grazing trial of 92 days in length, was evaluated using a herd of purebred Hereford cows and their calves. Thirty-seven cows were allocated to treatments of 0, 25 or 50 g CuON (79.9% Cu in CuON) on the basis of body weight and plasma Cu. Calves were dosed with 0 or 20 g CuON, within cow treatment group, with allocation on the basis of body weight, plasma Cu and sex of calf. Cows and calves were weighed and blood samples were taken on days 0, 28, 63 and 92, while liver biopsies were taken on days 0, 28 and 92. A combination of low Cu in the grass forage ($n = 4$) at $3.7 \text{ mg kg}^{-1} \text{ DM}$ (± 0.4), together with Mo and S at 3.9 mg (± 0.3) and 1.4 g (± 0.0) $\text{kg}^{-1} \text{ DM}$, respectively, plus water ($n = 4$) with $947 \text{ mg sulphate L}^{-1}$ (± 26.3) contributed to a low available Cu in the forage consumed by cows and calves. Body weight of cows and calves was not influenced ($P > 0.05$) by CuON. Liver Cu was higher ($P < 0.01$) in treated cows and calves. Liver Cu was not different ($P > 0.05$) between cows dosed with 25 or 50 g CuON. Treatments of cows and calves with CuON had no influence ($P > 0.05$) upon the concentration of Fe or Mo in liver or plasma, the Cu concentration and CpOx activity in plasma or the concentration of Zn in liver, in cows and calves. Plasma Zn of cows did not differ ($P > 0.05$). Plasma Zn of calves was higher ($P < 0.05$) in the calves suckling CuON treated cows. Dosing cows and calves with CuON resulted in a higher Cu content of liver, but did not adversely influence the metabolism of Fe or Zn, or modify the concentration of Mo in the plasma or liver of dosed animals.

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LIST OF ABBREVIATIONS

A	absorbance
(A)	Cu absorption coefficient
BW	body weight
CpOx	ceruloplasmin oxidase activity
CuON	cupric oxide needles
CuTM	copper-thiomolybdate
DM	dry matter
g	gram
kg	kilogram
L	litre
LW	live weight
LD ₅₀	lethal dose of 50% of population
LD ₅	lethal dose of 5% of population
mg	milligram
min	minute
MT	metallothionein
SO ₄	sulphate
SOD	superoxide dismutase
TCA	trichloroacetic acid
TM	thiomolybdate
TM ₄	tetrathiomolybdate
Δ	delta (change)
μg	microgram

INTRODUCTION

Copper deficiencies have been reported in grazing cattle throughout Canada (Brockman 1977; Smart et al. 1980; Boila et al. 1984a; Korsrud et al. 1985). In Manitoba, a Cu deficiency due to excessive levels of Mo in forages was first reported by Cunningham et al. (1953) in northwestern Manitoba. Drysdale (1979) and Boila et al. (1984b, d) confirmed that cattle in Manitoba may suffer from a Cu deficiency.

Cattle consuming forage low in Cu are susceptible to a primary Cu deficiency (Underwood 1977). High Mo levels in forage (Boila et al. 1984d) or sulphur in forage (Boila et al. 1987) or water (Boila et al. 1984d) are commonly found in northwestern Manitoba and may lead to an induced metabolic Cu insufficiency or a secondary Cu deficiency. Both primary and secondary Cu deficiencies are encountered in Manitoba (Drysdale et al. 1980). Cattle grazing pasture with adequate Cu levels of 10 to 14 mg Cu kg⁻¹ DM showed signs of Cu deficiency due to high Mo levels in the forage ranging from 3 to 20 mg Mo kg⁻¹ DM (Thorton et al. 1972). When dietary Cu appears to be adequate ruminants require a minimum Cu:Mo ratio of 2.0 to prevent a Cu deficiency condition (Miltimore and Mason 1971).

Additionally, S interacts with Cu and Mo in the rumen reducing Cu absorption. In the rumen, sulphate or dietary amino acids are reduced to sulphide and thiomolydates $(\text{MoO}_{4-n}\text{S}_n)^{2-}$, $n=1$ to 4) are formed. This leads to the formation of insoluble Cu thiomolybdates in the digestive tract (Dick et al. 1975) thereby reducing the availability of dietary Cu. Elements such as Zn, Fe and Cd may also inhibit Cu absorption and utilization (Campbell et al. 1974; Pringle et al. 1973; Mills 1980).

There are several methods available to prevent Cu deficiency. Cu salts in a free choice mineral supplement effectively provided supplemental Cu to grazing cattle (Cunningham et al. 1953); however, calves do not always appear to consume adequate mineral (Boila et al. 1984b). In addition mineral intake is extremely variable when mineral is available free-choice. Cu sulphate included as a dietary supplement in drinking water is of no practical use for grazing cattle (MacPherson 1982). Injectable Cu preparations provide an effective source of supplementary Cu, and are largely effective against secondary Cu deficiency because absorption from the alimentary canal is by-passed (Allen and Mallinson 1984). Injectable Cu glycinate (Cunningham 1959) and CuCaEDTA (Suttle 1981b) provided effective Cu supplementation; however there are disadvantages associated with this method. These include 1) difficulty in controlling the rate of Cu release, thereby resulting in a Cu toxicity (Mylrea and Byrne 1974); 2) slow absorption of the supplement resulting in a residue buildup and potential of carcass damage at the site of injection (Boila et al. 1984a) and 3) short-lived benefits requiring repeated injections (MacPherson et al. 1979). Cu containing glass boluses appear to provide a similar degree of protection as the injectable Cu source (Allen et al. 1984) but they predispose the animal to Cu toxicity through rapid bolus disintegration (Tengrove and Judson 1985).

Attention has focused on the effective use of Cu oxide needles (CuON) orally administered to grazing sheep and cattle (Suttle 1981a; Dewland et al. 1986). Dewey (1977) reported that CuON entered the rumen transiently before passing to the abomasum where some needles are retained in the folds. There is a release of Cu where conditions for

absorption of Cu are favourable. The nature of the diet and the availability and quantity of Cu in the oxide form may determine the quantity of Cu available to the animal. The optimum dosage rate under different conditions of dietary Cu, Mo and S intakes has yet to be determined (Langlands et al. 1986a, b).

This study was carried out to evaluate the effectiveness of a single dose of CuON administered to beef cows at 0,25 and 50 g and to suckling calves at 0 and 20 g at the start of the grazing season. Parameters measured included body weight, the concentration of Cu, Mo, Fe and Zn in liver and blood plasma and plasma ceruloplasmin activity (CpOx).

LITERATURE REVIEW

Copper nutrition and metabolism have been extensively studied for many years because of the major problems of copper deficiency and toxicity associated with ruminant diets. Copper is an element in Group 1B of the Periodic System. It is a transition element containing one electron in the outer shell. Copper has a variable valency forming cuprous (Cu^{+1}) and cupric (Cu^{+2}) compounds, but more commonly in the Cu^{+2} state (Georgievskii 1982).

Copper is a highly versatile and essential micronutrient in the mammalian body. Copper is most commonly used in a biological role as a copper metalloenzyme. These cuproenzymes are involved in numerous processes including the formation of melanin pigment in hair and skin (tyrosinase); synthesis of the structural subunits of collagen and elastin (lysyl oxidase); the tertiary structure of keratin proteins (monoamine oxidase); adrenal synthesis of catecholamine (dopamine B hydroxylase); killing bacteria by white blood cells (Zn-Cu superoxide dismutase); and mitochondrial energy production through oxidative phosphorylation (cytochrome C oxidase) (Allen and Solomons 1984). Bone formation; iron metabolism, myelination of the spinal cord and reproduction depend on Cu metabolism (Underwood 1977).

Copper Requirements

Before Cu requirements can be specified, it is necessary to determine the dietary conditions to which the requirements must apply. Interactions between Cu and other dietary components, such as Mo and S, in particular are of prime concern.

Cu requirements in sheep increased by 50% by raising the sulphur content of the diet from the lower to the upper limit of the normal range of forage S (Whitehead 1966 as cited in Underwood 1981). Thornton et al. (1972) reported cattle grazing pasture forage with adequate Cu levels of 10 to 14 mg Cu kg⁻¹ showed signs of Cu deficiency due to the high Mo (3 to 20 mg kg⁻¹) content of the pasture. From this study it was concluded that when Mo levels are greater than 1.0 mg kg⁻¹ DM, it is important to know the dietary ratio of Cu:Mo when considering dietary Cu requirements. Miltmore and Mason (1971) reported a Cu:Mo ratio lower than 2:1 can result in Cu deficiency in cattle. A Cu:Mo ratio of 4:1 was thought to be ideal (Alloway 1973; Stewart and Racz 1977; Fisher and Waldern 1978).

Dietary Cu requirements are based upon the need for Cu to meet endogenous body losses and the demands for growth, pregnancy and lactation. Preruminant calves at 40 kg body weight are reported to have an absorption coefficient for Cu of 0.7 while all other ruminating cattle have a coefficient of 0.04 (ARC 1980). When dietary S and Mo contents are known, Cu absorption coefficients can be calculated from the equation by Suttle and McLauchlan (1976). These researchers examined data from repletion experiments with sheep in which the true availability (A) for Cu was measured in diets differing in Mo and S

content. The availability was highly correlated ($r = 0.93$) with the dietary concentrations of S (g kg^{-1}) and Mo (mg kg^{-1}) as described in the equation:

$$\log (A) = -1.153 - 0.076 (S) - 0.013 (S \times \text{Mo})$$

Table 1 lists the Cu requirements of cattle through different classes (ARC 1980).

Smith (1982) reported that the maintenance requirement for a wide range of species fell within a narrow range of values when expressed on a per kg body weight (BW) basis. Dietary Cu requirement is approximately 0.1 mg kg^{-1} BW per day. Requirements show no general relationship to metabolic rate but do show some variation in relation to growth rate. During growth, differences in dietary Cu requirements are modified by large differences in availability of that Cu. Endogenous losses of trace elements are more logically related to body size than metabolic rate and energy needs.

Table 1. Estimated requirements of copper for cattle*

Class of animal	Live weight (kg)	Rate of gain stage of pregnancy or milk yield	Absolute requirement (mg Cu/day)		Relative requirement (mg Cu kg ⁻¹ diet DM)
			Net	Dietary	
Pre-ruminant calf	40	0.5 kg/day gain	0.81	116	1.12
Growing cattle	100	0.5	1.24(0.58) [†]	31	8-14(6.2)
		1.0	1.76(0.81)	44	10-16(7.5)
	200	0.5	1.94	48	8-14
		1.0	2.45	61	9-15
	300	0.5	2.64(1.25)	66	8-15(6.7)
		1.0	3.16(1.49)	79	9-15(7.2)
Adult	500	0	3.50	88	12-19
Pregnant cow	500	7 months	4.56	114	13-17
		9 months	5.64	141	13-20
Lactating cow	500	10 kg milk	4.50	113	10-14
		20	5.50	138	8-11
		30	6.50	163	8-11

*ARC (1980).

*Calculated from: endogenous loss, 7 $\mu\text{g Cu kg}^{-1}$ LW per day; growth, 1 mg Cu kg⁻¹ gain; milk, 0.1 mg Cu kg⁻¹; pregnancy, based upon predicted daily increments in the Cu content of the conceptus.

[†]Values in brackets reported by Suttle (1983). Endogenous loss, 3.4 $\mu\text{g Cu kg}^{-1}$; growth, 0.47 mg Cu kg⁻¹ gain; absorption coefficient of 0.03.

Absorption of Copper

The absorption of Cu in the body is influenced by dietary intake of Cu as well as genetic factors, age, dietary composition and the physiological state of the animal (Underwood 1977).

Copper absorption appears to be very rapid in the upper intestine; however, specific sites of absorption are not agreed upon. In most mammals, copper absorption appears to take place in the stomach or upper portion of the small intestine (Aspin and Sass-Kortsak 1981). Van Campen and Mitchell (1965) found copper absorption in rats to be greatest from the stomach with declining rates from the pylorus to the ileum, where absorption declined to 20% of that from the stomach. Owen (1964) reported copper absorption in the rat to be highest from the jejunum followed by the stomach. In everted intestinal sacs of the hamster, maximal copper absorption was found to be in the lower small intestine (Crampton et al. 1965). It is not clear whether differences in sites of absorption are due to a species effect. In sheep, Prokuchin et al. (1976 as cited in Georgievskii 1982) reported absorption occurred in the forestomach, while Grace (1975) found the large intestine was the site of considerable absorption.

The rapid absorption of copper in the small intestine was demonstrated by Marceau et al. (1970). The maximum rate of appearance of ^{64}Cu in the plasma of the rat occurred at 0.5 hours after dosing. In man serum ^{64}Cu , bound to albumin and partly to amino acids, peaks at 1 to 2 hours after dosing followed by a drop as the liver takes up the Cu. A slow secondary rise in ^{64}Cu in plasma appeared for 48 to 72 hours as the copper becomes incorporated into the copper protein, ceruloplasmin.

(Sternleib 1967).

In the low pH contents of the stomach, copper ions are readily soluble and available for absorption. As the gastric contents pass into the duodenum they are subject to an increasing pH environment. Copper ions tend to form insoluble precipitates in a basic medium. These precipitates were thought to inhibit copper absorption (Allen and Solomons 1984). However, Gollan et al. (1971) found substances that formed soluble complexes with copper under alkaline conditions in human alimentary secretions.

Cu is essential for the microflora of the forestomach. Rumen microorganisms take up cupric ions forming a water soluble complex. Rumen fluid contains Cu at a concentration between 0.1 and 1.0 mg L⁻¹. Cu in the rumen affects volatile fatty acid formation and bacterial protein synthesis (Georgievskii 1982).

Suckling animals have a more efficient absorption mechanism to ensure an adequate Cu supply. Bremner and Dalgarno (1973a, b) estimated that 50 to 60% of the Cu from a diet supplemented with cupric sulphate offered to calves at 3 to 14 weeks of age was retained in the liver. The Cu absorption coefficient predicted by ARC (1980) is 0.7 for calves and 0.04 for mature cattle. The high Cu uptake of the young animals was thought to be due to a high retention of immobile Cu in the intestinal walls which becomes mobile as the animal is weaned (Bremner 1980).

Efficiency of Cu absorption increases during pregnancy (Davies and Williams 1976). They found the absorption of an intragastric dose of ⁶⁴Cu to be 54% in pregnant rats versus 26% in non-pregnant rats.

Dietary copper varies in form and availability. Using labelled copper compounds with sheep, Lassiter and Bell (1960) found the

availability of cupric carbonate and copper salts to be greater than that of cupric oxide. The relative absorption of inorganic sources in cattle was shown to be $\text{CuCO}_3 > \text{Cu}(\text{NO}_3)_2 > \text{CuSO}_4 > \text{CuCl}_2 > \text{Cu}_2\text{O} > \text{CuO}$ (Chapman and Bell 1963).

Absorptive Mechanism

Few studies have been undertaken to identify the mechanism by which Cu is absorbed (Crampton et al. 1965; Marceau et al. 1970; Hall et al. 1979; Bremner 1980). It is suggested that Cu absorption from the mucosal to the serosal side of the intestine occurs by two separate steps; first movement through the mucosal membrane, and then transport across to the serosal side.

Both steps are dependent upon the concentration of Cu and are saturable. Mucosal uptake is not energy dependent, while transport out of the cell is. The second step operates at a rate lower than that of mucosal uptake and may limit the transfer rate of Cu (Crampton et al. 1965). Marceau et al. (1970) reported the entry rate of ^{64}Cu into rat plasma was linearly related to the amount of Cu given by a stomach tube up to doses of 12 ug Cu. Under these conditions, 30% of the metal was absorbed. The relationship was non-linear at doses between 12 and 36 ug Cu. The percent copper absorbed decreased to 13% at 36 ug Cu. Maximum absorption rates were achieved within 30 minutes, with absorption continuing at a slower rate for several hours thereafter. This could be due to the passage of ^{64}Cu into the lower small intestine where the rate of absorption of Cu was lower. However, it could be the result of a saturation of the rapid transport system, which in turn leads to an increased involvement of the slower transport system (Bremner 1980).

The slower rate of absorption system could result from the mucosal cell uptake of Cu and the subsequent slow Cu release from these cells (Crampton et al. 1965). Evans and Johnson (1978) proposed that the important step in the regulation of Cu absorption was the binding of the

Cu in mucosal cells to a specific protein, metallothionein (MT), when dietary copper was high. The oral administration of Cu induced MT synthesis in the rat intestine while administration of cycloheximide, an inhibitor of MT synthesis, increased Cu absorption due to the lack of storage of Cu by MT. Alternatively, Bremner (1980) found that the efficiency of Cu absorption decreased and no increase in the mucosal content of ^{64}Cu or Cu-MT was seen when dietary Cu was increased in rats. Hall et al. (1979) reported the concentrations of non-radioactive Cu and Cu-MT in the intestinal mucosal of rats were not greatly affected by oral or parenteral doses of Cu.

The proposal by Evans and Johnson (1978) that MT may act as a mucosal block may hold true in certain situations. Hall et al. (1979) reported that the intestinal absorption of ^{64}Cu was decreased in rats by 50% after increasing dietary zinc to 900 mg kg^{-1} . There was a major increase in the mucosal content of ^{64}Cu with most of this additional ^{64}Cu being bound to MT. The antagonistic effect of zinc on copper absorption appears to be related to its ability to increase the concentration of MT in the mucosa by up to 25 times. Copper has a greater affinity than zinc for binding sites on MT. Therefore, Cu can displace zinc from MT and form a copper-MT complex which is not readily transported into the blood (Bremner 1980).

Brewer (1987) appeared to agree with Evans and Johnson (1978) in that Cu can increase the MT content in tissues. An enzymatic function of MT has not been found but when large amounts of Cu are ingested, MT prevents the development of Cu toxicity by binding Cu and slowly releasing the Cu from the enterocytes to the bloodstream. People with Menke's disease and brindled mice have a strong retention of Cu in

enterocytes by MT. The genetic defect is thought to be caused by a mucosal block.

Copper Transport in the Circulatory System

About 90 to 95% of the total plasma Cu is tightly bound in the form of ceruloplasmin (CpOx) an oxidase synthesized in the liver. This complex is referred to as the "indirect-reacting Cu" since it will not react with diethyldithiocarbamate unless the Cu becomes ionic as a result of acid cleavage of the protein-Cu linkage (Aspin and Sass-Kortsak 1981). A small fraction (1 to 2%) of the Cu not bound to CpOx is associated with selected amino acids such as histidine, threonine and glutamine. The remaining non-CpOx bound Cu is loosely bound in its cupric form to albumin. This is referred to as the "direct-reacting Cu" since it reacts with diethyldithiocarbamate, without acid cleavage. The albumin- and amino acid-bound Cu are thought to be in equilibrium with each other (Bremner 1980).

The forms in which Cu passes to the serosal side of the intestinal mucosa have not been established (Bremner 1980). In the plasma, Cu is rapidly incorporated into complexes with albumin and amino acids. These complexes are rapidly taken up by the liver and other tissues. The liver is the main organ in the body for Cu storage and metabolism. Copper reaching the liver is incorporated into the mitochondria, microsomes, nuclei and soluble fraction of the parenchyma cells (Underwood 1977).

Copper Excretion

In the ruminant, Cu is released into the bile and ultimately into the feces. Trace amounts of Cu are also excreted in the urine, perspiration and milk (Underwood 1977).

The amount and rate of fecal Cu excretion depends upon the amount and form of dietary Cu. Lassiter and Bell (1960) fed various copper sources to sheep and examined Cu excretion. After oral dosing of the sulphate, chloride and nitrate Cu sources, cumulative fecal excretions ranged from 78% to 87% of the dose after 96 hours but only 3% of the dose for the cupric oxide needles.

Cumulative urinary excretion following intravenous injections of cupric sulphate, chloride and nitrate showed the chloride excretion to be greater than the sulphate or nitrate forms. Significantly more Cu was excreted in the urine and feces with the chloride source. The sulphate form has the lowest solubility in water followed by nitrate and chloride. Tissue uptake is greater with the sulphate form and less Cu from this source was seen in blood, plasma, urine and feces (Lassiter and Bell 1960). Cumulative fecal excretions averaged 7, 80 and 90% when oral doses of cupric oxide needle, cupric carbonate and cuprous oxide powder were given. Cumulative urinary excretion was greatest for the carbonate (3.1% of the dose) whereas it was only 0.19 to 0.25% for the other sources (Lassiter and Bell 1960).

Urinary Cu is excreted in low amounts due to the fact that ionic Cu does not exist in a free state in biological systems. It is usually bound to proteins or amino acids (Aspin and Sass-Kortsak 1981). Bremner and Young (1978) reported increases in urinary Cu excretion in ruminants

fed diets high in Mo and S. This was thought to be due to the presence of a Cu-Mo-sulphate complex in the plasma. This complex of minerals, not metabolically available to the animal, is taken up by the kidney and excreted.

Soli and Rambaek (1978) reported that Cu excretion in the bile is at very low amounts; however in their study, no bile was returned to the intestine. Brown (1967) reported the excretion of Cu in the bile of sheep is followed by a mechanism which involves restricted filtration. Cu excretion in bile was stable in sheep at 10 to 35 $\mu\text{g dL}^{-1}$. When large amounts of Cu are suddenly released in the body following destruction and reabsorption of blood coagula, Cu filtration increases, and concentrations of Cu in the bile can reach concentrations of Cu seen in plasma. Owen (1980) reported that in Cu deficient states there is an increase in the partitioning of liver Cu to the synthesis of CpOx at the expense of the biliary compartment. In pregnant rats, there is an almost discontinued biliary Cu excretion despite the fact that hepatic Cu remains normal.

Factors Affecting Cu Availability

Plant Factors

Mineral content of forages can be determined chemically but biological availability is more difficult to estimate. Biological availability of minerals in forages is a partial function of the extent to which they are solubilized in the rumen (Kincaid and Conrath 1983). Cu availability may be reduced by soluble plant proteins (Ivan and Velra 1981).

The mineral in forages consist of three fractions: a fast reacting, soluble fraction of high bioavailability to ruminants, a fraction which is available for absorption due to fibre and protein digestion and a bound fraction of low bioavailability (Kincaid and Conrath 1983). The percent of total Cu associated with acid detergent fibre in alfalfa hay and grass silage was approximately 40 and 60%, respectively. As forage quality declines, with a concomitant decrease in crude protein and an increase in fibre level, Ca, Mg and Cu in alfalfa, and P, Mg, Zn and Cu in grass silage decline. Ca and Cu in the neutral detergent fibre and acid detergent fibre fractions decrease when there are increased amounts of cell wall and lignin. This suggests there is a dilution or translocation effect (Kincaid and Conrath 1983).

Underwood (1981) reported that Cu and Mo levels fall to a small extent with advancing maturity of a forage. However, Jarvis and Austin (1983) reported grass samples fell to a low of $3.0 \text{ mg Cu kg}^{-1} \text{ DM}$ in July compared to an average value of approximately $7.5 \text{ mg Cu kg}^{-1} \text{ DM}$ for the rest of the grazing period.

There is evidence that availability of Cu to animals is higher in

mature as opposed to fresh herbage (Hartsman and Bosman 1970). The decline in hepatic reserves on turning cattle out to graze short grass is more rapid than when grass at a later stage of growth is consumed.

Hay has a higher Cu availability than fresh forage (Ferguson et al. 1943). Diarrhea was seen in cattle grazing forage high in Mo content. However, the diarrhea stopped when the forage was frosted or made into hay. The response was thought to be a result of a decrease in the water-soluble Mo content of the forage with little or no change in total Mo.

Fleming (1965 as cited by Reid and Horvath 1980) concluded legumes usually contain higher concentrations of Cu than grasses. The decline in concentration of Cu and Mo in grasses and legumes with advancing maturation is continuous in grasses but not with legumes (Reid and Horvath 1980). Legume species were generally found to be higher in Cu than grasses in Northwestern Manitoba although in some cases, grasses had higher Cu levels than legumes (Boila et al. 1984c).

Grasses differ in their Cu availability. Heifers fed quack grass were less susceptible to Cu deficiency than those fed tall fescue, although the fescue contained more Cu (Stoszek et al. 1986). Grains are lower than forages in Cu content. Plants containing high levels of Mo, S, phytate or lignin may be reduced in Cu availability (Underwood 1977).

Soil Type

Copper deficiency in livestock occurs on a wide variety of soil types (Underwood 1981). This relates to the fact that many forms of Cu imbalances are due to interference from other minerals. Rocks with the higher Cu concentrations are basic rocks such as basalt and dolerite

while lower levels are found in the acid rocks such as granite. Molybdenum levels are highest in the acid volcanic, metamorphic and sedimentary material. Molybdenum is concentrated in shales, clays, phosphorites, petroleum and coal. Soil contents of Mo are often higher than in parent rock (Reid and Horvath 1980).

Soils often associated with the Cu-Mo problem are the peats, muck and poorly drained soils with high organic matter content (Reid and Horvath 1980). This may be due to the strong binding of Cu in organic complexes or to the low total Cu content of organic soils. A high organic matter content in soils is often due to poor drainage and a high water table which favours the uptake of Mo in plants.

Available Cu in soils is in the cupric form, in the cuprous form chelated to a clay-humic complex and as ions in true solution (Reid and Horvath 1980). The availability of Cu to plants decreases as the soil pH increases. Molybdenum in soils appears as MoO_4^{2-} . At higher soil pH, high soil organic matter levels, and calcareous soils, Mo availability is enhanced. Liming increases Mo uptake due to an increase in soil pH (Mitchell et al. 1957).

The amount of water in the soil may affect the process of Cu becoming complexed with organic soils which form Cu compounds. Waterlogging results in the development of an anaerobic environment developing around the roots with a corresponding drop in soil pH which increases the rate of Cu mobilization and absorption (Reid and Horvath 1980). However, this condition may also increase the availability of Mo, antagonizing Cu at the roots (Beeson 1978).

Blood Copper

There is little difference in the concentrations of Cu in whole blood, serum and plasma because Cu is fairly uniformly distributed between erythrocytes and plasma, except in pigs (Georgievskii 1982). The normal range of Cu in whole blood, serum or plasma in ruminants is 80-120 $\mu\text{g dL}^{-1}$. Levels consistently below 60 $\mu\text{g dL}^{-1}$ in cattle or sheep are indicative of Cu deficiency (Underwood 1977).

Plasma Cu is recognized to be more sensitive measure of Cu status in animals than whole blood Cu (Dreosti and Quicke 1968). The Cu content of erythrocytes was unaffected even in severely depleted animals. It appears plasma Cu levels in the normal animal represent a saturated level of Cu transport. Increased Cu intake does not usually lead to an increased plasma Cu level in the normal animal.

For this reason, Price and Chesters (1985) developed a more sensitive bioassay of Cu deficiency. The bioassay measures cytochrome C oxidase in the duodenal mucosa and resulted in a linear response to Cu doses from CuSO_4 . They were interested in the effect of Mo upon availability of Cu. The method may be useful in assessing relative availabilities of Cu from various Cu sources.

Significant breed differences in plasma Cu concentrations have been reported in sheep and cattle (Suttle 1985). Welsh mountain sheep have higher plasma Cu levels than Scottish Blackface sheep. Sheep of the Welsh genotype had a lower incidence of swayback, a condition caused by Cu deficiency, than did Blackface sheep. These differences were correlated with plasma Cu concentration (Wiener and Field 1969).

Rowlands et al. (1974) reported significant differences in blood Cu

levels in Hereford x Friesian calves from 12 different sires. They found a heritability value of 0.34 for blood Cu. Angus-sired cows had lower plasma and liver Cu values than Hereford-sired cows but Simmental-sired calves had lower values than both breeds (Smart and Christensen 1985). Gooneratne (1986) reported that Simmental cattle have a higher dietary Cu requirement than Angus. It appears Simmental cattle excrete more Cu in their urine and bile.

For calcium, magnesium, phosphorus and Cu, heritability estimates ranging from 0.2 to 0.4 suggest the concentration in the blood could be changed by selection. However estimates of heritability may be influenced by the plane of nutrition leading to conflicting heritability reports. However, it is generally agreed that there is evidence for genetic variation in blood Cu concentration suggesting the possibility that the amount of Cu in the diet which is appropriate or safe may differ among breeds (Wiener 1979).

The concentration of Cu in blood decreases rapidly in cattle and sheep on Cu deficient diets (McMurray 1980). Values for these Cu parameters may be below the accepted normal limits, while the animal appears clinically normal. The level of plasma Cu must be maintained below 30 ug dL^{-1} for 16 to 24 weeks before growth rate or food conversion ratio is affected by Cu supplementation (Suttle and McMurray 1983).

Ceruloplasmin is a single-chain glycoprotein from the alpha-2 globulin fraction of plasma, named as such because of its deep cerulean blue color (Frieden 1980). It binds 6 Cu atoms, 4 of which must be present in order for the oxidase enzyme (CpOx) to be active. CpOx has a molecular weight of 132,000 and normally binds 90 to 95% of the plasma

Cu.

CpOx synthesis occurs in the liver where Cu is incorporated into the apoceruloplasmin to form holoceruloplasmin. All species have low CpOx levels at birth which rise until levels characteristic of mature animals are reached at one year of age (Aspin and Sass-Kortsak 1981).

Reductions in CpOx and plasma Cu concentrations were observed in cows (McMurray 1980) before calving, with immediate increases following calving. The decrease before birth most likely reflected an increased requirement for Cu by the dam for fetal Cu deposition. The increase after birth may reflect an increase in the availability of Cu for CpOx synthesis (McMurray 1980). Plasma Cu concentration increases rapidly in neonatal calves during the first 96 hours after birth (McMurray et al. 1978). An increase in CpOx activity has been observed in other species during an estrogenic stimulus but this increase is absent in cattle (Underwood 1977).

Studies have demonstrated that CpOx can mobilize plasma Fe from the storage sites for Fe in the liver. CpOx functions to oxidize Fe from the ferrous (Fe^{+2}) form to ferric (Fe^{+3}) form which then becomes bound to transferrin. In this form, Fe is delivered to all the cells, particularly the reticulocytes involved in haem biosynthesis in bone marrow (Frieden 1980).

An extremely important function of CpOx is that of Cu transport (Frieden 1980). It is thought that the transport of Cu from CpOx to a cell requires a specific receptor mechanism in the target tissues as well as a reductive step because of the greater ability of Cu in the Cu^{+1} form. The mechanism proposed by Frieden (1980) for the incorporation of Cu from CpOx calls for an intracellular Cu enzyme or

protein. Cu^{+2} from CpOx is reduced by endogenous substrates under anaerobic conditions. If this occurs at the cell membrane Cu^{+1} is transferred into an intracellular acceptor. However, CpOx may penetrate the cell without the aid of an acceptor. In the Cu^{+1} form, Cu is added to the apoenzyme where it is fixed into the holoenzyme with the aid of oxygen in the Cu^{+2} state. This mechanism explains the primary role of CpOx as a Cu transport and donor molecule, the high exchangeability seen with Cu^{+1} ligands and the greater stability of the Cu ion in Cu^{+2} proteins.

It is possible that CpOx catalyzes the oxidation of the biogenic amines 5-hydroxytryptamine and noradrenaline (Barrass and Coutt 1972). If the cerebral levels of these neurotransmitters are regulated by CpOx, then this Cu protein could function in the regulation of brain chemistry that is needed for mental function. Abnormalities in CpOx levels may lead to a chemical imbalance reflected in abnormal mental states.

CpOx functions as a prominent serum anti-oxidant by preventing deleterious oxidation of polyenoic acids (Frieden 1980). It acts as a scavenger for superoxide anion radicals in a mode of action similar to the enzyme superoxide dismutase but in a much weaker manner. Finally, CpOx acts as an acute-phase reactant in a role which may be related to its antioxidant function. As an endogenous modulator of the inflammatory response, serum CpOx is increased two to three times in response to inflammation (Frieden 1980).

In all species, CpOx activity has been used diagnostically to identify a Cu deficiency (Frieden 1980). Elevated CpOx levels were seen in Cu deficient sheep and cattle after being fed a Cu supplemented diet (Lorentz and Gibb 1975); Kincaid et al. 1986a). Blakely and Hamilton

(1985) found a linear response in CpOx levels throughout a range of deficient and adequate Cu status levels in cattle and sheep suggesting that CpOx levels are a reliable indicator of Cu status in both deficient and adequate Cu status animals.

Comparisons of ovine and bovine serum CpOx relationship indicate a significant species difference exists (Blakley and Hamilton 1985). Ovine CpOx activity increased more rapidly compared to the corresponding bovine activity over a range of dietary Cu concentrations investigated (Blakley and Hamilton 1985). CpOx activity in bovine serum was reported to be significantly lower than that in bovine plasma; serum CpOx activity was 18 to 35% lower than plasma CpOx activity. McMurray (1980) suggested that CpOx is sequestered during blood clotting resulting in relatively lower serum CpOx activity.

CpOx activity in bovine plasma and serum were found to be more variable than plasma Cu or serum Cu. Correlation coefficients of CpOx to plasma or serum Cu levels were lower than those for plasma Cu relative to serum Cu concentrations. Coefficients reported were plasma Cu to serum Cu ($r = .99$); serum CpOx to serum Cu ($r = .48$) plasma CpOx to plasma Cu ($r = .43$) (Kincaid et al. 1986a).

Higher correlation coefficient values were reported by Blakley and Hamilton (1985). They reported: bovine serum CpOx to serum Cu as $r = 0.83$, bovine plasma CpOx to plasma Cu as $r = 0.60$ and ovine serum CpOx to serum Cu as $r = 0.92$. Coefficients as high as 0.93 have been observed using bovine plasma samples (Bingley and Anderson 1972). It is not surprising such good correlations are observed since CpOx is reported to contain 90% of the circulating Cu in normal animals (Frieden 1980).

Since CpOx synthesis occurs in liver, a major site of Cu storage, it would be expected that CpOx activity would be a useful indicator of hepatic Cu concentration. However, studies with cattle and sheep have shown that correlations are generally poor at levels of 0.50 (Blakley and Hamilton 1985; Sriastura and Dwarak 1972 as cited in Blakley and Hamilton 1985). It appears that CpOx activity is poorly related to hepatic Cu concentrations. The activity of CpOx is probably maintained at the expense of liver Cu stores and would not necessarily reflect these reserves until liver stores have been depleted. Under normal Cu conditions, most of the Cu in the liver is probably sequestered in a storage pool and is unlikely to be correlated closely with the functional copper pool that is associated with CpOx (Blakely and Hamilton 1985).

The Cu containing fraction of the erythrocytes is divided into two parts: the enzyme superoxide dismutase (SOD) and a free labile pool (Hassan 1980). Approximately 60% of the Cu in erythrocytes is associated with SOD which contains 0.34% Cu. This form of Cu in the blood was originally known as erythrocuprein. Superoxide dismutase is a metalloenzyme which catalytically scavenges the superoxide radical. It is essential for the aerobic survival of life forms. SOD has a molecular weight of 34,000 and contains two Cu and two Zn atoms (Hassan 1980). Replacement of the Cu by other metals results in a loss of activity. However, Zn has been replaced by Co, Hg and Cd without a loss of activity (Forman and Fridovich 1973). Therefore, it is known that Cu plays a catalytic role, whereas the Zn plays a structural role. SOD functions to provide protection against oxygen toxicity, compounds that cause exacerbation of oxygen toxicity, radiation damage and the damaging

effects of prolonged inflammation. Cu-Zn-SOD has been isolated in many organs including the liver and brain (Hassan 1980).

Cu nutrition affects the activity of SOD in the erythrocytes of mammals. The activity of SOD in erythrocytes decreases in Cu depleted sheep and cattle and increases in repleted sheep and cattle. In both these species, the rate of response to a changing Cu status, as measured by SOD activity, is delayed with respect to the changes in plasma Cu. It was suggested that erythrocyte SOD reflects a wider range in the severity of Cu deficiency. Plasma and liver Cu concentrations approach minimum values before clinical symptoms of Cu deficiency occur in steers. Therefore, they are better indicators of Cu status than erythrocyte SOD activity. SOD activity does not respond in the same way as plasma Cu to physiological effects such as growth, pregnancy and lactation; it is basically an independent measure. Unlike CpOx, erythrocyte SOD is not correlated to plasma Cu concentrations (Suttle and McMurray 1983).

Paynter and Allen (1982) reported that for growth of cattle to be affected by a simple Cu deficiency, plasma CpOx activity must be near zero and erythrocyte SOD should be decreased to 75% of normal activity.

SOD activity differs in animals of different ages. Erythrocyte SOD concentration in the neonatal calf is twice ($1.2 \text{ mg SOD g}^{-1} \text{ Hb}$) the activity found in the adult ($0.6 \text{ mg SOD g}^{-1} \text{ Hb}$) (Hassan 1980).

A smaller proportion of the Cu in erythrocytes forms labile compounds with albumin and beta-globulins, forming a dialysable fraction which is in dynamic equilibrium with erythrocyte SOD. This fraction of Cu is used by SOD, ensuring an adequate supply of Cu to maintain SOD activity (Georgievskii 1982).

A reduction in SOD activity may have clinical consequences for the animal (Chance et al. 1979). An alteration in the positive effect of SOD on the leucocytes or other cells against superoxides produced during phagocytosis would result in a loss of control over the inflammatory response. As a result of a reduced phagocytosing efficiency, the Cu deficient animal would be less resistant to infection.

Liver Copper

Once Cu is absorbed from the intestine, ionic Cu is transported in the blood to the liver as an albumin or amino acid complex. The liver separates the Cu from the complex and the Cu is joined with a low-molecular weight protein in the liver. From here, Cu enters a number of pathways in the liver. Some of the Cu is rapidly excreted in the bile. Some Cu is slowly converted to CpOx and returned to the blood. The Cu which reaches the liver in amounts too great to be converted to CP, is stored for future use (Owen 1980).

Development of Cu deficiency in animals consuming diets low in Cu is slow, indicating many species possess appreciable stores of Cu. The liver is the main Cu storage site in the body, although large amounts of Cu also accumulate in other tissues. The concentration of Cu in liver strongly depends on the Cu concentration in the diet (Georgievskii 1982).

Claypool et al. (1975) estimated that in order to maintain a normal plasma Cu level, the minimum liver Cu level is $40 \text{ mg kg}^{-1} \text{ DM}$ in cattle. Plasma Cu was a poor indicator of liver Cu status unless plasma Cu was at levels of 50 ug dL^{-1} or less. Blakley and Hamilton (1985) reported serum Cu and CpOx activity were not highly correlated with hepatic Cu concentrations in cattle and sheep.

The normal Cu concentration in cattle and sheep is 100 to $400 \text{ mg kg}^{-1} \text{ DM}$ (Underwood 1977). The content of Cu in the liver varies with the age of the animals. A calf is born with high liver Cu concentration (approximately $300 \text{ mg kg}^{-1} \text{ DM}$).

When large amounts of Cu are fed to pregnant animals, excess Cu

concentrations are found in the livers of the newborn animals (Georgievskii 1982). Liver Cu levels of dams fed a low Cu diet were not correlated with fetal liver concentrations. Accumulation of Cu in the liver starts very early in fetal development and reaches levels higher than that of its dam, up to 180 days of gestation. After this time, liver Cu in the fetus of a Cu deficient dam declines but the liver Cu in the fetus of a Cu adequate dam continues to increase (Smart and Christensen 1985).

Animals appear to differ in their capacity to bind Cu in liver tissue. The concentrations of Cu in the livers of animals depends as follows: sheep>cattle>rabbits>pigs>ducks>hens (Georgievskii 1982). In cattle and sheep, an increase in dietary and liver Cu does not appear to lead to a rise in blood Cu concentrations (Underwood 1977).

There are three specific Cu storage proteins in the liver. They are hepatocuprein (SOD), MT and mitochondriocuprein. When liver Cu concentrations are low, SOD can account for 20 to 50% of the total Cu in the liver. As liver Cu levels decrease, the relative contribution of SOD decreases. In ruminant liver, as little as 4% of the Cu may be associated with SOD (Bremner 1980).

MT is thought to play a major role in the storage of liver Cu. In Cu supplemented pigs and newborn calves, a Cu protein was purified which had an amino acid composition identical to MT, with a cysteine content of 30%. There were up to 10 moles of Cu and Zn bound to each mole of protein giving the equivalent of one metal atom for every two cysteine residues. The Cu to Zn ratio was found to vary from 4:1 to 1:1 (Bremner and Young 1976).

The function of MT in the control of hepatic Cu metabolism is still

unclear. MT rapidly incorporates injected Cu suggesting the protein is involved in hepatic Cu uptake (Bremner 1980). However, inhibition of MT synthesis by cycloheximide had no effect on accumulation of hepatic Cu; therefore, MT may not be essential for this process (Bremner and Davies 1976). These authors suggested the alternative hypothesis that the protein functions as a temporary storage or cellular detoxification of Cu.

Evidence for a role of MT as a temporary reservoir for Cu which is transported to other cellular proteins was reported by Seagrave et al. (1986). They reported that the rate of Cu uptake by high- and medium-molecular weight fractions in cultured cells decreased as the Cu in MT was depleted. No mechanism of transfer was determined in the study but they concluded that the process of Cu transfer from MT to other components is a property of the living cell.

The proportion of hepatic Cu bound to MT in cattle and sheep is less than that in pigs (Bremner 1980). It is thought that the greater susceptibility of cattle and sheep to chronic Cu poisoning could be related to their inability to accumulate large amounts of Cu as MT in their livers. Cattle and sheep accumulate large amounts of hepatic Cu but it is probably stored in a protein which does not temporarily store the Cu as MT appears to do.

The pattern of Cu distribution in liver tissue may favour excretion rather than accumulation of Cu in some species (Bremner 1980). This may explain why hepatic Cu concentrations in the pig rapidly fall when the animals are changed from a high Cu to low Cu ration, whereas excretion of ovine hepatic Cu occurs more slowly and is less sensitive to changes in hepatic Cu concentrations.

