BIOCHEMICAL AND CELLULAR PROFILES IN FEEDLOT CATTLE DURING NORMAL FEEDING TRIALS FOLLOWING TRANSPORT AND DURING RESPIRATORY DISEASE

by

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GENERAL INTRODUCTION

Very little published data is available on blood constituents in feedlot cattle and how these parameters vary with nonspecific stress and time on feed. The lack of published normal values impairs the clinical use of biochemical profiles in disease states.

Metabolic profiling in ruminants has been suggested as a means of evaluating general health of populations, assessing nutritional adequacy of rations, screening individuals for overt and occult disease and as a predictor of milk production or weight gains.

Stress in cattle has been well recognized as contributing significantly to susceptibility to disease and reduced weight gains. Disease outbreaks have been induced by weaning, transport, dehorning, castration and altered rations. It has been recognized that intensive production practices have resulted in a marked increase in certain disease syndromes in feedlot cattle including bloat, lactic acidosis, laminitis and hepatic abscesses.

Lipid metabolism in feedlot cattle with the exception of plasma FFA has received little attention in the literature. The effect of stress-related hormonal release on lipid metabolism and efficiency of feed utilization remains to be evaluated. The objective of this study is to determine an extensive profile including serum lipids and serum cortisol on feedlot cattle as they progress through feeding trials, following transport and in acute respiratory disease.

CHANGES IN BIOCHEMICAL AND CELLULAR PROFILES

DETERMINED BY SERIAL SAMPLING OF NORMAL FEEDLOT CATTLE

SUMMARY

Ten healthy feedlot cattle were sampled at intervals throughout the fattening period. An Extensive biochemical and cellular profile was determined including serum lipids and serum cortisol. The packed cell volume (PCV) increased throughout the feeding trial. Total lipids, cholesterol, triglycerides increased with time on feed while plasma free fatty acids decreased. D(-) and L(+) lactate levels reached their highest level early in the feeding period and then declined suggesting an adaption to high grain feeding. Urea nitrogen levels increased with time on feed but mean total protein levels decreased slightly. No significant changes during the feeding period included increased serum sodium, inorganic phosphate and creatinine levels and decreased levels of potassium and calcium.

INTRODUCTION

Hemalogic and biochemical parameters have been determined for feedlot cattle under a variety of experimental conditions[5,10,11,17,21,30,32,40,42,47]. However, reports of serial profiles during the feeding period are few in number and normal values are not well established[11,32,34,47].

Values for total serum lipids, triglycerides, cholesterol and plasma free fatty acids (FFA) have been reported[12,32,34]. No reports of phospholipids or complete lipid profiles could be found.

Serum proteins, glucose and urea nitrogen have been evaluated in feedlot cattle[14,19,32,34] but in only one report[38] were they included in a biochemical profile. Other blood parameters reported for feedlot cattle

include serum calcium, inorganic phosphate, magnesium, creatinine, serum alkaline phosphatase, protein electrophoresis and L(+) lactate[32,34]. No reports of D(-) lactate, sodium or potassium could be found.

Stuffelbean[47] evaluated beef heifers on 3 rations varying in digestible energy. Low energy diets resulted in decreased total reducing substances, uric acid, creatinine, cholesterol, glucuronic acid, PCV, hemoglobin, total leukocytes, glutathione and saccharides, while inorganic phosphate and potassium increased.

Metabolic profiles in dairy herds have been reported[3,4,35,36,37,43,44]. These profiles have been directed toward detecting general health of populations, assessing nutritional adequacy of rations, screening herds for occult disease and selection of stock with improved growth potential and milk production. Statistical analysis was based on identification of biochemical deviations from a population mean selected on the basis of lactational group, herd and season of the year.

Serum cortisol has been reported for hereford bulls on pasture but not for cattle on a feeding ration[41]. Plasma samples were measured. fluorometrically after separation by thin layer chromatography. Mean plasma concentrations of cortisol was 31.9 ng/ml. No reports of serum cortisol determined by radioimmunoassay could be found.

The purpose of the present study was to determine the relationship of biochemical and cellular profiles in healthy feedlot cattle as they progress through feeding trials.

MATERIALS AND METHODS

Animals: Ten 400 to 500 pound mixed breed beef steers were selected from a group entering the Kansas State University Beef Research Unit.

The animals originated from Texarkansas, Arkansas and arrived in December. They were initially placed on an 85% silage, 15% concentrate ration and switched to a 60% silage, 40% concentrate ration on January 22, 1976. In May they were placed on a finishing ration consisting of 85% concentrate and 15% silage.

Analysis: Blood samples were collected by jugular venipuncture on days 35, 125, 169, 208, and 249 from time of arrival. Whole blood was preserved with EDTA and analyzed within 2 hours. Clotted samples were cooled to 4 C immediately, separated from cells and frozen within 2 hours. On each sample the following parameters were measured: total leukocytes, leukocyte differential, PCV, total lipids, phospholipids, cholesterol, triglycerides, plasma FFA, serum cortisol, D(-) lactate, L(+) lactate, sodium, potassium, total CO_2, serum urea nitrogen, alkaline phosphatase, GPT, total protein, albumin, calcium, inorganic phosphate, glucose and creatinine.

The PCV, total leukocytes and differential leukocyte counts were determined by standard methods[45]. Total lipid determination was based on the sulfo-phosphovanillin reaction[7]. Phospholipids were determined by measuring phosphate liberated after digestion of with percholoric acid[2]. A spectrophometric method was used to measure plasma FFA[13]. One step enzymatic procedures were used to determine total cholesterol[a] and triglycerides[b] on an automated batch-type analyzer[c]. An enzymatic method was used to measure D(-) lactate[15] with D(-) lactic dehydrogenase obtained

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[a]Abbott Labs, Pasadena, Calif.
[b]Calbiochem, LaJolla, Calif.
[c]ABA-100 Abbott Labs, Pasadena, Calif.

commercially[d]. The procedure was modified for determination on a batch-type analyzer[c]. L(+) lactate was determined by an enzymatic method[b]. Serum cortisol was determined by the radioimmunoassay method of Abraham[1] as modified by Carter et al[8] employing antisera S6#3.