Three Cu and Zn binding proteins were identified in bovine livers (Mjor-Grimsrud and Norheim 1980). The differences in the amounts of Cu and Zn among soluble proteins in bovine liver are dependent mainly upon the total hepatic zinc concentration and to a small extent, on the total Cu concentration. At liver Zn concentrations below 40 mg Zn kg⁻¹ DM most Cu and Zn is bound to a high molecular weight protein (M.W. greater than 65,000). At higher Zn concentrations, significantly higher percentages of both Cu and Zn are bound to a low molecular weight protein (M.W. = 10,000), which is thought to be MT. These results are similar to those found in the calf (Bremner and Marshall 1974) and in the goat (Mjor-Grimsrud et al. 1979) but not with those in sheep (Norheim and Soli 1977) or swine (Froslie and Norheim 1977). In sheep, the amount of Cu and Zn present in the low molecular weight MT-like protein is dependent upon the hepatic Cu level. In cattle and goats, the binding of Cu and Zn to this protein is primarily dependent on hepatic zinc levels. In swine, the distribution of the two elements is not interdependent. These differences clearly indicate that sheep differ not only from swine but also from other ruminants.

Copper and Molybdenum in Milk

The concentration of Cu in ruminant milk varies with the species, stage of lactation and Cu status of the animal (Underwood 1977). Copper concentration in cow and ewe colostrum ranges from 0.20 to 0.30 mg L⁻¹ and 0.50 to 1.40 mg L⁻¹, respectively. Milk contains Cu at 0.10 to 0.20 mg L⁻¹ and 0.25 to 0.40 mg L⁻¹ for cows and ewes, respectively (Lonnerdal et al. 1981).

Copper content in bovine milk declined from 0.18 mg L⁻¹ to 0.06 mg L⁻¹ by day 300 of lactation in dairy cows (Vanderveen and Keener 1964). The concentration of Cu in ovine milk also declines from 0.20 to 0.60 mg L⁻¹ in early lactation to 0.16 to 0.40 mg L⁻¹ in later months (Beck 1941).

Adding Cu to the diet to increase Cu in milk has not occurred in a predictable manner. Underwood (1981) concluded from varying reports that supplementary Cu did not increase the Cu content in milk. However, Dunkley et al (1963) reported milk Cu values rose for a period of at least 4 weeks following a 300 mg subcutaneous injection of Cu glycinate; however, this did not increase the incidence of spontaneous oxidized flavour in the milk, a reaction which requires Cu as a catalyst. It was suggested that the additional Cu had a low catalytic activity because it was bound in a different form from than in normal milk. The levels of other trace elements were not influenced by the additional Cu. The provision of an injection of sodium sulphate had no effect on the levels of minerals in the milk.

Competitive interactions between Zn and Cu occur in the mammary gland (Mills 1980). Dietary Zn depletion and repletion leads to a

decrease and increase in Zn content in bovine milk accompanied by a reciprocal change in Cu content. It is not known whether Cd acts in the same manner as Zn in lactating females.

Milk is considered to be a major excretory route for Mo. The levels of Mo in milk are influenced by dietary Mo (Underwood 1981). Increasing Mo as ammonium molybdate from 5 to 50 mg Kg⁻¹ DM in the diets of Holstein cows resulted in an increase of Mo content in milk from 0.08 to 0.87 mg Mo L⁻¹, respectively (Vanderveen and Keener 1964); milk Mo was 0.03 mg L⁻¹ in non-supplemented cows. Dietary Cu content was 2 mg kg⁻¹ DM. Milk Cu levels were not affected. Adding 3.0 g kg⁻¹ sulphate to the diets did not affect the milk Mo content in the control animals. With diets containing 5 mg and 50 mg kg⁻¹ added Mo and with 3.0 g kg⁻¹ added S, there was a drop in milk Mo levels to 0.05 and 0.22 mg L⁻¹, respectively. The addition of Mo and S to the diets of cows resulted in a decrease of milk Cu concentration.

Huber et al. (1971) fed dairy cattle 53 or 173 mg Mo kg⁻¹ DM as sodium molybdate with a basal Cu level of 6.4 mg Cu kg⁻¹ DM. Contrary to Vanderveen and Keener (1964), Huber et al. (1971) found that the Cu content in milk increased from 0.10 mg Cu L⁻¹ for control cows to 1.08 to 1.27 mg L⁻¹ for Mo supplemented cows.

Wittenberg (1985) reported that supplemental levels of Mo of 20 mg kg⁻¹ DM and 40 mg kg⁻¹ DM fed to cows and ewes did not change the Cu content of milk. Molybdenum levels in milk did increase with each increase in dietary Mo. Control cows and ewes had Mo milk levels of 0.10 and 0.14 mg L⁻¹ respectively. When fed supplementary Mo at 20 mg kg⁻¹ DM, cows and ewes produced milk containing 0.51 and 1.53 mg Mo L⁻¹, respectively, while cows and ewes fed 40 mg Mo kg⁻¹ DM produced milk

containing 1.19 and 2.69 mg Mo L⁻¹, respectively.

Molybdenum supplementation did not influence plasma Cu concentration or distribution of Cu in calves or lambs (Wittenberg 1985). Liver Cu levels for calves suckling Mo supplemented dams, tended to decline relative to initial concentration. No difference in decline was noted in suckling lambs. Molybdenum supplementation increased plasma Mo concentrations for ewes and their lambs.

Mineral Interactions

Metabolic interactions between Cu and other trace elements such as Mo, S, Fe and Zn affect Cu absorption and utilization. There are two principle interactions that affect the outcome of dietary deficiencies or excesses of Cu (Mills 1980). First, many of the various stages associated with Cu absorption, transport and tissue uptake are susceptible to interference from the simultaneous presence of ions that either reduce the solubility of Cu within the intestinal lumen or modify its fate after absorption. These competitive interactions may either increase the effects of a low Cu diet or increase the tolerance of a dietary Cu excess (Mills 1980). The second type of interaction seen is when the metabolism of one element is influenced by an enzyme-catalyzed step for which a second element is essential. An example is the participation of Cu in iron metabolism through the action of CpOx (Mills 1980).

Copper-Molybdenum-Sulphur Interactions

Cattle are the least tolerant species to high dietary Mo followed by sheep while horses and pigs are the most tolerant of domestic livestock (Underwood 1981). When Mo intakes are low ($0.8 \text{ mg kg}^{-1} \text{ DM}$) a minimum requirement of $10 \text{ mg Cu kg}^{-1} \text{ DM}$ was needed to prevent Cu deficiency in calves (Mills et al. 1976). An increase in dietary Mo from 2 to $4 \text{ mg kg}^{-1} \text{ DM}$ can decrease the efficiency of Cu utilization (Suttle 1974).

It is clear that the Cu:Mo ratio is important in avoiding a Cu deficiency. An ideal ratio of 4:1 for cattle has been suggested by Alloway (1973), Steward and Racz (1977) and Fisher and Waldern (1978)

when Cu levels are at least 10 mg kg^{-1} DM. Excess dietary Mo is thought to have an inhibitory effect in the gut on the activity of sulphide oxidase, a Cu requiring enzyme (Mason et al. 1978). This results in the accumulation of insoluble cupric sulphide (CuS) in the tissue due to depressed sulphide oxidase activity.

The availability of Cu in forages is profoundly influenced by the concentration of S in plants (Mason 1978) and in water (Smart et al. 1986). It is not clear whether S (inorganic and organic) affects Cu metabolism on its own or whether it is dependent upon Mo (Gooneratne 1986). However, Suttle (1974) showed that increasing S sources from 1 to 4 mg kg^{-1} DM in ewe diets reduced Cu absorption by 40%. This is thought to be due to the increase in the fraction of insoluble CuS in the rumen and the gastrointestinal tract beyond the rumen. This reaction is dependent upon the ruminal pool of sulphide. Huisingh et al. (1975) reported that molybdate may modify this form of Cu deficiency as molybdate inhibits sulphate reduction in the rumen. However, if the major dietary source of S is S containing amino acids, molybdate increases the concentration of sulphide formed from these amino acids, thereby aggravating the Cu deficiency by promoting sulphide formation.

Sulphur toxicity has rarely been seen at levels below 0.25% S added to the diets of ruminants (Bull 1984). For beef cattle, dietary S should not exceed 4 g kg^{-1} DM (NAS-NRC 1984). Unlike dietary sulphate, S in water is 100% available to the rumen (Gooneratne 1986). Water containing S as sulphates can cause a secondary Cu deficiency in cattle. Sulphur at 600 mg L^{-1} or higher increases the S in the total ration from 1 g kg^{-1} to 4 g kg^{-1} (Smart et al. 1986). The effect on Cu and Zn status of sulphate removed from drinking water of beef cows and their

calves was studied by Smart et al. (1986). At calving the plasma and liver Cu concentration had decreased in both groups but the decline was greatest in the cows on the sulphated drinking water. After calving, Cu concentrations in liver and plasma increase, with the greater increase seen in the cows drinking the desulphated water. Water treatment had no effect on the Cu status of the nursing calves and on the Zn status of the cows.

In addition to the separate effects of Mo and S on Cu, a complex interaction between Cu, Mo and S is thought to play a role in reducing Cu retention although its exact mechanism is not fully understood (Gooneratne 1986). Dick et al. (1975) proposed a three-step series of events: reduction of sulphate to sulphide in the rumen; reaction of this sulphide with Mo to form thiomolybdate (TM); reaction of the thiomolybdate to form insoluble copper-thiomolybdate Cu-TM. This complex is thought to be absorbed and eventually excreted in the feces. Rather than just one TM being formed, a series of TM are formed. These are oxythiomolybdates $[\text{MoO}_{4-n}\text{Sn}]^{2-}$ where $n = 1$ to 3 or tetrathiomolybdate $[\text{MoS}_4]^{2-}$. Of the TM, $[\text{MoS}_4]^{2-}$ is the most potent and stable in the rumen (Gooneratne 1986). The formation of MoS_4^{2-} depends upon the S:Mo ratio in the rumen (Clarke and Laurie 1980). At normal S and Mo intakes such as 1 to 5 g kg^{-1} DM and 1 mg Mo kg^{-1} DM, the formation of dithiomolybdate or trithiomolybdate is favoured. Extensive formation of tetrathiomolybdate (TM4) occurs after a relatively long period of feeding forage with a high S:Mo ratio (Gooneratne 1986).

Suttle and Field (1968) reported the addition of molybdate and sulphate to the diets of ewes led to the appearance of a Cu fraction which was not released by treatment with trichloroacetic acid (TCA).

TCA-insoluble Cu was concluded to reflect a systemic consequence of the molybdate x sulphate interaction and TM must therefore be absorbable.

The administration of TM4 to sheep in the duodenum led to the appearance in plasma of TCA-insoluble Cu and low molecular weight protein thought to be albumin, containing Cu and Mo (Dick et al. 1975). These conditions are similar to those produced by the addition of molybdate and sulphate to the diets of sheep (Bremner and Young 1978). In these sheep, the cytosol preparations of kidney cortex contained TCA-insoluble Cu and Cu and Mo accumulated in the protein fraction. A similar response was seen by Suttle and Field (1969) when TM4 was added to the diets of lambs, in that there was tissue depletion of Cu except in the kidney, where both Cu and Mo accumulated. It appears TM induces plasma protein complexes which accumulate in the kidney in an unsuccessful excretory route.

Grace and Suttle (1978) found that when $^{99}\text{MoO}_4^{2-}$ was continually infused into the blood of ewes on diets supplemented with molybdate and sulphate, it did not exchange with the Mo being absorbed from the gut, thus proving that the systemic effects of dietary molybdate originate in the gut.

Suttle (1979a) reported that when TM4 was added to the diets at 2.7 or 5.4 mg Mo kg⁻¹ DM, the rate of hepatic Cu decline was not affected when dietary Cu was low, but when Cu was supplemented in the diet at higher levels, hepatic Cu accumulation was decreased by 30% suggesting that TM4 reduced absorption but had no effect on the endogenous loss of Cu. During the Cu depletion phase, TCA-insoluble Cu accumulated in the plasma but decreased during the repletion phase, suggesting the addition of Cu limited TM absorption and its ability to exert a systemic effect

on Cu metabolism.

Mo must pass through the rumen to interact with S and impair its own absorption and that of Cu as the absorption of ^{99}Mo in sheep given via the duodenum was not impaired by dietary S (Suttle 1980). The involvement of Cu in the three-way interaction occurs beyond the rumen. Hypocupremic ewes on diets low or high in molybdate and sulphate were given Cu orally as Cu sulphate or as Cu oxide needles, which lodge in the abomasum and slowly release Cu (Dewey 1977). The molybdate and sulphate supplements reduced the protection period from hypocupremia offered by each Cu source by 65%. With both Cu sources, there was a clear correlation between fecal Cu and Mo excretion. It appears that when Cu is released post ruminally, molybdate and sulphate simultaneously lower absorption of Cu and Mo by inducing the formation of unabsorbable Cu:Mo complexes.

Copper-Zinc Interaction

Zn is widely spread throughout the body with no specific storage site (Underwood 1981). Higher concentrations of Zn are found in the pancreas, liver, pituitary gland, kidney and adrenal gland. The normal liver Zn concentration in cattle is 25 to 50 mg kg⁻¹ (wet basis) (Puls 1981). In the liver, MT and SOD bind Zn acting as a Zn storage site (Miller 1979). During rapid growth, Zn is mobilized from the skeletal muscle to the liver (Lichti et al. 1970).

Zn is present in blood plasma, erythrocytes, leucocytes and platelets (Underwood 1981). Zn in erythrocytes occurs as carbonic anhydrase. In plasma, Zn is bound firmly to globulins for enzymatic function and weakly bound to albumin for transport. The normal plasma Zn concentration is 80 to 120 $\mu\text{g dL}^{-1}$ (Underwood 1981). Severe lesions

characteristic of Zn deficiency corresponded with plasma Zn levels of 40 to 60 $\mu\text{g dL}^{-1}$ and levels of 60 to 100 $\mu\text{g dL}^{-1}$ were related to slight Zn deficiency signs (Spais and Papasteriadis 1974).

Zn levels are five times higher in colostrum versus normal milk (Underwood 1977). The normal Zn concentration in milk is low at 2.30 to 5.00 mg L^{-1} and these values decline with length of lactation. With high levels of dietary Zn, milk Zn rises until a plateau is reached at 8.00 mg L^{-1} , while cows on a low Zn diet conserve Zn by reducing the amount going into the milk (Miller 1979). Beef cows with a high milk production level have higher Zn requirements because of the high Zn content in milk (NAS-NRC 1984).

In plasma, Zn turnover is more rapid than turnover in the liver. In contrast, exchange rates are slow in hair, muscle and red blood cells (Underwood 1981). At calving plasma Zn levels decrease while liver levels increase due to stress. The reverse occurs after calving (Smart et al. 1986).

Young cattle fed a high Zn diet show an increase in liver Zn while this is not seen in mature cows. It is believed that mature cattle can tolerate higher dietary Zn levels than younger ones since Zn absorption decreases with age and as growth rate decreases (Miller 1979). Dietary requirements for Zn are listed as 20 to 40 mg Zn kg^{-1} (NAS-NRC 1984).

The maximum safe level of dietary Zn is 500 mg kg^{-1} for growing cattle and 1000 mg kg^{-1} for mature ones (Miller 1979). This maximum amount of Zn which can be tolerated by ruminants without adverse effects is influenced by the level of dietary Cu.

It is thought that the gastrointestinal tract is the major site of the Cu and Zn antagonism. A single metal binding protein was identified

in the chick duodenum that bound Cu, Zn and Cd (Starcher 1969). Excess Cd or Zn induces MT synthesis in a wide variety of tissues (Mills 1980). As the tissue content of MT increased, the proportion of total cytosolic Cu bound to this fraction increased with either Cd or Zn because Cu is incorporated during the de novo synthesis of MT or because Cu displaces Zn or Cd that was previously bound. This leaves the Cu bound to MT and unavailable for utilization.

Rats on a high Zn diet exhibited poor utilization of Cu (Hall et al. 1979). After an oral Cu dose, the incorporation of Cu into MT increased six fold. Up to 60% of the total tissue deficit of Cu could be accounted for by Cu bound to intestinal mucosal MT. This resulted in a decrease in Cu flux to other body tissues. Lower levels of dietary Zn in the range of 150 to 450 mg Zn kg⁻¹ DM did not increase MT concentration in the intestinal mucosa and did not have an effect on Cu absorption (Hall et al. 1979).

Heifer calves were fed a diet containing 4 mg Cu kg⁻¹ DM and supplemented with 60 mg Zn kg⁻¹ DM for 130 days (Pringle et al. 1973). The Zn supplemented diet resulted in a decreased amount of liver Cu but did not affect the level of liver Zn. Pringle et al. (1973) showed that for the length of the trial, heifers with liver Cu levels above 100 mg Cu kg⁻¹ DM can maintain normal metabolic balance when the ration is less than 4 mg Cu kg⁻¹ and 33 mg Zn kg⁻¹.

Zn supplementation can prevent a Cu toxicity in sheep (Bremner et al. 1976). Diets fed to sheep supplemented with Zn at 220 mg Zn kg⁻¹ DM resulted in liver Cu levels being reduced by 40%. Liver damage as indicated by plasma aminotransferase levels, due to high Cu levels, was reduced when dietary Zn levels were increased.

Sheep fed diets high in Cd ($12 \text{ mg Cd kg}^{-1} \text{ DM}$) during pregnancy had lambs with a low liver Cu content at birth (Mills 1980). During pregnancy, the placenta blocks the transfer of Cd to the fetus but this is done at the expense of Cu transfer. This finding is consistent with the evidence that cadmium, acting as a Cu antagonist, is involved in the demyelinating disease, neonatal ataxia, in newborn lambs. With respect to high dietary Zn, the placenta fails to discriminate against Zn transport. A decrease in fetal tissue Cu is accompanied by a rise in Zn content (Mills 1980).

Copper-Iron Interaction

Fe absorption is greater in younger than mature animals and deficient as opposed to Fe sufficient animals. In erythrocytes, iron is associated with hemoglobin and ferritin, while in plasma, the iron is present in transferrin, a beta-globulin which transports iron (Underwood 1981).

The function of Cu in Fe utilization is carried out by CpOx. CpOx functions as an oxidase and links Fe and Cu metabolism by facilitating the release of Fe from the intestinal mucosal cells and parenchymal cells of the liver (Underwood 1981). High Zn levels which depress CpOx activity, interfere with Fe metabolism (El-Shobaki and Rommel 1979). The principle carrier of Fe in the blood is transferrin. Some plasma Fe is free which makes animals capable of fixing Fe in the plasma (Bremner 1966). Serum Fe levels indicative of Fe deficiency in cattle are less than 11 ug dL^{-1} (McDowell 1985). Ferritin is the form of Fe found in the liver (Underwood 1981). Normal liver Fe levels are 45 to $300 \text{ mg Fe kg}^{-1}$ on a wet basis (Puls 1981). Milk is relatively low in Fe, containing 0.3 to 0.5 mg L^{-1} while colostrum contains three times the

normal milk level (Underwood 1981).

Fe is transported from the intestinal mucosa in transferrin. In rats, El-Shobaki and Rommel (1979) suggested that Cu and Zn compete with Fe for binding sites on mucosal transferrin. When Fe and Cu were fed in excess of requirements, Fe absorption decreased because Cu had a greater affinity for transferrin.

Pig rations commonly contain high levels of Cu as a growth promotant. The addition of 500 mg Zn kg⁻¹ or 750 mg Fe kg⁻¹ DM to pig rations containing 750 mg Cu kg⁻¹ eliminated the toxic effects of Cu (Suttle and Mills 1975). The addition of 150 mg Zn and 150 mg Fe to pig rations containing 250 mg Cu kg⁻¹ eliminated toxic effects of Cu (El-Shobaki and Rommel 1979).

Campbell et al. (1974) examined the effects that moderately excessive intakes of Fe over an extended period may have on grazing cattle. Liver Cu reserves in grazing calves were depleted by daily doses of Fe as ferric hydroxide which was equivalent to a dietary concentration of 1.4 g kg⁻¹ DM.

The Fe dosing imposed considerable uniformity of liver Cu values between animals. Fe dosed animals with higher initial liver Cu values lost more liver Cu than animals with lower liver Cu values. Near the end of the trial, when Cu levels in the treated animals were low, there appeared to be a resistance to further depletion of liver Cu stores.

Standish et al. (1969, 1971) fed bull calves at 200 kg, a basal diet of 77 to 100 mg Fe kg⁻¹ DM with supplements of 400 to 1600 mg Fe kg⁻¹ as ferrous sulphate. With 1000 to 1600 mg Fe kg⁻¹, plasma and liver Cu and liver Zn were reduced while Fe in the liver, heart, kidney and spleen were increased. Calves on treatment had a reduced feed

intake and average daily gain.

The decrease in Cu status seen in these experiments may be due to the reduced feed intake or to the S intake from the Fe source. To eliminate these factors, Humphries et al. (1983) studied the effect that a lower concentration of Fe, when included in a diet with a suboptimal Cu control, had on Cu. As well the effect of Mo and Fe plus Mo (FeMo) was examined. Heifer calves were fed a diet containing 4 mg Cu kg⁻¹ with supplements of 0 or 800 mg Fe kg⁻¹ as ferrous carbonate and 0 or 5 mg Mo kg⁻¹ as ammonium molybdate. Liver and plasma Cu levels, erythrocyte SOD and plasma CpOx activities decreased in all calves given Fe, Mo or FeMo after 8 weeks. There was no significant difference among the treated groups. Plasma and liver Fe increased in calves supplemented only with Fe while there was no increase with Mo or FeMo diets. Calves on Mo and FeMo diets showed clinical signs of Cu deficiency after 20 weeks. Growth rates decreased, skeletal lesions developed and hair texture changed. It was clear that the addition of 800 mg Fe kg⁻¹ DM to the diet of calves influenced their Fe and Cu status.

Soil ingestion reduces dietary Cu availability for ruminants which may be due to an increase in Fe intake. When pasture is the sole source of feed for cattle, the average soil intake ranged from 4 to 8% of DM intake for cows. The amount of soil ingested by cattle is variable and not predictable (Healy 1968). Concentrations of Fe in soils in northwestern Manitoba range from 13.2 g kg⁻¹ to 48.8 g kg⁻¹ with a mean of 16.7 g kg⁻¹ (Boila, Wittenberg and Devlin, unpublished). A significant proportion of the Fe in soil can be liberated upon incubation of soil with rumen fluid (Healy 1972). At the higher soil Fe

levels of soil Fe, Cu status would be adversely affected by soil ingestion. These levels of Fe intake influence Cu metabolism (Humphries et al. 1983). Suttle et al. (1984) proposed that soil and dietary Fe inhibit Cu absorption by trapping sulphide, normally absorbed from the rumen, as FeS. The sulphide is released in the abomasum where an insoluble and unavailable Cu-S complex is formed.

Copper Toxicity in the Ruminant

Ingestion of high doses of Cu leads to an accumulation of Cu in tissues, particularly the liver. Animal species differ in their ability to store Cu in the liver; therefore, tolerances to high dietary Cu differ among species. Pigs are highly tolerant, cattle less so, with sheep being the most intolerant to dietary Cu excesses (Underwood 1981).

Copper poisoning may be acute or chronic. Acute Cu poisoning occurs when large Cu doses are ingested. Chronic Cu poisoning occurs under natural grazing conditions, and is due to excessive Cu levels in forages consumed by ruminants. These plants are growing on cupriferous soils and the Cu is taken up by the animal through ingestion of the plants. Some plants have been reported to have Cu levels as high as $50-60 \text{ mg kg}^{-1} \text{ DM}$ (Underwood 1981) although this is not normally seen.

Intake of herbage with normal Cu levels but low Mo levels by animals may lead to Cu toxicity. These low levels of Mo are found in forages growing on acidic soils and are dominant in pastures with the clover, Trifolium subterraneum. The Cu levels in this plant are usually 10 to 15 mg Cu kg^{-1} with low levels of Mo at 0.1 to $0.2 \text{ mg Mo kg}^{-1}$. Low levels of Mo in herbage allow the accumulation of Cu in the animal's liver (Underwood 1981).

Chronic Cu poisoning may also be associated with liver damage due to the consumption of the plant Heliotropium europaeum which contains hepatotoxic alkaloids, heliotrine and lasiocarpine. This disease known as toxæmic jaundice or "yellows", leads to Cu accumulation in body tissues (Underwood 1981).

High Cu levels are commonly fed to pigs and poultry. Subsequently,

wastes from these animals are used as fertilizers or as dietary supplements (Cooke 1983). Broiler litter containing $125 \text{ mg Cu kg}^{-1}$ when fed to ewes increased liver Cu levels to $1425 \text{ mg Cu kg}^{-1} \text{ DM}$. Removing litter from the ration did not reduce liver Cu levels (Olson et al. 1982). Broiler litter fed to helper calves for 201 days at $593 \text{ mg Cu kg}^{-1}$, did not produce signs of toxicity or impaired performance (Westing et al. 1985). Liver Cu levels rose in litter fed calves.

Pig slurry from pigs fed high dietary Cu levels is commonly used as a fertilizer (Cooke 1983). The Cu in this fertilizer contaminates the surface of the plant while very little Cu is actually taken up by the plant. This surface Cu is available to the grazing animal (Price and Suttle 1975). Dalgarno and Mills (1975) reported that if the slurry from pigs made up 2% of the dry matter intake for sheep for six months, the sheep can be termed as suffering from a Cu toxicity.

Chronic Cu poisoning occurs in two phases. During the first phase Cu accumulates in the tissues and liver over several weeks until liver Cu reaches $1000 \text{ mg Cu kg}^{-1} \text{ DM}$ or more. At this liver Cu level, Cu is released into the blood from the liver and blood Cu levels increase to 10 times the normal level. The second phase of poisoning begins with the haemolytic crisis including haemoglobinuria, haemoglobinaemia and icterus, and death follows (Underwood 1981). The haemolytic crisis is preceded for several weeks by a rise in serum aminotransferases. The determination of serum aspartic aminotransferase is a useful aid in the early detection of chronic Cu poisoning (Thompson and Todd 1974).

Chronic Cu poisoning promotes renal tubular necrosis, spongy lesions in the white matter of the brain, dark kidneys and swollen livers (Stogdale 1978). Acute Cu poisoning is characterized by nausea,

vomiting, salivation, convulsions, paralysis and collapse. Anorexia, depression and dehydration follows the initial attack (Mylrea and Byrne 1974).

Housed lambs and sheep on high concentrate diets have shown chronic Cu poisoning. When diets have been stored or processed in contact with Cu salts used in pig rations or when Cu but not Mo is included in the mineral-vitamin premix, rations may contain a high Cu content that may contribute to a Cu toxicity (Underwood 1981). Buck (1970) reported complete feeds with Cu levels of 25 mg Cu kg^{-1} , when fed to sheep may cause Cu poisoning. It was reported for cattle that the toxic dose of Cu is 3 to 12 times the dose needed to overcome a Cu deficient state (Reed 1975). The maximum tolerable Cu level for cattle is $115 \text{ mg Cu kg}^{-1}$. This level is greater for mature than young, growing animals (NAS-NRC 1984).

Treating Cu toxic animals which have reached the second phase of poisoning is rarely successful (Underwood 1981). Preventative steps can be taken to ensure the condition does not arise. Providing molybdate-containing salts to sheep to improve the Cu:Mo dietary ratio reduced Cu storage and eliminated mortality (Dick and Bull 1945). Intravenous administration of ammonium tetrathiomolybdate two times a week to sheep dosed with Cu sulphate prevented hemolytic crisis in sheep and prevented further hemolytic crisis in sheep already in hemolysis (Gooneratne et al. 1981).

Copper Deficiency in the Ruminant

Dietary Cu deficiency occurs naturally in grazing livestock in many parts of the world under varying soil and climatic conditions. Primary Cu deficiency occurs as a single deficiency of Cu arising from a low dietary intake of Cu. Secondary or conditioned Cu deficiency occurs naturally by the presence of dietary factors, such as Mo and S, which interfere with the absorption and utilization of Cu by the animal. Copper deficiency is commonly seen in grazing animals, particularly in beef cattle on pasture, grass hay and silage, and feedstuffs commonly low in Cu (Underwood 1977).

As available dietary Cu becomes insufficient for the metabolic processes requiring the element, these metabolic processes fail or are diminished. The susceptibility of different Cu-dependent processes varies with animal species (Underwood 1977).

Neonatal ataxia arises rapidly in lambs and kids from Cu-deficient ewes and goats. The bone changes manifested by Cu-deficient pigs and chicks are only seen to a lesser extent in lambs, calves and foals.

In cattle, Cu deficiency is seen mostly in calves with the occurrence of diarrhea, anemia, bone abnormalities, loss of coat color and unthriftiness. "Falling disease", a condition of fibrosis of the myocardium, is seen in cattle (Underwood 1981).

A Cu deficiency is of economic importance because losses which include growth retardation, increased susceptibility to health problems, and infertility may not be readily definable in a herd of cattle.

Growth Response

Variable responses to Cu have been obtained with comparisons of the growth rates of Cu treated and untreated cattle under field conditions.

Suttle and Angus (1976) reported that after weaning, Cu depleted calves grew more slowly than Cu supplemented calves. They found the growth curves began to diverge at 12 to 16 weeks. The food conversion ratio increased in the Cu deficient animal. Plasma Cu was maintained at levels below 30 ug dL^{-1} for variable lengths of time before growth rate or food conversion rate was affected. Poole et al. (1974) found no effect on growth in Cu depleted cattle over one year of age.

Villus atrophy (Fell et al. 1975) and lesions (Mills et al. 1976) in the small intestine were seen in Cu deficient cattle. These abnormalities along the digestive tract may lead to impaired absorption of dietary constituents with a resultant reduced growth rate (McMurray 1980).

Anemia

Anemia is commonly seen in Cu deficient states in all species when the deficiency is severe or prolonged. In these circumstances, normal haematopoiesis cannot be sustained when the normal blood Cu in mammals decreases from 80 to 120 ug dL^{-1} to 10 to 20 ug dL^{-1} (Underwood 1981).

In ruminants, the characteristic anemia encountered is indistinguishable from an iron deficiency. This is due to the inability of Cu deficient animals to release iron from cells into the plasma at a normal rate (Underwood 1981).

Copper is essential in adult red cells (McMurray 1980). A minimum amount of Cu is needed for their protection and maintenance in circulation. It does not appear that Cu is essential for the haem biosynthetic pathway but it is needed for the absorption of Fe from the intestine, tissue mobilization of Fe and its utilization in haemoglobin synthesis. Ceruloplasmin is involved in these functions since it is

essential for the formation of transferrin, the transport vehicle for Fe. The Fe transferrin is transferred to the reticulocyte in the bone marrow (McMurray 1980).

The survival time of erythrocytes is shorter than normal in Cu deficient animals. This may be due to the lower activity of the SOD enzyme seen in Cu deficient animals (Suttle and McMurray 1983). The SOD enzyme at a reduced activity would result in an increase in the toxic effects of superoxide anions, leading to a destruction of cell membranes.

Bone Disorders

Copper is essential for the activity of the Cu containing enzyme lysyl oxidase which is needed in the biosynthesis of cross linkages in collagen (Suttle and Angus 1976). Collagen accounts for 95% of the protein in the bone matrix. An impairment in collagen synthesis leads to defective bone formation (Underwood 1981).

The clinical symptoms associated with joint and bone disorders in Cu deficient animals vary. They include stiffness of gait, swelling of joints, lameness and an increase in the incidence of bone fractures (Underwood 1977).

Suttle and Angus (1976) attributed the bone lesions seen in Cu deficient animals to a reduced activity of osteoblasts which produces thin bone and an overgrowth of cartilage in cattle. Alternatively, Mills et al. (1976) found some evidence of osteoporosis, where the tissue surrounding the metacarpophalangeal and carpometacarpal joints was swollen. The economic significance of skeletal lesions in cattle is likely to depend upon the period of time during which the animals are exposed to a Cu deficiency. In meat producing animals, the production

cycle is relatively short; therefore, the effects of a Cu deficiency would be insignificant. In animals living to older ages such as breeding stock, the effect could be significant (Underwood 1981).

Neonatal Ataxia

The classical Cu deficiency syndrome in sheep is neonatal ataxia, also known as swayback and lamkruis. This condition is the result of motor dysfunction caused by a defective synthesis of myelin. Two types of ataxia occur in the lamb: (1) the common form where a congenital neonatal form shows macroscopic cerebrospinal lesions; and (2) secondary form in which the clinical signs occur one to twelve weeks after birth. The syndrome is associated with uncoordinated movements of the hind limbs, a stiff and staggering gait and swaying of the hindquarters. Some lambs are paralyzed (ataxic) when born and soon die. Calves are rarely affected by this condition (Underwood 1981). The disease is associated with low levels of Cu in the fetal brain and liver (Underwood 1977) and can be prevented by Cu supplementation of the ewe lamb during late pregnancy (Allcroft et al. 1959).

The two forms are associated with the distinct pre- and postnatal phases of myelin degeneration. Myelin aplasia is caused by the degeneration of the motor neurons of the brain and spinal cord (Howell et al. 1964). The lesions are irreversible and may start at 6 weeks before birth and continue until parturition. Levels of cytochrome oxidase, the Cu containing terminal respiratory enzyme of the mitochondrial electron transfer chain, are low in the brain of ataxic lambs (Howell and Davidson 1959) and are at their lowest in the neurons of the grey matter of the spinal cord, which show the morphological lesions of the disease (Fell et al. 1965). Copper deficiency in rats

leads to a depressed cytochrome activity which impairs phospholipid synthesis. It is thought that since myelin is composed largely of phospholipids, Cu deficiency could lead to myelin aplasia (Gallagher and Reeve 1971).

O'Dell et al. (1976) reported that the catecholamine neurotransmitters, dopamine and norepinephrine, as well as plasma amine oxidase, are significantly lower in the brain stem of ataxic lambs compared to Cu treated controls. The locomotor disorders seen in the ataxic condition may be due to the impaired biosynthesis of these catecholamines. A depression in amine oxidase, a Cu containing enzyme, is seen in Cu deficient animals resulting in thicker vascular walls. This could lead to a reduced oxygen supply to the nerve cells, thereby interfering with myelination (Underwood 1981).

Diarrhea

Diarrhea has been observed in Cu deficient cattle (Mills et al. 1976; Suttle and Angus 1976). Diarrhea in young Fresian cattle was considered to be due to changes in the cellular structure of the small intestine, mainly villus atrophy. Additionally, loss of cytochrome C oxidase in intestinal cells occurred along with a destruction of the enterocytic mitochondria (Fell et al. 1975).

Profuse diarrhea is a clinical sign of Cu deficiency in cattle grazing pasture high in Mo. Horses and sheep grazing pastures with similar mineral content do not scour. However, scouring has been seen in goats grazing pastures that induce scouring in cattle.

Fertility

Copper deficiency resulting in poor fertility has been documented (Howell and Hall 1970; Peterson and Waldern 1977) while other studies

have shown no relationship (Phillippo et al. 1982).

Underwood (1981) reported that low fertility was evident as delayed or depressed estrus in cattle grazing pastures low in Cu. Infertility associated with aborted, small, dead fetuses has been cited with experimental Cu deficiency in ewes (Howell and Hall 1970). In British Columbia, Peterson and Waldern (1977) reported that in dairy herds fed silage, there was a positive association between the Cu:Mo ratio of the feed and fertility in the herd. Thomas and Moss (1951) noted that high levels of Mo have adverse effects on spermatogenesis in young bulls.

Phillippo et al. (1982) treated low Cu status cows in a beef suckler herd with 100 mg injectable Cu before mating. There was no improvement in conception rate in the low status animals compared with control animals, even when the serum Cu of the low status animals was below 30 ug dL^{-1} . The lack of effect may be due to the dose which only elevated the Cu status for a short time. Smart and Christensen (1985) found there was no difference in the bovine fetal Cu liver level during the first trimester of pregnancy when adequate and deficient dams were compared.

Hunter (1977) suggested Cu may have a pharmacological effect on ovulation and fertilization in cattle. This was due to the effect of the Cu dependent enzyme, dopamine beta hydroxylase, which is involved in the synthesis of hypothalamic transmitter substances.

Wool and Hair

One of the first changes seen in Cu deficient animals is achromotrichia, which is loss of hair color due to defective formation of melanin (McMurray 1980). Greying of hair around the eyes in cattle and lack of pigment production in black-woolled sheep are characteristic

of the condition. The conversion of tyrosine to melanin in hair or wool follicles is dependent upon the Cu containing enzyme, polyphenyl oxidase. Therefore, a diet low in Cu results in failure of the pigment for black being incorporated into the follicle resulting in a paler color of hair or wool (Underwood 1981).

As well as a color change, growth of hair and a change in physical appearance was noted as a thin, harsh coat. As the reserves of Cu are depleted and blood Cu falls in sheep, the crimp in the wool is less distinct until the fibres are almost straight, hair-like fibres known as "stringy" or "steely" wool. The elastic properties of this wool is abnormal along with a reduced tensile strength (Underwood 1981). This condition has been seen in sheep with an experimentally produced primary Cu deficiency (Suttle et al. 1970) and with sheep grazing pastures low in Cu (Whitelaw et al. 1979).

The physical properties of hair are dependent upon the presence of disulphide groups which provide the cross-linkages or bonding and also upon the alignment or orientation of the long chain fibrillae in the fibre (Underwood 1981). In steely wool, there is a lack of formation of the disulphide bridges and an increase in free sulphydryl groups (Marston 1952). The precise biochemical involvement of Cu in the lack of formation of the disulphide bridges is unknown.

Cardiovascular Disorders

Cardiac disorders due to Cu deficiency were first reported as a disease of cattle known as "falling disease" (Underwood 1981; Hesketh 1982). The disease is characterized by a slow and progressive degeneration of the myocardium with replacement fibrosis followed by sudden death due to acute heart failure usually after mild exercise or

excitement. Cytochrome oxidase is required by the heart to maintain its oxidative activity and energy metabolism. This enzyme is low in the Cu deficient animal; therefore, a deficiency of this enzyme appears to lead to the occurrence of characteristic cardiac disorders (Fell et al. 1975).

Cardiac lesions due to failure of elastin and collagen cross-linkage have been observed in Cu deficient cattle (Mills et al. 1976; Leith 1975). These lesions may account for the "falling disease" condition. Cross-linkages between polypeptide chains in collagen and elastin is dependent upon the Cu containing enzyme, lysyl oxidase. A Cu deficient animal shows reduced activity of this enzyme thus preventing normal formation of collagen and elastin (McMurray 1980). This is the same enzyme that is associated with the cartilage-bone relationship and the consequent bone disorders noted in cattle.

Copper Supplementation

In order to correct Cu deficiency in animals, supplementation of Cu is necessary. Methods of Cu supplementation include Cu containing fertilizers, mineral salts, parenteral injections and oral dosing.

The application of Cu containing fertilizers to pasture has been examined as an effective way of raising forage Cu levels and forage yields for grazing animals (Underwood 1981). These fertilizers include commercial preparations or a Cu-containing pig slurry. The beneficial effects that Cu fertilizers have on soil and forage uptake depends upon the Cu status of soils and upon soil antagonists.

Although it has been suggested that the ingestion by sheep of herbage heavily contaminated with slurry from pigs receiving added dietary Cu can cause toxicity problems (Dalgarno and Mills 1975), proper slurry management and animal husbandry can overcome this problem. The addition of $443 \text{ kg Cu ha}^{-1}$ to soil was found to increase the Cu in plants from $4.6 \text{ mg kg}^{-1} \text{ DM}$ to $6.8 \text{ mg kg}^{-1} \text{ DM}$ (Davies 1980 as cited by Cooke 1983). Because sheep store Cu in liver more readily than other animals, they are more prone to Cu toxicity levels than other species. However, with proper management, sheep can graze on pasture fertilized with Cu-containing pig slurry. It is unlikely that the use of Cu-containing pig slurry would solve Cu deficient problems in cattle as cattle do not store Cu in the liver as readily as sheep (Cooke 1983).

A single dressing of copper sulphate at $5 \text{ to } 7 \text{ kg ha}^{-1}$ or the equivalent amount of an inexpensive Cu ore, was found to significantly increase soil Cu levels for 3 to 4 years, except on calcareous soils (Underwood 1981). MacPherson et al. (1975) suggested the addition of

1.1 kg $\text{CuSO}_4 \text{ ha}^{-1}$ in dry conditions would raise the Cu concentration of hay to levels high enough to prevent a Cu deficiency in wintering cattle. The application of 5.6 to 11.2 kg $\text{CuSO}_4 \text{ ha}^{-1}$ in solution to pasture or hayland foliage, increased the Cu content of forage to 100 and 208 mg Cu kg^{-1} , respectively. At this level of forage Cu toxicity problems in ruminants would develop. Rainfall following the treatment reduced the Cu concentration to 24 and 41 mg Cu kg^{-1} , respectively.

Cu deficiency in grazing animals can be prevented by providing Cu containing salt licks. Mineral mixtures or salt licks containing 2.5 to 5.0% of CuSO_4 fed to cattle provided adequate Cu if enough of the mineral is consumed (Smart et al. 1980). However cattle may not eat enough mineral to provide enough dietary Cu to meet their requirements. Inclusion of CuSO_4 in the mineral supplement supplied enough Cu for cows, but calves did not appear to consume enough mineral (Boila et al. 1984b).

Copper sulphate added to the drinking water of grazing cattle at 2 to 3 mg Cu L^{-1} of water provided enough dietary Cu (MacPherson 1982). Water consumption varies with season, and intake of supplemental Cu could also vary. Differences in precipitation and evaporation result in a varying level of Cu consumed, thereby making this method of supplementation inadequate.

In intensive animal production, Cu supplements may be added in the concentrate. Chemical forms of available Cu that can be added to the diet include the sulphate, oxide, chelates and proteinate salts of Cu. Maro and Kategile (1980) fed dairy cows CuSO_4 at 10 mg Cu kg^{-1} . This level of CuSO_4 increased plasma Cu to normal levels. The bioavailability of Cu from Cu proteinate was found to be greater than

CuSO_4 in calves (Kincaid et al. 1986b). Plasma and liver Cu concentrations were significantly greater in calves supplemented with Cu proteinate compared to control or CuSO_4 supplemented calves.

A parenteral route of Cu administration is probably the most rapid method for restoring normal concentrations of trace elements in the body (Allen and Mallinson 1984). Parenteral supplements are effective against many secondary deficiencies, a situation where dietary factors convert Cu into an unavailable form. Absorption from the alimentary canal is by-passed. Another advantage is that a known amount of Cu is given to the animal. To be effective for grazing animals, the supplement must provide a source of Cu which increases body stores over a long period of time. This is accomplished by giving a large amount of Cu in a single dose; however, the amount given must not be toxic. Because a large amount of the biologically active form may be toxic, Cu is constrained. Over time the active supplement is released at a controlled rate (Allen and Mallinson 1984). Jamieson and Allcroft (1950) demonstrated the effectiveness of 50 mg Cu administered intravenously as CuSO_4 in maintaining normal Cu status in calves. Given orally, 500 mg CuSO_4 was needed to obtain equivalent results.

Although large intravenous doses can be tolerated, Harvey and Sutherland (1953 as cited in Allen and Mallinson 1984) pointed out that intravenous injections are impractical for a large number of animals. They examined a number of Cu supplements for suitability for subcutaneous or intramuscular injections in adult sheep. These supplements included sulphate, acetate, citrate, oxychloride, borogluconate, naphthenate, oleate, asparagenate, caseinate, ammonium sulphate, oxine, versenate (sodium ethylene diamine tetra-acetic acid

(EDTA)) and glycinate salts of Cu. They concluded that Cu glycinate given subcutaneously was the most satisfactory. A dose of 50 mg Cu as Cu glycinate which is about half of the toxic dose, caused no significant Cu toxic lesions and appeared to be stored in the liver.

Cunningham (1959) examined the effects of diethylamine oxyquinoline sulphonate as a Cu source for sheep. A dose of 30 mg Cu caused little or no reaction at the site of injection and increased liver Cu stores. However, this form of Cu had no advantage over the Cu glycinate in cattle. Whitelaw et al. (1979) found improved weight gains and plasma Cu in sheep injected with CuCaEDTA. Deaths have been reported in sheep (Sutherland et al. 1955; Wiener and Macleod 1970) and in calves (Mylrea and Byrne 1979) given these injectable compounds.

Ishmal et al. (1977) examined Cu methionate injections for the use in parenteral Cu therapy and concluded Cu methionate was safer than CuCaEDTA. Since a Cu toxicity problem was associated with CuCaEDTA in sheep, the commercial preparation of this Cu source is no longer available (Allen and Mallinson 1984).

Copper methionate was the safest Cu compound when administered at 30 mg Cu to sheep and 50 mg Cu to calves, but was the least effective in alleviating hypocupraemia in calves and sheep (Suttle 1981b). The most effective compound in sheep, Cu diethylamine oxyquinoline sulphonate, was the most toxic (Suttle 1981b). Therefore, the recommended dose for this compound is only 12.5 mg Cu for sheep. Copper CaEDTA was the most effective treatment in cattle (Suttle 1981b).

Bolla et al. (1984a) examined Cu methionate, CuCaEDTA and two forms of Cu glycinate as injectable Cu preparations for grazing cattle. Treatments were injected subcutaneously. All four treatments increased

serum Cu levels but did not influence weight gain. Copper methionate produced the most severe reaction at the injection site, followed by the two forms of Cu glycinate, with CuCaEDTA providing the least severe reaction at the injection site.

Poole (1980, 1981, 1982) compared the effects of injectable CuCaEDTA, Cu oxide and Cu glycinate in treating yearling bulls grazing high Mo pastures. Consistently over the three years. Cu oxide performed poorly in raising liver and plasma Cu levels compared to the other two Cu supplements.

Parenteral Cu administrations are commonly used in treating Cu deficient ruminants; however, some disadvantages are evident with this type of Cu supplementation. One of the dangers associated with injectable Cu is the difficulty in controlling the rate at which the preparation releases the active Cu. There is always a risk of acute general toxicity due to rapid release from the injection site (Wiener and Macleod 1970; Mylrea and Byrne 1974). Alternatively, the Cu preparation may be released so slowly that large residues persist at the injection site, damaging the carcass and costing the producer. Finally, the beneficial effects of Cu supplementation by injection may be so short lived that repeated injections are necessary if the deficiency persists (MacPherson et al. 1979; Gleed et al. 1983).

In sheep, parenteral Cu treatment provides protection for a maximum of two to three months because of the small dose of Cu administered. Copper methionate is inefficient in repleting body stores, and diethylamineoxyquinoline sulphate only provides an initial small dose at 12 mg. However, these Cu supplements are commonly used. In cattle, a larger dose per unit body weight can be safely administered up to 1 mg

kg^{-1} as either the EDTA complex or Cu methionate. This dose is usually not effective any longer than 4 months and repeated injections must be given if a Cu deficiency persists (Allen and Mallinson 1984).

Glass boluses or bullets containing Cu and other elements, have been examined. Allen et al. (1984) used Cu containing glass made from a mixture of sodium oxide, Cu oxide, phosphorous pentoxide and zinc oxide cast into molds approximately 50 mm x 25 mm for cattle and 30 mm x 15 mm for sheep. The resulting boluses weighed 75 g and 17 g respectively, had a density of 2.8 and 3.0 g mm^{-3} respectively and contained 18% Cu by weight. Steers grazing pasture containing an average 6.5 mg Cu kg^{-1} DM were treated with either 200 mg CuCaEDTA (twice the recommended dose), dosed with a Cu-containing glass bolus or left untreated as a control. Blood Cu concentration rose more rapidly in the injected group versus the bolus treated group but declined to a level just below that of the bolus treated group by 140 days. Liver Cu values were higher at 184 days in the injected group. The bolus provided the steers with 11 mg Cu per day, protecting them from hypocupremia and increasing their liver stores of Cu during the trial. A similar degree of protection was provided by the CuCaEDTA.

Lambs treated with a glass bolus (Allen et al. 1984) had significantly higher liver Cu levels than controls but no difference was observed in plasma Cu between the two groups. The glass dissolved at a rate of 17 mg glass per day giving a mean of 3.1 mg Cu per day. After slaughter, boluses were found and removed from the rumen or reticulum from six of the eleven treated steers and eighteen of the nineteen treated lambs. It was proposed that the non-recovered boluses may have been lost whole or may have disintegrated in the reticulorumen and then

been lost. In the event of the disintegration of the bolus and the rapid release of a large proportion of its Cu content, the animal is at risk to a Cu toxicity (Allen et al. 1984).

Tengrove and Judson (1985) reported two deaths in sheep which received a Cu glass bullet. The two glass bullets recovered from the sheep had lost 75% in 34 weeks and 85% at 39 weeks of their initial weight. Bullets used in the study were intended to last one year. The dissolution rate of the bullet may have been accelerated through contact with a cobalt bullet which was also administered. There was also a problem with fragments of the glass bullet or even whole bullet being regurgitated.

Different formulations of glass with a wide range of solubilities could be made which release their constituents at different rates. The daily requirements of sheep or cattle under different situations have been defined and it would therefore be possible to make a bolus of Cu-containing controlled release glass suitable for any condition (Allen and Samson 1985). The bolus offers the extra advantage that additional elements may be added such as selenium or cobalt. However, if an animal deficient in selenium is treated with a bullet containing selenium as well as Cu, a potential Cu toxicity problem may be present if the animal is adequate or above average in Cu status. Certainly further testing is required before safe and maximal effectiveness can be gained from the glass bolus treatment.

Lassiter and Bell (1960) reported only 2 to 20% of the dose of copper oxide needles (CuON) orally administered to sheep was recovered in the feces after four days. It was suggested that the needles had been retained in the alimentary tract and might provide a slow release

of absorbable Cu. This was confirmed by Dewey (1977) who reported a single dose of CuON in sheep caused a steady increase in liver Cu concentrations over a 16 day period to values of $1000 \text{ mg kg}^{-1} \text{ DM}$ above initial levels. The needles were described as fine rods of Cu oxide wire, fragmented to lengths of 10 mm and less.

The effectiveness of a Cu supplement may vary depending on whether the Cu is released within the reticulorumen where TM is active or elsewhere where conditions for Cu absorption may be more favourable. Dewey (1977) recovered considerable amounts of the CuON from the abomasum. CuON are believed to enter the rumen transiently before passing to the abomasum where some particles are retained in the folds. The slow release of Cu in the acid environment of the abomasum enabled the use of CuON as an effective oral treatment of Cu deficiency.

CuO needles are often referred to as wire, particles or granules. Cupric oxide is more commonly used as opposed to cuprous oxide, because of the cost of manufacturing. Cupric oxide is formed at 300 degrees whereas cuprous oxide is formed at about 1000 degrees. This is an oxidation method.

Whitelaw et al. (1983) treated grazing ewes and their lambs with CuON or injected Cu. Ewes were injected with 90 mg Cu as Cu methionate, orally dosed with 4 g CuON or left as a control. Their lambs were treated with repeated injections of CuCaEDTA, CuON or left as a control. The administration of Cu as Cu methionate or CuON to ewes at parturition increased ewe plasma Cu in a situation where ewes become hypocupremia very rapidly. The injection prevented hypocupremia for only 10 weeks while the CuON maintained plasma Cu levels at normal concentrations for more than 17 weeks. Copper treated ewes had increased Cu levels in the

milk in the initial week of lactation; however, this effect was transient. This was reflected in the control lambs from treated ewes which had higher plasma Cu values than control lambs from control ewes. These high plasma Cu levels decreased after two weeks. It appears that treatment of ewes with CuON is more effective in preventing the onset of hypocupremia than Cu methionate.

The largest increase in plasma Cu concentration was seen in lambs treated with 1.6 g CuON (Whitelaw et al. 1983). The needles maintained normal Cu levels until weaning, and resulted in higher liver Cu levels and higher live weight gains than lambs given four injections of CuCaEDTA.

Tengrove and Judson (1985) examined five different Cu supplements in grazing Merino sheep. Sheep receiving 2.5 g CuON, a Cu glass bullet or 50 mg Cu as CuCaEDTA in a subcutaneous injection, had significantly greater liver Cu concentrations than those of control sheep for up to 51 weeks; liver Cu levels for treated sheep were 205, 205 and 238 mg Cu kg⁻¹ DM, respectively with untreated sheep having liver Cu of 165 mg kg⁻¹. Oral doses of 30 mg Cu as Cu oxychloride or 300 mg Cu as CuSO₄ did not alter the concentration of Cu in liver. The relative effectiveness of the successful treatments is difficult to compare because they provided different amounts of Cu, through different routes of supplementation.

In a series of experiments aimed at evaluating CuON as a supplement Langlands et al. (1983) orally dosed grazing sheep with varying amounts of CuON ranging from 0 to 64 g. The toxicity level, rate of particle excretion in feces and Cu storage in the liver and other tissues was recorded. The breeds of sheep used were white and black Merinos.

In the first experiment the toxic level (lethal dose 50 (LD_{50})) of the CuON were 1.17 and 1.80 g CuON g kg^{-1} BW for black and white sheep respectively. Death usually occurred 88 to 96 days after dosing. Hepatic Cu concentrations of sheep dying from Cu toxicity were similar for blacks and whites (4122 and 4308 mg Cu kg^{-1} DM, respectively). The doses for black and white sheep where 5% of the population died from Cu toxicity (LD_5) were .50 and .77 g CuON kg^{-1} BW, respectively. The LD_5 dose of .77 for white sheep was approximately seven times the dose which the authors found gave a significant growth response in Merino lambs. Weaned lambs dosed with 2 g CuON (.13 g CuON kg^{-1} BW) grew significantly faster (+11 g per day) than unsupplemented lambs.

In the second experiment (Langlands et al. 1983) the rate of fecal Cu excretion in Cu supplemented sheep was less when 50 g CuON were given than when the dose was 5 or 10 g CuON; fecal excretion patterns were variable with the group which received the 50 g dose showing the greatest variability. Of the two sheep which survived this high Cu dose, sixty percent of the dose was excreted within 10 days of dosing. Those sheep which did not survive retained a greater proportion of the dose during the 30 days post dosing.

In experiment three (Langlands et al. 1983) the quantity of hepatic Cu stored per gram of CuON given, declined as the dose increased from 0 to 32 g, but increased again at higher doses. Hepatic Cu concentration reached a maximum 2 to 3 months after dosing. There was a rapid fall in liver Cu content after peak values were reached, the rate of decline being positively related to the dose of Cu. Between days 141 and 224, hepatic Cu concentrations declined by 0.8, 2.0 and 10.4 mg kg^{-1} DM per day in sheep dosed with 0.5, 16 and 64 g CuON, respectively. It is

therefore unlikely large doses will extend the elevated Cu status in sheep. In fact, large doses led to Cu poisoning indicated by elevated plasma aspartate transaminase activity.

Langlands et al. (1983) also examined the concentration of Cu in various tissues, since most sheep are ultimately consumed by humans. Copper content of the lungs and kidney increased with large doses (64 g) of CuON. The liver contained the greatest concentration of Cu. When dosing with 4 g CuON, 1.4 to 1.8% of the dose was retained in the liver after 109 days but when more than 32 g CuON were administered, less than 1% was retained. It is thought that the absorption mechanisms are saturated when large doses of Cu oxide particles are administered and the rate at which hepatic Cu reserves are depleted increases when hepatic Cu concentrations are elevated. The report concludes that CuON, when given at appropriate rates, do not endanger the sheep or excessively increase tissue Cu concentrations or depress productivity. At correct doses for lambs, growth rates could be increased.

Suttle (1981a) orally treated ewes with either 0.5 g Cu as CuON or as CuSO₄. One group on each Cu source was fed the basal diet (0.7 mg Mo kg⁻¹ DM, 1.2 g S kg⁻¹ DM) or a diet supplemented with Mo and S (3.7 mg Mo kg⁻¹ DM, 4.2 g S kg⁻¹ DM). Fecal Cu concentrations were monitored to assess the rate of passage of dosed Cu through the alimentary tract. The administration of CuON to hypocupremic Scottish blackface ewes resulted in a steady increase in plasma Cu concentration over 30 days whereas with CuSO₄, the initial response was more rapid. The advantage provided by CuSO₄ was short lived. On Mo-S supplemented diets, CuON provided protection for 111 days compared with 57 days for ewes treated with CuSO₄. When the Mo-S was omitted, the difference widened; the CuON

benefit lasted for 301 days while that of CuSO_4 lasted for 188 days. Copper absorption was calculated to be higher for CuON than for CuSO_4 and for the unsupplemented versus supplemented diet. The amount of Cu absorbed from the Cu sources were estimated by assuming that the daily deficit between the net maintenance requirement of the ewe and dietary provision of absorbable Cu had been met throughout the period of protection. The Cu absorbed from each source was the product of deficit $\times$ period of protection.

Fecal Cu excretion patterns differed between the CuON and CuSO_4 treated groups, but were not influenced by diet (Suttle 1981a). Fecal Cu values from CuSO_4 diets reached a maximum on the second day after dosing and returned to predose levels between the ninth and fifteenth days. With Cu as CuON , maximum values were not reached until six days and remained high until the thirteenth day after dosing. Values were twice the predosing level indicating the CuON passed slowly through the alimentary tract. Fecal Mo concentration followed a pattern similar to fecal Cu with both Cu sources, showing a rapid but short lived increase in ewes given CuSO_4 . Ewes given CuON showed an elevated Mo level which was sustained for 30 days.

The addition of Mo and S ($9.5 \text{ mg Mo kg}^{-1} \text{ DM}$, $3.9 \text{ g S kg}^{-1} \text{ DM}$) to diets fed to Merino cross wethers did not significantly effect the response to supplementation of 2.5 g CuON (Langlands et al. 1986a). These results conflict with those of Suttle (1981a). Copper deficiency is frequently associated with elevated concentrations of dietary Mo and S, which form TM in the rumen and complex with dietary Cu rendering it unavailable (Dick et al. 1975). In the study by Langlands et al. (1986a) CuON did not appear to be sensitive to such a reaction. They

proposed that little Cu is released in the rumen and the availability of Cu in the CuON when lodged in the abomasum is unaffected by dietary Mo and S. This is not consistent with the proposal by Suttle (1981a) that the mechanism initiated in the rumen can cause Cu and Mo to form insoluble complexes at sites further down the alimentary tract. The difference between the two sets of observations may be due to the difference in dosage rates, different techniques and breeds of animals used.

Langlands et al. (1986a) tried to establish if Cu is released during the period of residence in the rumen or whether Cu is rendered ineffective by an extended period in the rumen. CuON supplying 2.32 g Cu was administered to Merino wethers either orally, suspended in the rumen or surgically implanted to the wall of the abomasum. CuON increased hepatic Cu concentration when surgically implanted or administered orally but not when suspended in the rumen. Responses were greatest when the CuON were inserted directly into the abomasum. The CuON suspended in the rumen for six weeks were effective in raising hepatic Cu levels once they were taken from the rumen and released in the abomasum. Rate of passage of CuON is likely to cause differences in the response in liver Cu levels. Many factors influence the rate of rumen clearance of CuON; however, CuON retention in the rumen does not impair their ultimate effectiveness.

Differences in the effect of degree of oxidation were determined in sheep by Langlands et al. (1986a). The response in increasing hepatic Cu concentration was greatest with laboratory grade CuO (containing 86% Cu and 13.7% oxygen content) than oxidized Cu particles (containing 92.6% Cu and 7.7% oxygen content); elemental Cu was ineffective.

Manipulating the degree of particle oxidation during the manufacturing process provides one way of controlling the quantity of active material administered. This method may be preferable in administering the size of dose given since a minimum number of particles must be given if some of the dose is to be retained in the abomasum. Results indicate that the response in hepatic Cu concentration is proportional to both the quantity of particle retained in the abomasum and their duration in that organ.

Suttle and Valente (1981) investigated the retention of CuON in pigs receiving a concentrate diet with no supplemental Cu. Male pigs weighing 30 to 40 kg were given 1.6 g Cu as CuO in needle or powder form in a single oral dose after feeding. Fecal Cu levels in animals treated with CuO powder peaked two days after dosing and remained elevated for three days. Fecal Cu levels in CuON treated pigs peaked at day four and stayed elevated for 18 days. CuON retention was less pronounced than that found in sheep (Suttle 1981a). Species differences in the anatomy of the alimentary tract may influence the effectiveness of CuON.

The administration of 50 g CuON to mildly and severely hypocupremic cattle restored plasma Cu levels to normal values within 10 days (Suttle 1981a). There was a partial and slower recovery in erythrocyte SOD activity. Fecal Cu levels remained elevated for 40 days after dosing indicating CuON had been retained in the alimentary tract. After 41 days, cattle were left with a liver reserve ($428 \text{ mg Cu kg}^{-1} \text{ DM}$) equivalent to 4 conventional injection doses from a single dose of CuON.

Suttle and Valente (1981) dosed Jersey cows with $0.33 \text{ g CuON kg}^{-1} \text{ BW}$. Distribution of CuON in the alimentary tract was assessed 7 and 15

days after treatment by mixing the contents of the particular organ with water, decanting, sieving the heavy residue and hand picking the needles. Needles were predominantly found in the reticulum (29 to 53%) and a few were present in the abomasum (3 to 18%).

Three months after steers were dosed with 50 g CuON, the needles were found to be retained in all stomach compartments. The amount of needles remaining in the abomasum, reticulum, rumen and omasum after 3 months were 1.87, 1.24, 0.79 and 0.12 g, respectively and totalled 4.02 g or 8% of the dose. After 1 and 2 months a total of 20.2 g (40% of the dose) and 13.6 g (27% of the dose), respectively were recovered. At 1 month 38% of the retained needles were found in the abomasum while at 2 months, 41% were in the reticulum. Much of the CuON had lost the oxide coat, exposing the wire core; therefore the needles were not 100% oxidized. This was more noticeable in the abomasum (Dewland et al. 1986).

At higher doses of CuON, the specific region of retention and amount of dose retained was more variable. Heifers dosed with CuON at rates of 100, 200 or 300 g retained 45 to 68%, 6 to 68% and 46 to 95% of the dose, respectively. The abomasum appeared to be the main storage area followed by the reticulum (Dewland et al. 1986).

Plasma Cu levels were higher in cows dosed with 50 g CuON than control animals 30 weeks after treatment (Judson et al. 1982). Cows treated with 120 mg Cu glycinate had higher plasma levels than controls. In a second study (Judson et al. 1982) 25 g CuON in cows was a sufficient dose to sustain elevated plasma Cu levels similar to those in cows injected with Cu for periods up to 33 weeks post treatment.

MacPherson (1983a, b) showed single doses of 4, 10 and 20 g CuON

were effective in raising blood Cu levels in deficient calves, heifers and cows, respectively to normal and maintaining these levels for 4 to 6 months.

Doses of 0, 20 or 60 g CuON were administered to suckler cows to see if larger doses provided a longer protective period (MacPherson 1984). The initial plasma Cu level in all groups was approximately $31.8 \mu\text{g dL}^{-1}$. After 8 months, all three treated groups showed highly significant responses in plasma Cu with 40 g and 60 g groups having higher mean plasma Cu levels than the 20 g group. Over the last 4 months, the 60 g group was higher than the 40 g group, but plasma Cu levels in all four groups began to decline. At the end of the trial, all three groups had higher mean plasma Cu levels than the previous year, indicating some residual effects. By extrapolation, there appeared to be only a two month advantage conferred by a 3-fold increase in dosage rate from the 20 g to 60 g treatment. This type of response is thought to be due to the observation that the period of protection afforded by initially high liver Cu reserves is limited by an accelerated rate of loss from hepatic Cu stores (MacPherson 1984).

In an attempt to determine the appropriate CuON dose in Hereford cows and 6 month old heifers, Langlands et al. (1986b) administered one of six treatments to groups of cattle. The treatments were control, 5 g, 10 g, 15 g, 40 g CuON or 150 mg Cu injected as CuCaEDTA. The response in liver Cu was defined as the difference between the observed hepatic Cu concentration and the concentration prior to treatment adjusted for the increase in hepatic Cu concentration of the control group of the appropriate age. Liver Cu concentrations were higher initially for heifers, which is thought to be due to their smaller liver

size. As a result, 20 to 80% of heifers had liver Cu levels exceeding $320 \text{ mg Cu kg}^{-1} \text{ DM}$ at 50 and 100 days, respectively when given 5 to 15 g CuON. Both cows and heifers exceeded $320 \text{ mg Cu kg}^{-1} \text{ DM}$ when dosed with 40 g CuON.

The response to supplemental CuON varied inversely with body weight (Langlands et al. 1986b); therefore doses of CuON were expressed relative to live weights at dosing and related to the response in liver Cu concentration at 50 to 100 days from dosing. The average increase in hepatic Cu concentration in cattle at 50 to 100 days from dosing was predicted to be $70 \text{ to } 84 \text{ mg Cu kg}^{-1} \text{ DM}$, respectively for an animal given $0.045 \text{ g CuON kg}^{-1} \text{ BW}$. In Canada, approximately 90% of cattle have liver Cu values of less than $214 \text{ mg Cu kg}^{-1} \text{ DM}$, while the remaining 10% have liver Cu values ranging from $215 \text{ to } 764 \text{ mg Cu kg}^{-1} \text{ DM}$ (Korsrud et al. 1985). A dose of $0.045 \text{ g CuON kg}^{-1}$ or 20 g for an adult cow weighing 450 kg would result in sufficient stores of Cu in liver. Problems arise when animals with adequate Cu reserves are treated as a precautionary measure in the incorrect belief that the animals are deficient.

Similar calculations were made by Langlands et al. (1986b) for sheep which predicted an average increase in liver Cu to $0.045 \text{ g CuON kg}^{-1} \text{ BW}$ of 179, 177 and $106 \text{ mg Cu kg}^{-1} \text{ DM}$ at 49, 101 and 150 days from dosing, respectively.

Cows given large doses (300 g CuON) did not show any signs of Cu toxicity despite high ($1016 \text{ mg Cu kg}^{-1} \text{ DM}$) concentrations of Cu in liver. There was no evidence of higher concentrations of Cu in muscle, but kidney concentrations were higher in cows treated with 300 CuON than cows dosed with 50 g CuON (32.4 mg kg^{-1} versus 24.2 mg kg^{-1}) (Dewland et al. 1986).

Grazing cows and calves dosed with 20 g and 4 g CuON, respectively had significantly higher liver Cu values than control animals at the end of 85 days (Boila 1987a). Serum Cu, serum Zn, liver Fe and body weight responses were not affected by Cu treatment.

Calves on pasture received 25 g CuON or 60 mg injected Cu glycinate (Judson et al. 1982). Pasture Cu levels over the season decreased from 7.6 to 4.5 mg kg⁻¹ DM while Mo levels increased from 5.7 to 7.0 mg kg⁻¹ DM. After 35 weeks, calves treated with CuON had higher plasma Cu levels (71.1 ug dL⁻¹) than injected calves (45.1 ug dL⁻¹). Trichloroacetic acid (TCA)-soluble Cu and total plasma Cu were similar across the control, injected and CuON treated groups suggesting that little blood Cu was tied up as a Cu-Mo complex, a complex which is insoluble in 10% (w/v) TCA. Fecal Cu concentrations were raised in CuON treated calves for up to 12 weeks showing that particles are retained in the alimentary tract after dosing for several weeks. Dietary zinc absorption was not reduced in CuON treated animals.

Dewland et al. (1979) examined CuO granules for use as a long acting Cu supplement in grazing calves without the effects of carcass damage associated with injections. Shorthorn x Hereford calves at 3 months and 200 kg were treated with a 120 mg Cu glycinate injection or 50 g of CuO granules at a dose rate of 0.2 g kg⁻¹ LW. The granules were administered by a tube inserted into the esophagus. Plasma Cu rose in both groups by 40 days to 75.3 ug dL⁻¹ and then began to decline. By day 81, the oxide treated group had significantly higher plasma Cu levels than the injected group (57.2 vs 28.6 ug Cu dL⁻¹ plasma). There was no difference in calf weights.

Suttle (1979b) dosed hypocupremic calves with 0.2 g CuON kg⁻¹ LW.

The needles alleviated all clinical symptoms of Cu deficiency and increased Cu concentration in liver, plasma and hair. After 41 days, fecal Cu levels were elevated, indicating the CuON were well retained in the alimentary tract and still releasing absorbable Cu. Liver Cu rose to 342 mg kg^{-1} Cu but this represented only 1% of the dose.

Grazing calves were orally dosed with 0.1 g CuON or injected with 100 mg CuCaEDTA. At the end of the 133 day grazing period, calves treated with CuON had significantly higher plasma Cu levels than injected or control calves. Plasma Cu concentrations for oxide treated, injected and control calves at the end of the trial were 90, 66 and 57 ug dL^{-1} , respectively. The CuON calves had significantly higher LW gains than the control group and higher LW gains than the injected group. It appears that a dose rate of 0.1 g kg^{-1} LW to calves provides, at the end of the grazing period, an adequate but not excessive concentration of Cu in liver (Richards et al. 1985).

Whitelaw et al. (1984) dosed Aberdeen Angus cross calves with $0.1 \text{ g CuON kg}^{-1}$ LW. At the end of the 73 day grazing trial, CuON treated calves had a higher mean plasma Cu level (79.5 ug dL^{-1}) than the control group (14.8 ug dL^{-1}) and a higher mean daily LW gain (0.86 kg day^{-1}) as compared to the control (0.68 kg day^{-1}). The dose of $0.1 \text{ g CuON kg}^{-1}$ LW prevented growth retardation and hypocupremia in treated calves.

Potential exists for the use of CuON in the treatment and prevention of Cu deficiency but present difficulties must be overcome. With an element such as Cu which is cumulative and poisonous, the risk of chronic Cu poisoning is positively related to its effectiveness in alleviating Cu deficiency. CuON require a new approach to the estimation of dose rate and frequency, especially for sheep which are

more prone to Cu toxicity than cattle.

The optimum dose rate affecting the absorbability of Cu will vary from farm to farm. Increasing dietary S and Mo to the upper limits found in herbage more than halved the period of protection given by CuON (Suttle 1981a). The optimum dose will also vary from breed to breed, influenced by genetic factors which affect the efficiency of Cu absorption (Wiener 1979). Most importantly, the present Cu status of the animal will determine the toxic risk or value of CuON in alleviating deficiency. Langlands et al. (1986a) suggested the incorporation of CuON into a matrix which slowly disintegrates within the rumen releasing CuON over several months, may provide a way of extending the period of protection against Cu deficiency without risking Cu toxicity.

MATERIALS AND METHODS

Animals and Management

Thirty-seven purebred Hereford cow-calf pairs were used in a trial of 92 days in length. At the start of the trial, cows ranged in age from two to twelve years and calves ranged in age from 59 to 122 days. Before being moved onto pasture, cows had been fed a mixed grain/barley straw ration. Suckling calves were on an oat and barley creep feed, and barley straw. Cows were allocated into one of the three treatment groups and calves in one of two calf treatment groups based on sex of calf, age, weight and plasma Cu obtained on May 16 (Table 2). Cupric oxide needle (CuON) treatments for cows were 0, 25 and 50 g while CuON treatments for calves within cow groups were 0 and 20 g. Group 3B has only 1 heifer calf as there was a need for a cow-calf substitution on day -1.

The CuON contained 79.88 g Cu per 100 g CuON (Fisher Scientific) and were approximately 5 mm in length and 0.5 mm in width. Calves receiving CuON were dosed with 20 g CuON (16.0 g Cu) administered in 5 gelatin capsules (size 000, Parke-Davis), each capsule containing 4 g CuON. Cows receiving Cu were dosed with 25 g CuON (20.0 g Cu) or 50 g CuON (39.9 g Cu) administered in a Whatman cellulose thimble packed with a piece of filter paper. The doses were orally administered using a balling gun on day 0 (June 4) at the home farm. All animals received a subcutaneous injection of Covexine-8, an anticlostridial vaccine (Wellcome Veterinary Division), calves received an intramuscular injection of Dystosel, a selenium source (Rogar/STD), on day 0. All injections were given according to manufacturer's recommendations.

Table 2. Allocation of cows and calves to treatment groups

Cows

Dose, g CuON	<u>0</u>	<u>25</u>	<u>50</u>
Number	12	12	13

Calves (within cow groups)

Dose, g CuON	<u>0</u>	<u>20</u>	<u>0</u>	<u>20</u>	<u>0</u>	<u>20</u>
Number	6	6	6	6	7	6
<u>Total bull calves</u>	3	4	3	4	4	5
1st calf heifers	0	1	1	1	1	1
2nd calf heifers	1	0	0	0	0	1
Mature cows	2	3	2	3	3	3
<u>Total heifer calves</u>	3	2	3	2	3	1
1st calf heifers	0	0	0	0	0	0
2nd calf heifers	1	1	1	1	1	0
Mature cows	2	1	2	1	2	1

On day 1 (June 5) animals were transported to the 80 acre pasture which was approximately 5 miles north of the home farm. While on pasture, cattle were provided with a 1:1 mixture of loose cobalt-iodized salt and a 1:1 mineral (zero Cu, Co-op 1:1 Sheep Mineral; Appendix Table D1). The water sources on pasture consisted of a reservoir and runoff from a spring, neither of which provided Cu. The only source of supplemental Cu available to the animals was CuON.

Sampling Procedure

Cows and calves were weighed and blood samples and liver tissue were obtained from all animals in a combination squeeze chute-scale. Cattle were weighed on days -1, 0, 28, 63, 91 and 92. Blood samples were taken on days 0, 28, 63 and 92. Liver samples were taken on days 0, 28 and 92. Cow #65 from group 3 and her calf 21U from group 3A, have missing weight, plasma and tissue liver values on day 63 as they were removed from pasture for 5 days due to pinkeye infection.

Blood samples for plasma Cu, Mo, Fe and Zn were taken from the jugular vein, collected into heparinized vacutainer tubes and kept cooled overnight. Blood plasma was separated from whole blood within 24 hours of collection by centrifugation for 20 minutes at 2000 rpm. Three ml of plasma was transferred into a glass digestion vial for wet ash digestion. The remaining plasma was stored frozen in a capped plastic vial for CpOx analysis.

Liver samples were obtained by a biopsy procedure described by Smart and Northcote (1985) and stored frozen in capped, plastic vials for analysis of Cu, Mo, Fe and Zn contents.

Forage samples consisted mainly of grasses (mainly a mixture of brome, timothy and some kentucky) with a small amount of legume (red

clover). The pasture consisted mainly of grasses. Legume was consumed by cattle only in very minor amounts but was analyzed in order to examine the pasture potential if a higher yield of legume was grown. Forage samples for grasses were obtained on days 0, 28, 63 and 92 by a clipping. Legume samples were obtained only on days 28 and 63 due to insufficient amount of sample on days 0 and 92. Forage samples were dried in a 60 C oven and then ground using a Wiley hammer mill with a 1 mm stainless steel sieve. Samples were analyzed for dry matter (DM), crude protein (CP), acid detergent fibre (ADF), Cu, Mo, Fe, Zn, Ca, P, Mg, Mn and total S. Water samples were obtained from the banks of the pasture reservoir and spring on days 0, 28, 63 and 92. Water samples were analyzed for Cu, Mo, Fe, Zn, Ca, P, Mg, Mn and total S content.

Chemical Analysis

Liver and blood plasma were wet ashed using the method of Thompson and Blanchflower (1971) with some modifications. These modifications included: 1) liver size was approximately 0.5 g, while blood plasma was 3.0 ml and 2) there was an overnight predigestion of the samples in the 4:1 acid mixture.

A 0.5 g sample of liver tissue was placed in a clean, tarred, weighed glass vial and freeze dried overnight. To each of the dried liver samples, 3 ml plasma samples and blanks, a 5 ml volume of concentrated nitric-perchloric acid mixture (4:1 vol/vol), and a glass bead were added. The vials were placed in a slotted aluminum heating block and left overnight at room temperature. The following day the vials were heated and a clear colourless solution developed. The wet ashing procedure was completed when the clear colourless solution was boiled to dryness. After cooling, 10 ml of 5% HCl was added to each

vial to dissolve the residue and left to stand overnight. The solutions were aspirated directly or dilutions were made for Cu, Mo, Fe and Zn analyses using an Instrumentation Laboratory Model 551 Atomic Absorption Spectrophotometer (AOAC 1984; and Perkin Elmer 1973). The concentrations of Cu, Mo, Fe and Zn were reported as mg kg^{-1} DM for liver tissue and ug dL^{-1} for blood plasma.

CpOx activity in plasma was determined using the procedure of Robinson et al. (1987). Plasma CpOx activity was expressed as $\text{A min}^{-1} \text{L}^{-1}$ of plasma.

Forage samples

The dry ashing procedure for Cu, Mo, Fe, Zn, Mn, Mg and Ca analyses in forage samples was that of AOAC (1984) and Perkin Elmer (1973). Approximately 1.0 g of ground sample was added to a 50 ml Kimax borosilicate glass screw-top vial. Samples were ashed for 12 h at 550 C and slowly cooled to room temperature. Added to the ash residue was 10 ml 5 N HCl. Capped vials were placed in an ultrasonic 65 C water bath for 1 h, removed and left standing overnight. The Cu, Mo, Fe, Zn, Mn, Mg and Ca contents were determined using the Atomic Absorption Spectrophotometer mentioned above. Phosphorous was determined colorimetrically (AOAC 1984) using a Bausch and Lomb Spectronic 20 colormeter. The total S was determined by the method of Boila et al. (1984d). Dry matter of ground samples, crude protein (macrokjeldahl nitrogen $\times 6.25$) and ADF were analyzed using the procedure of AOAC (1984).

Water samples

Water samples were analyzed for Cu, Mo, Fe, Zn, Mn, Ca and Mg contents, directly or after dilution, by Atomic Absorption

Spectrophotometry while P and total S content were determined according to procedures mentioned above.

Statistical analysis

All data were subjected to a split-plot analysis of variance with repeated measures over time (Gill and Hafs 1971). The data were analyzed using the Statistical Analysis System (SAS), General Linear Models (GLM) procedure for unbalanced data (SAS 1985). To test the effects of experimental treatments for cows the error term used (Error A) was cow (cow treatment). For calves, Error A was calf (calf treatment x cow treatment). To test the effect of sampling day and the treatments by sampling day interaction, the error term used (Error B) for cows was cow (sampling day x cow treatment). For calves, Error B was calf (sampling day x calf treatment x cow treatment). Analysis of variance tables for body weight, plasma and liver parameters are reported in Appendix Tables A1, A2 and A3 for cows and Appendix Tables B1, B2 and B3 for calves.

Mean comparisons were undertaken with the Newman-Keuls procedure (Snedecor and Cochran 1980). Linear contrast analysis was performed when there was a significant ($P < 0.05$) treatment x sampling day interaction (Snedecor and Cochran 1980). Correlations among liver Cu, plasma Cu, plasma CpOx and plasma Mo were determined (Snedecor and Cochran 1980).

Sex effect was not significant ($P > 0.05$) for all calf parameters and therefore was removed to make the experimental design more uniform with respect to number of calves per cow treatment x calf treatment combinations.

RESULTS

Cows

Body weight

Mean body weight of all cows at the start of the trial was 616 ± 2.3 kg. Treatment with CuON had no effect ($P > 0.05$) on body weight (Table 3). Sub plot analysis showed a significant ($P < 0.01$) effect of sampling day on body weight (Table 4, Figure 1). Body weight of cows on day 92 had increased ($P < 0.05$) to 627 kg.

Liver Cu

Mean liver Cu concentration of all cows on day 0 was 159 ± 7.8 mg kg^{-1} DM (Table 4). Treatment had no effect ($P > 0.05$) on liver Cu (Table 3). Sub plot analysis revealed a sampling day effect ($P < 0.01$) on liver Cu (Table 4); liver Cu had increased in all cows over the trial. The analysis of variance for liver Cu (Appendix Table A3) showed a significant ($P < 0.01$) cow treatment x sampling day effect.

Based on a linear contrast analysis (Appendix Table A3) mean liver Cu on day 28 for 25 Cu cows was 78 mg kg^{-1} DM higher ($P < 0.01$) compared to untreated cows (Figure 2A). On day 92 the difference between the two groups was 85 mg kg^{-1} DM ($P < 0.01$). Liver Cu levels of 50 Cu cows were higher than 25 Cu cows on days 0, 28 and 92 but the difference was not significant ($P > 0.05$). On day 92, mean liver Cu concentrations were 207, 265 and 314 mg kg^{-1} for 0 Cu, 25 Cu and 50 Cu cows, respectively (Figure 2A, Appendix Table C1).

Plasma Cu

Mean plasma Cu concentration of all cows for all treatments on day 0 was $104 \pm 3.0 \text{ ug dL}^{-1}$ (Table 4). Cow treatment had no effect ($P > 0.05$) on plasma Cu (Table 3). Sub plot analysis showed a significant ($P < 0.01$)

Table 3. Main plot least square means for cow liver and plasma parameters

Parameter	Cow treatment (g CuON)			MES _A [†]
	0	25	50	
Number	12	12	13	
Body weight ¹	617	607	624	20900.1
Copper				
Liver Cu ²	182	210	254	23565.2
Plasma Cu ³	104	106	96.5	827.0
Plasma CpOx ⁴	23.0	21.1	22.6	107.1
Molybdenum				
Liver Mo	8.1	7.8	7.1	11.6
Plasma Mo	34	33	34	418.4
Iron				
Liver Fe	371	377	357	4033.1
Plasma Fe	211	196	177	9217.0
Zinc				
Liver Zn	175	146	148	9543.7
Plasma Zn	122	123	123	1992.2

[†]MSE_A - Mean square error (Error A) from the main plot.

¹Body weight (kg).

²Liver Cu, Mo, Fe, Zn mg kg⁻¹ DM.

³Plasma Cu, Mo, Fe, Zn µg dL⁻¹.

⁴Plasma CpOx ΔA min⁻¹ L⁻¹.

Table 4. Least square means of cow liver and plasma parameters over sampling days

	Days				MSE _B [†]
	0	28	63 [‡]	92	
Number	37	36	37	37	
Body weight ¹	616B	609B	610B	627A	192.4
Copper					
Liver Cu ²	159B	226A	-	262A	2225.3
Plasma Cu ³	106B	118A	95.2C	92.0C	321.8
Plasma CpOx ⁴	15.5C	20.1B	26.9A	26.4A	19.3
Molybdenum					
Liver Mo	5.8B	11A	-	5.5B	318.6
Plasma Mo	35B	47A	33B	22C	196.6
Iron					
Liver Fe	356	380	-	370	249367.9
Plasma Fe	255A	209B	139D	178C	2702.6
Zinc					
Liver Zn	190A	140B	-	139B	6663.8
Plasma Zn	124B	146A	147A	75C	1365.2

[†]MSE_B - Mean squares error (Error B) from the sub plot.

[‡]No liver biopsies were taken on day 63.

A,B,C,D, Means in the same row with a different letter are significantly different (P<0.05) using NK test.

¹Body weight (kg).

²Liver Cu, Mo, Fe, Zn mg kg⁻¹ DM.

³Plasma Cu, Mo, Fe, Zn µg dL⁻¹.

⁴Plasma CpOx ΔA min⁻¹ L⁻¹.