Other biochemical determinations were performed on a 12 channel sequential multiple analyzer[e] by following standard method[16], albumin by the bromcresol green method[20], calcium by complexing with cresolphthalein[28], inorganic phosphate by the phosphomolybdic acid method[26], creatinine by reaction with alkaline picrate[27], serum urea nitrogen by the diactyl-monoxine reaction[31], GPT by kinetic analysis[29], serum alkaline phosphatase enzymatically with p-Nitrophenyl phosphate as the substrate[33], total venous CO_2 by the phenolphthalein method[46], total protein by standard biuret method and sodium and potassium by flame photometry with an internal lithium standard.

RESULTS

Hemogram: The PCV increased ($P < .05$) during the feeding trial supporting previous data (Table 1)[23]. Total and differential leukocyte counts showed no significant change, however, mean neutrophil values increased with time on feed. Most results were consistent with published normal values except neutrophils and monocytes which were higher than previously reported.

Serum Lipids: Total serum lipids increased ($P < .01$) until the last sampling period when they decreased. These values did not agree with those

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[d] Sigma Chemical Co., St. Louis, Mo.
[e] Technicon Corporation, Tarrytown, N.Y.

obtained by the summation of component lipids (Cholesterol, Triglycerides, and Phospholipids). Cholesterol triglycerides and phospholipids decreased at day 167 and increased thereafter. Plasma FFA decreased throughout the feeding trial ($P < .01$) (Fig. 5).

Lactate: L(+) lactate increased significantly ($P < .01$) reaching its highest level at the second sampling and decreased thereafter (Fig. 2). D(-) lactate was not present in measurable quantities in most animals at the first sampling but increased to its highest level at the second sampling. A significant correlation was found between D(-) and L(+) lactate ($R = .95$).

Electrolytes and Total CO_2: Serum sodium increased significantly at day 125 ($P < .05$) and decreased thereafter while potassium values decreased ($P < .01$) throughout the feeding trial (Fig. 2). Mean serum calcium levels decreased ($P < .05$) from a mean of 9.74 on day 37 to a mean of 9.24 on day 249. Serum calcium showed a highly significant correlation with total serum protein and albumin. Inorganic phosphate increased ($P < .01$) during the feeding trial (Fig. 2). Venous total CO_2 decreased between day 167 and 208 ($P < .01$) and increased thereafter (Fig. 2). There was a significant negative correlation with D(-) lactate but not with L(+) lactate.

Serum Protein: Mean values for serum total protein decreased from 7.63 mg/dl to 7.17 mg/dl during the feeding trial, however, this was not significant at the .05 level of probability ($P = .10$). (Fig. 4). Little

change was noted in albumin values with mean values at the beginning and end of the trial almost identical (Fig. 4).

Serum Cortisol: No statistically significant change occurred in serum cortisol levels but mean values increased slightly throughout the trial (Fig. 5).

Other Biochemical Parameters: Serum urea nitrogen increased significantly during the feeding trial (Fig. 3). A marked increase was noted between the first and second sampling with mean values increasing from 4.7 to 22.5 mg/dl with no change in the creatinine values (Fig. 3). Creatinine increased significantly with time on feed ($P < .01$). No significant change was noted in serum glucose levels, however, mean values were considerably higher than published normal values[45] (Fig. 3). Mean values for serum alkaline phosphatase and serum GPT increased ($P < .01$) early but decreased later in the feeding trial (Fig. 4).

DISCUSSION

Significant changes have been found for total lipids, triglycerides, cholesterol, phospholipids, plasma FFA, D(-) lactate, L(+) lactate, sodium, potassium, total CO_2, urea nitrogen, alkaline phosphatase, GPT, calcium, inorganic phosphate, and creatinine in blood samples from mixed breed beef steers under feedlot conditions during a 249 day feeding trial. All serum lipids measured (total lipids, triglycerides, cholesterol, phospholipids) except for plasma FFA increased significantly. Total lipids determined by the sulfophospho-vanillin reaction greatly exceeded the sum of triglycerides, cholesterol, phospholipids and plasma FFA

although the results of this reaction correlate well with gravimetric and summation techniques in man. Since the color measured in the phospho-vanillin reaction is apparently directly related to the concentration of lipid compounds containing at least one double bond, it appears some unidentified compound of bovine serum reacts to yield a falsely high concentration.

The significance of the decrease in triglycerides, cholesterol and phospholipids on day 16 is unknown but was noted in previously reported feeding trial data[32]. The increase in triglycerides, cholesterol, and phospholipids after day 167 may represent increased hepatic synthesis from precursors supplied in the fattening ration.

Plasma FFA have been extensively investigated in the bovine species[11,24,34,40]. The present data provides further support for the hypothesis that as the level of feeding increases plasma FFA decreases. This reflects reduced mobilization and increased esterification of fatty acids in adipose tissue.

The sharp initial rise in L(+) lactate levels has been reported by others[34]. Olumeyan[34] suggested this rise is due to increased production of this metabolite by rumen micro-organisms in cattle placed on a high grain ration. Decline in lactate levels later in the feeding trial may represent an adaptive mechanism on the part of the animal[34]. It has been found that high concentrate rations stimulate the proliferation of lactate-utilizing micro-organisms and also increased tissue metabolism of L(+) lactate[6,25]. L(+) lactate is readily converted to glucose by the liver but D(-) lactate is not metabolized and is excreted in the urine[6].