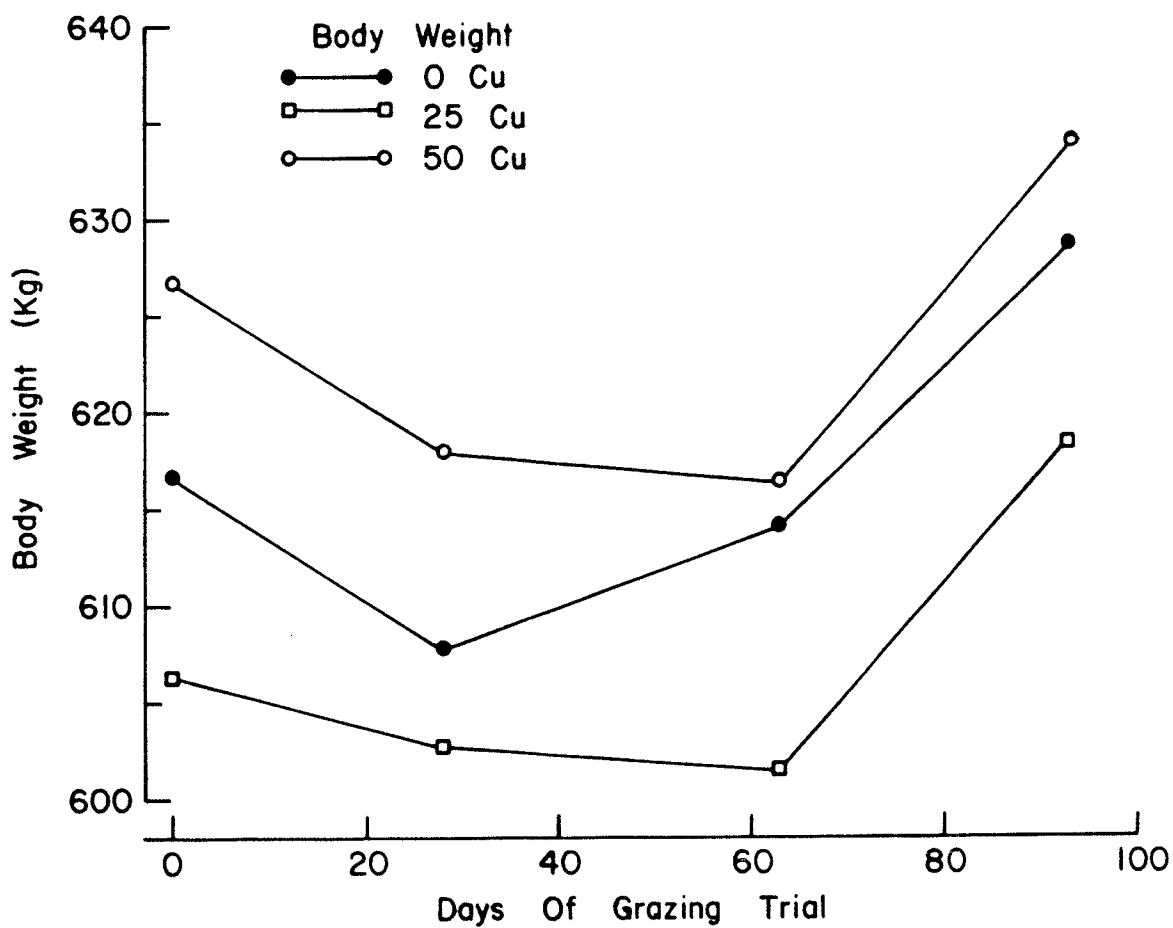


Figure 1. Least square means for body weight of cows treated with CuON.

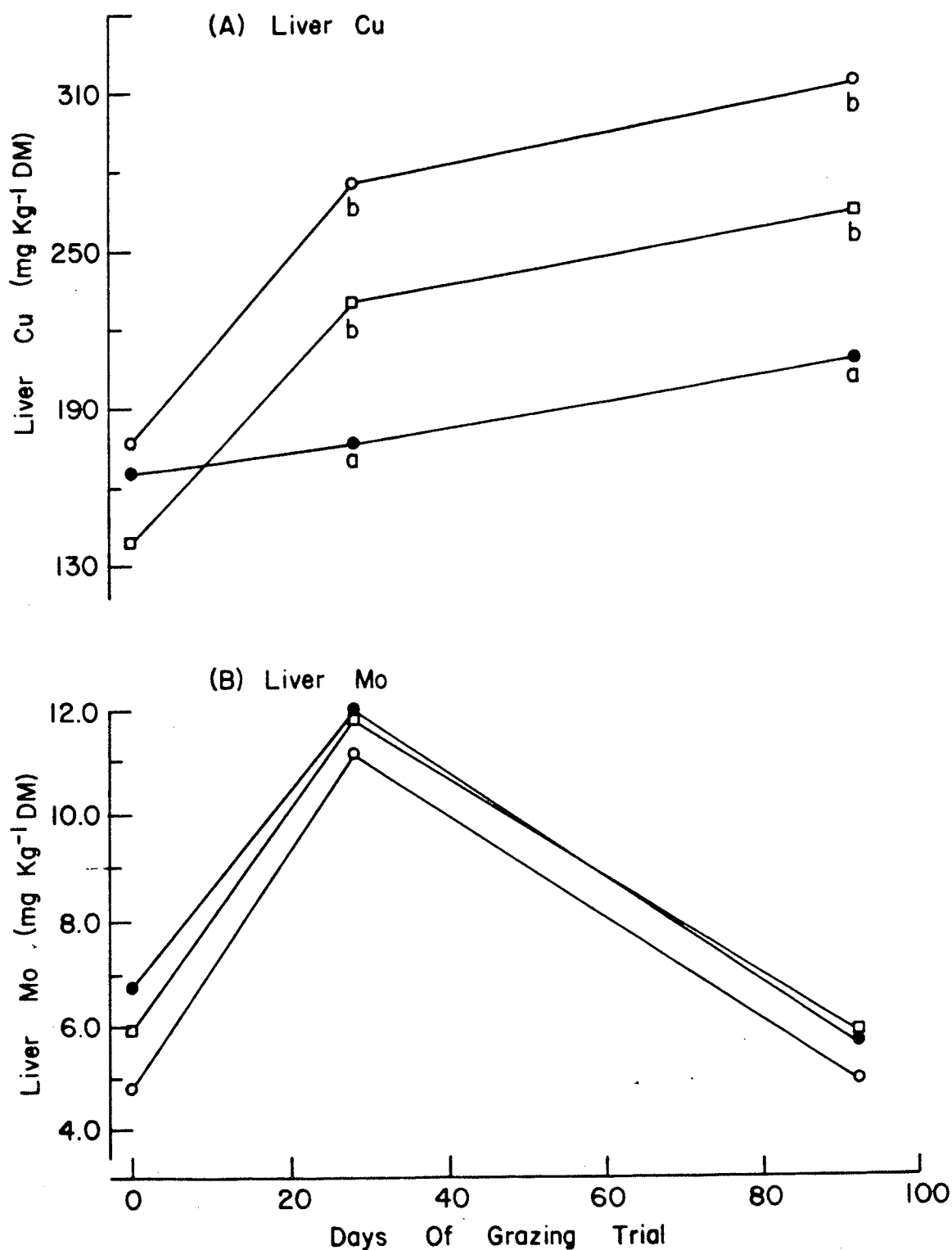


Figure 2. Least square means for liver Cu and Mo of cows treated with CuON: 0 Cu (●—●), 25 Cu (□—□) or 50 Cu (○—○), (A) liver Cu; (B) liver Mo. Means within a sampling day with different letters (a,b) are significantly different ($P < 0.01$) based on a linear contrast of days 92 or 28 relative to day 0.

sampling day effect (Table 4, Figure 3A). Mean plasma Cu for cows was higher ($P < 0.05$) on day 28 (Table 4). By day 63, all cows had lower ($P < 0.05$) plasma Cu levels. On day 92, plasma Cu for 0 Cu, 25 Cu and 50 Cu cows was 94, 94 and 88 $\mu\text{g dL}^{-1}$, respectively.

Plasma CpOx

Cow treatment did not affect ($P > 0.05$) CpOx activity (Table 3). On day 0, mean CpOx activity for all cows was $15.5 \pm 0.7 \Delta\text{A min}^{-1} \text{L}^{-1}$ (Table 4). Sub plot analysis showed a sampling day effect ($P < 0.01$) on plasma CpOx (Table 4, Figure 3B). Plasma CpOx activity was higher ($P < 0.05$) in all groups by day 28 (Table 4). Plasma CpOx activity was higher still ($P < 0.05$) at day 63, and was not different ($P > 0.05$) at day 92.

Liver Mo

On day 0, mean liver Mo for all cows was $5.8 \pm 0.4 \text{ mg kg}^{-1} \text{DM}$ (Table 4). Treatment with CuON had no effect ($P > 0.05$) on liver Mo levels (Table 3). There was a significant ($P < 0.01$) sampling day effect (Table 4, Figure 2B). In all cows, mean liver Mo increased ($P < 0.05$) to $11 \text{ mg kg}^{-1} \text{DM}$ by day 28 (Table 4). By the end of the trial, liver Mo was lower ($P < 0.05$) at $5.5 \text{ mg kg}^{-1} \text{DM}$.

Plasma Mo

Mean plasma Mo on day 0 for all cows was $35 \pm 2.3 \mu\text{g dL}^{-1}$ (Table 4). Cow treatment had no effect ($P > 0.05$) on plasma Mo levels (Table 3). There was a significant ($P < 0.01$) sampling day effect (Table 4, Figure 3C). For all cows, plasma Mo was higher ($P < 0.05$) at $47 \mu\text{g dL}^{-1}$ on day 28 (Table 4). Plasma Mo was lower ($P < 0.05$) at $22 \mu\text{g dL}^{-1}$ on day 92.

Liver Fe

There were no significant ($P > 0.05$) main plot or sub plot effects on liver Fe levels in cows (Appendix Tables A1, A2, A3). The mean liver Fe

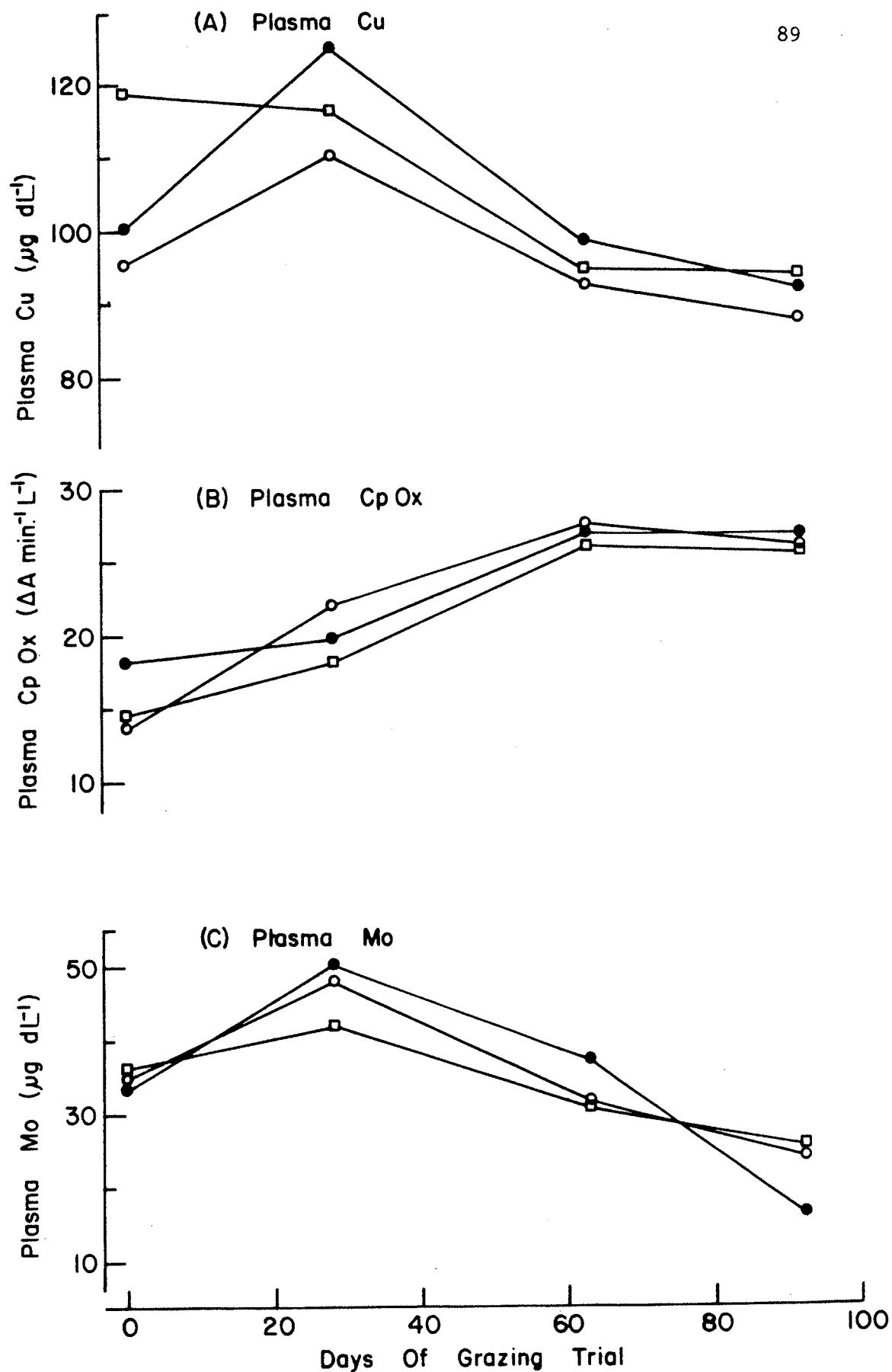


Figure 3. Least square means for plasma Cu, CpOx and Mo of cows treated with CuON: 0 Cu ($\bullet$ — $\bullet$), 25 Cu ($\square$ — $\square$), 50 Cu ($\circ$ — $\circ$). (A) plasma Cu; (B) plasma CpOx; (C) plasma Mo.

levels for all cows on days 0, 28 and 92 were 356, 380 and 370 ± 10.1 mg kg^{-1} DM (Table 4).

Plasma Fe

Cow treatment had no effect ($P > 0.05$) on cow plasma Fe. Mean plasma Fe at day 0 was 255 ± 8.6 $\mu\text{g dL}^{-1}$ for all cows (Table 4). Plasma Fe across sampling day was different ($P < 0.01$) for cows (Table 4). Plasma Fe was lower ($P < 0.05$) at day 63 (Table 4). At day 92, plasma Fe was lower ($P < 0.05$) than day 28 but greater than day 63.

Liver Zn

Mean liver Zn concentration at day 0 for all cows was 190 ± 13.4 mg kg^{-1} DM (Table 4). Treatment with CuON had no effect ($P > 0.05$) on liver Zn (Table 3). Sub plot analysis showed a significant ($P < 0.05$) sampling day effect (Table 4). On day 28, liver Zn was lower at 140 mg kg^{-1} and remained at this level on day 92.

Plasma Zn

Cow treatment had no effect ($P > 0.05$) on plasma Zn (Table 3). Mean plasma Zn for all cows on day 0 was 124 ± 6.1 $\mu\text{g dL}^{-1}$ (Table 4). Sampling day had a significant effect ($P < 0.01$) on plasma Zn across all treatments (Table 4). By day 28, plasma Zn was higher ($P < 0.05$) at 146 $\mu\text{g dL}^{-1}$ and remained at this level on day 63. On day 92, plasma Zn was lower ($P < 0.05$) at 75 $\mu\text{g dL}^{-1}$.

Correlations

Overall correlation coefficients and treatment correlation coefficients for cow plasma Cu, CpOx, Mo and liver Cu are reported in Table 5. There was a significant ($P < 0.01$) negative correlation between plasma Cu and plasma CpOx for 25 Cu cows ($r = -0.42$). Plasma Cu was correlated ($P < 0.01$) with plasma Mo in cows overall ($r = 0.26$) and in 0

Table 5. Correlation coefficients (r) between parameters measured over 92 days for cows treated with CuO_N (25 Cu, 50 Cu) or not treated (0 Cu)

	Number	Plasma Cu	Plasma CpOx	Plasma Mo
Liver Cu				
All cows	37	-0.02	0.04	-0.11
0 Cu	12	-0.29	-0.15	-0.17
25 Cu	12	0.14	0.07	-0.06
50 Cu	13	-0.01	0.18	-0.09
Plasma Cu				
All cows	37		-0.18	0.26**
0 Cu	12		0.09	0.45**
25 Cu	12		-0.42**	0.19
50 Cu	13		-0.02	0.26
Plasma CpOx				
All cows	37			-0.10
0 Cu	12			0.09
25 Cu	12			-0.41**
50 Cu	13			-0.14

**P<0.01)

Cu cows ($r = 0.45$). Plasma CpOx activity was significantly correlated ($P < 0.01$) with plasma Mo in 25 Cu cows.

Calves

Calves were divided into a calf treatment group (0 or 20 g CuON) within a cow treatment group (0, 25 or 50 g CuON) (Table 1). Therefore when considering calf main plot treatment effects, three treatment effects may exist which include: 1) calf treatment, 2) cow treatment (the effect of the cow treatment upon her calf) and 3) a calf treatment x cow treatment interaction).

Body weight

At day 0, mean body weight for all calves was 114 ± 1.8 kg. Calf treatment had no effect ($P > 0.05$) on body weight (Table 6). In the subplot analysis, sampling day affected ($P < 0.01$) body weight (Table 7, Figure 4). All calves gained weight over the grazing trial. On day 92, mean calf weight was 210 kg (Table 7).

Liver Cu

Main plot analysis showed a significant calf treatment effect ($P < 0.01$) on liver Cu levels (Table 6). Cow treatment had no effect ($P > 0.05$) on calf liver Cu. On day 0 mean liver Cu for all calves was 156 ± 7.3 mg kg⁻¹ DM (Table 7). On day 92, 0 Cu and 20 Cu calves had mean liver Cu values of 214 and 866 mg kg⁻¹ respectively. Subplot analysis showed a sampling day effect ($P < 0.01$; Table 7, Figure 5A) and a calf treatment x sampling day effect ($P < 0.01$).

Based on a linear contrast (Appendix Table B4), mean liver Cu on day 28 and 92 was 254 and 131 mg kg⁻¹ higher ($P < 0.01$), respectively, for 20 Cu calves compared to 0 Cu calves (Figure 5A). Liver Cu least square means for calf treatment are reported in Appendix Table C2. Liver Cu

Table 6. Main plot least square means for calf liver and plasma parameters

Parameter	Calf treatment		Cow treatment			MSE _A ⁺
	0	20	0	25	50	
Number	18	19	12	12	13	
Body weight ¹	160	163	161	161	162	2727.5
Copper						
Liver Cu ²	121B	246A	183	179	189	9106.6
Plasma Cu ³	87.1	87.1	85.8	88.3	87.3	588.1
Plasma CpOx ⁴	16.4	17.1	17.4	15.8	17.1	42.2
Molybdenum						
Liver Mo	4.3	4.3	4.2	4.6	4.1	3.0
Plasma Mo	20	23	20	21	25	210.4
Iron						
Liver Fe	310	301	298	297	322	18062.8
Plasma Fe	226	213	227	210	222	6118.5
Zinc						
Liver Zn	135	145	140	130	150	6035.9
Plasma Zn	145	150	124B	158A	160A	4064.6

⁺MSE_A - Mean squares error (Error A) in the main plot.

A,B, Means in the same row within cow or calf treatment with a different letter are significantly different (P<0.05) using NK test.

¹Body weight (kg).

²Liver Cu, Mo, Fe, Zn mg kg⁻¹ DM.

³Plasma Cu, Mo, Fe, Zn µg dL⁻¹.

⁴Plasma CpOx ΔA min⁻¹ L⁻¹.

Table 7. Least square means of calf liver and plasma parameters over sampling days

Parameter	Days				MSE _B [†]
	0	28	63 [‡]	92	
Number	37	36	37	37	
Body weight ¹	114D	142C	179B	210A	115.4
Copper					
Liver Cu ²	156B	245A	-	150B	1941.9
Plasma Cu ³	81.9B	111A	78.1B	77.3B	211.6
Plasma CpOx ⁴	9.1C	16.2B	20.0A	21.7A	14.6
Molybdenum					
Liver Mo	3.0C	5.5A	-	4.4B	2.9
Plasma Mo	8.9B	54A	14B	11B	210.4
Iron					
Liver Fe	279B	318A	-	320A	3848.0
Plasma Fe	255A	266A	178B	181B	3600.9
Zinc					
Liver Zn	158	129	-	133	5017.2
Plasma Zn	141B	186A	153B	111C	1584.5

[†]MSE_B - Mean squares error (Error B) from the sub plot.

[‡]No liver biopsies were taken on day 63.

A,B, Means in the same row with a different letter are significantly different (P<0.05) using NK test.

¹Body weight (kg).

²Liver Cu, Mo, Fe, Zn mg kg⁻¹ DM.

³Plasma Cu, Mo, Fe, Zn µg dL⁻¹.

⁴Plasma CpOx ΔA min⁻¹ L⁻¹.

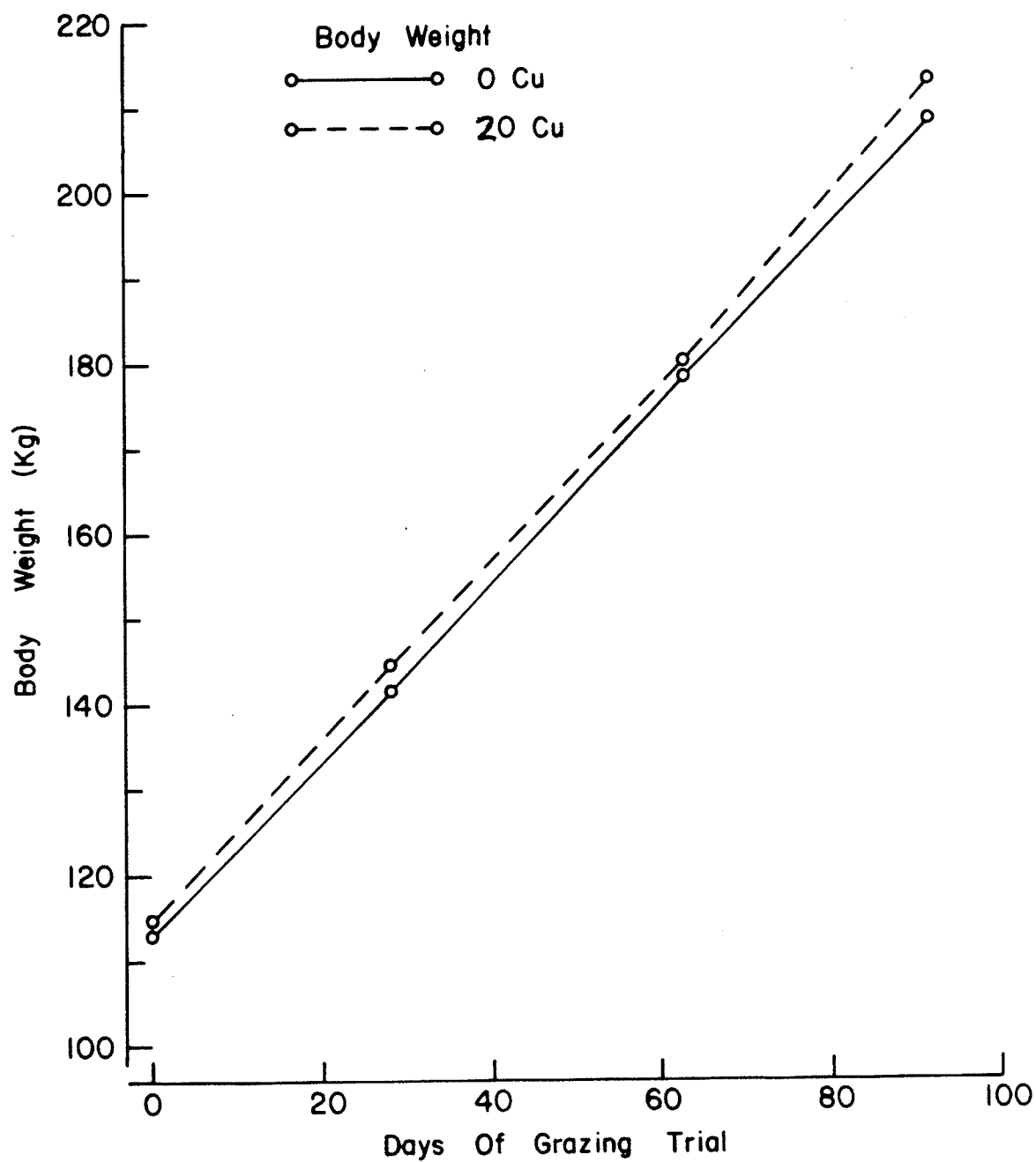


Figure 4. Least square means for body weight of calves treated with CuON.

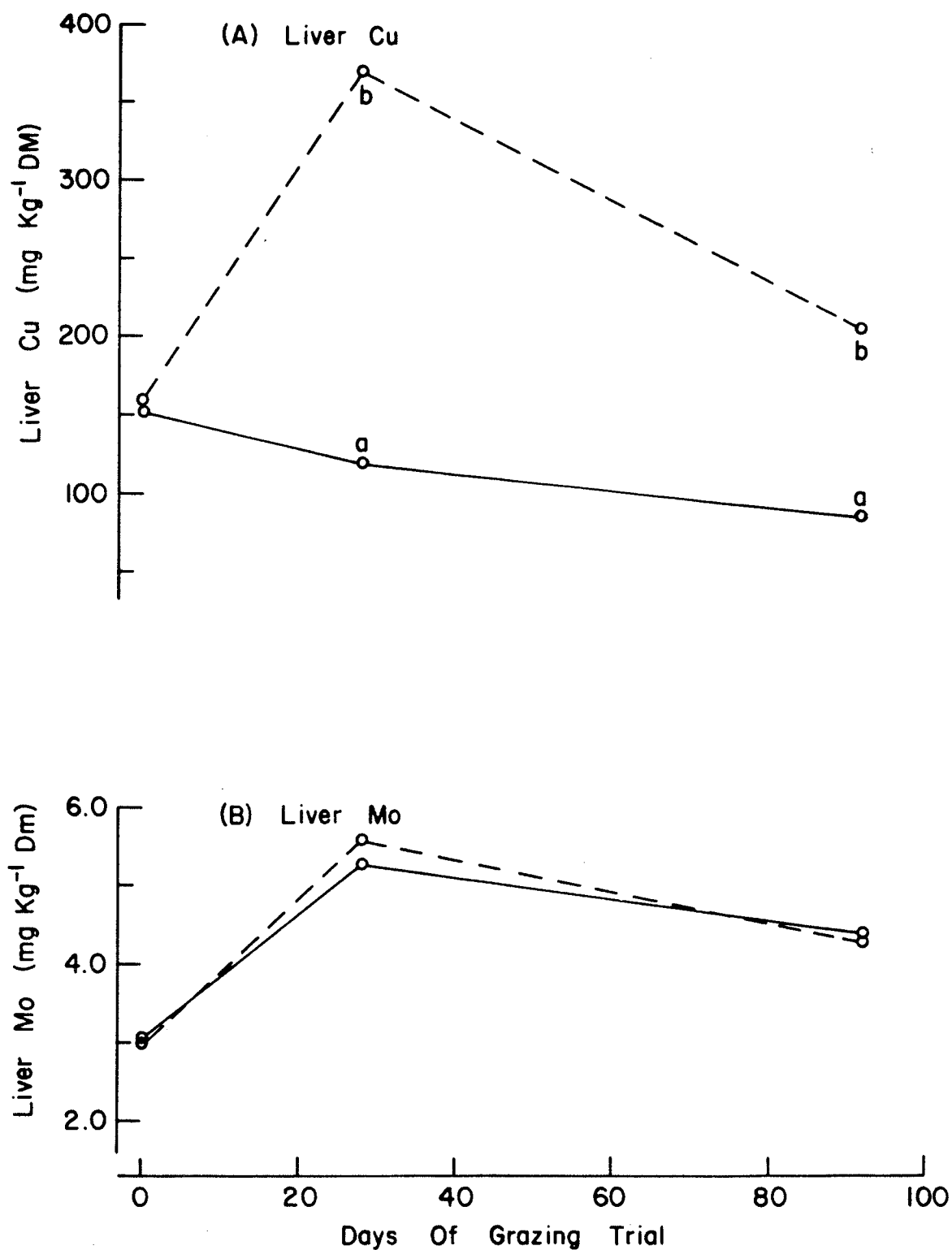


Figure 5. Least square means for liver Cu and Mo of calves treated with CuON: 0 Cu (o—o) or 20 Cu (o---o). (A) liver Cu; (B) liver Mo. Means within a sampling day with different letters (a,b) are significantly different ($P < 0.01$) based on a linear contrast of days 92 or 28 relative to day 0.

levels for treated calves were higher ($P < 0.05$) at day 28 (Figure 5A, Appendix Table C2), a pattern similar to that for all cows (Figure 2A, Appendix Table C1). However after day 28, liver Cu for treated cows (Figure 2A) continued to rise, while for treated calves (Figure 5A), liver Cu declined. Additionally, liver Cu of 0 Cu calves declined over the trial (Figure 5A) while liver Cu of 0 Cu cows increased (Figure 2A).

Plasma Cu

Plasma Cu for all calves on day 0 was $81.9 \pm 2.4 \text{ ug dL}^{-1}$ (Table 7). Cow treatment and calf treatment had no influence ($P > 0.05$) on plasma Cu (Table 6). Sampling day had a significant ($P < 0.01$) effect on plasma Cu (Table 7, Figure 6A). In all calves, plasma Cu was higher ($P < 0.05$) on day 28 at 111 ug dL^{-1} (Table 7). By day 63, plasma Cu was 78 ug dL^{-1} and was not different ($P > 0.05$) on day 92.

Plasma CpOx

Plasma CpOx activity for all calves on day 0 was $9.06 \pm 0.64 \Delta \text{A min}^{-1} \text{ L}^{-1}$. Calf or cow treatment had no significant ($P > 0.05$) effect on plasma CpOx (Table 6). The sub plot analysis showed a significant ($P < 0.01$) sampling day effect on plasma CpOx (Table 7, Figure 6B). Plasma CpOx activity increased ($P < 0.05$) in all calves over days 0, 28 to 63 and did not change ($P > 0.05$) thereafter (Table 7).

Liver Mo

Treatment with CuON had no effect ($P > 0.05$) on liver Mo (Table 6). Sampling day had a significant effect ($P < 0.01$) on liver Mo (Table 7, Figure 6C). The mean liver Mo level at day 0 for all calves was $3.0 \pm 0.3 \text{ mg kg}^{-1} \text{ DM}$ (Table 7). By day 28, the level was higher ($P < 0.05$) at 5.5 in all calves ($P < 0.05$). Liver Mo was subsequently lower ($P < 0.05$) on day

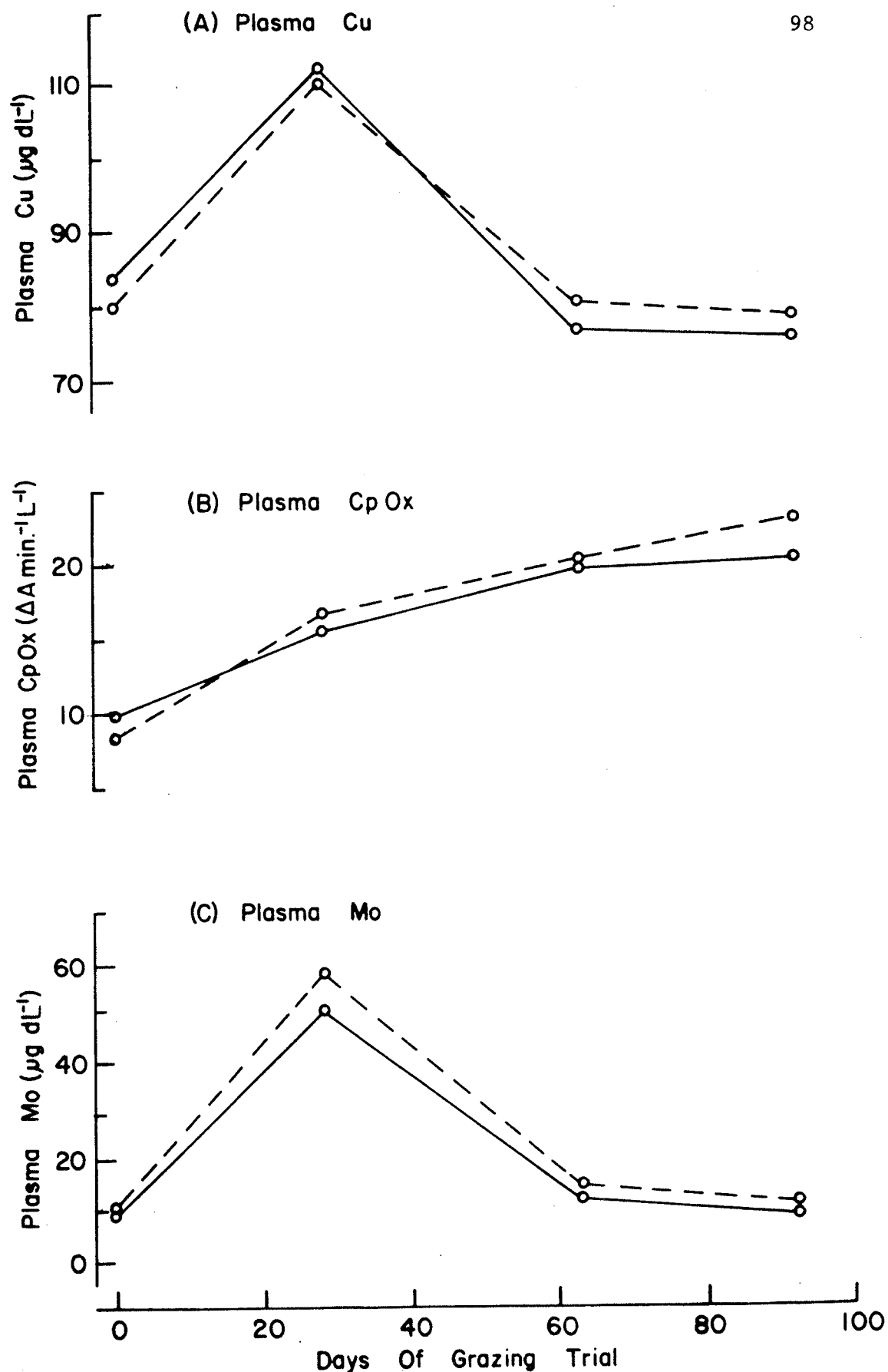


Figure 6. Least square means for plasma Cu, CpOx and Mo of calves treated with CuON: 0 Cu (o—o) or 20 Cu (o---o). (A) plasma Cu; (B) plasma CpOx; (C) plasma Mo.

92 at 4.4 mg kg^{-1} DM, but was not as low ($P < 0.05$) as at day 0 (Table 7).

Plasma Mo

Mean plasma Mo for all calves on day 0 was $8.9 \pm 2.6 \text{ ug dL}^{-1}$ (Table 7). Calf treatment or cow treatment had no effect ($P > 0.05$) on plasma Mo (Table 6, Figure 6C). On day 28, plasma Mo was higher ($P < 0.05$) at 54 ug dL^{-1} . By day 63, plasma Mo levels had returned to levels comparable to day 0 and were at this level on day 92 (Table 7). Plasma Mo level was higher ($P < 0.05$) on day 28 and lower thereafter, a pattern similar to that of calf liver Mo (Table 7) and that of Mo in liver and blood plasma of cows (Table 5).

Liver Fe

Liver Fe for all calves on day 0 was $279 \pm 10.2 \text{ mg kg}^{-1}$ DM. Treatment had no effect ($P > 0.05$) on calf liver Fe (Table 6); however there was a significant ($P < 0.05$) sampling day effect (Table 7). Liver Fe levels were higher ($P < 0.05$) on day 28 at 318 mg kg^{-1} and remained ($P > 0.05$) at this level on day 92.

Plasma Fe

Plasma Fe on day 0 for all calves was $255 \pm 9.9 \text{ ug dL}^{-1}$. Calf or cow treatment had no effect ($P > 0.05$) on plasma Fe levels (Table 6). Calf plasma Fe was not different ($P > 0.05$) for the first two sampling days (Table 7), however, on day 63 the mean plasma Fe was lower ($P < 0.05$) at 179 ug dL^{-1} and remained at this level at the end of the trial.

Liver Zn

Calf or cow treatment had no effect ($P > 0.05$) on liver Zn (Table 6). In the split plot analysis, no significant ($P > 0.05$) effects were reported. Mean liver Zn values for all calves on days 0, 28 and 92 were

158, 129 and 133 ± 11.7 mg kg⁻¹ DM, respectively (Table 7).

Plasma Zn

Plasma Zn for all calves on day 0 was 141 ± 6.6 ug dL⁻¹ (Table 7). Calf treatment had no effect ($P > 0.05$) on plasma Zn; cow treatment did have an effect ($P < 0.01$) on calf plasma Zn. The sub plot analysis revealed a sampling day effect ($P < 0.01$; Table 7), treatment cow x sampling day effect ($P < 0.01$; Figure 7) and a significant ($P < 0.05$) treatment calf x treatment cow x sampling day interaction (Appendix Table C4). The three-way interaction may be due to the variation among calf groups on day 0, as calves were not allocated into treatment groups based on plasma Zn (Appendix Table C4).

Based on a linear contrast analysis (Figure 7, Appendix Tables B3 and C3), mean plasma Zn of calves suckling 25 Cu cows was 71 ug dL⁻¹ higher ($P < 0.01$) on day 28 than calves suckling 0 Cu cows. Plasma Zn from calves on 50 Cu and 25 Cu cows was not different ($P > 0.05$). On day 63, there were no significant differences ($P > 0.05$) for plasma Zn of calves among the three cow groups (Figure 7). Plasma Zn was 111 ug dL⁻¹ on day 92 for all calves (Table 7).

Correlations

Overall correlation coefficients and treatment correlation coefficients for calf plasma Cu, CpOx, Mo and liver Cu are reported in Table 8. Unlike cow data, there were no significant ($P > 0.05$) correlations among these calf parameters.

Forage analysis

Forage consumed by cows and calves throughout the grazing trial consisted mainly of a mixture of brome and timothy grasses with a bit of kentucky grass. There was a very small amount of red clover estimated

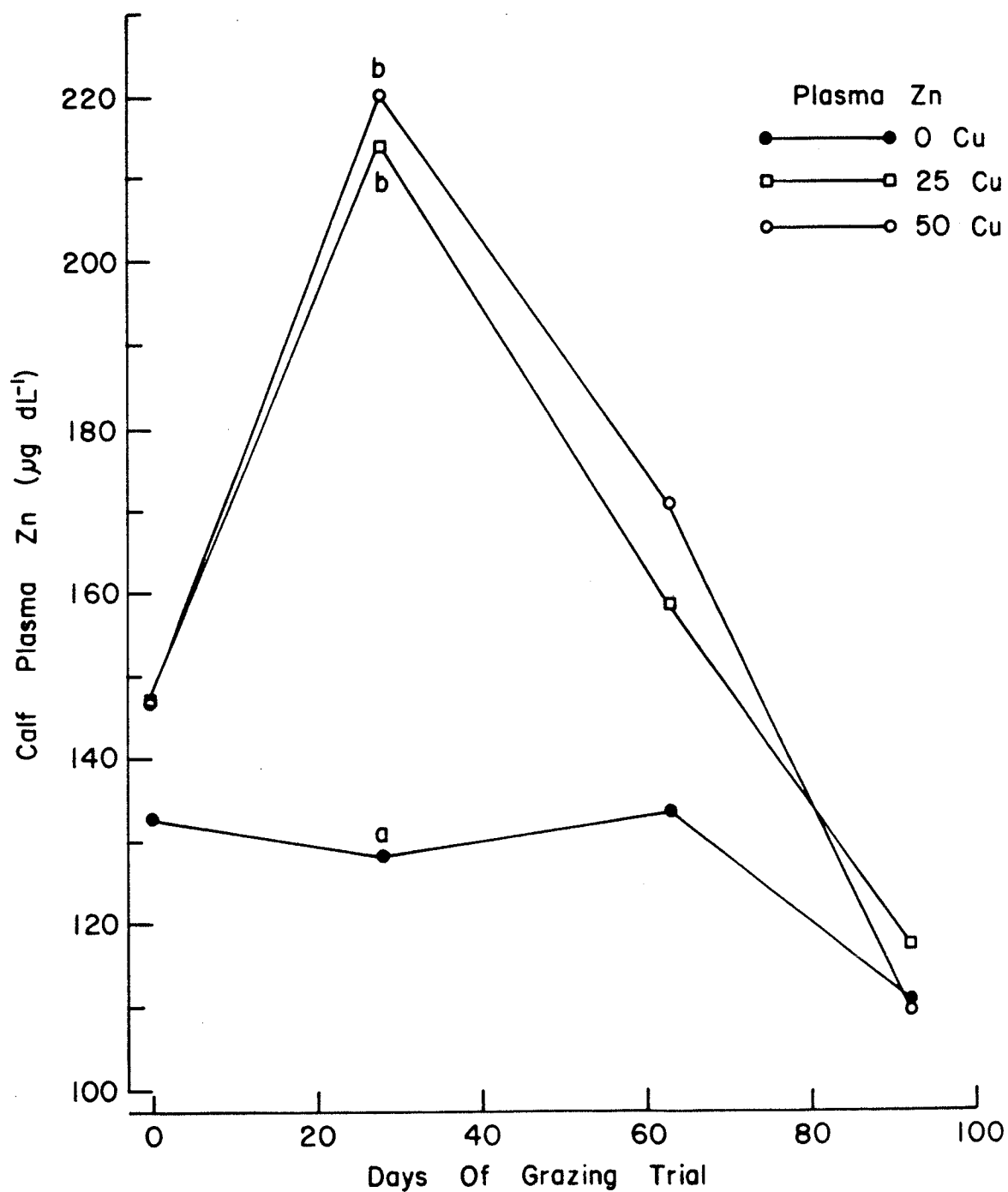


Figure 7. Least square means for calf plasma Zn in relation to cow treatment. Means within a sampling day with a different letter are significantly different ($P < 0.01$) based on a linear contrast on days 92, 63 or 28 relative to day 0.

Table 8. Correlation coefficients (r) between parameters measured over 92 days for calves treated with CuON (20 Cu) or not treated (0 Cu)

	Number	Plasma Cu	Plasma CpOx	Plasma Mo
Liver Cu				
All calves	37	-0.01	-0.08	0.06
0 Cu	18	-0.15	-0.10	-0.11
20 Cu	19	0.06	-0.12	0.07
Plasma Cu				
All calves	37		0.02	0.45
0 Cu	18		0.01	0.40
20 Cu	19		0.03	0.58
Plasma CpOx				
All calves	37			-0.01
0 Cu	18			-0.07
20 Cu	19			0.03

as less than 1% of total growth on pasture. Grasses were considered the main forage consumed. The legume is reported only to show the pasture potential if legume growth was encouraged. The pasture had excellent forage coverage and at no time throughout the trial did there appear to be a shortage of forage cover. The means for Cu, Mo and S for the grasses by sampling day and overall average, and for the legume overall average are reported in Table 9. Analysis of the water samples showed that the only mineral present was sulphide. The estimate of the effect of sulphide in the water upon the total sulphur content of the diet was calculated in Table 10. Water and dry matter intake of the cows was taken from NAS-NRC (1984). The estimate of the amount of water from the reservoir or spring consumed by calves was derived from Baily and Lawson (1981). They reported that at a calf weight of 130 to 175 kg, 75% of the total water intake was derived from the pasture source while 25% came from the cow's milk. The means for Fe, Zn, Mn, Ca, P, Mg, ADF and CP in the forage are listed in Table 11.

For the grasses, the mean Cu concentration on the sampling days ranged from 1.6 to 5.5 mg kg⁻¹ DM (Table 9). Cu content in grasses decreased over the trial resulting in an overall Cu average of 3.7 mg kg⁻¹ over the 92 days of the trial. The mean Mo concentration in grasses on sampling days ranged from 2.5 to 5.5 mg Mo kg⁻¹ DM. Mo levels were higher on day 63, but subsequently lower on day 92. Mean total S concentration in the forage remained relatively constant. The overall mean total S concentration over the season was 1.4 g kg⁻¹ DM. The Cu:Mo ratio decreased in the grasses throughout the trial and ranged from 0.5 to 1.4 with a mean overall average of 1.0.

On days 0 and 92 there was no red clover available for sampling.

Table 9. Cu, Mo and S content of forage¹ consumed by cows and calves

Parameter	Grasses								Legume			
	Day											
	3	28	63	92	Overall average		Overall average					
Number	3	6	3	3	15		2					
	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE
Cu mg kg ⁻¹	5.5	0.3	4.5	0.3	2.6	0.3	1.6	0.4	3.7	0.4	6.4	2.2
Mo mg kg ⁻¹	4.0	0.4	3.7	0.3	5.5	0.4	2.5	0.2	3.9	0.3	6.8	1.4
Total S g kg ⁻¹	1.5	0.0	1.5	0.0	1.3	0.1	1.3	0.4	1.4	0.0	1.8	0.5
Cu:Mo ratio	1.4		1.2		0.5		0.6		1.0		0.9	
Cu absorption coefficient ²	0.045		0.046		0.045		0.051		0.047		0.036	
Available Cu ³	0.25		0.21		0.12		0.08		0.17		0.23	
Final dietary ⁴ total S g kg ⁻¹												
Cows	3.5		3.4		3.2		3.2		3.3		3.7	
Calves	3.3		3.2		3.1		3.1		3.2		3.6	
Final dietary Cu absorption coefficient												
Cows	0.025		0.027		0.024		0.032		0.027		0.017	
Calves	0.026		0.028		0.024		0.032		0.028		0.018	
Final available Cu mg kg ⁻¹												
Cows	0.14		0.12		0.06		0.05		0.10		0.11	
Calves	0.14		0.13		0.06		0.05		0.10		0.12	

¹Expressed on a 100% DM basis. Grasses are a mixture of brome and timothy. Legume is red clover sampled on days 28 and 63.

²Calculated using the equation of Suttle and McLauchlan (1976) $\log(A) = -1.153 - 0.076(S) - 0.013(S \times Mo)$.
A = absorption coefficient, S = g kg⁻¹ DM, Mo = mg kg⁻¹ DM.

³Available Cu = mg kg⁻¹ Cu in forage, DM x (A).

⁴All final dietary data calculated using the final dietary total S calculated from Table 10.

Table 10. Estimation of the effect of sulphate in water consumed by grazing cows and calves upon the total sulphur content of the diet¹

	Day	Number	Concentration of sulphate in water (mg L ⁻¹)	Water sulphate ² added to diet (g kg ⁻¹ DM)	Water sulphur ³ added to diet ³ (g kg ⁻¹ DM)	Final dietary total S (g kg ⁻¹)	
						Grasses	Legume
<u>Cows</u>	0	2	985	6.0	2.0	3.5	-
	28	2	924	5.6	1.9	3.4	-
	63	2	942	5.7	1.9	3.2	-
	92	1	938	5.7	1.9	3.2	-
	Overall	7	947	5.8	1.9	3.3	3.7
<u>Calves</u>	0	2	985	6.7	1.8	3.3	-
	28	2	924	6.2	1.7	3.2	-
	63	2	942	6.4	1.8	3.1	-
	92	1	938	6.3	1.8	3.1	-
	Overall	7	947	6.4	1.8	3.2	3.6

¹ Assumptions - 600 kg cow consuming 11.2 kg DM with a S content from Table 9, and drinking 68 litres of water per day (NAS-NRC 1984).
- 175 kg calf consuming 5.5 kg DM with a S content from Table 9, and drinking 10.3 litres of water per day (Bailey and Lawson 1981).

² g sulphate per kg DM = litres of water x $\frac{\text{mg sulphate per litre} \times 1}{1000 \text{ mg per g}}$ DM intake (kg)

³ Sulphate = 33% sulphur.

⁴ Final sulphur content of diet = feed input (Table 9) + water input.

Table 11. Nutrient composition of forage¹ consumed by grazing cows and calves

Parameter		Grasses										Legume	
		Day										Overall average	
		0		28		63		92					
Number		3		6		3		3		15		2	
		$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE
Fe	mg kg ⁻¹	88.5	0.8	73.0	0.8	66.6	0.87	65.2	0.76	73.3	2.25	165.2	35.07
Zn	mg kg ⁻¹	25.9	0.7	21.7	0.85	14.2	0.65	13.8	0.82	19.3	1.36	27.0	3.17
Mn	mg kg ⁻¹	37.4	1.36	34.6	1.14	26.1	1.06	24.2	1.64	31.4	1.51	30.9	5.90
Ca	g kg ⁻¹	3.7	0.2	2.7	0.1	2.5	0.5	3.4	0.1	3.0	0.2	13.9	1.84
P	g kg ⁻¹	2.4	0.6	1.7	0.1	1.4	0.2	1.5	0.1	1.8	0.1	3.1	0.7
Mg	g kg ⁻¹	2.0	0.3	1.3	0.1	1.4	0.2	1.5	0.1	1.5	0.1	3.7	0.2
ADF	%	29.7	0.92	33.7	0.6	36.4	0.35	37.7	0.8	34.2	0.8	29.3	0.90
CP	%	13.6	0.55	9.1	0.8	7.4	0.1	7.3	0.3	9.3	0.7	17.8	1.90

¹Expressed on a 100% DM basis. Grasses is a mixture of brome and timothy. Legume is red clover sampled on days 28 and 63.

The concentration of Cu, Mo and total S in legume tended to be higher than that in grasses overall. The Cu:Mo ratio was similar for legume (0.9) and grasses (1.0).

The Cu absorption coefficient was calculating using the equation by Suttle and McLauchlan (1976) (Table 9). Including the water S into the calculation decreased the coefficient and the available Cu. The coefficient remained relatively constant during the grazing trial except on day 92 when it was higher. The final available Cu was similar for grasses and legumes.

Consumption of Pasture Mineral

The daily intake of the Co-op Sheep Mineral Supplement by the cattle was estimated on the basis of a cow-calf unit. It was assumed that the cows ate the mineral and the calves did not although this may vary over the period of the grazing trial. Mineral consumption was 26.5 g per cow-calf unit.

DISCUSSION

Body Weight

Mean body weight of all cows on day 0 was 616 kg (Table 3): cows were in good body condition at the start of the trial. While the body weight response of cows was not different among treatments, there was an improved final body weight gain for all cows (Figure 1). Body weight increased after day 63. The delayed increase in weight was probably due to a combination of factors. At the start of the trial the cows were already at a good body weight and it would not be expected that they would become heavier. Early in the season milk production is still relatively high as peak milk production is reached 30 to 60 days after calving (Streeter et al. 1974) followed by a continuous decline as the grazing season advances. Additionally early in the season grasses have a high moisture content (Church 1986). It is because of this high moisture content that cows have difficulty in consuming enough forage dry matter to meet their energy intake requirement even though early in the season energy digestibility may be quite high at 70 to 85% (Church 1986).

In situations where Cu is known to be deficient, positive responses to Cu supplementation have not always been obtained (Drysdale 1979). Variable body weight responses to Cu supplementation have been reported by Bingley and Anderson (1972), Thorton et al. (1972), Millis et al. (1976) and Boila et al. (1984a, b).

Other dietary components may affect Cu utilization by cattle, thereby contributing to a lack of response to Cu supplementation. Zn and Mn are known to be deficient in northwestern Manitoba (Drysdale et al. 1980). Forage consumed by grazing cows and calves was low in Zn. Mn

and CP. The overall nutritional requirement of the animal must be met before Cu supplementation may be able to give a positive response in body weight.

Cu supplementation had no effect on calf weight; all calves gained weight over the season (Figure 4). The age of animal is known to influence the response to Cu supplementation. Suttle and Angus (1976) reported that only after weaning did Cu supplementation have an effect upon body weight of calves. Plasma Cu levels below $30 \mu\text{g dL}^{-1}$ had to be maintained in order for this effect to be shown (Suttle and Angus 1976). Poole et al. (1974) found no effect of Cu supplementation on growth in cattle over one year of age. They reported the critical time for cattle to receive Cu supplementation was at less than 5 to 6 months of age. Beyond this age, Cu status of the animal and not body weight would be influenced by Cu supplementation.

Liver Copper

The normal liver Cu concentration in cattle is 100 to 400 mg kg^{-1} DM (Underwood 1977). To maintain a normal plasma Cu level, the minimum liver Cu concentration needed is 40 mg kg^{-1} DM (Claypool et al. 1975). All cows had initial liver Cu levels above 40 mg kg^{-1} except for one cow in group 25 Cu and one cow in group 50 Cu. Liver Cu levels in these two animals were 39 and 33 mg kg^{-1} DM. Therefore, based on liver Cu levels most of the cattle were in good Cu status at the start of the trial.

Over the grazing season liver Cu was different between CuON treated and untreated cows (Figure 2A). CuON were effective in increasing hepatic Cu concentrations. The higher dose of CuON had no beneficial effect over the 25 g dose (Figure 2A). It appears that liver Cu reached a plateau by day 28 and remained at that level until the end of the

trial.

Langlands et al. (1986b) reported that a dose of 0.045 g CuON per kg body weight provided sufficient hepatic Cu to Hereford cows and heifers. They predicted that at 50 to 100 days after dosing, the average increase in hepatic Cu concentration was 70 to 84 mg kg⁻¹ DM, respectively for an animal given 0.045 g CuON kg⁻¹ body weight. This dose is equivalent to the 25 g dose administered to the cows used in this experiment. Additionally, the increases in hepatic Cu in cows treated with 25 g CuON (Figure 2A) were similar to those reported by Langlands et al. (1986b). At days 28 and 92, cows in group 25 Cu had 78 and 85 mg kg⁻¹ DM more liver Cu than untreated cows.

Langlands et al. (1983) reported that as the dose rate of CuON increased in sheep, the quantity of hepatic Cu stored per gram of CuON given declined as the dose increased from 0 to 32 g. Two to three months after dosing peak hepatic Cu values were reached, followed by a rate of decline positively related to the dose given. MacPherson (1984) found there appeared to be only a two month advantage conferred by an increase in dosage rate in cows from 20 to 60 g CuON. It is therefore not surprising that the higher dose of 50 g CuON used in this study did not increase the hepatic Cu content above that of the 25 g CuON treated cows (Figure 2A). Liver Cu content of 0 Cu cows appeared to increase indicating dietary intake of forage Cu appeared to be sufficient, although final available Cu was lower as the trial advanced (Table 9). Cows were in good body Cu status when the trial began and cows may have been efficient at conserving liver Cu.

Treatment of 20 g CuON to calves increased liver Cu (Figure 5A). CuON provided an effective method of raising liver Cu levels in this

trial when administered at $0.18 \text{ g CuON kg}^{-1}$ body weight. This dose of 20 g was reported by others to be effective in raising liver Cu levels in calves (Dewland et al. 1979; Whitelaw et al. 1984; Richards et al. 1985).

Unlike treated cows, treated calves had liver Cu levels that were lower at the end than the beginning of the trial (Figure 5A). Calves (Figure 5A) compared to cows (Figure 2A) were not efficient in maintaining hepatic Cu stores. At the start of the grazing trial, calves obtained most of their dietary Cu from milk, which is very low in Cu (Underwood 1981) at 0.10 to 0.20 mg L^{-1} (Lonnerdal et al. 1981). The efficiency of Cu absorption in calves is high (ARC 1980), but at a time when dietary Cu from milk is low. As the season progresses more of the calf dietary Cu comes from the forage, also low in available Cu (Table 9). In addition the calf becomes less efficient in absorbing Cu as the efficiency of Cu absorption decreases as the calf grows (ARC 1980).

Additionally, the Cu requirement of the calf increases over time therefore liver reserves are depleted in order to meet this added Cu demand. In contrast, as the season progresses, lactation in the cow declines and her Cu demand decreases therefore the liver reserves can be replenished as Cu intake remains constant or increases.

There was no beneficial effect of providing supplemental Cu to the cow relative to increasing calf liver Cu levels (Table 6). This is in agreement with Underwood (1981) who reported that milk Cu, although low in Cu content, did not increase with Cu supplementation.

Plasma Copper

Treatment with CuON had no effect on plasma Cu levels in cows (Figure 3A) or calves (Figure 6A). Calves appeared to gain no benefit

when suckling cows dosed with CuON. This is in agreement with Boila et al. (1984b) who reported calves suckling cows injected with Cu glycinate showed no improvement in serum Cu levels over control calves.

The mean plasma Cu concentration for all cows and all calves on day 0 was 106 and 82 $\mu\text{g dL}^{-1}$, respectively. The normal range of Cu in plasma in ruminants is 80 to 120 $\mu\text{g dL}^{-1}$ (Underwood 1977). Levels below 60 $\mu\text{g dL}^{-1}$ were reported as indicative of a Cu deficiency. All cows and calves had initial plasma Cu levels above the deficient level throughout the trial. The ability of cows and calves to maintain adequate plasma Cu levels were related to adequate liver Cu stores at the start of the trial. In cows (Table 4) and calves (Table 7) plasma Cu concentrations were higher on day 28 and lower by day 63.

Dewland et al. (1986) reported plasma Cu levels increased in cows treated with 50 g CuON and in untreated cows, but to a smaller extent. After 80 days they found that plasma Cu was different between the treated and untreated cows. Judson et al. (1982) reported plasma Cu levels in suckling calves treated with 25 g CuON were not different from untreated calves at 20 weeks; however, by 41 weeks the treated calves had higher plasma Cu levels. It may therefore be possible that had this trial run longer than 92 days, a difference in plasma Cu between treated and untreated cattle might have been measured.

Plasma Cu was not correlated with liver Cu in cows (Table 5) or calves (Table 8) indicating that plasma Cu did not accurately reflect body Cu status. Blakley and Hamilton (1985) also reported that serum Cu was not correlated with hepatic Cu in cattle and sheep. The concentration of Cu in blood is not necessarily indicative of the Cu body status of the animal. Claypool et al. (1975) reported that only

when plasma Cu was less than 50 ug dL^{-1} was there a linear relationship at liver Cu of less than $40 \text{ mg kg}^{-1} \text{ DM}$. Throughout the trial reported herein, plasma Cu levels did not fall below 60 ug dL^{-1} and therefore it is not surprising that plasma Cu levels do not reflect the concentration of Cu in the liver.

Plasma CpOx

Since CpOx synthesis occurs in the liver, a major Cu storage site, CpOx activity may be a useful indicator of Cu status. Elevated CpOx levels were observed in Cu deficient sheep and cattle that were fed Cu supplemented diets (Lorentz and Gibb 1975; Kincaid et al. 1986a). Feeding Mo and S to ruminants has resulted in varying reports in CpOx activity. A decrease in CpOx was seen in Mo and S supplemented sheep (Suttle and Field 1968) while others have reported no reduction in CpOx in sheep (Bremner 1976; Bremner and Young 1978). CuON had no effect on the activity of CpOx in cows (Figure 3B) or calves (Figure 6B). Activity was higher on day 63 in cows (Figure 3B) and calves (Figure 6B) indicating circulating levels of metabolically active Cu were higher in all cattle. CpOx is reported to comprise 90 to 95% of the plasma Cu (Frieden 1980).

Since CpOx is synthesized in the liver, it would be expected that liver Cu and CpOx activity may be highly correlated. However, in cows (Table 5) and calves (Table 8), liver Cu and plasma CpOx were not correlated. This is in agreement with the results of Blakely and Hamilton (1985). They proposed that the correlation is poor because CpOx activity is likely maintained at the expense of hepatic Cu stores and would not be a useful indicator of liver Cu stores until liver Cu had been depleted. Under conditions associated with normal Cu status,

as appears with cows and calves in this trial, most of the liver Cu stores are thought to be sequestered in a storage pool, unlikely to correlate closely with the functional Cu pool that is associated with CpOx.

As CpOx activity changed over sampling days in all cows (Table 4) and calves (Table 7), a different pattern was observed in plasma Cu in cows (Table 4) and calves (Table 7). Plasma Cu in all cows (Table 4) and calves (Table 7) was higher on day 28 and lower by day 63. Correlation coefficients were poor for calves (Table 8) between plasma Cu and CpOx and poor for cows (Table 5) except for 25 Cu cows. Surprisingly there was a significant negative correlation between the two parameters in 25 Cu cows. This may be due to the fact that plasma Cu appeared to be higher at day 0 in 25 Cu cows (Figure 3A) compared to 0 Cu or 50 Cu cows. Cows were not allocated into treatment groups based on plasma CpOx.

Since CpOx is reported to comprise 90 to 95% of the plasma Cu (Frieden 1980), it would be expected that a good correlation would exist between the two parameters, as reported by Bingley and Anderson (1972). However, results of this trial agree with those of Kincaid et al. (1986a) who reported a poor correlation between plasma Cu and CpOx in cattle. Connor and Poole (1981) reported there was a dramatic drop in the correlation between plasma Cu and CpOx activity in yearling bulls treated with CuON, CuCaEDTA or Cu glycinate which coincided with the appearance of a significant quantity of TCA-insoluble Cu. They thought this was due to a seasonal rise in S levels in an area with elevated pasture Mo levels. In this trial, high water S (Table 10) and high forage Mo levels (Table 9) remained constant throughout the season.

This S and Mo might have contributed to an increase in TCA-insoluble Cu resulting in a poor correlation between plasma Cu and CpOx.

Molybdenum

Lesperance and Bohman (1983) reported that plasma Mo was the best indicator of a high dietary intake of Mo; liver Mo increased at a slower rate relative to plasma Mo. In this trial, liver Mo levels peak at the same time as plasma Mo (Table 4, Figures 2B and 3C). Liver Mo was low in calves and cows indicating that the liver is not a site of Mo accumulation.

Liver Mo in cows (Figure 2B) and calves (Figure 5B), and plasma Mo in cows (Figure 3C) and calves (Figure 6C) was higher on day 28 and was subsequently lower on day 63. Higher plasma Mo coincided with a higher plasma Cu concentration in cows (Figure 3A and 3C) and calves (Figure 6A and 6C), followed by a lower concentration for both parameters. Clawson et al. (1972) reported that plasma Cu in Hereford helpers was higher at day 30 in animals receiving supplemental Cu and Mo. They proposed that the higher plasma Cu levels in the helpers represented the plasma accumulation of an insoluble Cu-Mo complex. Mason et al. (1986) suggested a three-way interaction between albumin, Cu and TM occurs in plasma leading to an increase in the plasma Cu measure. Therefore, the coincident peaks observed in this grazing trial may be due to the formation of an insoluble Cu-Mo complex in the plasma. The high level of Mo in the forage consumed by the cattle appears to have this effect whether cattle were treated with CuON or left untreated. The fact that there was no difference in plasma Cu or Mo between treatment groups in cows and calves indicates that CuON did not modify the Cu-TM relationship as reported by Langlands et al. (1986a). They proposed

that little Cu is released in the rumen and the availability of Cu in the CuON when lodged in the abomasum is unaffected by dietary Mo and S. Therefore Cu from the CuON would not be available in the rumen for the formation of insoluble Cu-TM.

Correlations between plasma Cu and Mo were significant in all cows and 0 Cu cows (Table 5). Mills et al. (1978) and Moshtaghi-Nia (1985) reported that a high correlation between serum Cu and serum Mo was associated with a high level of serum TCA-insoluble Cu.

Lesperance and Bohman (1963) reported the addition of inorganic Mo at 100 mg kg^{-1} DM to the diets of Hereford heifers increased plasma Mo concentrations. However, 12 months after the start of the trial, a sudden decrease in plasma Mo occurred which was not accounted for by animals going off feed. This decline in plasma Mo was much greater and appeared later than the decline observed in cows (Figure 3C) and calves (Figure 6C) in this trial. This may be due to the extreme differences in dietary Mo levels. Monty and Click (1961) reported that rats maintained on diets containing toxic levels of Mo developed the ability to adjust homeostatically to high Mo levels. This phenomenon may explain the reduction seen in plasma Mo and liver Mo in cows (Figures 2B and 3C) and calves (Figures 5B and 6C) as they become more efficient in excreting excess dietary Mo.

Iron

Liver Fe levels in cattle were well above the liver Fe content of 169 mg kg^{-1} DM measured by Ammerman et al. (1967) in what they termed Fe-depleted calves. Supplementation with CuON had no influence on the concentration of Fe in the liver or plasma of cows or calves. Over the grazing season, liver Fe levels remained constant in cows (Table 4)

while in calves (Table 7) levels were higher on day 28. Milk is relatively low in Fe, containing 0.3 to 0.5 mg L⁻¹ (Underwood 1981). The increase seen in calf liver Fe (Table 7) may reflect a decrease in milk consumption by calves along with an increase in forage (Table 11) and soil consumption (Healy 1968) with a resultant increase of dietary Fe content. Following the increased hepatic Fe deposition, calf plasma Fe was lower (Table 7) indicating that less Fe was circulating in the plasma. Charpentier (1966 as cited in ARC 1980) reported that with Fe deficient calves, an increase in Fe in the blood is accompanied by a release of Fe from the liver. The reverse occurs with calves in this trial (Table 7) indicating that calves are in an Fe sufficient state since Fe is taken up from the plasma and deposited in the liver.

Liver Fe levels in cows remained constant throughout the trial (Table 4). Older cattle appear to be more efficient in maintaining Fe body stores as the Fe requirement in older cattle is less critical than young animals (ARC 1980). Reports of Fe deficiency of nutritional origin are rare in cattle (ARC 1980). The reduction seen in plasma Fe in cows (Table 4) may reflect the change in Fe content in the forage (Table 11).

Zinc

Treatment of cows with CuON had no effect on cow Zn status (Table 4). Underwood (1981) reported that the normal plasma Zn concentration is 80 to 120 ug dL⁻¹. Plasma Zn in cows was higher on day 28 accompanied by a lower concentration in liver Zn (Table 4). It appeared that plasma Zn of cows was higher at the expense of reserves of Zn in liver. Underwood (1977) reported changes in concentration of serum Zn may be indicative of changes in tissue turnover of Zn, stress, infection

or dietary availability. Plasma and liver Zn in cows remained stable until the end of the trial (Table 4) when plasma Zn was at levels indicative of a Zn deficiency based on criteria established by Spais and Papasteriadis (1974). This possibly reflects the low Zn content of the forage consumed by cattle (Table 11). Zn levels in forage on day 63 were approximately half that on day 0. Pasture mineral intake was calculated to be 26.5 g per cow-calf unit; therefore each cow-calf unit was calculated to be consuming $39.8 \text{ mg Zn day}^{-1}$. The Zn requirement of cattle (NAS-NRC 1984) should have been met; however mineral consumption is variable and the mineral may have been consumed in greater amounts by grazing cattle that were not on trial.

Treatment of calves with CuON had no effect on Zn status of calves (Table 6); however plasma Zn was higher in calves suckling cows treated with CuON (Figure 7). The only link that exists between a cow and her calf is the cow's milk. Zn content in milk is high at 2.30 to 5.00 mg L^{-1} (Underwood 1977). It is assumed that there was an increase in Zn excretion in the milk of cows treated with CuON. However, there was no difference for plasma Zn or liver Zn between treated and untreated cows (Table 4). There may have been an increase in the Zn turnover, without changes in Zn content in the body of cows treated with CuON, resulting in an increase of Zn excretion in the milk. Miller (1979) reported that the mammary gland excretes Zn at a higher rate when excess Zn is present in the body, while Underwood (1981) reported an increase of Cu content in milk was not seen with excess dietary Cu. Additionally, Mills (1980) reported a depletion and repletion in dietary Zn leads to a decrease and increase, respectively, in Zn content in milk. The decrease in plasma Zn seen in calves after day 28 may be due to a decrease in Zn content in

the milk. Forage consumed by cows was lower in Zn content by day 63 (Table 11) and cows on a low Zn diet conserve Zn by reducing the amount of Zn going into milk (Miller 1979). Additionally Zn content in the milk decreases as lactation progresses (Underwood 1977). Calves consumed less milk, lower in Zn content, and more forage, also lower in Zn (Table 11) as the season progressed.

Judson et al. (1982) reported plasma Zn did not differ in calves treated with CuON or untreated calves. Bolla (1987a) reported that serum Zn from cows and calves did not differ between CuON treated and untreated cows and calves. In both studies the effect of calves suckling cows treated with CuON was not examined.

Minerals in the Forage

Forage nutrient contents (Table 9 and 11) are defined as adequate or deficient for cattle as recommended by NAS-NRC (1984). The Ca, Fe, Mg and total S concentrations were adequate in the grasses and legume. In the grasses and legume, Cu, Mn, Zn and CP were deficient. P was also low in the grasses. Supplementary minerals in the free-choice mineral preparation were provided to correct mineral deficiencies. Based on a mineral intake of the Co-op supplement of 26.5 g per cow-calf unit, consumption of Zn and Mn was 39.8 and 17.0 mg kg⁻¹ DM, respectively, from the Co-op sheep mineral supplement. Therefore this mineral supplement in addition to the Zn and Mn in the forage should have been sufficient to meet the animal's requirements.

Calculation of the Cu absorption coefficient (A) provides an estimate of the fraction of dietary Cu available for absorption from the digestive tract (Table 9). The equation used in this study by Suttle and McLauchlan (1976) was criticized by Suttle (1983) in that the former

study used semi-purified diets with sheep. Suttle (1983) suggested the use of an equation derived from pasture animals, however at high S levels, the calculations do not "make sense". The percentage of available Cu should decrease with increasing S content in the forage but with the equation by Suttle (1983), this value increased. Therefore, the equation by Suttle and McLauchlan (1976) was thought to give a more reliable estimate of available Cu. Including water S into the calculation as a source of dietary S caused a dramatic drop in the value of (A) as water S was quite high (Table 10). The absorption coefficient and available Cu was calculated to be similar for cows and calves. The available Cu appeared to decline over the grazing season (Table 9), as did the Cu measure in the grasses.

The absorption coefficient was calculated for legume and appeared to be lower than in grasses (Table 9); however, available Cu in legume was approximately the same as in grasses overall (Table 9). It does not appear that encouragement of legume growth in the pasture would not change the available Cu content of forage consumed by cattle. However, an increase in the amount of legume on pasture would increase the CP intake of the cattle as in general, legumes have a higher protein content than grasses (Church 1986). The Cu:Mo ratio in grasses and legume are lower than those recommended by Alloway (1973), Stewart and Racz (1977) and Fisher and Waldern (1978); suggesting that the high Mo content of the forage exerted adverse effects upon Cu utilization in ruminants and had contributed to the declining liver Cu concentration observed in calves (Figure 5A). More importantly, the Cu content in the forage (Table 9) was much lower than the absolute Cu requirement, based on dietary levels for cattle reported by ARC (1980). This low Cu level

by itself would lead to a primary Cu deficiency if these animals had lower body Cu levels when grazing began.

SUMMARY

Copper oxide needles were evaluated as a source of supplemental Cu for cows and their calves. Doses of CuON were given, 0, 25 or 50 g to cows and 0 or 20 g to calves, at the start of the grazing season. Effects upon body weight; and measures of Cu, Zn, Fe and Mo status in cattle were evaluated.

1. The body weight response of cows and calves was not influenced ($P>0.05$) by supplementation with CuON. Cow weight was higher after day 63 and calves grew continually throughout the trial.
2. There was a significant ($P<0.01$) cow treatment x sampling day interaction for liver Cu. CuON increased ($P<0.01$) liver Cu concentration of 25 Cu and 50 Cu cows when measured on sampling days 28 and 63. Liver Cu concentration of 50 Cu cows was not higher ($P>0.05$) than the liver Cu concentration of the 25 Cu cows.
3. Calves treated with 20 g CuON had higher ($P<0.01$) liver Cu concentrations than untreated calves at days 28 and 92. By day 92, liver Cu levels were lower ($P<0.01$) than on day 28 in all calves.
4. Treatment of cows with CuON had no effect ($P>0.05$) on the Cu status of calves.
5. CuON had no effect ($P>0.05$) on the concentration of Mo or Fe in liver or plasma, the Cu concentration and CpOx activity in plasma or the concentration of Zn in liver in cows and calves.

6. Treatment of cows or calves did not influence ($P>0.05$) plasma Zn of cows or calves, respectively. Treatment of cows with CuON did modify ($P<0.05$) calf plasma Zn. On day 28 plasma Zn was higher ($P<0.01$) in calves suckling 25 Cu and 50 Cu cows compared to calves suckling 0 Cu cows.
7. Forage analysis showed Cu was deficient in grasses relative to the absolute requirement of cattle. In addition, Mo levels were high in the grasses, resulting in a low Cu:Mo ratio of 0.9. Water consumed by grazing cattle contained a high sulphate level, contributing to a decrease in the absorption coefficient for Cu, resulting in a low available Cu in grasses.
8. Legume was present in the pasture but contributed to less than 1% of the ground cover. The legume Cu absorption coefficient appeared to be lower than that of grasses while available Cu and the Cu:Mo ratio in legume was approximately the same as in grasses overall. It does not appear that encouragement of legume growth in the pasture would not change the available Cu content of forage consumed by cattle; however, an increase in legume pasture coverage would increase the protein intake of the cattle which was low.

CONCLUSIONS

CuON did not modify the body weight response of grazing cows or calves. This lack of response was possibly due to the good body condition and Cu status of the animals at the start of the trial. Liver Cu concentrations were improved in treated cows and calves over the season indicating CuON were an effective method of providing supplemental Cu to grazing cattle. A dose of 50 g CuON did not increase the hepatic Cu content above that of the 25 g CuON treated cows.

Plasma Cu and CpOx activity and Mo in plasma and liver were not influenced by dosing cows or calves with CuON. There were no adverse effects of a high dose of CuON upon Zn or Fe in plasma or liver, as indicators of Zn or Fe status. Plasma Zn was higher in calves suckling CuON treated cows, thereby indicating a beneficial effect of CuON supplementation in cows upon Zn status in calves.

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A P P E N D I X

Appendix Table A1: Analysis of variance table for cow weight

	Source	DF	Type III MSE
<u>Main plot</u>	Trt cow	2	3452.0
	Error A = cow (trt cow)	34	20900.1
<u>Sub plot</u>	Sampling day	3	2319.3**
	Trt cow x sampling day	6	67.0
	Error B = cow (trt cow x sampling day)	101	192.4

**P<0.01

Appendix Table A2: Analysis of variance for cow plasma parameters

Source	DF	Type III MSE				
		Cu	CpOx	Mo	Fe	Zn
<u>Main plot</u>						
Trt cow	2	1291.4	47.3	18.1	14620.6	28.4
Error A = cow (trt cow)	34	827.0	107.1	418.4	9217.0	1992.2
<u>Sub plot</u>						
Sampling day	3	4647.6**	1098.1**	3785.2**	87464.9**	42734.8**
Trt cow x sampling day	6	559.0	25.3	241.0	3743.0	783.9
Error B = cow (trt cow x sampling day)	101	321.8	19.3	196.6	2702.6	1365.2

**P<0.01

Appendix Table A3: Analysis of variance table for cow liver parameters together with linear contrast analysis and estimate of the difference in liver Cu of CuO_N treated cows (50 Cu, 25 Cu) and untreated cows (0 Cu) at days 28 or 92 relative to day 0

Source		DF	Type III MSE			
			Cu	Mo	Fe	Zn
<u>Main plot</u>	Trt cow	2	48511.9	11.6	4033.1	9543.7
	Error A = cow (trt cow)	34	23565.2	6.2	14387.3	8909.6
<u>Sub plot</u>	Sampling day	2	100170.9**	473.7**	5166.0	30199.7*
	Trt cow x sampling day	4	10245.5**	1.0	4387.8	11378.0
	Error B = cow (trt cow x sampling day)	67	2225.3	318.6	249367.9	6663.8
<u>Contrast liver Cu</u>						
50 Cu - 25 Cu	Day 92-0	1	365.0			
25 Cu - 0 Cu	Day 92-0	1	21463.8**			
50 Cu - 25 Cu	Day 28-0	1	239.9			
25 Cu - 0 Cu	Day 28-0	1	18365.3**			
<u>Estimate liver Cu</u>	<u>Estimated difference</u>	<u>S.E. of estimate</u>				
50 Cu - 25 Cu	Day 92-0	10.8	26.7			
25 Cu - 0 Cu	Day 92-0	84.6	27.2**			
50 Cu - 25 Cu	Day 28-0	8.9	27.1			
25 Cu - 0 Cu	Day 28-0	78.2	27.2**			

**P<0.01

*P<0.05

Appendix Table B1: Analysis of variance table for calf weight

	Source	DF	Type III MSE
<u>Main plot</u>	Trt calf	1	303.0
	Trt cow	2	9.3
	Trt calf x trt cow	2	6793.8
	Error A = calf (trt calf x trt cow)	31	2727.5
<u>Sub plot</u>	Sampling day	3	65330.5**
	Trt calf x sampling day	3	14.3
	Trt cow x sampling day	6	38.7
	Trt calf x trt cow x sampling day	6	54.1
	Error B = calf (trt calf x trt cow x sampling day)	92	115.4

**P<0.01

Appendix Table B2: Analysis of variance for calf plasma parameters

Source	DF	Type III MSE				
		Cu	CpOx	Mo	Fe	Zn
<u>Main plot</u>						
Trt calf	1	0.1	20.1	508.5	6105.2	678.0
Trt cow	2	146.5	36.3	321.9	3606.2	18700.6**
Trt calf x trt cow	2	258.7	16.4	458.7	3762.7	183.7
Error A = calf (trt calf x trt cow)	31	588.1	42.2	210.4	6118.5	4064.6
<u>Sub plot</u>						
Sampling day	3	9341.7**	1167.6**	16890.0**	79888.7**	34472.6**
Trt calf x sampling day	3	97.7	23.7	66.6	2083.1	3281.0
Trt cow x sampling day	6	108.4	15.8	176.2	2944.5	5918.4**
Trt calf x trt cow x sampling day	6	217.6	5.3	93.7	1822.4	3491.1*
Error B = calf (trt calf x trt cow x sampling day)	92	211.6	14.6	257.2	3600.9	1584.5

**P<0.01

*P<0.05

Appendix Table B3: Linear contrast analysis and estimate of the difference in calf plasma Zn within cow treatment on days 28, 63 or 92 in relation to day 0

Contrast	DF	MSE	
50 Cu - 25 Cu Day 92-0	1	164.1	
25 Cu - 0 Cu Day 92-0	1	211.8	
50 Cu - 25 Cu Day 63-0	1	542.1	
25 Cu - 0 Cu Day 63-0	1	293.7	
50 Cu - 25 Cu Day 28-0	1	123.3	
25 Cu - 0 Cu Day 28-0	1	15494.6	**

Estimate	Estimated difference	S.E. of estimate	Significance
50 Cu - 25 Cu Day 92-0	-7.3	22.7	
25 Cu - 0 Cu Day 92-0	-8.3	22.7	
50 Cu - 25 Cu Day 63-0	13.3	22.7	
25 Cu - 0 Cu Day 63-0	9.8	22.7	
50 Cu - 25 Cu Day 28-0	6.4	23.0	
25 Cu - 0 Cu Day 28-0	71.1	22.7	**

**P<0.01

Appendix Table B4: Analysis of variance table for calf liver parameters together with linear contrast analysis and estimate of the difference in liver Cu of CuO_N treated calves (20 Cu) and untreated calves (0 Cu) at days 28 or 92 relative to day 0

		Type III MSE				
	Source	DF	Cu	Mo	Fe	Zn
<u>Main plot</u>	Trt calf	1	422030.9**	0.1	1744.4	2582.0
	Trt cow	2	975.1	2.5	7423.0	3455.7
	Trt calf x trt cow	2	19433.7	6.9	24977.5	3050.7
	Error A = calf (trt calf x trt cow)	31	9106.6	3.0	18062.8	6035.9
<u>Sub plot</u>	Sampling day	2	102840.1**	55.5**	19959.2**	9142.6
	Trt calf x sampling day	2	145559.4**	0.4	11660.0	10295.5
	Trt cow x sampling day	4	1787.0	2.5	4069.9	5912.1
	Trt calf x trt cow x sampling day	4	1559.0	5.8	2270.7	7423.2
	Error B = calf (trt calf x cow x sampling day)	61	1941.9	2.9	3848.0	5071.2
<u>Contrast liver Cu</u>						
20 Cu - 0 Cu	Day 92-0	1	79393.0**			
20 Cu - 0 Cu	Day 28-0	1	290838.0**			
<u>Estimate liver Cu</u>	<u>Estimated difference</u>	<u>S.E. of estimate</u>				
20 Cu - 0 Cu	Day 92-0	131.2	20.5**			
20 Cu - 0 Cu	Day 28-0	253.5	20.7**			

**P<0.01

Appendix Table C1: Least square means of cow liver Cu based on a linear contrast on days 92 or 28 relative to day 0†

Liver Cu (mg kg ⁻¹ DM)				
	Day			
	0	28	92	MSE _B †
Number	37	36	37	
0 Cu	164	175a	207a	2225.3
25 Cu	138	227b	265b	2225.3
50 Cu	175	273b	314b	2225.3

†MSE_B - Mean squares error (Error B) from the sub plot.

#Figure 2A.

a,b Means in the same column with a different letter are significantly different (P<0.01) using linear contrast.

Appendix Table C2: Least square means of calf liver Cu based on a linear contrast on days 92 or 28 relative to day 0†

	Liver Cu (mg kg ⁻¹ DM)			
	Day			
	0	28	92	MSE _B †
Number	37	36	37	
0 Cu	158	120a	86.6a	1941.9
20 Cu	154	370b	213b	1941.9

†MSE_B - Mean squares error (Error B) from the sub plot.

‡Figure 5A.

a,b Means in the same column with a different letter are significantly different (P<0.01) using linear contrast.

Appendix Table C3: Least square means of calf plasma Zn within cow treatment based on a linear contrast on days 92, 63 or 28 relative to day 0†

	Plasma Zn ($\mu\text{g dL}^{-1}$)				MSE _B ⁺
	Day				
	0	28	63	92	
Number	37	36	37	37	
0 Cu	131	127a	132	109	1584.5
25 Cu	146	213b	156	116	1584.5
50 Cu	145	219b	169	108	1584.5

†MSE_B - Mean squares error (Error B) from the sub plot.

‡Figure 7.

a,b Means in the same column with a different letter are significantly different ($P < 0.01$) using linear contrast.

Appendix Table C4: Least square means of calf plasma Zn within cow treatment†

Cow treatment	Calf treatment	Plasma Zn ($\mu\text{g dL}^{-1}$)			
		Day			
		0	28	63	92
0	0	127	109	122	124
	20	136	145	142	94
25	0	137	193	164	132
	20	155	234	150	100
50	0	163	229	142	106
	20	127	209	198	110

†MSE_B - Mean squares error (Error B) from sub plot was 1584.5.

Appendix Table D1: Analysis of pasture mineral† consumed by cattle

Constituent		Concentration per kg	
Ca	g	Act.	160
P	g	Act.	160
Na	g	Act.	4.0
Fl	mg	Max.	3000
Zn	mg	Act.	1500
Fe	mg	Act.	1000
Mn	mg	Act.	640
I	mg	Act.	25
Co	mg	Act.	14
Vitamin A	IU	Min.	151,800
Vitamin D ₃	IU	Min.	151,180
Vitamin E	IU	Min.	500

†Co-op 1:1 Sheep mineral (Co-op Feeds).
Guaranteed analysis by the manufacturer.