A positive correlation of urea nitrogen with time on feed and dietary protein intake has been reported[12,14,19,39]. Harrison[19] suggested that urea nitrogen in feedlot cattle greater than 10 mg/dl indicated adequate protein intake whereas levels between 7 and 9 mg/dl represented inadequate intake. Our data shows a mean level of urea nitrogen on day 37 of $4.7 \pm .82$ with a 5 fold increase at the second sampling. It is not known whether this was a progressive increase over time or a transient change on the day of sampling. The animals had normal access to water on that day and the weather was mild. No evidence of hemoconcentration was present. The protein source was soybean oil meal and no urea was fed.

Several investigators have reported a positive correlation of total serum protein with time on feed and with protein content of ration. Although changes in total protein were not significant in the present study ($P = .10$). Mean values decreased throughout the feeding trial with the exception of day 208. Hemoconcentration may have occurred on this date due to high environmental temperatures (note increase PCV-Table 1).

Statistically significant changes were not observed in serum cortisol levels but mean values increased throughout the feeding trial. No normal values for cortisol determined by radioimmunoassay could be found and normal values by other methods vary quite widely[41,49]. In this study acute stress induced by herding, crowding, weighing and bleeding of the animals over a one to two hour period may have elevated serum values. Willette et al[49] found as much as a five fold increase in plasma cortisol levels over a 10 minute period due to acute stress, however his baseline values were considerably lower than those reported elsewhere. Results

in the present study probably reflect more closely normal values for cattle sampled under field conditions.

The present study provides some normal values for a variety of biochemical tests in feedlot cattle and shows how these parameters vary with time on feed. Future work should be directed toward using specific tests or groups of tests to detect inadequacy of rations, occult and overt disease, production stress and as a predictor of growth potential.

TABLES AND FIGURES

PAPER I

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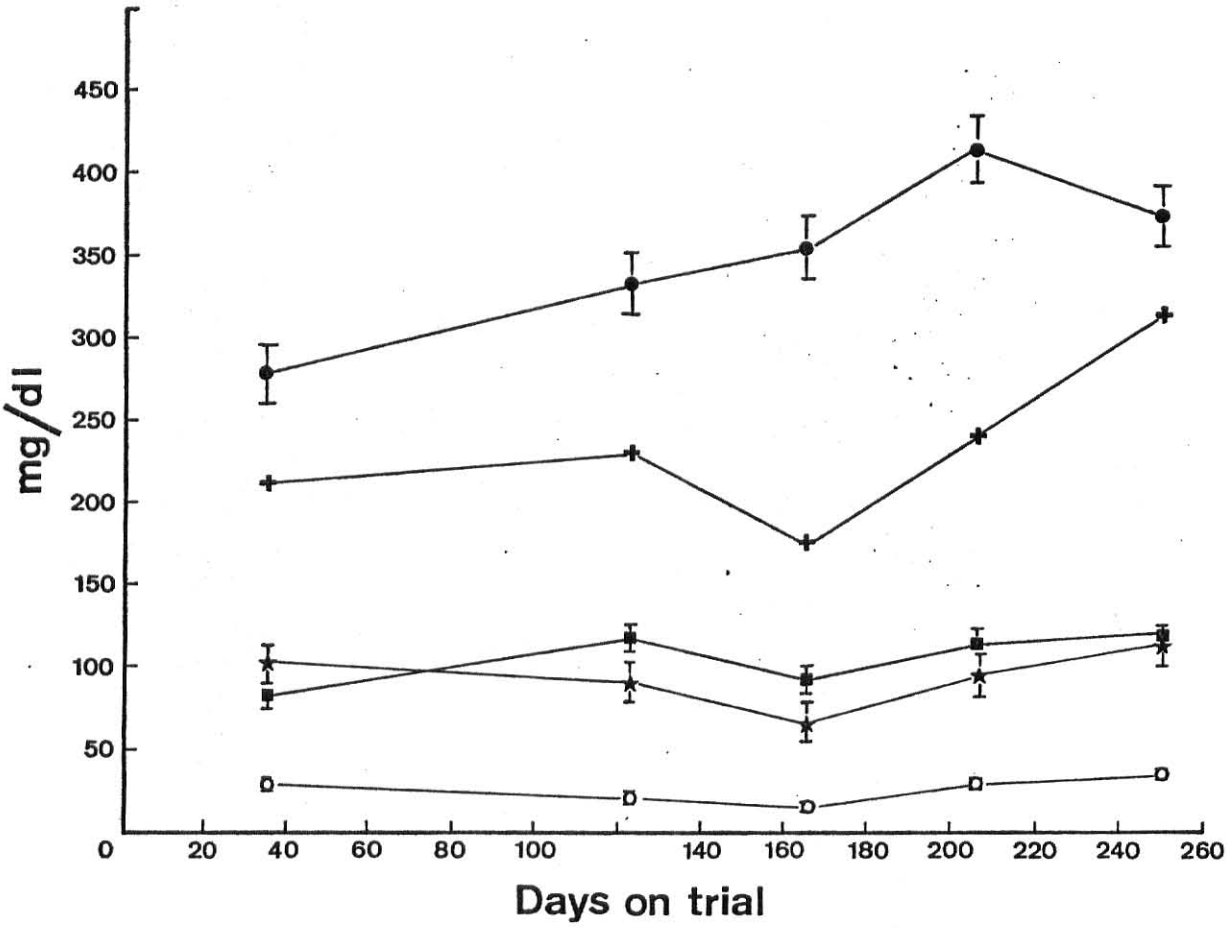

FIGURE 1 SERIAL MEANS AND STANDARD DEVIATIONS

DURING FEEDING TRIALS FOR TOTAL LIPIDS ●——●
TRIGLYCERIDES o——o CHOLESTEROL ■——■
PHOSPHOLIPIDS ★——★ AND SUMMATION +——+

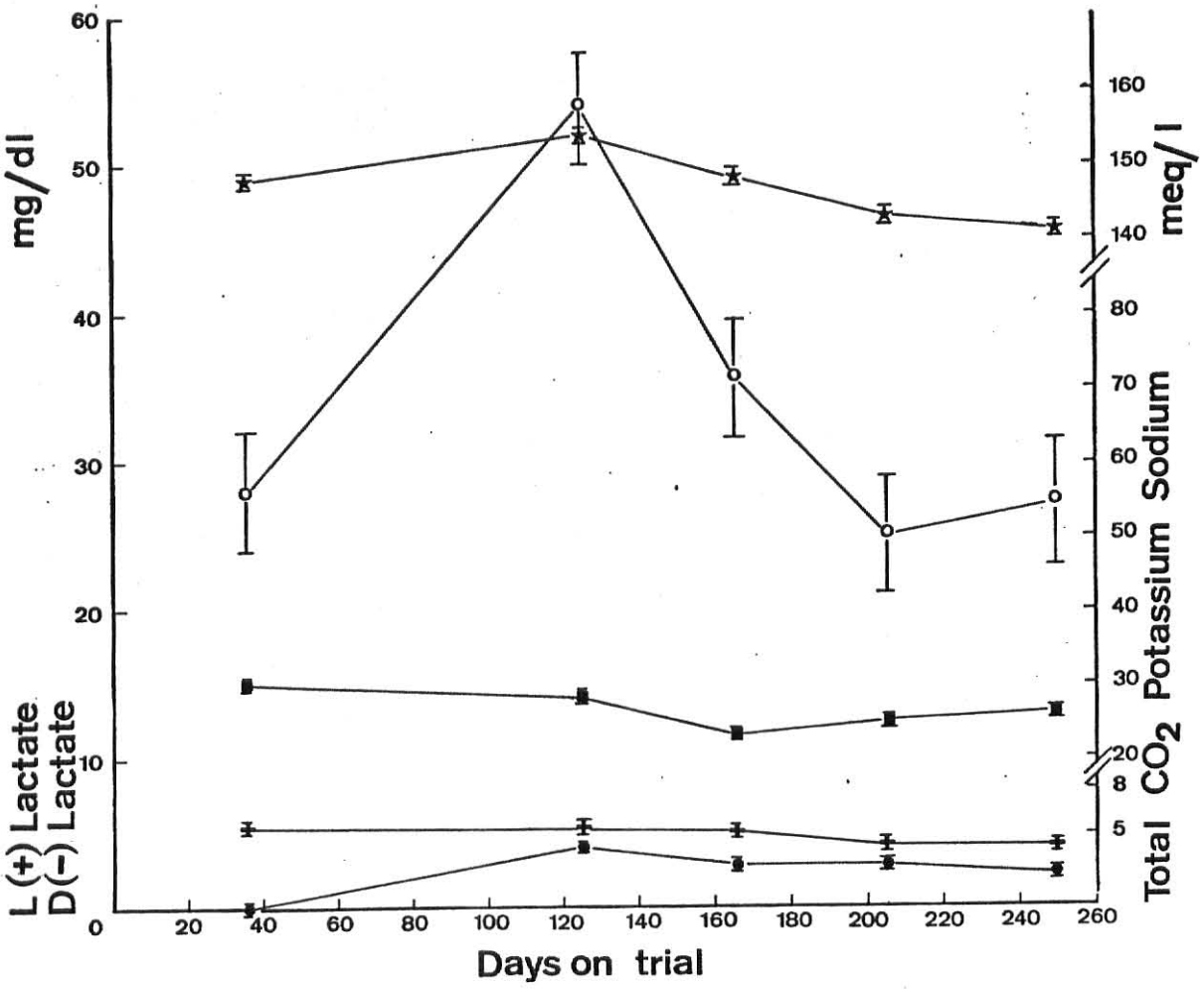

FIGURE 2 SERIAL MEANS AND STANDARD DEVIATIONS