Appendix Table E1: Raw data of concentration of nutrients in forage and water consumed by cows and calves during the grazing trial

Abbreviations:

Month refers to sampling day: Jun - day 0
 Jly - day 28
 Aug - day 63
 Sep - day 92

Plant refers to speceis: tim - timothy
 bro - brome
 Kty - kentucky
 clo - clover
 H₂O - water

DMPCT - dry matter % (all forage analyses expressed on a 100% DM basis)
 Cu - copper mg kg⁻¹
 Mo - molybdenum mg kg⁻¹
 Fe - iron mg kg⁻¹
 Zn - zinc mg kg⁻¹
 Mn - manganese mg kg⁻¹
 Ca - calcium g kg⁻¹
 P - phosphorous g kg⁻¹
 Mg - magnesium g kg⁻¹
 S - total S forages: g kg⁻¹; water: mg SO₄² L⁻¹
 ADF - acid detergent fibre %
 CP - crude protein %

OBS	MONTH	PLANT	LOCATION	DMPCT	CU	MO	FE	ZN	MN	CA	P	MG	S	ADF	CP	SOURCE
1	JUN	TIM	NEP	94	5.94	4.46	87.23	24.64	35.24	3.7	1.7	1.6	1.54	31.4	12.5	FEEDS
2	JUN	BRO	NWP	96	5.67	4.39	88.37	26.17	36.97	3.4	2.0	1.7	1.52	28.3	13.9	FEEDS
3	JUN	KTY	NEP	95	4.97	3.22	90.00	26.86	39.91	3.9	3.6	2.6	1.54	29.3	14.3	FEEDS
4	JLY	TIM	NEP	94	4.85	3.91	72.86	23.42	31.75	2.3	1.8	1.4	1.44	33.7	8.3	FEEDS
5	JLY	TIM	SWP	94	4.31	4.07	73.72	20.64	38.07	3.5	1.6	1.5	1.43	32.4	8.8	FEEDS
6	JLY	TIX	NEP	94	4.44	4.09	70.98	21.98	36.67	2.5	1.5	1.3	1.47	32.0	7.9	FEEDS
7	JLY	TIW	NWP	94	3.13	2.48	73.95	19.20	32.52	2.9	1.6	1.1	1.42	33.1	8.4	FEEDS
8	JLY	BRO	NEP	96	5.44	4.65	70.68	20.16	31.94	1.8	2.0	1.3	1.44	36.0	12.8	FEEDS
9	JLY	KBR	NWP	94	4.53	2.81	75.96	24.66	36.39	2.9	1.8	1.3	1.54	34.7	8.4	FEEDS
10	AUG	TIM	NEP	96	2.44	4.75	65.42	13.48	24.08	1.9	1.4	1.3	1.19	37.1	7.5	FEEDS
11	AUG	TIM	SWP	96	2.32	5.92	68.29	15.51	27.64	3.4	1.2	1.2	1.23	36.1	7.5	FEEDS
12	AUG	BRO	NWP	95	3.16	5.76	66.08	13.65	26.63	2.3	1.7	1.7	1.34	36.0	7.3	FEEDS
13	SEP	TIM	SWP	95	2.36	2.90	65.69	14.54	26.55	3.2	1.4	2.1	1.36	37.7	7.8	FEEDS
14	SEP	TBR	NEP	96	1.38	2.27	63.73	11.71	24.94	3.6	1.5	1.2	1.23	36.4	7.4	FEEDS
15	SEP	TBR	NWP	96	1.06	2.24	66.22	13.20	21.03	3.4	1.7	1.3	1.26	39.0	6.8	FEEDS
16	JLY	CLO	NWP	95	7.96	7.74	189.99	29.28	26.78	15.2	3.6	3.5	2.13	28.7	19.1	FEEDS
17	AUG	CLO	SWP	95	4.87	5.80	140.39	24.90	35.08	12.6	2.6	3.8	1.50	29.9	16.4	FEEDS
18	AUG	CLO	SWP	95	4.87	5.80	140.39	24.90	35.08	12.6	2.6	3.8	1.50	29.9	16.4	FEEDS
19	JUN	H2O	SPR	.	.	.	.	.	.	.	.	.	938.00	.	.	FEEDS
20	JUN	H2O	RES	.	.	.	.	.	.	.	.	.	994.00	.	.	FEEDS
21	JLY	H2O	SPR	.	.	.	.	.	.	.	.	.	928.00	.	.	FEEDS
22	JLY	H2O	RES	.	.	.	.	.	.	.	.	.	919.00	.	.	FEEDS
23	AUG	H2O	SPR	.	.	.	.	.	.	.	.	.	947.00	.	.	FEEDS
24	AUG	H2O	RES	.	.	.	.	.	.	.	.	.	938.00	.	.	FEEDS
25	SEP	H2O	RES	.	.	.	.	.	.	.	.	.	938.00	.	.	FEEDS

Appendix Table E2: Raw data of weight, plasma and liver parameters collected from cows and calves over the 92 day grazing trial

Abbreviations:

Month refers to sampling day: Jun - day 0
 Jly - day 28
 Aug - day 63
 Sep - day 92

Sex calf: B - bull calf
 H - heifer calf

Trt cow: 1 - 0 Cu
 2 - 25 Cu
 3 - 50 Cu

Trt calf: A - 0 Cu
 B - 20 Cu

Wt weight (lb.)
 KGWT weight (kg)
 BCUP plasma Cu $\mu\text{g dL}^{-1}$
 BCP plasma CpOx $\Delta\text{A min}^{-1} \text{L}^{-1}$
 BMOL plasma Mo $\mu\text{g dL}^{-1}$
 BFE plasma Fe $\mu\text{g dL}^{-1}$
 BZN plasma Zn $\mu\text{g dL}^{-1}$
 LCUP liver Cu $\text{mg kg}^{-1} \text{DM}$
 LMOL liver Mo $\text{mg kg}^{-1} \text{DM}$
 LFE liver Fe $\text{mg kg}^{-1} \text{DM}$
 LZN liver Zn $\text{mg kg}^{-1} \text{DM}$

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWWT	MAYCAFWT	MONTH	COWWT	CALFWT	KGCOWWT	KGCALFWT
1	5C	24U	B	1	A	1A	1580	310	JUNE	1500	350	681.818	159.091
2	5C	24U	B	1	A	1A	1580	310	JULY	1530	440	695.455	200.000
3	5C	24U	B	1	A	1A	1580	310	AUGU	1505	545	684.091	247.727
4	5C	24U	B	1	A	1A	1580	310	SEPT	1535	640	697.727	290.809
5	6C	29U	B	1	A	1A	1345	170	JUNE	1268	235	576.364	106.818
6	6C	29U	B	1	A	1A	1345	170	JULY	1235	295	561.364	134.091
7	6C	29U	B	1	A	1A	1345	170	AUGU	1245	395	565.909	179.545
8	6C	29U	B	1	A	1A	1345	170	SEPT	1300	475	590.909	215.909
9	12C	28U	B	1	A	1A	1430	190	JUNE	1403	218	637.727	99.091
10	12C	28U	B	1	A	1A	1430	190	JULY	1375	265	625.000	120.455
11	12C	28U	B	1	A	1A	1430	190	AUGU	1310	330	595.455	150.000
12	12C	28U	B	1	A	1A	1430	190	SEPT	1378	395	626.364	179.545
13	56C	13U	H	1	A	1A	1250	230	JUNE	1213	273	551.364	124.091
14	56C	13U	H	1	A	1A	1250	230	JULY	1220	330	554.545	150.000
15	56C	13U	H	1	A	1A	1250	230	AUGU	1225	405	556.818	184.091
16	56C	13U	H	1	A	1A	1250	230	SEPT	1233	465	560.455	211.364
17	74C	34U	H	1	A	1A	1470	175	JUNE	1448	203	658.182	92.273
18	74C	34U	H	1	A	1A	1470	175	JULY	1420	280	645.455	127.273
19	74C	34U	H	1	A	1A	1470	175	AUGU	1380	355	627.273	161.364
20	74C	34U	H	1	A	1A	1470	175	SEPT	1405	438	638.636	199.091
21	11C	2U	H	1	A	1A	1435	270	JUNE	1425	315	647.727	143.182
22	11C	2U	H	1	A	1A	1435	270	JULY	1440	375	654.545	170.455
23	11C	2U	H	1	A	1A	1435	270	AUGU	1395	465	634.091	211.364
24	11C	2U	H	1	A	1A	1435	270	SEPT	1420	425	645.455	193.182
25	33C	41U	B	1	B	1B	1630	190	JUNE	1548	227	703.636	103.182
26	33C	41U	B	1	B	1B	1630	190	JULY	1370	300	622.727	136.364
27	33C	41U	B	1	B	1B	1630	190	AUGU	1535	400	697.727	181.818
28	33C	41U	B	1	B	1B	1630	190	SEPT	1570	495	713.636	225.000
29	16C	17U	B	1	B	1B	1430	225	JUNE	1330	278	604.545	126.364
30	16C	17U	B	1	B	1B	1430	225	JULY	1330	340	604.545	154.545
31	16C	17U	B	1	B	1B	1430	225	AUGU	1325	420	602.273	190.909
32	16C	17U	B	1	B	1B	1430	225	SEPT	1350	500	613.636	227.273
33	17C	9U	B	1	B	1B	1415	180	JUNE	1110	200	504.545	90.909
34	17C	9U	B	1	B	1B	1415	180	JULY	1115	260	506.818	118.182
35	17C	9U	B	1	B	1B	1415	180	AUGU	1265	340	575.000	154.545
36	17C	9U	B	1	B	1B	1415	180	SEPT	1255	413	570.455	187.727
37	2P	10U	B	1	B	1B	1280	165	JUNE	1245	187	565.909	85.000
38	2P	10U	B	1	B	1B	1280	165	JULY	1245	225	565.909	102.273
39	2P	10U	B	1	B	1B	1280	165	AUGU	1315	280	597.727	127.273
40	2P	10U	B	1	B	1B	1280	165	SEPT	1318	305	599.091	138.636
41	32C	30U	H	1	B	1B	1415	225	JUNE	1393	285	633.182	129.545
42	32C	30U	H	1	B	1B	1415	225	JULY	1390	345	631.818	156.818
43	32C	30U	H	1	B	1B	1415	225	AUGU	1335	435	606.818	197.727

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWWT	MAYCAFWT	MONTH	COWWT	CALFWT	KGCOWWT	KGCALFWT
44	32C	30U	H	1	B	1B	1415	225	SEPT	1393	505	633.182	229.545
45	72C	15U	H	1	B	1B	1380	205	JUNE	1395	243	634.081	110.455
46	72C	15U	H	1	B	1B	1380	205	JULY	1375	300	625.000	136.864
47	72C	15U	H	1	B	1B	1380	205	AUGU	1375	390	625.000	177.273
48	72C	15U	H	1	B	1B	1380	205	SEPT	1440	458	654.545	208.182
49	29C	36U	B	2	A	2A	1610	200	JUNE	1568	260	712.727	118.182
50	29C	36U	B	2	A	2A	1610	200	JULY	1555	340	706.818	154.545
51	29C	36U	B	2	A	2A	1610	200	AUGU	1600	440	727.273	200.000
52	29C	36U	B	2	A	2A	1610	200	SEPT	1613	533	733.182	242.273
53	35C	39U	B	2	A	2A	1435	210	JUNE	1378	255	626.364	115.909
54	35C	39U	B	2	A	2A	1435	210	JULY	1310	305	595.455	138.636
55	35C	39U	B	2	A	2A	1435	210	AUGU	1385	390	629.545	177.273
56	35C	39U	B	2	A	2A	1435	210	SEPT	1353	468	615.000	212.727
57	18C	12U	B	2	A	2A	1190	190	JUNE	1160	220	527.273	100.000
58	18C	12U	B	2	A	2A	1190	190	JULY	1180	375	536.364	170.455
59	18C	12U	B	2	A	2A	1190	190	AUGU	1155	460	525.000	209.091
60	18C	12U	B	2	A	2A	1190	190	SEPT	1200	540	545.455	245.455
61	10C	25U	H	2	A	2A	1320	260	JUNE	1313	300	596.818	136.364
62	10C	25U	H	2	A	2A	1320	260	JULY	1265	275	575.000	125.000
63	10C	25U	H	2	A	2A	1320	260	AUGU	1250	345	568.182	156.818
64	10C	25U	H	2	A	2A	1320	260	SEPT	1314	403	597.273	183.182
65	19C	19U	H	2	A	2A	1285	215	JUNE	1220	245	554.545	111.364
66	19C	19U	H	2	A	2A	1285	215	JULY	1305	310	593.182	140.909
67	19C	19U	H	2	A	2A	1285	215	AUGU	1260	400	572.727	181.818
68	19C	19U	H	2	A	2A	1285	215	SEPT	1315	465	597.727	211.364
69	25C	23U	H	2	A	2A	1605	200	JUNE	1590	248	722.727	112.727
70	25C	23U	H	2	A	2A	1605	200	JULY	1560	305	709.091	138.636
71	25C	23U	H	2	A	2A	1605	200	AUGU	1570	365	713.636	165.909
72	25C	23U	H	2	A	2A	1605	200	SEPT	1575	425	715.909	193.182
73	14C	22U	B	2	B	2B	1605	150	JUNE	1467	243	666.818	110.455
74	14C	22U	B	2	B	2B	1605	150	JULY	1490	295	677.273	134.091
75	14C	22U	B	2	B	2B	1605	150	AUGU	1460	370	663.636	168.182
76	14C	22U	B	2	B	2B	1605	150	SEPT	1518	425	690.000	193.182
77	66C	18U	B	2	B	2B	1435	180	JUNE	1425	220	647.727	100.000
78	66C	18U	B	2	B	2B	1435	180	JULY	1405	385	638.636	175.000
79	66C	18U	B	2	B	2B	1435	180	AUGU	1375	365	625.000	165.909
80	66C	18U	B	2	B	2B	1435	180	SEPT	1448	440	658.182	200.000
81	19S	20U	B	2	B	2B	1130	205	JUNE	1090	240	495.455	109.091
82	19S	20U	B	2	B	2B	1130	205	JULY	1115	295	506.818	134.091
83	19S	20U	B	2	B	2B	1130	205	AUGU	1090	385	495.455	175.000
84	19S	20U	B	2	B	2B	1130	205	SEPT	1118	463	508.182	210.455
85	60C	11U	B	2	B	2B	1335	235	JUNE	1303	288	592.273	130.909
86	60C	11U	B	2	B	2B	1335	235	JULY	1295	345	588.636	156.818
87	60C	11U	B	2	B	2B	1335	235	AUGU	1310	425	595.455	193.182

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWWT	MAYCAFWT	MONTH	COWWT	CALFWT	KGCOWWT	KGCALFWT
88	60C	11U	B	2	B	2B	1335	235	SEPT	1353	505	615.000	229.545
89	63C	27U	H	2	B	2B	1310	175	JUNE	1263	225	574.091	102.273
90	63C	27U	H	2	B	2B	1310	175	JULY	1235	295	561.364	134.091
91	63C	27U	H	2	B	2B	1310	175	AUGU	1200	375	545.455	170.455
92	63C	27U	H	2	B	2B	1310	175	SEPT	1245	450	565.909	204.545
93	6R	3U	H	2	B	2B	1255	210	JUNE	1230	238	559.091	108.182
94	6R	3U	H	2	B	2B	1255	210	JULY	1200	290	545.455	131.818
95	6R	3U	H	2	B	2B	1255	210	AUGU	1230	355	559.091	161.364
96	6R	3U	H	2	B	2B	1255	210	SEPT	1265	428	575.000	194.545
97	65C	21U	B	3	A	3A	1585	205	JUNE	1550	215	704.545	97.727
98	65C	21U	B	3	A	3A	1585	205	JULY	.	.	.	.
99	65C	21U	B	3	A	3A	1585	205	AUGU	1470	355	668.182	161.364
100	65C	21U	B	3	A	3A	1585	205	SEPT	1503	438	683.182	199.091
101	2N	5U	B	3	A	3A	1430	300	JUNE	1375	358	625.000	162.727
102	2N	5U	B	3	A	3A	1430	300	JULY	1405	410	638.636	186.364
103	2N	5U	B	3	A	3A	1430	300	AUGU	1380	510	627.273	231.818
104	2N	5U	B	3	A	3A	1430	300	SEPT	1430	580	650.000	263.636
105	40C	45U	B	3	A	3A	1120	100	JUNE	1093	130	496.818	59.091
106	40C	45U	B	3	A	3A	1120	100	JULY	1065	165	484.091	75.000
107	40C	45U	B	3	A	3A	1120	100	AUGU	1055	245	479.545	111.364
108	40C	45U	B	3	A	3A	1120	100	SEPT	1095	290	497.727	131.818
109	64C	14U	B	3	A	3A	1590	270	JUNE	1543	203	701.364	92.273
110	64C	14U	B	3	A	3A	1590	270	JULY	1520	265	690.909	120.455
111	64C	14U	B	3	A	3A	1590	270	AUGU	1515	345	688.636	156.818
112	64C	14U	B	3	A	3A	590	270	SEPT	1543	408	701.364	185.455
113	21C	38U	H	3	A	3A	1425	185	JUNE	1365	230	620.455	104.545
114	21C	38U	H	3	A	3A	1425	185	JULY	1410	310	640.909	140.909
115	21C	38U	H	3	A	3A	1425	185	AUGU	1330	410	604.545	186.364
116	21C	38U	H	3	A	3A	1425	185	SEPT	1358	478	617.273	217.273
117	93C	40U	H	3	A	3A	1250	155	JUNE	1260	203	572.727	92.273
118	93C	40U	H	3	A	3A	1250	155	JULY	1235	255	561.364	115.909
119	93C	40U	H	3	A	3A	1250	155	AUGU	1250	320	568.182	145.455
120	93C	40U	H	3	A	3A	1250	155	SEPT	1285	413	584.091	187.727
121	67C	6U	H	3	A	3A	1560	190	JUNE	1525	240	693.182	109.091
122	67C	6U	H	3	A	3A	1560	190	JULY	1505	275	684.091	125.000
123	67C	6U	H	3	A	3A	1560	190	AUGU	1525	345	693.182	156.818
124	67C	6U	H	3	A	3A	1560	190	SEPT	1595	408	725.000	185.455
125	13C	4U	B	3	B	3B	1640	290	JUNE	1595	343	725.000	155.909
126	13C	4U	B	3	B	3B	1640	290	JULY	1580	395	718.182	179.545
127	13C	4U	B	3	B	3B	1640	290	AUGU	1570	500	713.636	227.273
128	13C	4U	B	3	B	3B	1640	290	SEPT	1610	580	731.818	263.636

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWWT	MAYCAFWT	MONTH	COWWT	CALFWT	KGCOWWT	KGCALFWT
129	22C	37U	B	3	B	3B	1535	200	JUNE	1490	250	677.273	113.636
130	22C	37U	B	3	B	3B	1535	200	JULY	1470	315	668.182	143.182
131	22C	37U	B	3	B	3B	1535	200	AUGU	1490	420	677.273	190.909
132	22C	37U	B	3	B	3B	1535	200	SEPT	1465	495	665.909	225.000
133	6S	1U	B	3	B	3B	1000	270	JUNE	1023	343	465.000	155.909
134	6S	1U	B	3	B	3B	1000	270	JULY	1030	410	468.182	186.364
135	6S	1U	B	3	B	3B	1000	270	AUGU	1065	505	484.091	229.545
136	6S	1U	B	3	B	3B	1000	270	SEPT	1078	585	490.000	265.909
137	52C	8U	B	3	B	3B	1335	200	JUNE	1293	235	587.727	106.818
138	52C	8U	B	3	B	3B	1335	200	JULY	1210	300	550.000	136.364
139	52C	8U	B	3	B	3B	1335	200	AUGU	1240	370	563.636	168.182
140	52C	8U	B	3	B	3B	1335	200	SEPT	1298	428	590.000	194.545
141	23C	7U	B	3	B	3B	1150	255	JUNE	1130	303	513.636	137.727
142	23C	7U	B	3	B	3B	1150	255	JULY	1105	372	502.273	169.091
143	23C	7U	B	3	B	3B	1150	255	AUGU	1110	450	504.545	204.545
144	23C	7U	B	3	B	3B	1150	255	SEPT	1183	543	537.727	246.818
145	75C	35U	H	3	B	3B	1690	165	JUNE	1688	203	767.273	92.273
146	75C	35U	H	3	B	3B	1690	165	JULY	1655	255	752.273	115.909
147	75C	35U	H	3	B	3B	1690	165	AUGU	1630	340	740.909	154.545
148	75C	35U	H	3	B	3B	1690	165	SEPT	1695	395	770.455	179.545

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBC	MAYCAFBC	SOURCE	MONTH	COWBCUP	CALFBCUP
			B	1	A	1A	1.04	0.74	BLOOD	JUNE	98	63
1	5C	24U	B	1	A	1A	1.04	0.74	BLOOD	JULY	146	81
2	5C	24U	B	1	A	1A	1.04	0.74	BLOOD	AUGU	96	66
3	5C	24U	B	1	A	1A	1.04	0.74	BLOOD	SEPT	98	69
4	5C	24U	B	1	A	1A	1.45	0.84	BLOOD	JUNE	130	81
5	6C	29U	B	1	A	1A	1.45	0.84	BLOOD	JULY	144	127
6	6C	29U	B	1	A	1A	1.45	0.84	BLOOD	AUGU	99	62
7	6C	29U	B	1	A	1A	1.45	0.84	BLOOD	SEPT	105	66
8	6C	29U	B	1	A	1A	1.09	0.81	BLOOD	JUNE	98	104
9	12C	28U	B	1	A	1A	1.09	0.81	BLOOD	JULY	143	91
10	12C	28U	B	1	A	1A	1.09	0.81	BLOOD	AUGU	111	84
11	12C	28U	B	1	A	1A	1.09	0.81	BLOOD	SEPT	96	68
12	12C	28U	B	1	A	1A	1.02	0.74	BLOOD	JUNE	122	73
13	56C	13U	H	1	A	1A	1.02	0.74	BLOOD	JULY	112	105
14	56C	13U	H	1	A	1A	1.02	0.74	BLOOD	AUGU	90	93
15	56C	13U	H	1	A	1A	1.02	0.74	BLOOD	SEPT	95	77
16	56C	13U	H	1	A	1A	1.06	0.70	BLOOD	JUNE	101	86
17	74C	34U	H	1	A	1A	1.06	0.70	BLOOD	JULY	121	102
18	74C	34U	H	1	A	1A	1.06	0.70	BLOOD	AUGU	102	91
19	74C	34U	H	1	A	1A	1.06	0.70	BLOOD	SEPT	95	76
20	74C	34U	H	1	A	1A	0.92	0.98	BLOOD	JUNE	77	101
21	11C	2U	H	1	A	1A	0.92	0.98	BLOOD	JULY	123	111
22	11C	2U	H	1	A	1A	0.92	0.98	BLOOD	AUGU	89	78
23	11C	2U	H	1	A	1A	0.92	0.98	BLOOD	SEPT	84	82
24	11C	2U	H	1	A	1A	1.14	1.13	BLOOD	JUNE	112	85
25	33C	41U	B	2	B	2B	1.14	1.13	BLOOD	JULY	157	89
26	33C	41U	B	2	B	2B	1.14	1.13	BLOOD	AUGU	104	79
27	33C	41U	B	2	B	2B	1.14	1.13	BLOOD	SEPT	113	65
28	33C	41U	B	2	B	2B	1.11	0.84	BLOOD	JUNE	117	76
29	16C	17U	B	1	B	1B	1.11	0.84	BLOOD	JULY	125	105
30	16C	17U	B	1	B	1B	1.11	0.84	BLOOD	AUGU	112	68
31	16C	17U	B	1	B	1B	1.11	0.84	BLOOD	SEPT	134	69
32	16C	17U	B	1	B	1B	1.04	0.82	BLOOD	JUNE	79	86
33	17C	9U	B	1	B	1B	1.04	0.82	BLOOD	JULY	114	110
34	17C	9U	B	1	B	1B	1.04	0.82	BLOOD	AUGU	96	94
35	17C	9U	B	1	B	1B	1.04	0.82	BLOOD	SEPT	79	80
36	17C	9U	B	1	B	1B	1.03	0.80	BLOOD	JUNE	95	66
37	2P	10U	B	1	B	1B	1.03	0.80	BLOOD	JULY	146	131
38	2P	10U	B	1	B	1B	1.03	0.80	BLOOD	AUGU	110	88
39	2P	10U	B	1	B	1B	1.03	0.80	BLOOD	SEPT	93	94
40	2P	10U	B	1	B	1B	1.04	0.81	BLOOD	JUNE	94	78
41	32C	30U	H	1	B	1B	1.04	0.81	BLOOD	JULY	96	126
42	32C	30U	H	1	B	1B	1.04	0.81	BLOOD	AUGU	95	88
43	32C	30U	H	1	B	1B	1.04	0.81	BLOOD	SEPT	81	84
44	32C	30U	H	1	B	1B	0.94	0.73	BLOOD	JUNE	81	66
45	72C	15U	H	1	B	1B	0.94	0.73	BLOOD	JULY	109	81
46	72C	15U	H	1	B	1B	0.94	0.73	BLOOD	AUGU	86	68
47	72C	15U	H	1	B	1B						

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBC	MAYCAFBC	SOURCE	MONTH	COWBCUP	CALFBCUP
48	72C	15U	H	1	B	1B	0.84	0.73	BLOOD	SEPT	74	77
49	29C	36U	B	2	A	2A	1.07	0.88	BLOOD	JUNE	104	126
50	29C	36U	B	2	A	2A	1.07	0.88	BLOOD	JULY	123	102
51	29C	36U	B	2	A	2A	1.07	0.88	BLOOD	AUGU	111	94
52	29C	36U	B	2	A	2A	1.07	0.88	BLOOD	SEPT	100	87
53	35C	39U	B	2	A	2A	1.14	0.73	BLOOD	JUNE	163	65
54	35C	39U	B	2	A	2A	1.14	0.73	BLOOD	JULY	139	134
55	35C	39U	B	2	A	2A	1.14	0.73	BLOOD	AUGU	100	76
56	35C	39U	B	2	A	2A	1.14	0.73	BLOOD	SEPT	94	78
57	18C	12U	B	2	A	2A	0.99	0.88	BLOOD	JUNE	82	73
58	18C	12U	B	2	A	2A	0.99	0.88	BLOOD	JULY	82	114
59	18C	12U	B	2	A	2A	0.99	0.88	BLOOD	AUGU	84	67
60	18C	12U	B	2	A	2A	0.99	0.88	BLOOD	SEPT	79	69
61	10C	25U	H	2	A	2A	0.86	0.79	BLOOD	JUNE	85	59
62	10C	25U	H	2	A	2A	0.86	0.79	BLOOD	JULY	76	119
63	10C	25U	H	2	A	2A	0.86	0.79	BLOOD	AUGU	70	71
64	10C	25U	H	2	A	2A	0.86	0.79	BLOOD	SEPT	76	64
65	19C	19U	H	2	A	2A	0.95	0.70	BLOOD	JUNE	85	68
66	19C	19U	H	2	A	2A	0.95	0.70	BLOOD	JULY	106	125
67	19C	19U	H	2	A	2A	0.95	0.70	BLOOD	AUGU	88	69
68	19C	19U	H	2	A	2A	0.95	0.70	BLOOD	SEPT	75	81
69	25C	23U	H	2	A	2A	0.92	0.68	BLOOD	JUNE	79	70
70	25C	23U	H	2	A	2A	0.92	0.68	BLOOD	JULY	101	122
71	25C	23U	H	2	A	2A	0.92	0.68	BLOOD	AUGU	81	90
72	25C	23U	H	2	A	2A	0.92	0.68	BLOOD	SEPT	82	74
73	14C	24U	B	2	B	2B	1.24	0.88	BLOOD	JUNE	142	82
74	14C	24U	B	2	B	2B	1.24	0.88	BLOOD	JULY	123	118
75	14C	24U	B	2	B	2B	1.24	0.88	BLOOD	AUGU	102	88
76	14C	24U	B	2	B	2B	1.24	0.88	BLOOD	SEPT	122	74
77	66C	18U	B	2	B	2B	0.89	0.67	BLOOD	JUNE	90	111
78	66C	18U	B	2	B	2B	0.89	0.67	BLOOD	JULY	97	116
79	66C	18U	B	2	B	2B	0.89	0.67	BLOOD	AUGU	82	79
80	66C	18U	B	2	B	2B	0.89	0.67	BLOOD	SEPT	83	85
81	19S	20U	B	2	B	2B	1.07	0.70	BLOOD	JUNE	92	83
82	19S	20U	B	2	B	2B	1.07	0.70	BLOOD	JULY	176	109
83	19S	20U	B	2	B	2B	1.07	0.70	BLOOD	AUGU	107	71
84	19S	20U	B	2	B	2B	1.07	0.70	BLOOD	SEPT	105	79
85	60C	11U	B	2	B	2B	1.18	0.89	BLOOD	JUNE	197	80
86	60C	11U	B	2	B	2B	1.18	0.89	BLOOD	JULY	120	124
87	60C	11U	B	2	B	2B	1.18	0.89	BLOOD	AUGU	100	86
88	60C	11U	B	2	B	2B	1.18	0.89	BLOOD	SEPT	113	98
89	63C	27U	H	2	B	2B	1.03	0.98	BLOOD	JUNE	221	77
90	63C	27U	H	2	B	2B	1.03	0.98	BLOOD	JULY	108	108
91	63C	27U	H	2	B	2B	1.03	0.98	BLOOD	AUGU	99	65
92	63C	27U	H	2	B	2B	1.03	0.98	BLOOD	SEPT	88	76

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBC	MAYCAFCB	SOURCE	MONTH	COWBCUP	CALFBCUP
93	6R	3U	H	2	B	2B	1.03	1.09	BLOOD	JUNE	92	87
94	6R	3U	H	2	B	2B	1.03	1.09	BLOOD	JULY	107	130
95	6R	3U	H	2	B	2B	1.03	1.09	BLOOD	AUGU	97	70
96	6R	3U	H	2	B	2B	1.03	1.09	BLOOD	SEPT	96	83
97	65C	21U	B	3	A	3A	1.10	1.03	BLOOD	JUNE	98	109
98	65C	21U	B	3	A	3A	1.10	1.03	BLOOD	JULY	.	.
99	65C	21U	B	3	A	3A	1.10	1.03	BLOOD	AUGU	93	70
100	65C	21U	B	3	A	3A	1.10	1.03	BLOOD	SEPT	78	67
101	2N	5U	B	3	A	3A	0.98	0.64	BLOOD	JUNE	88	77
102	2N	5U	B	3	A	3A	0.98	0.64	BLOOD	JULY	139	115
103	2N	5U	B	3	A	3A	0.98	0.64	BLOOD	AUGU	82	68
104	2N	5U	B	3	A	3A	0.98	0.64	BLOOD	SEPT	87	69
105	40C	45U	B	3	A	3A	1.12	1.04	BLOOD	JUNE	100	108
106	40C	45U	B	3	A	3A	1.12	1.04	BLOOD	JULY	127	124
107	40C	45U	B	3	A	3A	1.12	1.04	BLOOD	AUGU	118	67
108	40C	45U	B	3	A	3A	1.12	1.04	BLOOD	SEPT	109	90
109	64C	14U	B	3	A	3A	1.04	0.97	BLOOD	JUNE	86	61
110	64C	14U	B	3	A	3A	1.04	0.97	BLOOD	JULY	104	79
111	64C	14U	B	3	A	3A	1.04	0.97	BLOOD	AUGU	91	76
112	64C	14U	B	3	A	3A	1.04	0.97	BLOOD	SEPT	95	91
113	21C	38U	H	3	A	3A	1.18	0.74	BLOOD	JUNE	95	85
114	21C	38U	H	3	A	3A	1.18	0.74	BLOOD	JULY	108	69
115	21C	38U	H	3	A	3A	1.18	0.74	BLOOD	AUGU	82	63
116	21C	38U	H	3	A	3A	1.18	0.74	BLOOD	SEPT	78	70
117	93C	40U	H	3	A	3A	0.95	1.10	BLOOD	JUNE	92	126
118	93C	40U	H	3	A	3A	0.95	1.10	BLOOD	JULY	102	197
119	93C	40U	H	3	A	3A	0.95	1.10	BLOOD	AUGU	91	97
120	93C	40U	H	3	A	3A	0.95	1.10	BLOOD	SEPT	91	90
121	67C	6U	H	3	A	3A	1.17	0.85	BLOOD	JUNE	98	64
122	67C	6U	H	3	A	3A	1.17	0.85	BLOOD	JULY	112	98
123	67C	6U	H	3	A	3A	1.17	0.85	BLOOD	AUGU	97	73
124	67C	6U	H	3	A	3A	1.17	0.85	BLOOD	SEPT	81	78
125	13C	4U	B	3	B	3B	0.97	0.88	BLOOD	JUNE	88	75
126	13C	4U	B	3	B	3B	0.97	0.88	BLOOD	JULY	96	120
127	13C	4U	B	3	B	3B	0.97	0.88	BLOOD	AUGU	83	66
128	13C	4U	B	3	B	3B	0.97	0.88	BLOOD	SEPT	88	69
129	22C	37U	B	3	B	3B	0.91	0.87	BLOOD	JUNE	91	77
130	22C	37U	B	3	B	3B	0.91	0.87	BLOOD	JULY	97	62
131	22C	37U	B	3	B	3B	0.91	0.87	BLOOD	AUGU	85	70
132	22C	37U	B	3	B	3B	0.91	0.87	BLOOD	SEPT	83	64
133	6S	1U	B	3	B	3B	1.03	1.06	BLOOD	JUNE	78	97
134	6S	1U	B	3	B	3B	1.03	1.06	BLOOD	JULY	89	170
135	6S	1U	B	3	B	3B	1.03	1.06	BLOOD	AUGU	99	92
136	6S	1U	B	3	B	3B	1.03	1.06	BLOOD	SEPT	99	81
137	52C	8U	B	3	B	3B	1.05	1.16	BLOOD	JUNE	99	83
138	52C	8U	B	3	B	3B	1.05	1.16	BLOOD	JULY	134	97

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBC	MAYCAFBC	SOURCE	MONTH	COWBCUP	CALFBCUP
									BLOOD	AUGU	92	91
							1.05	1.16	BLOOD	SEPT	94	79
139	52C	8U	B	3	B	3B	1.05	1.16	BLOOD	JUNE	71	70
140	52C	8U	B	3	B	3B	1.07	0.71	BLOOD	JULY	120	91
141	23C	7U	B	3	B	3B	1.07	0.71	BLOOD	AUGU	87	73
142	23C	7U	B	3	B	3B	1.07	0.71	BLOOD	SEPT	74	68
143	23C	7U	B	3	B	3B	1.07	0.71	BLOOD	JUNE	156	72
144	23C	7U	B	3	B	3B	1.00	0.88	BLOOD	JULY	96	103
145	75C	35U	H	3	B	3B	1.00	0.88	BLOOD	AUGU	105	89
146	75C	35U	H	3	B	3B	1.00	0.88	BLOOD	SEPT	84	90
147	75C	35U	H	3	B	3B	1.00	0.88	BLOOD			
148	75C	35U	H	3	B	3B						

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWBCP	CALFBCP
1	5C	24U	B	1	A	1A	BLOOD	JUNE	14	12
2	5C	24U	B	1	A	1A	BLOOD	JULY	29	13
3	5C	24U	B	1	A	1A	BLOOD	AUGU	22	16
4	5C	24U	B	1	A	1A	BLOOD	SEPT	25	18
5	6C	29U	B	1	A	1A	BLOOD	JUNE	23	8
6	6C	29U	B	1	A	1A	BLOOD	JULY	24	15
7	6C	29U	B	1	A	1A	BLOOD	AUGU	30	14
8	6C	29U	B	1	A	1A	BLOOD	SEPT	27	18
9	12C	28U	B	1	A	1A	BLOOD	JUNE	33	19
10	12C	28U	B	1	A	1A	BLOOD	JULY	30	20
11	12C	28U	B	1	A	1A	BLOOD	AUGU	23	25
12	12C	28U	B	1	A	1A	BLOOD	SEPT	31	17
13	56C	13U	H	1	A	1A	BLOOD	JUNE	10	15
14	56C	13U	H	1	A	1A	BLOOD	JULY	15	27
15	56C	13U	H	1	A	1A	BLOOD	AUGU	27	32
16	56C	13U	H	1	A	1A	BLOOD	SEPT	24	20
17	74C	34U	H	1	A	1A	BLOOD	JUNE	14	3
18	74C	34U	H	1	A	1A	BLOOD	JULY	17	7
19	74C	34U	H	1	A	1A	BLOOD	AUGU	24	11
20	74C	34U	H	1	A	1A	BLOOD	SEPT	25	16
21	11C	2U	H	1	A	1A	BLOOD	JUNE	10	11
22	11C	2U	H	1	A	1A	BLOOD	JULY	19	18
23	11C	2U	H	1	A	1A	BLOOD	AUGU	26	20
24	11C	2U	H	1	A	1A	BLOOD	SEPT	24	20
25	33C	41U	B	1	B	1B	BLOOD	JUNE	36	13
26	33C	41U	B	1	B	1B	BLOOD	JULY	32	17
27	33C	41U	B	1	B	1B	BLOOD	AUGU	32	22
28	33C	41U	B	1	B	1B	BLOOD	SEPT	34	23
29	16C	17U	B	1	B	1B	BLOOD	JUNE	12	11
30	16C	17U	B	1	B	1B	BLOOD	JULY	10	17
31	16C	17U	B	1	B	1B	BLOOD	AUGU	27	20
32	16C	17U	B	1	B	1B	BLOOD	SEPT	41	20
33	17C	9U	B	1	B	1B	BLOOD	JUNE	13	5
34	17C	9U	B	1	B	1B	BLOOD	JULY	12	18
35	17C	9U	B	1	B	1B	BLOOD	AUGU	27	18
36	17C	9U	B	1	B	1B	BLOOD	SEPT	20	24
37	2P	10U	B	1	B	1B	BLOOD	JUNE	25	16
38	2P	10U	B	1	B	1B	BLOOD	JULY	26	20
39	2P	10U	B	1	B	1B	BLOOD	AUGU	33	23
40	2P	10U	B	1	B	1B	BLOOD	SEPT	24	19
41	32C	30U	H	1	B	1B	BLOOD	JUNE	11	13
42	32C	30U	H	1	B	1B	BLOOD	JULY	11	20

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWBCP	CALFBCP
43	32C	30U	H	1	B	1B	BLOOD	AUGU	30	24
44	32C	30U	H	1	B	1B	BLOOD	SEPT	24	28
45	72C	15U	H	1	B	1B	BLOOD	JUNE	16	5
46	72C	15U	H	1	B	1B	BLOOD	JULY	14	17
47	72C	15U	H	1	B	1B	BLOOD	AUGU	23	21
48	72C	15U	H	1	B	1B	BLOOD	SEPT	23	27
49	29C	36U	B	2	A	2A	BLOOD	JUNE	14	5
50	29C	36U	B	2	A	2A	BLOOD	JULY	21	14
51	29C	36U	B	2	A	2A	BLOOD	AUGU	29	25
52	29C	36U	B	2	A	2A	BLOOD	SEPT	34	23
53	35C	39U	B	2	A	2A	BLOOD	JUNE	10	14
54	35C	39U	B	2	A	2A	BLOOD	JULY	20	16
55	35C	39U	B	2	A	2A	BLOOD	AUGU	25	20
56	35C	39U	B	2	A	2A	BLOOD	SEPT	27	20
57	18C	12U	B	2	A	2A	BLOOD	JUNE	12	13
58	18C	12U	B	2	A	2A	BLOOD	JULY	18	13
59	18C	12U	B	2	A	2A	BLOOD	AUGU	21	18
60	18C	12U	B	2	A	2A	BLOOD	SEPT	22	21
61	10C	25U	H	2	A	2A	BLOOD	JUNE	8	4
62	10C	25U	H	2	A	2A	BLOOD	JULY	13	16
63	10C	25U	H	2	A	2A	BLOOD	AUGU	19	17
64	10C	25U	H	2	A	2A	BLOOD	SEPT	21	17
65	19C	19U	H	2	A	2A	BLOOD	JUNE	14	16
66	19C	19U	H	2	A	2A	BLOOD	JULY	17	8
67	19C	19U	H	2	A	2A	BLOOD	AUGU	25	16
68	19C	19U	H	2	A	2A	BLOOD	SEPT	19	19
69	25C	23U	H	2	A	2A	BLOOD	JUNE	15	2
70	25C	23U	H	2	A	2A	BLOOD	JULY	14	16
71	25C	23U	H	2	A	2A	BLOOD	AUGU	22	18
72	25C	23U	H	2	A	2A	BLOOD	SEPT	21	19
73	14C	22U	B	2	B	2B	BLOOD	JUNE	35	10
74	14C	22U	B	2	B	2B	BLOOD	JULY	34	14
75	14C	22U	B	2	B	2B	BLOOD	AUGU	32	20
76	14C	22U	B	2	B	2B	BLOOD	SEPT	34	28
77	66C	18U	B	2	B	2B	BLOOD	JUNE	8	2
78	66C	18U	B	2	B	2B	BLOOD	JULY	13	13
79	66C	18U	B	2	B	2B	BLOOD	AUGU	25	22
80	66C	18U	B	2	B	2B	BLOOD	SEPT	21	21
81	19S	20U	B	2	B	2B	BLOOD	JUNE	27	13
82	19S	20U	B	2	B	2B	BLOOD	JULY	22	16
83	19S	20U	B	2	B	2B	BLOOD	AUGU	32	18
84	19S	20U	B	2	B	2B	BLOOD	SEPT	29	19

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWBCP	CALFBCP
85	60C	11U	B	2	B	2B	BLOOD	JUNE	14	6
86	60C	11U	B	2	B	2B	BLOOD	JULY	17	22
87	60C	11U	B	2	B	2B	BLOOD	AUGU	28	26
88	60C	11U	B	2	B	2B	BLOOD	SEPT	24	27
89	63C	27U	H	2	B	2B	BLOOD	JUNE	4	5
90	63C	27U	H	2	B	2B	BLOOD	JULY	10	15
91	63C	27U	H	2	B	2B	BLOOD	AUGU	23	17
92	63C	27U	H	2	B	2B	BLOOD	SEPT	21	17
93	6R	3U	H	2	B	2B	BLOOD	JUNE	13	4
94	6R	3U	H	2	B	2B	BLOOD	JULY	21	15
95	6R	3U	H	2	B	2B	BLOOD	AUGU	31	17
96	6R	3U	H	2	B	2B	BLOOD	SEPT	33	20
97	65C	21U	B	3	A	3A	BLOOD	JUNE	9	17
98	65C	21U	B	3	A	3A	BLOOD	JULY	.	.
99	65C	21U	B	3	A	3A	BLOOD	AUGU	28	19
100	65C	21U	B	3	A	3A	BLOOD	SEPT	25	29
101	2N	5U	B	3	A	3A	BLOOD	JUNE	8	10
102	2N	5U	B	3	A	3A	BLOOD	JULY	13	19
103	2N	5U	B	3	A	3A	BLOOD	AUGU	22	19
104	2N	5U	B	3	A	3A	BLOOD	SEPT	29	27
105	40C	45U	B	3	A	3A	BLOOD	JUNE	20	10
106	40C	45U	B	3	A	3A	BLOOD	JULY	31	15
107	40C	45U	B	3	A	3A	BLOOD	AUGU	36	17
108	40C	45U	B	3	A	3A	BLOOD	SEPT	39	22
109	64C	14U	B	3	A	3A	BLOOD	JUNE	15	6
110	64C	14U	B	3	A	3A	BLOOD	JULY	27	13
111	64C	14U	B	3	A	3A	BLOOD	AUGU	30	22
112	64C	14U	B	3	A	3A	BLOOD	SEPT	29	25
113	21C	38U	H	3	A	3A	BLOOD	JUNE	9	9
114	21C	38U	H	3	A	3A	BLOOD	JULY	17	13
115	21C	38U	H	3	A	3A	BLOOD	AUGU	26	17
116	21C	38U	H	3	A	3A	BLOOD	SEPT	16	17
117	93C	40U	H	3	A	3A	BLOOD	JUNE	6	4
118	93C	40U	H	3	A	3A	BLOOD	JULY	16	24
119	93C	40U	H	3	A	3A	BLOOD	AUGU	20	30
120	93C	40U	H	3	A	3A	BLOOD	SEPT	23	24
121	67C	6U	H	3	A	3A	BLOOD	JUNE	12	6
122	67C	6U	H	3	A	3A	BLOOD	JULY	13	10
123	67C	6U	H	3	A	3A	BLOOD	AUGU	24	19
124	67C	6U	H	3	A	3A	BLOOD	SEPT	22	20
125	13C	4U	B	3	B	3B	BLOOD	JUNE	22	3
126	13C	4U	B	3	B	3B	BLOOD	JULY	27	7