DURING FEEDING TRIALS FOR D(-) LACTATE ●——● L(+) LACTATE o——o TOTAL CO_2 ■——■ SODIUM ★——★ AND POTASSIUM +——+

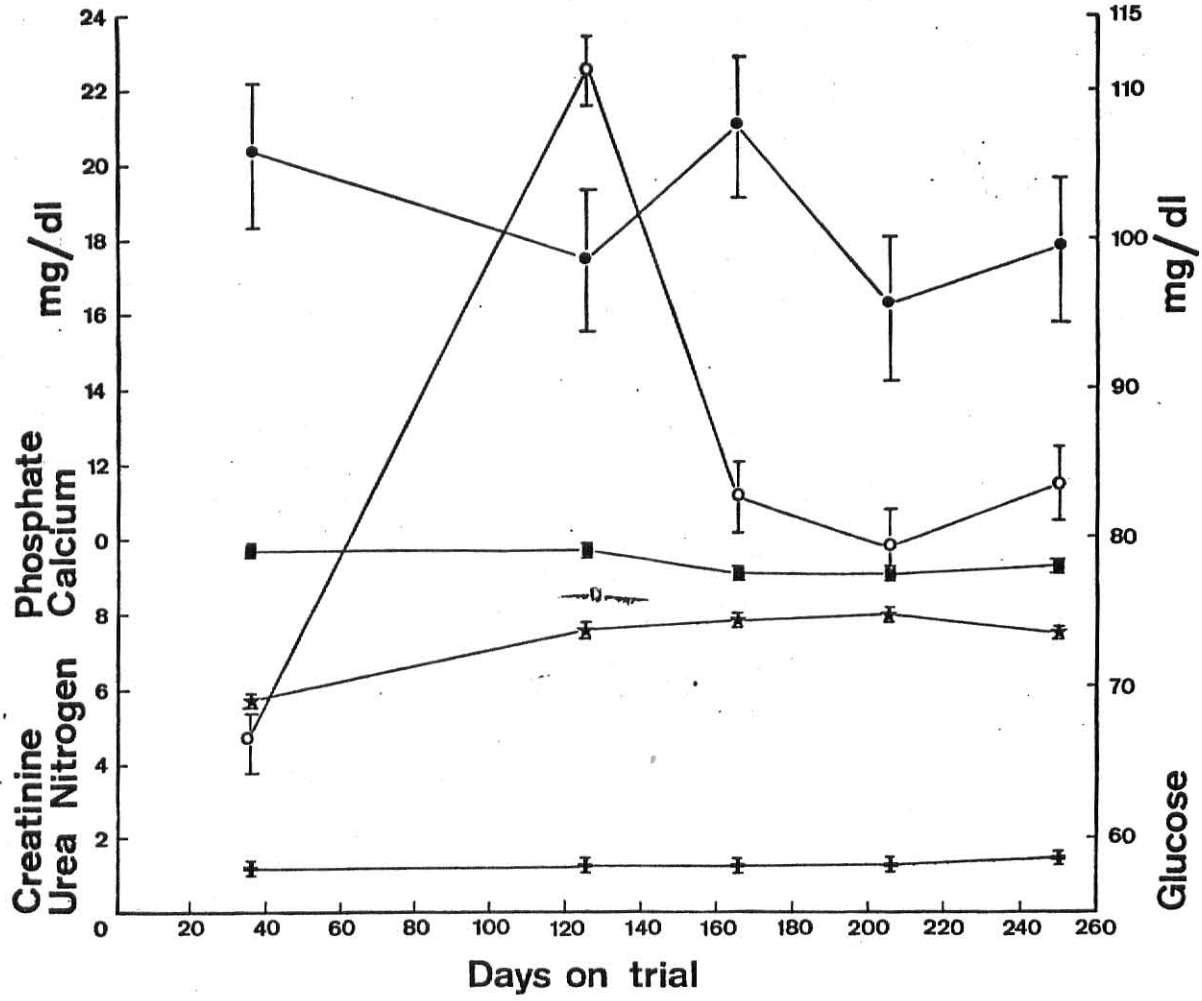

FIGURE 3 SERIAL MEANS AND STANDARD DEVIATIONS

DURING FEEDING TRIALS FOR GLUCOSE ●——●
UREA NITROGEN o——o SERUM CALCIUM ■——■
INORGANIC PHOSPHATE ★——★ AND CREATININE +——+