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWBCP	CALFBCP
127	13C	4U	B	3	B	3B	BLOOD	AUGU	34	13
128	13C	4U	B	3	B	3B	BLOOD	SEPT	32	15
129	22C	37U	B	3	B	3B	BLOOD	JUNE	21	1
130	22C	37U	B	3	B	3B	BLOOD	JULY	28	13
131	22C	37U	B	3	B	3B	BLOOD	AUGU	25	18
132	22C	37U	B	3	B	3B	BLOOD	SEPT	29	27
133	6S	1U	B	3	B	3B	BLOOD	JUNE	23	6
134	6S	1U	B	3	B	3B	BLOOD	JULY	22	24
135	6S	1U	B	3	B	3B	BLOOD	AUGU	29	25
136	6S	1U	B	3	B	3B	BLOOD	SEPT	36	26
137	52C	8U	B	3	B	3B	BLOOD	JUNE	12	15
138	52C	8U	B	3	B	3B	BLOOD	JULY	29	20
139	52C	8U	B	3	B	3B	BLOOD	AUGU	27	18
140	52C	8U	B	3	B	3B	BLOOD	SEPT	26	20
141	23C	7U	B	3	B	3B	BLOOD	JUNE	12	15
142	23C	7U	B	3	B	3B	BLOOD	JULY	29	20
143	23C	7U	B	3	B	3B	BLOOD	AUGU	27	18
144	23C	7U	B	3	B	3B	BLOOD	SEPT	26	20
145	75C	35U	H	3	B	3B	BLOOD	JUNE	10	8
146	75C	35U	H	3	B	3B	BLOOD	JULY	15	14
147	75C	35U	H	3	B	3B	BLOOD	AUGU	30	26
148	75C	35U	H	3	B	3B	BLOOD	SEPT	18	33

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBM	MAYCAFBM	SOURCE	MONTH	COWBMOL	CALFBMOL
1	5C	24U	B	1	A	1A	0.30	0.17	BLOOD	JUNE	28	18
2	5C	24U	B	1	A	1A	0.30	0.17	BLOOD	JULY	93	53
3	5C	24U	B	1	A	1A	0.30	0.17	BLOOD	AUGU	42	15
4	5C	24U	B	1	A	1A	0.30	0.17	BLOOD	SEPT	27	29
5	6C	29U	B	1	A	1A	0.56	0.15	BLOOD	JUNE	37	7
6	6C	29U	B	1	A	1A	0.56	0.15	BLOOD	JULY	73	32
7	6C	29U	B	1	A	1A	0.56	0.15	BLOOD	AUGU	67	2
8	6C	29U	B	1	A	1A	0.56	0.15	BLOOD	SEPT	37	8
9	12C	28U	B	1	A	1A	0.32	0.26	BLOOD	JUNE	77	7
10	12C	28U	B	1	A	1A	0.32	0.26	BLOOD	JULY	83	40
11	12C	28U	B	1	A	1A	0.32	0.26	BLOOD	AUGU	21	24
12	12C	28U	B	1	A	1A	0.32	0.26	BLOOD	SEPT	12	4
13	56C	13U	H	1	A	1A	0.72	0.17	BLOOD	JUNE	69	12
14	56C	13U	H	1	A	1A	0.72	0.17	BLOOD	JULY	44	85
15	56C	13U	H	1	A	1A	0.72	0.17	BLOOD	AUGU	21	6
16	56C	13U	H	1	A	1A	0.72	0.17	BLOOD	SEPT	26	1
17	74C	34U	H	1	A	1A	0.24	0.19	BLOOD	JUNE	35	6
18	74C	34U	H	1	A	1A	0.24	0.19	BLOOD	JULY	31	48
19	74C	34U	H	1	A	1A	0.24	0.19	BLOOD	AUGU	27	13
20	74C	34U	H	1	A	1A	0.24	0.19	BLOOD	SEPT	16	10
21	11C	2U	H	1	A	1A	0.41	0.21	BLOOD	JUNE	26	8
22	11C	2U	H	1	A	1A	0.41	0.21	BLOOD	JULY	46	46
23	11C	2U	H	1	A	1A	0.41	0.21	BLOOD	AUGU	88	23
24	11C	2U	H	1	A	1A	0.41	0.21	BLOOD	SEPT	31	18
25	33C	41U	B	1	B	1B	0.42	0.14	BLOOD	JUNE	27	4
26	33C	41U	B	1	B	1B	0.42	0.14	BLOOD	JULY	46	51
27	33C	41U	B	1	B	1B	0.42	0.14	BLOOD	AUGU	9	20
28	33C	41U	B	1	B	1B	0.42	0.14	BLOOD	SEPT	9	27
29	16C	17U	B	1	B	1B	0.56	0.33	BLOOD	JUNE	30	8
30	16C	17U	B	1	B	1B	0.56	0.33	BLOOD	JULY	40	78
31	16C	17U	B	1	B	1B	0.56	0.33	BLOOD	AUGU	18	6
32	16C	17U	B	1	B	1B	0.56	0.33	BLOOD	SEPT	8	6
33	17C	9U	B	1	B	1B	0.68	0.93	BLOOD	JUNE	21	6
34	17C	9U	B	1	B	1B	0.68	0.93	BLOOD	JULY	27	21
35	17C	9U	B	1	B	1B	0.68	0.93	BLOOD	AUGU	29	21
36	17C	9U	B	1	B	1B	0.68	0.93	BLOOD	SEPT	2	6
37	2P	10U	B	1	B	1B	0.49	0.10	BLOOD	JUNE	25	6
38	2P	10U	B	1	B	1B	0.49	0.10	BLOOD	JULY	46	55
39	2P	10U	B	1	B	1B	0.49	0.10	BLOOD	AUGU	75	14
40	2P	10U	B	1	B	1B	0.49	0.10	BLOOD	SEPT	17	4
41	32C	30U	H	1	B	1B	0.27	0.02	BLOOD	JUNE	9	7
42	32C	30U	H	1	B	1B	0.27	0.02	BLOOD	JULY	38	24
43	32C	30U	H	1	B	1B	0.27	0.02	BLOOD	AUGU	29	24
44	32C	30U	H	1	B	1B	0.27	0.02	BLOOD	SEPT	1	7
45	72C	15U	H	1	B	1B	0.20	0.04	BLOOD	JUNE	15	10

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBM	MAYCAFBM	SOURCE	MONTH	COWBMOL	CALFBMOL
46	72C	15U	H	1	B	1B	0.20	0.04	BLOOD	JULY	41	41
47	72C	15U	H	1	B	1B	0.20	0.04	BLOOD	AUGU	26	5
48	72C	15U	H	1	B	1B	0.20	0.04	BLOOD	SEPT	8	4
49	29C	36U	B	2	A	2A	0.74	0.12	BLOOD	JUNE	62	12
50	29C	36U	B	2	A	2A	0.74	0.12	BLOOD	JULY	46	73
51	29C	36U	B	2	A	2A	0.74	0.12	BLOOD	AUGU	39	18
52	29C	36U	B	2	A	2A	0.74	0.12	BLOOD	SEPT	36	7
53	35C	39U	B	2	A	2A	0.43	0.11	BLOOD	JUNE	25	7
54	35C	39U	B	2	A	2A	0.43	0.11	BLOOD	JULY	69	8
55	35C	39U	B	2	A	2A	0.43	0.11	BLOOD	AUGU	33	2
56	35C	39U	B	2	A	2A	0.43	0.11	BLOOD	SEPT	34	3
57	18C	12U	B	2	A	2A	0.33	0.16	BLOOD	JUNE	33	11
58	18C	12U	B	2	A	2A	0.33	0.16	BLOOD	JULY	36	6
59	18C	12U	B	2	A	2A	0.33	0.16	BLOOD	AUGU	28	1
60	18C	12U	B	2	A	2A	0.33	0.16	BLOOD	SEPT	28	4
61	10C	25U	H	2	A	2A	0.44	0.11	BLOOD	JUNE	53	10
62	10C	25U	H	2	A	2A	0.44	0.11	BLOOD	JULY	39	97
63	10C	25U	H	2	A	2A	0.44	0.11	BLOOD	AUGU	28	16
64	10C	25U	H	2	A	2A	0.44	0.11	BLOOD	SEPT	6	8
65	19C	19U	H	2	A	2A	0.53	0.25	BLOOD	JUNE	63	1
66	19C	19U	H	2	A	2A	0.53	0.25	BLOOD	JULY	37	99
67	19C	19U	H	2	A	2A	0.53	0.25	BLOOD	AUGU	30	3
68	19C	19U	H	2	A	2A	0.53	0.25	BLOOD	SEPT	28	9
69	25C	23U	H	2	A	2A	0.62	0.16	BLOOD	JUNE	34	7
70	25C	23U	H	2	A	2A	0.62	0.16	BLOOD	JULY	40	31
71	25C	23U	H	2	A	2A	0.62	0.16	BLOOD	AUGU	35	14
72	25C	23U	H	2	A	2A	0.62	0.16	BLOOD	SEPT	32	9
73	14C	22U	B	2	B	2B	0.42	0.23	BLOOD	JUNE	13	24
74	14C	22U	B	2	B	2B	0.42	0.23	BLOOD	JULY	29	13
75	14C	22U	B	2	B	2B	0.42	0.23	BLOOD	AUGU	17	18
76	14C	22U	B	2	B	2B	0.42	0.23	BLOOD	SEPT	18	1
77	66C	18U	B	2	B	2B	0.54	0.20	BLOOD	JUNE	25	14
78	66C	18U	B	2	B	2B	0.54	0.20	BLOOD	JULY	36	86
79	66C	18U	B	2	B	2B	0.54	0.20	BLOOD	AUGU	26	3
80	66C	18U	B	2	B	2B	0.54	0.20	BLOOD	SEPT	15	5
81	19S	20U	B	2	B	2B	0.37	0.19	BLOOD	JUNE	29	16
82	19S	20U	B	2	B	2B	0.37	0.19	BLOOD	JULY	45	38
83	19S	20U	B	2	B	2B	0.37	0.19	BLOOD	AUGU	24	5
84	19S	20U	B	2	B	2B	0.37	0.19	BLOOD	SEPT	18	1
85	60C	11U	B	2	B	2B	0.65	0.20	BLOOD	JUNE	37	5
86	60C	11U	B	2	B	2B	0.65	0.20	BLOOD	JULY	38	92
87	60C	11U	B	2	B	2B	0.65	0.20	BLOOD	AUGU	36	17
88	60C	11U	B	2	B	2B	0.65	0.20	BLOOD	SEPT	37	4
89	63C	27U	H	2	B	2B	0.46	0.20	BLOOD	JUNE	24	5
90	63C	27U	H	2	B	2B	0.46	0.20	BLOOD	JULY	54	84
91	63C	27U	H	2	B	2B	0.46	0.20	BLOOD	AUGU	46	7

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBM	MAYCALFBM	SOURCE	MONTH	COWBMOL	CALFBMOL
92	63C	27U	H	2	3	2B	0.46	0.20	BLOOD	SEPT	47	5
93	6R	3U	H	2	B	2B	0.56	0.29	BLOOD	JUNE	37	12
94	6R	3U	H	2	B	2B	0.56	0.29	BLOOD	JULY	39	56
95	6R	3U	H	2	B	2B	0.56	0.29	BLOOD	AUGU	28	20
96	6R	3U	H	2	B	2B	0.56	0.29	BLOOD	SEPT	6	19
97	65C	21U	B	3	A	3A	0.51	0.40	BLOOD	JUNE	54	9
98	65C	21U	B	3	A	3A	0.51	0.40	BLOOD	JULY	.	.
99	65C	21U	B	3	A	3A	0.51	0.40	BLOOD	AUGU	20	6
100	65C	21U	B	3	A	3A	0.51	0.40	BLOOD	SEPT	40	4
101	2N	5U	B	3	A	3A	0.26	0.06	BLOOD	JUNE	25	5
102	2N	5U	B	3	A	3A	0.26	0.06	BLOOD	JULY	82	9
103	2N	5U	B	3	A	3A	0.26	0.06	BLOOD	AUGU	41	24
104	2N	5U	B	3	A	3A	0.26	0.06	BLOOD	SEPT	36	13
105	40C	45U	B	3	A	3A	0.23	0.07	BLOOD	JUNE	13	6
106	40C	45U	B	3	A	3A	0.23	0.07	BLOOD	JULY	36	57
107	40C	45U	B	3	A	3A	0.23	0.07	BLOOD	AUGU	39	10
108	40C	45U	B	3	A	3A	0.23	0.07	BLOOD	SEPT	33	7
109	64C	14U	B	3	A	3A	0.44	0.08	BLOOD	JUNE	29	8
110	64C	14U	B	3	A	3A	0.44	0.08	BLOOD	JULY	33	38
111	64C	14U	B	3	A	3A	0.44	0.08	BLOOD	AUGU	18	13
112	64C	14U	B	3	A	3A	0.44	0.08	BLOOD	SEPT	5	7
113	21C	38U	H	3	A	3A	0.22	0.01	BLOOD	JUNE	56	7
114	21C	38U	H	3	A	3A	0.22	0.01	BLOOD	JULY	45	64
115	21C	38U	H	3	A	3A	0.22	0.01	BLOOD	AUGU	31	11
116	21C	38U	H	3	A	3A	0.22	0.01	BLOOD	SEPT	18	6
117	93C	40U	H	3	A	3A	0.54	0.07	BLOOD	JUNE	27	9
118	93C	40U	H	3	A	3A	0.54	0.07	BLOOD	JULY	30	41
119	93C	40U	H	3	A	3A	0.54	0.07	BLOOD	AUGU	27	19
120	93C	40U	H	3	A	3A	0.54	0.07	BLOOD	SEPT	17	18
121	67C	6U	H	3	A	3A	0.32	0.28	BLOOD	JUNE	37	13
122	67C	6U	H	3	A	3A	0.32	0.28	BLOOD	JULY	29	91
123	67C	6U	H	3	A	3A	0.32	0.28	BLOOD	AUGU	16	15
124	67C	6U	H	3	A	3A	0.32	0.28	BLOOD	SEPT	13	8
125	13C	4U	B	3	B	3B	0.55	0.28	BLOOD	JUNE	37	13
126	13C	4U	B	3	B	3B	0.55	0.28	BLOOD	JULY	80	97
127	13C	4U	B	3	B	3B	0.55	0.28	BLOOD	AUGU	30	22
128	13C	4U	B	3	B	3B	0.55	0.28	BLOOD	SEPT	32	23
129	22C	37U	B	3	B	3B	0.39	0.02	BLOOD	JUNE	31	5
130	22C	37U	B	3	B	3B	0.39	0.02	BLOOD	JULY	40	23
131	22C	37U	B	3	B	3B	0.39	0.02	BLOOD	AUGU	40	29
132	22C	37U	B	3	B	3B	0.39	0.02	BLOOD	SEPT	33	47
133	6S	1U	B	3	B	3B	0.47	0.03	BLOOD	JUNE	26	13
134	6S	1U	B	3	B	3B	0.47	0.03	BLOOD	JULY	77	59
135	6S	1U	B	3	B	3B	0.47	0.03	BLOOD	AUGU	67	22

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBM	MAYCAFBM	SOURCE	MONTH	COWBMOL	CALFBMOL
136	6S	1U	B	3	B	3B	0.47	0.03	BLOOD	SEPT	31	16
137	52C	8U	B	3	B	3B	0.60	0.12	BLOOD	JUNE	43	3
138	52C	8U	B	3	B	3B	0.60	0.12	BLOOD	JULY	37	74
139	52C	8U	B	3	B	3B	0.60	0.12	BLOOD	AUGU	29	28
140	52C	8U	B	3	B	3B	0.60	0.12	BLOOD	SEPT	25	16
141	23C	7U	B	3	B	3B	0.78	0.27	BLOOD	JUNE	46	9
142	23C	7U	B	3	B	3B	0.78	0.27	BLOOD	JULY	41	95
143	23C	7U	B	3	B	3B	0.78	0.27	BLOOD	AUGU	34	14
144	23C	7U	B	3	B	3B	0.78	0.27	BLOOD	SEPT	30	13
145	75C	35U	H	3	B	3B	0.48	0.24	BLOOD	JUNE	39	6
146	75C	35U	H	3	B	3B	0.48	0.24	BLOOD	JULY	39	56
147	75C	35U	H	3	B	3B	0.48	0.24	BLOOD	AUGU	16	18
148	75C	35U	H	3	B	3B	0.48	0.24	BLOOD	SEPT	7	15

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBF	MAYCAFBF	SOURCE	MONTH	COWBFE	CALFBFE
1	5C	24U	B	1	A	1A	1.89	2.21	BLOOD	JUNE	212	269
2	5C	24U	B	1	A	1A	1.89	2.21	BLOOD	JULY	217	200
3	5C	24U	B	1	A	1A	1.89	2.21	BLOOD	AUGU	135	75
4	5C	24U	B	1	A	1A	1.89	2.21	BLOOD	SEPT	150	133
5	6C	29U	B	1	A	1A	1.76	2.91	BLOOD	JUNE	282	299
6	6C	29U	B	1	A	1A	1.76	2.91	BLOOD	JULY	150	333
7	6C	29U	B	1	A	1A	1.76	2.91	BLOOD	AUGU	90	165
8	6C	29U	B	1	A	1A	1.76	2.91	BLOOD	SEPT	183	200
9	12C	28U	B	1	A	1A	1.95	3.28	BLOOD	JUNE	266	209
10	12C	28U	B	1	A	1A	1.95	3.28	BLOOD	JULY	300	267
11	12C	28U	B	1	A	1A	1.95	3.28	BLOOD	AUGU	90	270
12	12C	28U	B	1	A	1A	1.95	3.28	BLOOD	SEPT	180	217
13	56C	13U	H	1	A	1A	2.08	2.61	BLOOD	JUNE	239	357
14	56C	13U	H	1	A	1A	2.08	2.61	BLOOD	JULY	150	392
15	56C	13U	H	1	A	1A	2.08	2.61	BLOOD	AUGU	90	225
16	56C	13U	H	1	A	1A	2.08	2.61	BLOOD	SEPT	133	167
17	74C	34U	H	1	A	1A	2.16	2.03	BLOOD	JUNE	389	274
18	74C	34U	H	1	A	1A	2.16	2.03	BLOOD	JULY	341	292
19	74C	34U	H	1	A	1A	2.16	2.03	BLOOD	AUGU	105	165
20	74C	34U	H	1	A	1A	2.16	2.03	BLOOD	SEPT	300	233
21	11C	2U	H	1	A	1A	2.11	2.19	BLOOD	JUNE	401	212
22	11C	2U	H	1	A	1A	2.11	2.19	BLOOD	JULY	400	392
23	11C	2U	H	1	A	1A	2.11	2.19	BLOOD	AUGU	435	180
24	11C	2U	H	1	A	1A	2.11	2.19	BLOOD	SEPT	333	233
25	33C	41U	B	1	B	1B	2.27	2.27	BLOOD	JUNE	271	192
26	33C	41U	B	1	B	1B	2.27	2.27	BLOOD	JULY	210	175
27	33C	41U	B	1	B	1B	2.27	2.27	BLOOD	AUGU	90	165
28	33C	41U	B	1	B	1B	2.27	2.27	BLOOD	SEPT	183	100
29	16C	17U	B	1	B	1B	2.77	2.16	BLOOD	JUNE	209	279
30	16C	17U	B	1	B	1B	2.77	2.16	BLOOD	JULY	187	241
31	16C	17U	B	1	B	1B	2.77	2.16	BLOOD	AUGU	.	90
32	16C	17U	B	1	B	1B	2.77	2.16	BLOOD	SEPT	167	167
33	17C	9U	B	1	B	1B	1.57	1.23	BLOOD	JUNE	177	151
34	17C	9U	B	1	B	1B	1.57	1.23	BLOOD	JULY	133	258
35	17C	9U	B	1	B	1B	1.57	1.23	BLOOD	AUGU	90	195
36	17C	9U	B	1	B	1B	1.57	1.23	BLOOD	SEPT	150	183
37	2P	10U	B	1	B	1B	2.13	1.75	BLOOD	JUNE	221	296
38	2P	10U	B	1	B	1B	2.13	1.75	BLOOD	JULY	275	258
39	2P	10U	B	1	B	1B	2.13	1.75	BLOOD	AUGU	165	135
40	2P	10U	B	1	B	1B	2.13	1.75	BLOOD	SEPT	233	100
41	32C	30U	H	1	B	1B	2.21	2.03	BLOOD	JUNE	319	356
42	32C	30U	H	1	B	1B	2.21	2.03	BLOOD	JULY	285	308
43	32C	30U	H	1	B	1B	2.21	2.03	BLOOD	AUGU	120	90

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBF	MAYCABF	SOURCE	MONTH	COWBFE	CALFBFE
44	32C	30U	H	1	B	1B	2.21	2.03	BLOOD	SEPT	250	267
45	72C	15U	H	1	B	1B	2.08	1.95	BLOOD	JUNE	234	326
46	72C	15U	H	1	B	1B	2.08	1.95	BLOOD	JULY	205	320
47	72C	15U	H	1	B	1B	2.08	1.95	BLOOD	AUGU	90	300
48	72C	15U	H	1	B	1B	2.08	1.95	BLOOD	SEPT	250	217
49	29C	36U	B	2	A	2A	2.29	3.17	BLOOD	JUNE	216	329
50	29C	36U	B	2	A	2A	2.29	3.17	BLOOD	JULY	175	233
51	29C	36U	B	2	A	2A	2.29	3.17	BLOOD	AUGU	165	155
52	29C	36U	B	2	A	2A	2.29	3.17	BLOOD	SEPT	117	133
53	35C	39U	B	2	A	2A	1.63	1.47	BLOOD	JUNE	304	262
54	35C	39U	B	2	A	2A	1.63	1.47	BLOOD	JULY	250	310
55	35C	39U	B	2	A	2A	1.63	1.47	BLOOD	AUGU	150	240
56	35C	39U	B	2	A	2A	1.63	1.47	BLOOD	SEPT	350	233
57	18C	12U	B	2	A	2A	1.55	1.36	BLOOD	JUNE	186	249
58	18C	12U	B	2	A	2A	1.55	1.36	BLOOD	JULY	133	208
59	18C	12U	B	2	A	2A	1.55	1.36	BLOOD	AUGU	240	75
60	18C	12U	B	2	A	2A	1.55	1.36	BLOOD	SEPT	167	183
61	10C	25U	H	2	A	2A	2.43	1.55	BLOOD	JUNE	234	199
62	10C	25U	H	2	A	2A	2.43	1.55	BLOOD	JULY	308	283
63	10C	25U	H	2	A	2A	2.43	1.55	BLOOD	AUGU	60	240
64	10C	25U	H	2	A	2A	2.43	1.55	BLOOD	SEPT	167	233
65	19C	19U	H	2	A	2A	2.40	2.11	BLOOD	JUNE	242	122
66	19C	19U	H	2	A	2A	2.40	2.11	BLOOD	JULY	160	292
67	19C	19U	H	2	A	2A	2.40	2.11	BLOOD	AUGU	75	120
68	19C	19U	H	2	A	2A	2.40	2.11	BLOOD	SEPT	183	117
69	25C	23U	H	2	A	2A	2.19	2.21	BLOOD	JUNE	326	239
70	25C	23U	H	2	A	2A	2.19	2.21	BLOOD	JULY	292	208
71	25C	23U	H	2	A	2A	2.19	2.21	BLOOD	AUGU	225	105
72	25C	23U	H	2	A	2A	2.19	2.21	BLOOD	SEPT	150	200
73	14C	22U	B	2	B	2B	2.05	2.21	BLOOD	JUNE	152	199
74	14C	22U	B	2	B	2B	2.05	2.21	BLOOD	JULY	192	225
75	14C	22U	B	2	B	2B	2.05	2.21	BLOOD	AUGU	195	240
76	14C	22U	B	2	B	2B	2.05	2.21	BLOOD	SEPT	117	150
77	66C	18U	B	2	B	2B	2.56	1.89	BLOOD	JUNE	177	167
78	66C	18U	B	2	B	2B	2.56	1.89	BLOOD	JULY	192	225
79	66C	18U	B	2	B	2B	2.56	1.89	BLOOD	AUGU	105	275
80	66C	18U	B	2	B	2B	2.56	1.89	BLOOD	SEPT	150	217
81	19S	20U	B	2	B	2B	1.87	1.12	BLOOD	JUNE	274	229
82	19S	20U	B	2	B	2B	1.87	1.12	BLOOD	JULY	125	242
83	19S	20U	B	2	B	2B	1.87	1.12	BLOOD	AUGU	45	150
84	19S	20U	B	2	B	2B	1.87	1.12	BLOOD	SEPT	300	217
85	60C	11U	B	2	B	2B	1.97	2.13	BLOOD	JUNE	306	206
86	60C	11U	B	2	B	2B	1.97	2.13	BLOOD	JULY	208	300
87	60C	11U	B	2	B	2B	1.97	2.13	BLOOD	AUGU	180	105

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBF	MAYCABF	SOURCE	MONTH	COWBFE	CALFBFE
88	60C	11U	U	2	B	2B	1.97	2.13	BLOOD	SEPT	133	33
89	63C	27U	H	2	B	2B	1.79	1.55	BLOOD	JUNE	291	237
90	63C	27U	H	2	B	2B	1.79	1.55	BLOOD	JULY	258	342
91	63C	27U	H	2	B	2B	1.79	1.55	BLOOD	AUGU	90	135
92	63C	27U	H	2	B	2B	1.79	1.55	BLOOD	SEPT	183	300
93	6R	3U	H	2	B	2B	2.11	2.99	BLOOD	JUNE	336	311
94	6R	3U	H	2	B	2B	2.11	2.99	BLOOD	JULY	258	283
95	6R	3U	H	2	B	2B	2.11	2.99	BLOOD	AUGU	165	225
96	6R	3U	H	2	B	2B	2.11	2.99	BLOOD	SEPT	117	133
97	65C	21U	B	3	A	3A	2.27	1.41	BLOOD	JUNE	312	167
98	65C	21U	B	3	A	3A	2.27	1.41	BLOOD	JULY	.	.
99	65C	21U	B	3	A	3A	2.27	1.41	BLOOD	AUGU	230	90
100	65C	21U	B	3	A	3A	2.27	1.41	BLOOD	SEPT	150	283
101	2N	5U	B	3	A	3A	1.39	3.77	BLOOD	JUNE	187	331
102	2N	5U	B	3	A	3A	1.39	3.77	BLOOD	JULY	167	383
103	2N	5U	B	3	A	3A	1.39	3.77	BLOOD	AUGU	60	180
104	2N	5U	B	3	A	3A	1.39	3.77	BLOOD	SEPT	100	183
105	40C	45U	B	3	A	3A	2.21	1.41	BLOOD	JUNE	244	334
106	40C	45U	B	3	A	3A	2.21	1.41	BLOOD	JULY	174	337
107	40C	45U	B	3	A	3A	2.21	1.41	BLOOD	AUGU	240	345
108	40C	45U	B	3	A	3A	2.21	1.41	BLOOD	SEPT	200	167
109	64C	14U	B	3	A	3A	1.65	1.28	BLOOD	JUNE	209	184
110	64C	14U	B	3	A	3A	1.65	1.28	BLOOD	JULY	125	225
111	64C	14U	B	3	A	3A	1.65	1.28	BLOOD	AUGU	165	210
112	64C	14U	B	3	A	3A	1.65	1.28	BLOOD	SEPT	100	83
113	21C	38U	H	3	A	3A	1.89	2.24	BLOOD	JUNE	261	322
114	21C	38U	H	3	A	3A	1.89	2.24	BLOOD	JULY	175	258
115	21C	38U	H	3	A	3A	1.89	2.24	BLOOD	AUGU	150	105
116	21C	38U	H	3	A	3A	1.89	2.24	BLOOD	SEPT	183	133
117	93C	40U	H	3	A	3A	2.00	2.82	BLOOD	JUNE	254	302
118	93C	40U	H	3	A	3A	2.00	2.82	BLOOD	JULY	117	292
119	93C	40U	H	3	A	3A	2.00	2.82	BLOOD	AUGU	135	235
120	93C	40U	H	3	A	3A	2.00	2.82	BLOOD	SEPT	167	217
121	67C	6U	H	3	A	3A	2.32	2.45	BLOOD	JUNE	339	226
122	67C	6U	H	3	A	3A	2.32	2.45	BLOOD	JULY	208	233
123	67C	6U	H	3	A	3A	2.32	2.45	BLOOD	AUGU	105	270
124	67C	6U	H	3	A	3A	2.32	2.45	BLOOD	SEPT	150	183
125	13C	4U	B	3	B	3B	2.00	3.12	BLOOD	JUNE	257	174
126	13C	4U	B	3	B	3B	2.00	3.12	BLOOD	JULY	233	200
127	13C	4U	B	3	B	3B	2.00	3.12	BLOOD	AUGU	145	255
128	13C	4U	B	3	B	3B	2.00	3.12	BLOOD	SEPT	250	150
129	22C	37U	B	3	B	3B	2.05	1.33	BLOOD	JUNE	217	280
130	22C	37U	B	3	B	3B	2.05	1.33	BLOOD	JULY	117	225

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBF	MAYCAFBF	SOURCE	MONTH	COWBFE	CALFBFE
131	22C	37U	B	3	B	3B	2.05	1.33	BLOOD	AUGU	115	240
132	22C	37U	B	3	B	3B	2.05	1.33	BLOOD	SEPT	117	150
133	6S	1U	B	3	B	3B	3.12	2.84	BLOOD	JUNE	197	284
134	6S	1U	B	3	B	3B	3.12	2.84	BLOOD	JULY	292	242
135	6S	1U	B	3	B	3B	3.12	2.84	BLOOD	AUGU	165	180
136	6S	1U	B	3	B	3B	3.12	2.84	BLOOD	SEPT	117	250
137	52C	8U	B	3	B	3B	2.88	1.44	BLOOD	JUNE	214	351
138	52C	8U	B	3	B	3B	2.88	1.44	BLOOD	JULY	142	258
139	52C	8U	B	3	B	3B	2.88	1.44	BLOOD	AUGU	165	90
140	52C	8U	B	3	B	3B	2.88	1.44	BLOOD	SEPT	150	217
141	23C	7U	B	3	B	3B	2.40	1.57	BLOOD	JUNE	284	286
142	23C	7U	B	3	B	3B	2.40	1.57	BLOOD	JULY	175	142
143	23C	7U	B	3	B	3B	2.40	1.57	BLOOD	AUGU	75	90
144	23C	7U	B	3	B	3B	2.40	1.57	BLOOD	SEPT	83	100
145	75C	35U	H	3	B	3B	2.29	3.57	BLOOD	JUNE	184	244
146	75C	35U	H	3	B	3B	2.29	3.57	BLOOD	JULY	125	225
147	75C	35U	H	3	B	3B	2.29	3.57	BLOOD	AUGU	110	225
148	75C	35U	H	3	B	3B	2.29	3.57	BLOOD	SEPT	150	217

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBZ	MAYCAFBZ	SOURCE	MONTH	COWBZN	CALFBZN
1	5C	24U	B	1	A	1A	1.35	1.31	BLOOD	JUNE	109	121
2	5C	24U	B	1	A	1A	1.35	1.31	BLOOD	JULY	158	129
3	5C	24U	B	1	A	1A	1.35	1.31	BLOOD	AUGU	227	89
4	5C	24U	B	1	A	1A	1.35	1.31	BLOOD	SEPT	51	102
5	6C	29U	B	1	A	1A	1.83	1.36	BLOOD	JUNE	147	165
6	6C	29U	B	1	A	1A	1.83	1.36	BLOOD	JULY	228	113
7	6C	29U	B	1	A	1A	1.83	1.36	BLOOD	AUGU	123	148
8	6C	29U	B	1	A	1A	1.83	1.36	BLOOD	SEPT	72	133
9	12C	28U	B	1	B	1B	0.96	1.37	BLOOD	JUNE	139	102
10	12C	28U	B	1	B	1B	0.96	1.37	BLOOD	JULY	232	98
11	12C	28U	B	1	B	1B	0.96	1.37	BLOOD	AUGU	185	135
12	12C	28U	B	1	B	1B	0.96	1.37	BLOOD	SEPT	70	68
13	56C	13U	H	1	A	1A	0.97	1.17	BLOOD	JUNE	122	140
14	56C	13U	H	1	A	1A	0.97	1.17	BLOOD	JULY	47	102
15	56C	13U	H	1	A	1A	0.97	1.17	BLOOD	AUGU	72	146
16	56C	13U	H	1	A	1A	0.97	1.17	BLOOD	SEPT	70	127
17	74C	34U	H	1	A	1A	2.80	1.12	BLOOD	JUNE	144	109
18	74C	34U	H	1	A	1A	2.80	1.12	BLOOD	JULY	111	96
19	74C	34U	H	1	A	1A	2.80	1.12	BLOOD	AUGU	74	114
20	74C	34U	H	1	A	1A	2.80	1.12	BLOOD	SEPT	195	112
21	11C	2U	H	1	A	1A	1.64	1.37	BLOOD	JUNE	111	98
22	11C	2U	H	1	A	1A	1.64	1.37	BLOOD	JULY	199	106
23	11C	2U	H	1	A	1A	1.64	1.37	BLOOD	AUGU	180	113
24	11C	2U	H	1	A	1A	1.64	1.37	BLOOD	SEPT	48	147
25	33C	41U	B	1	B	1B	1.40	1.08	BLOOD	JUNE	88	176
26	33C	41U	B	1	B	1B	1.40	1.08	BLOOD	JULY	114	105
27	33C	41U	B	1	B	1B	1.40	1.08	BLOOD	AUGU	186	138
28	33C	41U	B	1	B	1B	1.40	1.08	BLOOD	SEPT	77	70
29	16C	17U	B	1	B	1B	1.90	1.51	BLOOD	JUNE	104	107
30	16C	17U	B	1	B	1B	1.90	1.51	BLOOD	JULY	150	223
31	16C	17U	B	1	B	1B	1.90	1.51	BLOOD	AUGU	.	120
32	16C	17U	B	1	B	1B	1.90	1.51	BLOOD	SEPT	121	87
33	17C	9U	B	1	B	1B	1.00	0.95	BLOOD	JUNE	131	191
34	17C	9U	B	1	B	1B	1.00	0.95	BLOOD	JULY	116	115
35	17C	9U	B	1	B	1B	1.00	0.95	BLOOD	AUGU	90	98
36	17C	9U	B	1	B	1B	1.00	0.95	BLOOD	SEPT	92	88
37	2P	10U	B	1	B	1B	2.33	1.86	BLOOD	JUNE	115	82
38	2P	10U	B	1	B	1B	2.33	1.86	BLOOD	JULY	134	139
39	2P	10U	B	1	B	1B	2.33	1.86	BLOOD	AUGU	69	185
40	2P	10U	B	1	B	1B	2.33	1.86	BLOOD	SEPT	60	113
41	32C	30U	H	1	B	1B	1.40	1.63	BLOOD	JUNE	88	182
42	32C	30U	H	1	B	1B	1.40	1.63	BLOOD	JULY	116	161
43	32C	30U	H	1	B	1B	1.40	1.63	BLOOD	AUGU	186	159
44	32C	30U	H	1	B	1B	1.40	1.63	BLOOD	SEPT	77	113
45	72C	15U	H	1	B	1B	1.42	2.76	BLOOD	JUNE	126	109

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBZ	MAYCAFBZ	SOURCE	MONTH	COWBZN	CALFBZN
46	72C	15U	H	1	B	1B	1.42	2.76	BLOOD	JULY	168	173
47	72C	15U	H	1	B	1B	1.42	2.76	BLOOD	AUGU	135	161
48	72C	15U	H	1	B	1B	1.42	2.76	BLOOD	SEPT	63	122
49	29C	36U	B	2	A	2A	1.81	1.36	BLOOD	JUNE	121	165
50	29C	36U	B	2	A	2A	1.81	1.36	BLOOD	JULY	206	113
51	29C	36U	B	2	A	2A	1.81	1.36	BLOOD	AUGU	132	148
52	29C	36U	B	2	A	2A	1.81	1.36	BLOOD	SEPT	49	133
53	35C	39U	B	2	A	2A	2.61	0.97	BLOOD	JUNE	124	134
54	35C	39U	B	2	A	2A	2.61	0.97	BLOOD	JULY	140	271
55	35C	39U	B	2	A	2A	2.61	0.97	BLOOD	AUGU	161	250
56	35C	39U	B	2	A	2A	2.61	0.97	BLOOD	SEPT	100	142
57	18C	12U	B	2	A	2A	1.38	0.96	BLOOD	JUNE	192	97
58	18C	12U	B	2	A	2A	1.38	0.96	BLOOD	JULY	206	111
59	18C	12U	B	2	A	2A	1.38	0.96	BLOOD	AUGU	261	101
60	18C	12U	B	2	A	2A	1.38	0.96	BLOOD	SEPT	78	135
61	10C	25U	H	2	A	2A	1.98	1.40	BLOOD	JUNE	124	123
62	10C	25U	H	2	A	2A	1.98	1.40	BLOOD	JULY	99	113
63	10C	25U	H	2	A	2A	1.98	1.40	BLOOD	AUGU	162	171
64	10C	25U	H	2	A	2A	1.98	1.40	BLOOD	SEPT	49	103
65	19C	19U	H	2	A	2A	1.17	1.25	BLOOD	JUNE	139	114
66	19C	19U	H	2	A	2A	1.17	1.25	BLOOD	JULY	168	250
67	19C	19U	H	2	A	2A	1.17	1.25	BLOOD	AUGU	195	116
68	19C	19U	H	2	A	2A	1.17	1.25	BLOOD	SEPT	56	142
69	25C	23U	H	2	A	2A	1.34	1.65	BLOOD	JUNE	126	190
70	25C	23U	H	2	A	2A	1.34	1.65	BLOOD	JULY	107	298
71	25C	23U	H	2	A	2A	1.34	1.65	BLOOD	AUGU	99	198
72	25C	23U	H	2	A	2A	1.34	1.65	BLOOD	SEPT	82	137
73	14C	22U	B	2	B	2B	1.40	1.13	BLOOD	JUNE	113	92
74	14C	22U	B	2	B	2B	1.40	1.13	BLOOD	JULY	194	121
75	14C	22U	B	2	B	2B	1.40	1.13	BLOOD	AUGU	143	131
76	14C	22U	B	2	B	2B	1.40	1.13	BLOOD	SEPT	48	82
77	66C	18U	B	2	B	2B	1.25	1.13	BLOOD	JUNE	87	183
78	66C	18U	B	2	B	2B	1.25	1.13	BLOOD	JULY	156	270
79	66C	18U	B	2	B	2B	1.25	1.13	BLOOD	AUGU	234	210
80	66C	18U	B	2	B	2B	1.25	1.13	BLOOD	SEPT	65	130
81	19S	20U	B	2	B	2B	0.18	0.39	BLOOD	JUNE	128	119
82	19S	20U	B	2	B	2B	0.18	0.39	BLOOD	JULY	141	240
83	19S	20U	B	2	B	2B	0.18	0.39	BLOOD	AUGU	215	104
84	19S	20U	B	2	B	2B	0.18	0.39	BLOOD	SEPT	70	152
85	60C	11U	B	2	B	2B	2.18	1.26	BLOOD	JUNE	110	144
86	60C	11U	B	2	B	2B	2.18	1.26	BLOOD	JULY	81	224
87	60C	11U	B	2	B	2B	2.18	1.26	BLOOD	AUGU	102	203
88	60C	11U	B	2	B	2B	2.18	1.26	BLOOD	SEPT	45	42
89	63C	27U	H	2	B	2B	1.36	1.45	BLOOD	JUNE	76	160
90	63C	27U	H	2	B	2B	1.36	1.45	BLOOD	JULY	45	232
91	63C	27U	H	2	B	2B	1.36	1.45	BLOOD	AUGU	93	119