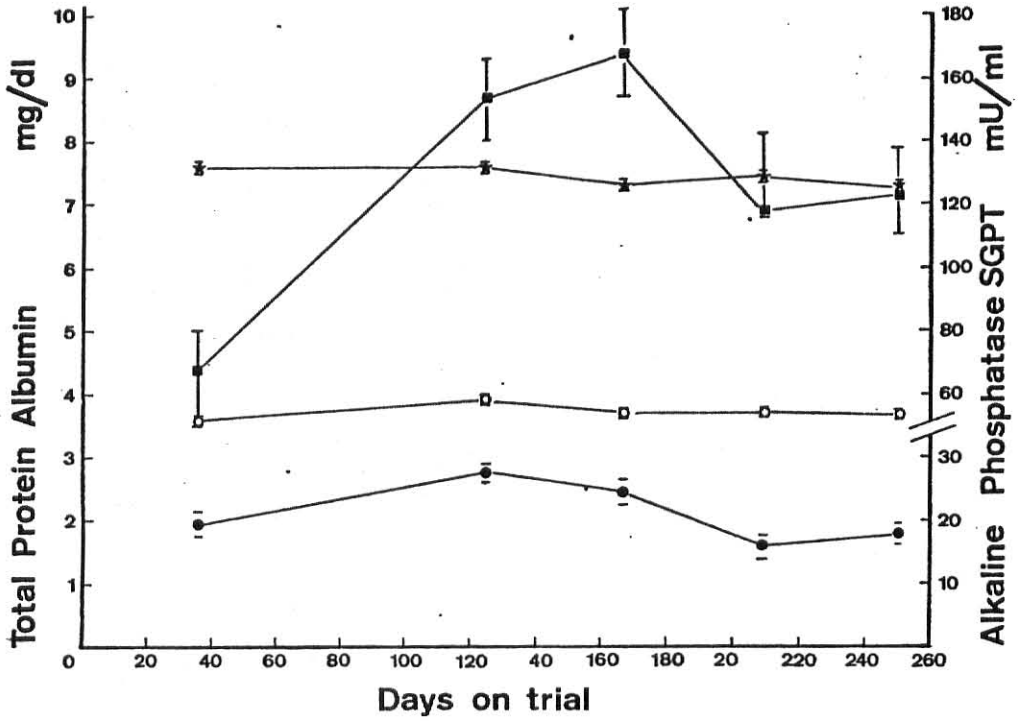

FIGURE 4 SERIAL MEANS AND STANDARD DEVIATIONS

DURING FEEDING TRIALS FOR GPT ●——●
ALBUMEN o——o ALKALINE PHOSPHATASE ■——■
AND TOTAL PROTEIN ★——★

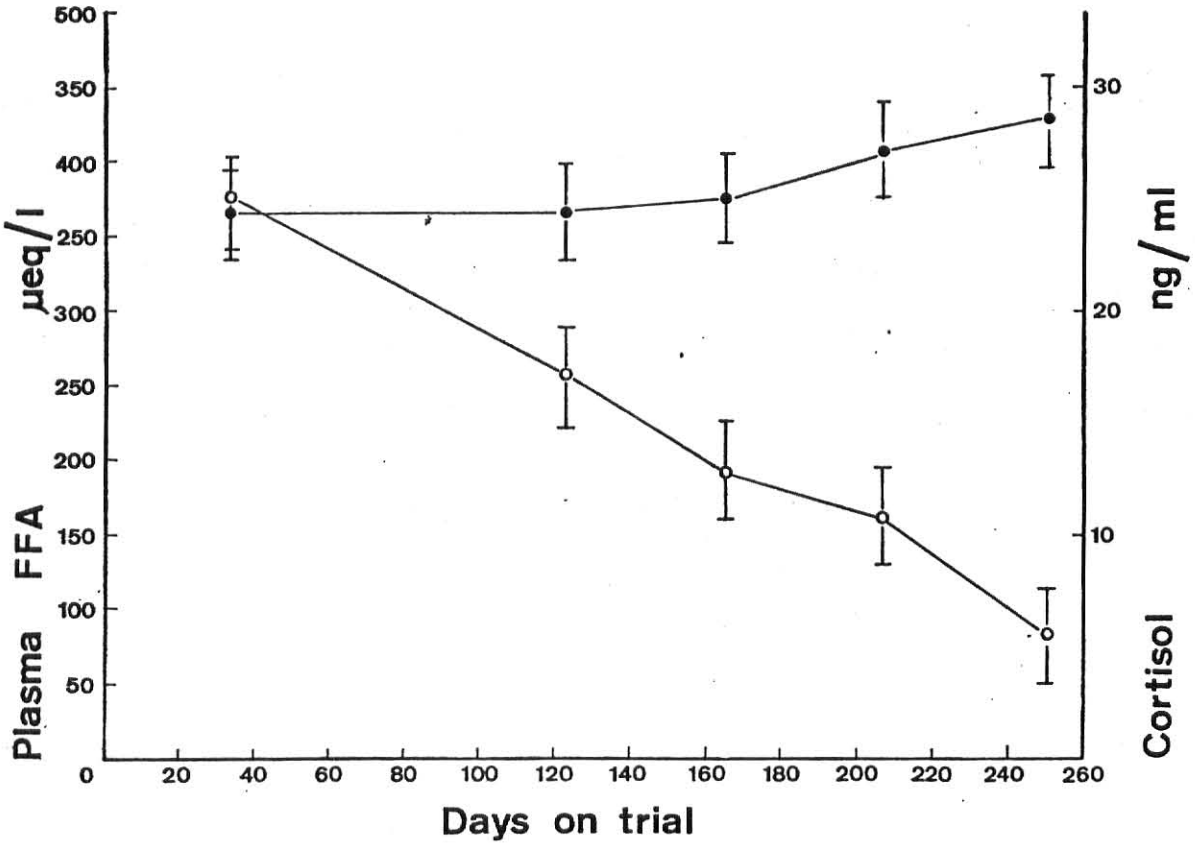

FIGURE 5 SERIAL MEANS AND STANDARD DEVIATIONS

DURING FEEDING TRIALS FOR SERUM CORTISOL ●——● AND PLASMA FFA o——o

TABLE 1

SERIAL MEANS AND STANDARD DEVIATIONS
FOR HEMATOLOGIC VALUES FOR 10 FEEDLOT STEERS

	Units	Day 37	Day 125	Day 167	Day 208	Day 249
PCV	Vol %	36.8 ± .75	37.4 ± .74	36.5 ± .83	39.7 ± .72	37.6 ± .72
Leukocytes	/ul	9,071 ± 2,200	11,313 ± 2,350	11,919 ± 2,000	10,680 ± 2,100	10,630 ± 2,050
Bands	/ul	30 ± 42	27 ± 40	15 ± 42	0	28 ± 0-
Segmented Neutrophils	/ul	3,037 ± 2,190	4,030 ± 1,670	4,111 ± 1,420	2,647 ± 1,300	2,822 ± 1,015
Lymphocytes	/ul	5,880 ± 1,460	5,857 ± 1,750	6,089 ± 1,870	6,548 ± 1,390	6,370 ± 1,450
Monocytes	/ul	738 ± 400	1,056 ± 330	1,023 ± 630	1,158 ± 600	1,214 ± 415
Eosinophils	/ul	61 ± 60	87 ± 130	153 ± 120	272 ± 215	191 ± 146
Basophils	/ul	53 ± 30	40 ± 48	46 ± 56	42 ± 43	0 ± 42

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TRANSPORT AND RESPIRATORY DISEASE AMONG FEEDLOT CATTLE RELATED TO CHANGES IN BIOCHEMICAL AND CELLULAR PROFILES

SUMMARY

Feedlot steers were sampled post-transport and in naturally occurring respiratory disease and an extensive biochemical and cellular profile was performed. Sampling was repeated one month later and results compared to initial samples and to values for normal feedlot cattle. Hematologic changes included signs of hemoconcentration in the post-transport group and signs of an inflammatory response in the sick group. Phospholipids were significantly decreased in both groups in the initial sampling. Mean serum cortisol levels were not different from normal in the post-transport group perhaps due to the delay in sampling. Serum cortisol in the sick group was significantly increased suggesting that cortisol concentration is a good indicator of acute stress in feedlot cattle.

INTRODUCTION

Transport and disease as causes of stress in feedlot cattle have been well recognized. The observable effects include depression, weight loss, or diminished weight gain, anorexia, and excitability[4,18]. Relatively little data is available on hematologic and biochemical parameters in stress.

Multiple stressors would appear to be involved in transport. Trauma, surgical procedures and drugs or biologicals administered before, during, or after transport may be stressful. Dietary changes including fasting due to innanition or failure to provide feed are commonly encountered[11]. Stressful physical agents encountered in transport include noise,

vibration, air blasts, smoke and dust. Stress due to muscular exercise, restraint and physiologic changes such as estrus and pregnancy also appear involved[18,19]. Environmental stresses include crowding, captivity, relocation, accidents, fighting, wind, cold, heat, and rain. Neuropsychologic stimuli are difficult to evaluate but would appear to include pain, fear, and sleep deprivation.

Biochemical alterations reported in cattle during transport stress include increased activity of glutamic-oxalacetic transaminase (7-38%), glutamic-pyruvic transaminase (29-83%), lactic dehydrogenase (14-42%), and creatine phosphokinase (30-308%). Wohler[23] reported a decrease in plasma total protein and an increase in sodium but no change in serum calcium, phosphorus or chloride. Early in transport calves showed a rapid rise in serum cortisol and corticosterone levels[17]. In another report 17 hydroxycorticosteroid levels evaluated in calves after transport showed either elevated or depressed levels after prolonged transport[15]. Depressed levels usually remained so during the first week post-transport and then increased to above normal values before returning to normal.

Hematologic and biochemical changes associated with disease stress vary with the pathobiology of the specific disease process however nonspecific responses to stress such as glucocorticoid and catecholamine levels may be consistent[13]. Carter et al[1] demonstrated a 2-3 fold increase in serum cortisol levels in sheep experimentally infected with Listeria monocytogenes.

The major mediators of stress include catecholamines (epinephrine and norepinephrine), glucocorticoids and growth horomone[13]. Urinary

excretion of epinephrine and nonrepinephrine was increased in sheep exposed to cold environmental temperatures[20]. Hyperglycemia, hyperlipidemia, mature neutrophilia, lymphopenia, eosinopenia and increased levels of serum alkaline phosphatase and amylase are associated with elevated glucocorticoid levels in stress[13]. Increased growth hormone levels in man have been associated with hyperglycemia and increased plasma free fatty acids (FFA)[13].

The purpose of the present study was to determine changes in biochemical and hematologic profiles induced by transport and respiratory disease in beef steers.

MATERIALS AND METHODS

Animals: Fifteen mixed breed beef steers ranging in weight from 300-600 pounds were purchased from a sales barn and held up to five days before being transported to a private feedlot. Blood was collected by jugular venipuncture approximately 20 hours after arrival. All animals were free of overt signs of illness at the time of sampling.

Ten animals not included in the initial group were sampled four days later, two days after developing a respiratory illness. All animals were partially anorectic, had increased rectal temperatures, rapid respiration and nasal discharge. They had been treated with tetracycline and sulfonamides for two days prior to sampling. Both groups were resampled one month from date of arrival.

Analysis: Blood samples were cooled immediately to $4^{\circ}C$ and serum separated and frozen within two hours. Analysis included PCV, total leukocytes, leukocyte differential, plasma FFA, serum cortisol, triglycerides, cholesterol, L(+) lactate, D(-) lactate, sodium, potassium,

venous total CO_2, alkaline phosphatase, urea nitrogen, GPT, total protein, albumin, calcium, inorganic phosphate, glucose and creatinine. All tests were performed by previously described methods[21].

RESULTS

Transport group: The post-transport PCV was increased but decreased to the normal range as determined in another group of feedlot cattle at the second sampling[21]. This may have been due to hemoconcentration post-transport. No significant changes in total leukocytes or differential leukocytes counts were noted (Table 1). There was no evidence of stress response in the post-transport differential leukocyte count and mean eosinophil counts were higher post-transport than at the later sampling.

No significant changes were noted for total lipids, triglycerides, and cholesterol although both total lipids and cholesterol values were higher than for normal feedlot cattle (Table 3). Phospholipids values were very low post-transport as compared to normal steers and increased to above normal values at the second sampling ($P < .00001$). Mean values for plasma FFA were increased post-transport and decreased to the normal range at the second sampling (Table 3). L(+) lactate levels were increased post-transport ($P < .05$) while D(-) lactate showed no change (Table 3). The L(+) lactate values were similar to results presented by Olumeyan[9]. Serum urea nitrogen values were increased at the second sampling ($P < .05$) and were considerably higher than the normal group (Table 3).

Other statistically significant changes included an increased glucose, calcium and alkaline phosphatase and a decreased inorganic

phosphate and total protein post-transport as compared to the second sampling. Compared to the normal group the inorganic phosphate was increased and calcium and glucose decreased at the second sampling.

Respiratory Disease Group: The mean PCV was slightly lower during illness ($P < .01$) and was even further decreased at the second sampling (Table 2). A band response was present during illness but no other changes were noted in the hemogram.

Total lipids, cholesterol and triglycerides values were not significantly different between the two samplings but values were generally higher than those of the normal group (Table 4). Phospholipids were very low during illness but increased to greater than normal at the second sampling ($P < .001$). Plasma FFA were high during illness and decreased but remained above normal values at the second sampling (Table 4).

Serum cortisol was increased during illness with mean values of 64 ng/ml. Normal values for feedlot cattle in our laboratory was 26.2 ± 3 ng/ml. At the second sampling the mean cortisol value was significantly less than normal.

Other significant changes between the two sampling periods included increased urea nitrogen and inorganic phosphate values at the second sampling with values at both samplings being greater than the normal, increased serum alkaline phosphatase at the second sampling compared to the first sampling and to normal values, decreased serum calcium and total protein during illness which increased to the normal range at the second sampling and a decreased glucose between the two periods with both values less than normal.

Comparisons Among Groups: At the second sampling statistically significant variations between transport and disease groups were found for PCV, L(+) lactate, cholesterol, plasma FFA, serum alkaline phosphatase, serum cortisol, and glucose. The transport group had higher mean values for PCV, cholesterol and serum cortisol while the disease group had higher L(+) lactate, plasma FFA, glucose and alkaline phosphatase.