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBZ	MAYCAFBZ	SOURCE	MONTH	COWBZN	CALFBZN
92	63C	27U	H	2	B	2B	1.36	1.45	BLOOD	SEPT	48	103
93	6R	3U	H	2	B	2B	1.00	1.51	BLOOD	JUNE	131	271
94	6R	3U	H	2	B	2B	1.00	1.51	BLOOD	JULY	216	330
95	6R	3U	H	2	B	2B	1.00	1.51	BLOOD	AUGU	129	204
96	6R	3U	H	2	B	2B	1.00	1.51	BLOOD	SEPT	78	108
97	65C	21U	B	3	A	3A	2.17	1.93	BLOOD	JUNE	139	210
98	65C	21U	B	3	A	3A	2.17	1.93	BLOOD	JULY	.	.
99	65C	21U	B	3	A	3A	2.17	1.93	BLOOD	AUGU	216	137
100	65C	21U	B	3	A	3A	2.17	1.93	BLOOD	SEPT	65	67
101	2N	5U	B	3	A	3A	1.83	1.11	BLOOD	JUNE	130	141
102	2N	5U	B	3	A	3A	1.83	1.11	BLOOD	JULY	199	241
103	2N	5U	B	3	A	3A	1.83	1.11	BLOOD	AUGU	69	111
104	2N	5U	B	3	A	3A	1.83	1.11	BLOOD	SEPT	95	88
105	40C	45U	B	3	A	3A	1.47	0.93	BLOOD	JUNE	135	103
106	40C	45U	B	3	A	3A	1.47	0.93	BLOOD	JULY	151	141
107	40C	45U	B	3	A	3A	1.47	0.93	BLOOD	AUGU	137	183
108	40C	45U	B	3	A	3A	1.47	0.93	BLOOD	SEPT	75	107
109	64C	14U	B	3	A	3A	2.02	1.29	BLOOD	JUNE	124	143
110	64C	14U	B	3	A	3A	2.02	1.29	BLOOD	JULY	120	276
111	64C	14U	B	3	A	3A	2.02	1.29	BLOOD	AUGU	164	141
112	64C	14U	B	3	A	3A	2.02	1.29	BLOOD	SEPT	99	95
113	21C	28U	H	3	A	3A	0.78	0.64	BLOOD	JUNE	129	146
114	21C	28U	H	3	A	3A	0.78	0.64	BLOOD	JULY	144	248
115	21C	28U	H	3	A	3A	0.78	0.64	BLOOD	AUGU	136	144
116	21C	28U	H	3	A	3A	0.78	0.64	BLOOD	SEPT	85	155
117	93C	40U	H	3	A	3A	1.73	1.22	BLOOD	JUNE	107	127
118	93C	40U	H	3	A	3A	1.73	1.22	BLOOD	JULY	147	207
119	93C	40U	H	3	A	3A	1.73	1.22	BLOOD	AUGU	79	144
120	93C	40U	H	3	A	3A	1.73	1.22	BLOOD	SEPT	92	100
121	67C	6U	H	3	A	3A	1.79	2.30	BLOOD	JUNE	146	273
122	67C	6U	H	3	A	3A	1.79	2.30	BLOOD	JULY	92	260
123	67C	6U	H	3	A	3A	1.79	2.30	BLOOD	AUGU	159	131
124	67C	6U	H	3	A	3A	1.79	2.30	BLOOD	SEPT	58	132
125	13C	4U	B	3	B	3B	1.25	1.81	BLOOD	JUNE	140	87
126	13C	4U	B	3	B	3B	1.25	1.81	BLOOD	JULY	178	165
127	13C	4U	B	3	B	3B	1.25	1.81	BLOOD	AUGU	161	153
128	13C	4U	B	3	B	3B	1.25	1.81	BLOOD	SEPT	82	153
129	22C	37U	B	3	B	3B	1.92	1.35	BLOOD	JUNE	161	113
130	22C	37U	B	3	B	3B	1.92	1.35	BLOOD	JULY	143	154
131	22C	37U	B	3	B	3B	1.92	1.35	BLOOD	AUGU	149	150
132	22C	37U	B	3	B	3B	1.92	1.35	BLOOD	SEPT	62	102
133	6S	1U	B	3	B	3B	2.00	1.12	BLOOD	JUNE	84	137
134	6S	1U	B	3	B	3B	2.00	1.12	BLOOD	JULY	93	180

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	MAYCOWBZ	MAYCAFBZ	SOURCE	MONTH	COWBZN	CALFBZN
				3	B	3B	2.00	1.12	BLOOD	AUGU	185	116
135	6S	1U	B	3	B	3B	2.00	1.12	BLOOD	SEPT	55	90
136	6S	1U	B	2	B	2B	2.14	0.82	BLOOD	JUNE	133	116
137	52C	8U	B	2	B	2B	2.14	0.82	BLOOD	JULY	134	218
138	52C	8U	B	2	B	2B	2.14	0.82	BLOOD	AUGU	130	78
139	52C	8U	B	2	B	2B	2.14	0.82	BLOOD	SEPT	83	83
140	52C	8U	B	3	B	3B	1.22	1.58	BLOOD	JUNE	135	174
141	23C	7U	B	3	B	3B	1.22	1.58	BLOOD	JULY	137	336
142	23C	7U	B	3	B	3B	1.22	1.58	BLOOD	AUGU	114	336
143	23C	7U	B	3	B	3B	1.22	1.58	BLOOD	SEPT	68	107
144	23C	7U	B	3	B	3B	1.35	1.17	BLOOD	JUNE	118	128
145	75C	35U	H	3	B	3B	1.35	1.17	BLOOD	JULY	171	210
146	75C	35U	H	3	B	3B	1.35	1.17	BLOOD	AUGU	183	234
147	75C	35U	H	3	B	3B	1.35	1.17	BLOOD	SEPT	63	98
148	75C	35U										

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLCUP	CALFLCUP
1	5C	24U	B	1	A	1A	LIVER	JUNE	80.2	103.1
2	5C	24U	B	1	A	1A	LIVER	JULY	60.6	92.4
3	5C	24U	B	1	A	1A	LIVER	SEPT	130.8	51.4
4	6C	29U	B	1	A	1A	LIVER	JUNE	123.2	212.0
5	6C	29U	B	1	A	1A	LIVER	JULY	159.0	75.0
6	6C	29U	B	1	A	1A	LIVER	SEPT	165.4	70.9
7	12C	28U	B	1	A	1A	LIVER	JUNE	257.4	320.7
8	12C	28U	B	1	A	1A	LIVER	JULY	254.7	259.5
9	12C	28U	B	1	A	1A	LIVER	SEPT	281.0	186.5
10	56C	13U	H	1	A	1A	LIVER	JUNE	60.4	105.6
11	56C	13U	H	1	A	1A	LIVER	JULY	67.9	52.4
12	56C	13U	H	1	A	1A	LIVER	SEPT	96.4	45.3
13	74C	34U	H	1	A	1A	LIVER	JUNE	284.9	156.0
14	74C	34U	H	1	A	1A	LIVER	JULY	319.3	109.0
15	74C	34U	H	1	A	1A	LIVER	SEPT	419.0	96.7
16	11C	2U	H	1	A	1A	LIVER	JUNE	246.7	204.5
17	11C	2U	H	1	A	1A	LIVER	JULY	264.3	336.9
18	11C	2U	H	1	A	1A	LIVER	SEPT	217.6	101.9
19	33C	41U	B	1	B	1B	LIVER	JUNE	142.8	78.8
20	33C	41U	B	1	B	1B	LIVER	JULY	151.3	347.7
21	33C	41U	B	1	B	1B	LIVER	SEPT	201.0	140.3
22	16C	17U	B	1	B	1B	LIVER	JUNE	69.0	188.1
23	16C	17U	B	1	B	1B	LIVER	JULY	77.0	354.2
24	16C	17U	B	1	B	1B	LIVER	SEPT	143.4	212.5
25	17C	9U	B	1	B	1B	LIVER	JUNE	116.3	102.3
26	17C	9U	B	1	B	1B	LIVER	JULY	109.4	270.3
27	17C	9U	B	1	B	1B	LIVER	SEPT	118.6	109.7
28	2P	10U	B	1	B	1B	LIVER	JUNE	114.4	139.6
29	2P	10U	B	1	B	1B	LIVER	JULY	120.5	333.6
30	2P	10U	B	1	B	1B	LIVER	SEPT	139.3	264.9
31	32C	30U	H	1	B	1B	LIVER	JUNE	241.3	237.5
32	32C	30U	H	1	B	1B	LIVER	JULY	296.4	343.8
33	32C	30U	H	1	B	1B	LIVER	SEPT	374.3	282.5
34	72C	15U	H	1	B	1B	LIVER	JUNE	241.3	132.0
35	72C	15U	H	1	B	1B	LIVER	JULY	227.8	348.8
36	72C	15U	H	1	B	1B	LIVER	SEPT	204.8	152.4
37	29C	36U	B	2	A	2A	LIVER	JUNE	69.0	149.8
38	29C	36U	B	2	A	2A	LIVER	JULY	144.3	120.5
39	29C	36U	B	2	A	2A	LIVER	SEPT	223.9	69.2
40	35C	39U	B	2	A	2A	LIVER	JUNE	101.5	159.0
41	35C	39U	B	2	A	2A	LIVER	JULY	198.4	61.0
42	35C	39U	B	2	A	2A	LIVER	SEPT	193.4	64.1
43	18C	12U	B	2	A	2A	LIVER	JUNE	192.1	165.6
44	18C	12U	B	2	A	2A	LIVER	JULY	315.5	92.9

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLCUP	CALFLCUP
45	18C	12U	B	2	A	2A	LIVER	SEPT	437.5	88.6
46	10C	25U	H	2	A	2A	LIVER	JUNE	196.5	105.0
47	10C	25U	H	2	A	2A	LIVER	JULY	302.6	84.8
48	10C	25U	H	2	A	2A	LIVER	SEPT	385.5	56.4
49	19C	19U	H	2	A	2A	LIVER	JUNE	147.1	136.1
50	19C	19U	H	2	A	2A	LIVER	JULY	192.2	72.1
51	19C	19U	H	2	A	2A	LIVER	SEPT	207.1	53.8
52	25C	23U	H	2	A	2A	LIVER	JUNE	38.5	81.4
53	25C	23U	H	2	A	2A	LIVER	JULY	150.9	61.4
54	25C	23U	H	2	A	2A	LIVER	SEPT	196.2	38.7
55	14C	22U	B	2	B	2B	LIVER	JUNE	213.1	192.3
56	14C	22U	B	2	B	2B	LIVER	JULY	252.5	385.2
57	14C	22U	B	2	B	2B	LIVER	SEPT	280.6	244.7
58	66C	18U	B	2	B	2B	LIVER	JUNE	115.5	207.8
59	66C	18U	B	2	B	2B	LIVER	JULY	158.2	437.0
60	66C	18U	B	2	B	2B	LIVER	SEPT	211.6	188.6
61	19S	20U	B	2	B	2B	LIVER	JUNE	164.2	306.9
62	19S	20U	B	2	B	2B	LIVER	JULY	318.2	368.4
63	19S	20U	B	2	B	2B	LIVER	SEPT	344.7	344.8
64	60C	11U	B	2	B	2B	LIVER	JUNE	86.3	126.3
65	60C	11U	B	2	B	2B	LIVER	JULY	149.8	378.3
66	60C	11U	B	2	B	2B	LIVER	SEPT	248.4	180.6
67	63C	27U	H	2	B	2B	LIVER	JUNE	67.8	85.6
68	63C	27U	H	2	B	2B	LIVER	JULY	109.4	345.7
69	63C	27U	H	2	B	2B	LIVER	SEPT	165.3	148.9
70	6R	3U	H	2	B	2B	LIVER	JUNE	265.2	171.9
71	6R	3U	H	2	B	2B	LIVER	JULY	434.0	394.5
72	6R	3U	H	2	B	2B	LIVER	SEPT	291.3	274.6
73	65C	21U	B	3	A	3A	LIVER	JUNE	100.3	73.1
74	65C	21U	B	3	A	3A	LIVER	JULY		
75	65C	21U	B	3	A	3A	LIVER	SEPT	178.7	215.9
76	2N	5U	B	3	A	3A	LIVER	JUNE	175.8	137.4
77	2N	5U	B	3	A	3A	LIVER	JULY	269.5	111.7
78	2N	5U	B	3	A	3A	LIVER	SEPT	279.1	86.8
79	40C	45U	B	3	A	3A	LIVER	JUNE	375.5	351.9
80	40C	45U	B	3	A	3A	LIVER	JULY	533.0	226.3
81	40C	45U	B	3	A	3A	LIVER	SEPT	451.8	148.9
82	64C	14U	B	3	A	3A	LIVER	JUNE	210.3	142.3
83	64C	14U	B	3	A	3A	LIVER	JULY	333.3	119.2
84	64C	14U	B	3	A	3A	LIVER	SEPT	380.8	76.3
85	21C	38U	H	3	A	3A	LIVER	JUNE	37.7	77.7
86	21C	38U	H	3	A	3A	LIVER	JULY	164.7	62.4
87	21C	38U	H	3	A	3A	LIVER	SEPT	273.3	56.9
88	93C	40U	H	3	A	3A	LIVER	JUNE	127.1	138.0
89	93C	40U	H	3	A	3A	LIVER	JULY	180.8	82.4

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLCUP	CALFLCUP
					A	3A	LIVER	SEPT	314.2	46.4
90	93C	40U	H	3	A	3A	LIVER	JUNE	157.8	185.0
91	67C	6U	H	3	A	3A	LIVER	JULY	273.0	143.3
92	67C	6U	H	3	A	3A	LIVER	SEPT	170.5	110.5
93	67C	6U	H	3	B	3B	LIVER	JUNE	180.1	224.9
94	13C	4U	B	3	B	3B	LIVER	JULY	273.5	492.1
95	13C	4U	B	3	B	3B	LIVER	SEPT	244.9	250.8
96	13C	4U	B	3	B	3B	LIVER	JUNE	287.3	112.6
97	22C	37U	B	3	B	3B	LIVER	JULY	255.5	312.3
98	22C	37U	B	3	B	3B	LIVER	SEPT	514.7	119.2
99	22C	37U	B	3	B	3B	LIVER	JUNE	185.0	105.6
100	6S	1U	B	3	B	3B	LIVER	JULY	268.6	289.0
101	6S	1U	B	3	B	3B	LIVER	SEPT	247.9	218.7
102	6S	1U	B	3	B	3B	LIVER	JUNE	182.3	95.5
103	52C	8U	B	3	B	3B	LIVER	JULY	468.5	437.3
104	52C	8U	B	3	B	3B	LIVER	SEPT	458.0	230.0
105	52C	8U	B	3	B	3B	LIVER	JUNE	206.8	178.8
106	23C	7U	B	3	B	3B	LIVER	JULY	265.7	483.4
107	23C	7U	B	3	B	3B	LIVER	SEPT	424.1	326.3
108	23C	7U	B	3	B	3B	LIVER	JUNE	61.0	88.7
109	75C	35U	H	3	B	3B	LIVER	JULY	106.5	345.6
110	75C	35U	H	3	B	3B	LIVER	SEPT	145.7	160.9
111	75C	35U	H	3	B	3B	LIVER			

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLMOL	CALFLMOL
1	5C	24U	B	1	A	1A	LIVER	JUNE	13.7	3.1
2	5C	24U	B	1	A	1A	LIVER	JULY	13.3	4.9
3	5C	24U	B	1	A	1A	LIVER	SEPT	5.7	5.8
4	6C	29U	B	1	A	1A	LIVER	JUNE	6.1	0.6
5	6C	29U	B	1	A	1A	LIVER	JULY	15.1	1.1
6	6C	29U	B	1	A	1A	LIVER	SEPT	7.2	3.3
7	12C	28U	B	1	A	1A	LIVER	JUNE	4.9	3.1
8	12C	28U	B	1	A	1A	LIVER	JULY	13.2	7.0
9	12C	28U	B	1	A	1A	LIVER	SEPT	6.2	7.3
10	56C	13U	H	1	A	1A	LIVER	JUNE	5.3	2.9
11	56C	13U	H	1	A	1A	LIVER	JULY	10.1	2.6
12	56C	13U	H	1	A	1A	LIVER	SEPT	5.9	3.6
13	74C	34U	H	1	A	1A	LIVER	JUNE	9.8	2.0
14	74C	34U	H	1	A	1A	LIVER	JULY	9.1	4.6
15	74C	34U	H	1	A	1A	LIVER	SEPT	3.5	4.2
16	11C	2U	H	1	A	1A	LIVER	JUNE	6.3	3.8
17	11C	2U	H	1	A	1A	LIVER	JULY	9.9	7.5
18	11C	2U	H	1	A	1A	LIVER	SEPT	6.6	4.9
19	33C	41U	B	1	B	1B	LIVER	JUNE	7.0	2.4
20	33C	41U	B	1	B	1B	LIVER	JULY	15.2	8.9
21	33C	41U	B	1	B	1B	LIVER	SEPT	8.4	5.4
22	16C	17U	B	1	B	1B	LIVER	JUNE	5.5	5.0
23	16C	17U	B	1	B	1B	LIVER	JULY	12.5	4.6
24	16C	17U	B	1	B	1B	LIVER	SEPT	8.3	6.6
25	17C	9U	B	1	B	1B	LIVER	JUNE	4.0	2.6
26	17C	9U	B	1	B	1B	LIVER	JULY	13.7	4.7
27	17C	9U	B	1	B	1B	LIVER	SEPT	3.2	3.5
28	2P	10U	B	1	B	1B	LIVER	JUNE	5.6	4.0
29	2P	10U	B	1	B	1B	LIVER	JULY	9.5	4.9
30	2P	10U	B	1	B	1B	LIVER	SEPT	5.2	2.9
31	32C	30U	H	1	B	1B	LIVER	JUNE	5.7	2.2
32	32C	30U	H	1	B	1B	LIVER	JULY	8.0	2.4
33	32C	30U	H	1	B	1B	LIVER	SEPT	6.3	5.9
34	72C	15U	H	1	B	1B	LIVER	JUNE	5.9	1.9
35	72C	15U	H	1	B	1B	LIVER	JULY	13.9	7.1
36	72C	15U	H	1	B	1B	LIVER	SEPT	3.2	2.9
37	29C	36U	B	2	A	2A	LIVER	JUNE	5.0	3.8
38	29C	36U	B	2	A	2A	LIVER	JULY	13.5	9.0
39	29C	36U	B	2	A	2A	LIVER	SEPT	6.9	1.4
40	35C	39U	B	2	A	2A	LIVER	JUNE	13.6	5.9
41	35C	39U	B	2	A	2A	LIVER	JULY	8.7	5.1
42	35C	39U	B	2	A	2A	LIVER	SEPT	3.5	5.5
43	18C	12U	B	2	A	2A	LIVER	JUNE	6.0	3.8
44	18C	12U	B	2	A	2A	LIVER	JULY	15.3	8.0

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLMOL	CALFLMOL
45	18C	12U	B	2	A	2A	LIVER	SEPT	6.3	5.6
46	10C	25U	H	2	A	2A	LIVER	JUNE	7.2	3.3
47	10C	25U	H	2	A	2A	LIVER	JULY	13.7	5.5
48	10C	25U	H	2	A	2A	LIVER	SEPT	8.2	4.8
49	19C	19U	H	2	A	2A	LIVER	JUNE	6.1	4.4
50	19C	19U	H	2	A	2A	LIVER	JULY	9.5	10.6
51	19C	19U	H	2	A	2A	LIVER	SEPT	4.7	3.0
52	25C	23U	H	2	A	2A	LIVER	JUNE	2.4	3.2
53	25C	23U	H	2	A	2A	LIVER	JULY	11.3	4.4
54	25C	23U	H	2	A	2A	LIVER	SEPT	2.7	3.3
55	14C	22U	B	2	B	2B	LIVER	JUNE	3.7	4.6
56	14C	22U	B	2	B	2B	LIVER	JULY	13.6	4.0
57	14C	22U	B	2	B	2B	LIVER	SEPT	7.7	4.8
58	66C	18U	B	2	B	2B	LIVER	JUNE	5.3	3.6
59	66C	18U	B	2	B	2B	LIVER	JULY	13.3	6.3
60	66C	18U	B	2	B	2B	LIVER	SEPT	8.6	4.2
61	19S	20U	B	2	B	2B	LIVER	JUNE	5.5	5.3
62	19S	20U	B	2	B	2B	LIVER	JULY	12.1	3.6
63	19S	20U	B	2	B	2B	LIVER	SEPT	6.9	4.6
64	60C	11U	B	2	B	2B	LIVER	JUNE	4.5	2.5
65	60C	11U	B	2	B	2B	LIVER	JULY	.	3.4
66	60C	11U	B	2	B	2B	LIVER	SEPT	2.4	3.2
67	63C	27U	H	2	B	2B	LIVER	JUNE	5.4	2.0
68	63C	27U	H	2	B	2B	LIVER	JULY	9.6	4.7
69	63C	27U	H	2	B	2B	LIVER	SEPT	7.6	4.1
70	6R	3U	H	2	B	2B	LIVER	JUNE	6.0	1.0
71	6R	3U	H	2	B	2B	LIVER	JULY	12.5	7.0
72	6R	3U	H	2	B	2B	LIVER	SEPT	3.0	4.9
73	65C	21U	B	3	A	3A	LIVER	JUNE	4.2	1.7
74	65C	21U	B	3	A	3A	LIVER	JULY	.	.
75	65C	21U	B	3	A	3A	LIVER	SEPT	5.6	2.8
76	2N	5U	B	3	A	3A	LIVER	JUNE	3.4	1.8
77	2N	5U	B	3	A	3A	LIVER	JULY	12.1	2.9
78	2N	5U	B	3	A	3A	LIVER	SEPT	5.1	7.6
79	40C	45U	B	3	A	3A	LIVER	JUNE	3.9	1.3
80	40C	45U	B	3	A	3A	LIVER	JULY	5.8	4.1
81	40C	45U	B	3	A	3A	LIVER	SEPT	2.8	5.3
82	64C	14U	B	3	A	3A	LIVER	JUNE	4.1	2.7
83	64C	14U	B	3	A	3A	LIVER	JULY	12.6	3.1
84	64C	14U	B	3	A	3A	LIVER	SEPT	6.5	4.2
85	21C	38U	H	3	A	3A	LIVER	JUNE	3.9	4.3
86	21C	38U	H	3	A	3A	LIVER	JULY	11.8	6.9
87	21C	38U	H	3	A	3A	LIVER	SEPT	2.6	3.6
88	93C	40U	H	3	A	3A	LIVER	JUNE	7.6	2.8
89	93C	40U	H	3	A	3A	LIVER	JULY	10.7	3.6
90	93C	40U	H	3	A	3A	LIVER	SEPT	6.6	2.1

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLMOL	CALFLMOL
91	67C	6U	H	3	A	3A	LIVER	JUNE	3.1	2.6
92	67C	6U	H	3	A	3A	LIVER	JULY	8.0	5.9
93	67C	6U	H	3	A	3A	LIVER	SEPT	2.4	5.5
94	13C	4U	B	3	B	3B	LIVER	JUNE	2.6	2.9
95	13C	4U	B	3	B	3B	LIVER	JULY	10.3	9.5
96	13C	4U	B	3	B	3B	LIVER	SEPT	6.9	3.5
97	22C	37U	B	3	B	3B	LIVER	JUNE	3.7	2.6
98	22C	37U	B	3	B	3B	LIVER	JULY	14.3	2.6
99	22C	37U	B	3	B	3B	LIVER	SEPT	4.6	5.6
100	6S	1U	B	3	B	3B	LIVER	JUNE	9.6	3.7
101	6S	1U	B	3	B	3B	LIVER	JULY	14.5	5.0
102	6S	1U	B	3	B	3B	LIVER	SEPT	4.4	2.8
103	52C	8U	B	3	B	3B	LIVER	JUNE	7.0	2.7
104	52C	8U	B	3	B	3B	LIVER	JULY	13.7	4.7
105	52C	8U	B	3	B	3B	LIVER	SEPT	7.1	3.2
106	23C	7U	B	3	B	3B	LIVER	JUNE	6.6	3.0
107	23C	7U	B	3	B	3B	LIVER	JULY	11.7	10.2
108	23C	7U	B	3	B	3B	LIVER	SEPT	3.3	2.6
109	75C	35U	H	3	B	3B	LIVER	JUNE	4.5	1.0
110	75C	35U	H	3	B	3B	LIVER	JULY	9.2	7.3
111	75C	35U	H	3	B	3B	LIVER	SEPT	6.9	6.2

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLFE	CALFLFE
1	5C	24U	B	1	A	1A	LIVER	JUNE	366.6	200.8
2	5C	24U	B	1	A	1A	LIVER	JULY	456.0	212.0
3	5C	24U	B	1	A	1A	LIVER	SEPT	323.0	219.7
4	6C	29U	B	1	A	1A	LIVER	JUNE	383.0	309.4
5	6C	29U	B	1	A	1A	LIVER	JULY	483.9	359.7
6	6C	29U	B	1	A	1A	LIVER	SEPT	399.3	310.4
7	12C	28U	B	1	A	1A	LIVER	JUNE	361.4	473.7
8	12C	28U	B	1	A	1A	LIVER	JULY	467.8	466.0
9	12C	28U	B	1	A	1A	LIVER	SEPT	514.8	244.2
10	56C	13U	H	1	A	1A	LIVER	JUNE	630.5	361.2
11	56C	13U	H	1	A	1A	LIVER	JULY	604.0	391.2
12	56C	13U	H	1	A	1A	LIVER	SEPT	503.1	318.2
13	74C	34U	H	1	A	1A	LIVER	JUNE	341.6	287.0
14	74C	34U	H	1	A	1A	LIVER	JULY	300.8	354.4
15	74C	34U	H	1	A	1A	LIVER	SEPT	298.4	354.5
16	11C	2U	H	1	A	1A	LIVER	JUNE	287.9	288.1
17	11C	2U	H	1	A	1A	LIVER	JULY	451.6	293.4
18	11C	2U	H	1	A	1A	LIVER	SEPT	240.5	268.0
19	33C	41U	B	1	B	1B	LIVER	JUNE	218.8	367.5
20	33C	41U	B	1	B	1B	LIVER	JULY	230.9	316.4
21	33C	41U	B	1	B	1B	LIVER	SEPT	290.7	135.3
22	16C	17U	B	1	B	1B	LIVER	JUNE	304.4	261.4
23	16C	17U	B	1	B	1B	LIVER	JULY	338.7	266.8
24	16C	17U	B	1	B	1B	LIVER	SEPT	469.8	353.9
25	17C	9U	B	1	B	1B	LIVER	JUNE	395.5	220.2
26	17C	9U	B	1	B	1B	LIVER	JULY	343.9	296.3
27	17C	9U	B	1	B	1B	LIVER	SEPT	321.7	334.8
28	2P	10U	B	1	B	1B	LIVER	JUNE	272.1	318.1
29	2P	10U	B	1	B	1B	LIVER	JULY	407.1	317.0
30	2P	10U	B	1	B	1B	LIVER	SEPT	243.6	417.9
31	32C	30U	H	1	B	1B	LIVER	JUNE	403.4	227.9
32	32C	30U	H	1	B	1B	LIVER	JULY	468.3	115.7
33	32C	30U	H	1	B	1B	LIVER	SEPT	324.6	229.8
34	72C	15U	H	1	B	1B	LIVER	JUNE	359.9	173.4
35	72C	15U	H	1	B	1B	LIVER	JULY	267.8	296.5
36	72C	15U	H	1	B	1B	LIVER	SEPT	312.2	370.5
37	29C	36U	B	2	A	2A	LIVER	JUNE	293.2	319.0
38	29C	36U	B	2	A	2A	LIVER	JULY	294.0	375.6
39	29C	36U	B	2	A	2A	LIVER	SEPT	410.5	375.3
40	35C	39U	B	2	A	2A	LIVER	JUNE	271.2	308.7
41	35C	39U	B	2	A	2A	LIVER	JULY	429.2	391.3
42	35C	39U	B	2	A	2A	LIVER	SEPT	417.2	278.8
43	18C	12U	B	2	A	2A	LIVER	JUNE	281.1	133.2
44	18C	12U	B	2	A	2A	LIVER	JULY	353.9	238.5

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLFE	CALFLFE
45	18C	12U	B	2	A	2A	LIVER	SEPT	271.7	264.7
46	10C	25U	H	2	A	2A	LIVER	JUNE	328.5	271.8
47	10C	25U	H	2	A	2A	LIVER	JULY	371.4	375.1
48	10C	25U	H	2	A	2A	LIVER	SEPT	380.0	344.9
49	19C	19U	H	2	A	2A	LIVER	JUNE	486.1	281.7
50	19C	19U	H	2	A	2A	LIVER	JULY	361.5	306.0
51	19C	19U	H	2	A	2A	LIVER	SEPT	386.3	340.0
52	25C	23U	H	2	A	2A	LIVER	JUNE	412.5	434.4
53	25C	23U	H	2	A	2A	LIVER	JULY	484.2	302.2
54	25C	23U	H	2	A	2A	LIVER	SEPT	451.1	352.1
55	14C	22U	B	2	B	2B	LIVER	JUNE	446.4	316.1
56	14C	22U	B	2	B	2B	LIVER	JULY	479.7	326.6
57	14C	22U	B	2	B	2B	LIVER	SEPT	479.4	325.3
58	66C	18U	B	2	B	2B	LIVER	JUNE	379.0	211.8
59	66C	18U	B	2	B	2B	LIVER	JULY	335.2	240.1
60	66C	18U	B	2	B	2B	LIVER	SEPT	344.2	341.3
61	19S	20U	B	2	B	2B	LIVER	JUNE	356.3	138.7
62	19S	20U	B	2	B	2B	LIVER	JULY	349.0	225.3
63	19S	20U	B	2	B	2B	LIVER	SEPT	422.0	318.0
64	60C	11U	B	2	B	2B	LIVER	JUNE	526.9	170.7
65	60C	11U	B	2	B	2B	LIVER	JULY	.	341.0
66	60C	11U	B	2	B	2B	LIVER	SEPT	400.9	356.3
67	63C	27U	H	2	B	2B	LIVER	JUNE	265.6	191.8
68	63C	27U	H	2	B	2B	LIVER	JULY	256.7	349.4
69	63C	27U	H	2	B	2B	LIVER	SEPT	380.6	314.2
70	6R	3U	H	2	B	2B	LIVER	JUNE	417.2	268.8
71	6R	3U	H	2	B	2B	LIVER	JULY	251.7	214.4
72	6R	3U	H	2	B	2B	LIVER	SEPT	364.1	360.8
73	65C	21U	B	3	A	3A	LIVER	JUNE	475.7	345.9
74	65C	21U	B	3	A	3A	LIVER	JULY	.	.
75	65C	21U	B	3	A	3A	LIVER	SEPT	453.6	311.9
76	2N	5U	B	3	A	3A	LIVER	JUNE	454.7	257.9
77	2N	5U	B	3	A	3A	LIVER	JULY	560.6	426.7
78	2N	5U	B	3	A	3A	LIVER	SEPT	453.9	242.5
79	40C	45U	B	3	A	3A	LIVER	JUNE	376.8	378.2
80	40C	45U	B	3	A	3A	LIVER	JULY	268.3	364.2
81	40C	45U	B	3	A	3A	LIVER	SEPT	394.5	329.4
82	64C	14U	B	3	A	3A	LIVER	JUNE	323.3	250.5
83	64C	14U	B	3	A	3A	LIVER	JULY	341.6	263.7
84	64C	14U	B	3	A	3A	LIVER	SEPT	348.5	293.5
85	21C	38U	H	3	A	3A	LIVER	JUNE	256.1	183.7
86	21C	38U	H	3	A	3A	LIVER	JULY	282.0	159.5
87	21C	38U	H	3	A	3A	LIVER	SEPT	330.5	337.2
88	93C	40U	H	3	A	3A	LIVER	JUNE	374.1	225.4
89	93C	40U	H	3	A	3A	LIVER	JULY	502.7	291.6

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLFE	CALFLFE
90	93C	40U	H	3	A	3A	LIVER	SEPT	357.0	267.4
91	67C	6U	H	3	A	3A	LIVER	JUNE	284.1	326.1
92	67C	6U	H	3	A	3A	LIVER	JULY	389.2	295.7
93	67C	6U	H	3	A	3A	LIVER	SEPT	385.6	332.5
94	13C	4U	B	3	B	3B	LIVER	JUNE	302.6	223.7
95	13C	4U	B	3	B	3B	LIVER	JULY	246.4	246.4
96	13C	4U	B	3	B	3B	LIVER	SEPT	280.1	243.5
97	22C	37U	B	3	B	3B	LIVER	JUNE	349.6	274.5
98	22C	37U	B	3	B	3B	LIVER	JULY	276.3	424.0
99	22C	37U	B	3	B	3B	LIVER	SEPT	442.4	346.4
100	6S	1U	B	3	B	3B	LIVER	JUNE	269.6	200.7
101	6S	1U	B	3	B	3B	LIVER	JULY	309.5	330.1
102	6S	1U	B	3	B	3B	LIVER	SEPT	378.9	351.6
103	52C	8U	B	3	B	3B	LIVER	JUNE	220.8	335.0
104	52C	8U	B	3	B	3B	LIVER	JULY	356.1	390.4
105	52C	8U	B	3	B	3B	LIVER	SEPT	269.2	328.2
106	23C	7U	B	3	B	3B	LIVER	JUNE	288.9	113.5
107	23C	7U	B	3	B	3B	LIVER	JULY	429.8	290.6
108	23C	7U	B	3	B	3B	LIVER	SEPT	350.5	238.9
109	75C	35U	H	3	B	3B	LIVER	JUNE	393.2	677.2
110	75C	35U	H	3	B	3B	LIVER	JULY	372.3	571.9
111	75C	35U	H	3	B	3B	LIVER	SEPT	291.3	686.2

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLZN	CALFLZN
1	5C	24U	B	1	A	1A	LIVER	JUNE	304.0	205.0
2	5C	24U	B	1	A	1A	LIVER	JULY	86.4	93.5
3	5C	24U	B	1	A	1A	LIVER	SEPT	231.4	103.8
4	6C	29U	B	1	A	1A	LIVER	JUNE	666.0	438.4
5	6C	29U	B	1	A	1A	LIVER	JULY	144.9	58.8
6	6C	29U	B	1	A	1A	LIVER	SEPT	116.0	100.0
7	12C	28U	B	1	A	1A	LIVER	JUNE	398.1	144.7
8	12C	28U	B	1	A	1A	LIVER	JULY	318.4	98.9
9	12C	28U	B	1	A	1A	LIVER	SEPT	154.9	120.8
10	56C	13U	H	1	A	1A	LIVER	JUNE	485.7	24.7
11	56C	13U	H	1	A	1A	LIVER	JULY	307.8	99.0
12	56C	13U	H	1	A	1A	LIVER	SEPT	117.2	116.4
13	74C	34U	H	1	A	1A	LIVER	JUNE	201.2	195.8
14	74C	34U	H	1	A	1A	LIVER	JULY	95.6	119.5
15	74C	34U	H	1	A	1A	LIVER	SEPT	129.2	127.6
16	11C	2U	H	1	A	1A	LIVER	JUNE	45.0	192.2
17	11C	2U	H	1	A	1A	LIVER	JULY	106.1	115.8
18	11C	2U	H	1	A	1A	LIVER	SEPT	118.9	135.0
19	33C	41U	B	1	B	1B	LIVER	JUNE	126.1	241.4
20	33C	41U	B	1	B	1B	LIVER	JULY	113.4	149.0
21	33C	41U	B	1	B	1B	LIVER	SEPT	121.1	125.3
22	16C	17U	B	1	B	1B	LIVER	JUNE	22.4	148.4
23	16C	17U	B	1	B	1B	LIVER	JULY	76.9	76.1
24	16C	17U	B	1	B	1B	LIVER	SEPT	115.9	103.1
25	17C	9U	B	1	B	1B	LIVER	JUNE	126.3	116.2
26	17C	9U	B	1	B	1B	LIVER	JULY	118.8	82.9
27	17C	9U	B	1	B	1B	LIVER	SEPT	123.5	138.0
28	2P	10U	B	1	B	1B	LIVER	JUNE	156.6	144.6
29	2P	10U	B	1	B	1B	LIVER	JULY	83.5	124.7
30	2P	10U	B	1	B	1B	LIVER	SEPT	123.3	170.7
31	32C	30U	H	1	B	1B	LIVER	JUNE	212.8	169.4
32	32C	30U	H	1	B	1B	LIVER	JULY	155.8	114.8
33	32C	30U	H	1	B	1B	LIVER	SEPT	124.1	144.3
34	72C	15U	H	1	B	1B	LIVER	JUNE	186.6	147.5
35	72C	15U	H	1	B	1B	LIVER	JULY	118.5	172.9
36	72C	15U	H	1	B	1B	LIVER	SEPT	186.6	195.9
37	29C	36U	B	2	A	2A	LIVER	JUNE	96.3	22.3
38	29C	36U	B	2	A	2A	LIVER	JULY	103.8	79.3
39	29C	36U	B	2	A	2A	LIVER	SEPT	147.3	110.8
40	35C	39U	B	2	A	2A	LIVER	JUNE	45.7	105.0
41	35C	39U	B	2	A	2A	LIVER	JULY	144.6	108.6
42	35C	39U	B	2	A	2A	LIVER	SEPT	163.3	173.3
43	18C	12U	B	2	A	2A	LIVER	JUNE	161.9	219.5
44	18C	12U	B	2	A	2A	LIVER	JULY	110.1	71.3
45	18C	12U	B	2	A	2A	LIVER	SEPT	200.7	132.1
46	10C	25U	H	2	A	2A	LIVER	JUNE	152.2	81.5

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLZN	CALFLZN
47	10C	25U	H	2	A	2A	LIVER	JULY	103.9	78.6
48	10C	25U	H	2	A	2A	LIVER	SEPT	185.9	103.3
49	19C	19U	H	2	A	2A	LIVER	JUNE	259.5	105.3
50	19C	19U	H	2	A	2A	LIVER	JULY	77.1	125.0
51	19C	19U	H	2	A	2A	LIVER	SEPT	119.9	128.1
52	25C	23U	H	2	A	2A	LIVER	JUNE	131.0	127.2
53	25C	23U	H	2	A	2A	LIVER	JULY	187.9	109.7
54	25C	23U	H	2	A	2A	LIVER	SEPT	168.2	199.0
55	14C	22U	B	2	B	2B	LIVER	JUNE	236.9	83.2
56	14C	22U	B	2	B	2B	LIVER	JULY	107.2	82.7
57	14C	22U	B	2	B	2B	LIVER	SEPT	129.5	122.8
58	66C	18U	B	2	B	2B	LIVER	JUNE	114.9	124.6
59	66C	18U	B	2	B	2B	LIVER	JULY	131.0	179.1
60	66C	18U	B	2	B	2B	LIVER	SEPT	115.2	123.7
61	19S	20U	B	2	B	2B	LIVER	JUNE	106.1	415.8
62	19S	20U	B	2	B	2B	LIVER	JULY	176.7	105.4
63	19S	20U	B	2	B	2B	LIVER	SEPT	235.8	115.3
64	60C	11U	B	2	B	2B	LIVER	JUNE	184.9	60.0
65	60C	11U	B	2	B	2B	LIVER	JULY	.	77.0
66	60C	11U	B	2	B	2B	LIVER	SEPT	122.6	155.6
67	63C	27U	H	2	B	2B	LIVER	JUNE	100.1	137.6
68	63C	27U	H	2	B	2B	LIVER	JULY	140.2	91.3
69	63C	27U	H	2	B	2B	LIVER	SEPT	161.5	151.8
70	6R	3U	H	2	B	2B	LIVER	JUNE	212.7	170.2
71	6R	3U	H	2	B	2B	LIVER	JULY	169.2	304.8
72	6R	3U	H	2	B	2B	LIVER	SEPT	133.5	122.6
73	65C	21U	B	3	A	3A	LIVER	JUNE	40.7	247.2
74	65C	21U	B	3	A	3A	LIVER	JULY	.	.
75	65C	21U	B	3	A	3A	LIVER	SEPT	102.7	110.8
76	2N	5U	B	3	A	3A	LIVER	JUNE	425.6	110.4
77	2N	5U	B	3	A	3A	LIVER	JULY	129.9	128.8
78	2N	5U	B	3	A	3A	LIVER	SEPT	117.9	124.0
79	40C	45U	B	3	A	3A	LIVER	JUNE	48.2	379.3
80	40C	45U	B	3	A	3A	LIVER	JULY	133.2	146.9
81	40C	45U	B	3	A	3A	LIVER	SEPT	191.9	156.6
82	64C	14U	B	3	A	3A	LIVER	JUNE	195.7	82.6
83	64C	14U	B	3	A	3A	LIVER	JULY	193.6	115.4
84	64C	14U	B	3	A	3A	LIVER	SEPT	122.9	141.6
85	21C	38U	H	3	A	3A	LIVER	JUNE	102.0	232.5
86	21C	38U	H	3	A	3A	LIVER	JULY	152.4	105.7
87	21C	38U	H	3	A	3A	LIVER	SEPT	103.0	54.4
88	93C	40U	H	3	A	3A	LIVER	JUNE	190.5	195.3
89	93C	40U	H	3	A	3A	LIVER	JULY	76.2	149.9
90	93C	40U	H	3	A	3A	LIVER	SEPT	128.9	142.4
91	67C	6U	H	3	A	3A	LIVER	JUNE	103.7	143.8
92	67C	6U	H	3	A	3A	LIVER	JULY	103.3	105.3

OBS	COWID	CALFID	SEXCALF	TRTCOW	TRTCALF	GROUP	SOURCE	MONTH	COWLZN	CALFLZN
93	67C	6U	H	3	A	3A	LIVER	SEPT	111.2	189.2
94	13C	4U	B	3	B	3B	LIVER	JUNE	283.6	48.7
95	13C	4U	B	3	B	3B	LIVER	JULY	158.0	115.9
96	13C	4U	B	3	B	3B	LIVER	SEPT	103.6	114.2
97	22C	37U	B	3	B	3B	LIVER	JUNE	320.7	56.3
98	22C	37U	B	3	B	3B	LIVER	JULY	120.7	112.8
99	22C	37U	B	3	B	3B	LIVER	SEPT	135.4	127.8
100	6S	1U	B	3	B	3B	LIVER	JUNE	186.7	125.2
101	6S	1U	B	3	B	3B	LIVER	JULY	226.8	486.2
102	6S	1U	B	3	B	3B	LIVER	SEPT	118.6	134.0
103	52C	8U	B	3	B	3B	LIVER	JUNE	121.6	58.6
104	52C	8U	B	3	B	3B	LIVER	JULY	166.6	86.6
105	52C	8U	B	3	B	3B	LIVER	SEPT	128.8	127.9
106	23C	7U	B	3	B	3B	LIVER	JUNE	137.2	267.5
107	23C	7U	B	3	B	3B	LIVER	JULY	265.1	247.7
108	23C	7U	B	3	B	3B	LIVER	SEPT	131.3	161.8
109	75C	35U	H	3	B	3B	LIVER	JUNE	124.7	136.1
110	75C	35U	H	3	B	3B	LIVER	JULY	96.3	122.6
111	75C	35U	H	3	B	3B	LIVER	SEPT	116.4	124.3