DISCUSSION

A typical leukocyte stress response was noted in none of the transport group and only two of the ill animals despite the significantly increased cortisol levels in the latter group. Sick animals number 7, 8, 9 and 10 had increased immature neutrophils while animal number 8 had a leukocytosis and increased neutrophils and decreased lymphocytes. Each of these changes have been associated with inflammatory responses in cattle[12]. The increase in PCV was probably due to hemoconcentration post-transport. Initial increase in mean creatinine levels in each group suggests decreased glomerular filtration rate probably related to dehydration. Significant increases in urea nitrogen and total protein at the second sampling are probably related to increased protein and energy intake during the period[5,10].

Consistent changes in serum cortisol levels were not found following transport but mean values were increased almost three fold in ill animals. The normal levels post-transport may have been related to a several hour delay after transport prior to sampling of the animals or to a decrease in serum cortisol as previously reported in chronic stress[15]. In one study mean serum glucocorticoid levels in beef cattle transported long

distances were almost identical to control levels but concentrations in many animals were above and below the mean with about equal frequency[15]. In the present study only two animals in the post-transport group were outside the 95% confidence limits of the normal group.

Phospholipid levels were very low post-transport and during illness but returned to the normal range at the second sampling in both groups. McCarthy et al[7] found that low density lipoproteins were almost devoid of phospholipids in dairy cattle with ketosis. Treatment with methionine resulted in a rapid return of phospholipids to normal. Methionine is needed as a mentyl donor in the formation of phospholipids and perhaps in the coupling of protein and lipid moieties in lipoproteins[7]. The present group was purchased through a sales barn on an individual or small group basis so analysis of methionine content of the ration was impossible however methionine was almost certainly deficient in the ration the week between purchase and entry into the feedlot.

TABLES AND FIGURES

PAPER II

TABLE 1

HEMATOLOGIC VALUES FOR 15 FEEDLOT STEERS
IMMEDIATELY AND 1 MONTH AFTER TRANSPORT

	UNITS	IMMEDIATE MEAN + S.D.	1 MONTH MEAN + S.D.	NORMAL VALUES* MEAN + S.D.
PCV	Vol %	38.2 ± .7[a]	35,3 ± .7[b]	36.9 ± .75[ab]
Leukocytes	/ul	8,940 ± 2,600	10,954 ± 2,200	9,071 ± 2,200
Bands	/ul	30 ± 80	206 ± 200	30 ± 42
Neutrophils	/ul	2,471 ± 1,050	3,937 ± 2,800	3,037 ± 2,190
Lymphocytes	/ul	5,227 ± 990	5,856 ± 2,050	5,880 ± 1,450
Monocytes	/ul	835 ± 480	904 ± 285	738 ± 400
Eosinophils	/ul	493 ± 405	165 ± 175	61 ± 60
Basophils	/ul	104 ± 98	55 ± 68	53 ± 30

*Normal values for 10 healthy feedlot steers 37 days after entry into feedlot.

[a,b]Values for each parameter with different superscripts are significantly different ($P < .05$).

TABLE 2

HEMATOLOGIC VALUES FOR 10 FEEDLOT STEERS
DURING RESPIRATORY ILLNESS AND 2 WEEKS AFTER RECOVERY

	UNITS	ILLNESS MEAN + S.D.	2 WEEKS AFTER RECOVERY MEAN + S.D.	NORMAL* VALUES MEAN + S.D.
PCV	Vol %	34.2 ± .85[a]	28.6 ± 1.2[b]	36.0 ± .75[a]
Leukocytes	/ul	10,050 ± 2,600	10,836 ± 2,500	9,071 ± 2,200
Bands	/ul	208 ± 228	0	30 ± 42
Neutrophils	/ul	3,225 ± 2,400	2,317 ± 1,850	3,037 ± 2,190
Lymphocytes	/ul	5,476 ± 2,500	7,389 ± 1,950	5,880 ± 1,460
Monocytes	/ul	854 ± 365	1,000 ± 333	738 ± 400
Eosinophils	/ul	210 ± 210	153 ± 220	61 ± 60
Basophils	/ul	12.4 ± 40	34 ± 65	53 ± 30

*Normal values for 10 healthy feedlot steers 37 days after arrival in the Feedlot.

[a,b]Values for each parameter with different superscripts are significantly different ($P < .05$).

TABLE 3

Biochemical Values For 15 Feedlot Steers

Immediately and 1 Month After Transport

	Units	Immediate Mean ± S.D.	1 Month Mean ± S.D.	Normal Values* Mean ± S.D.
Total lipids	mg/dl	369 ± 60	357 ± 60	279 ± 14.2
Triglycerides	mg/dl	23.7 ± 2.2	30.1 ± 2.2	30 ± 1.8
Cholesterol	mg/dl	121 ± 7.9[a]	115 ± 7.9[a]	84 ± 6.5[b]
Phospholipids	mg/dl	59.5 ± 6.3[a]	149.7 ± 6.3[b]	104 ± 9.7[c]
Plasma FFA	µEq/L	495 ± 23[a]	384 ± 23[b]	378.4 ± 28.7[b]
D (-) Lactate	mg/dl	0	.2 ± .1	0
L (+) Lactate	mg/dl	46.5 ± 4.3[a]	33.9 ± 4.3[b]	28.4 ± 4.2[c]
Sodium	mEq/L	139 ± .8[a]	136 ± .8[a]	148.0 ± 1.2[b]
Potassium	mEq/L	4.4 ± .1[a]	4.5 ± .1[a]	5.3 ± .1[b]
Total CO_2	mEq/L	26.1 ± 1.6[a]	21.8 ± 1.6[a]	30.9 ± 1.0[b]
Urea Nitrogen	mg/dl	6.7 ± .8[a]	9.5 ± .8[a]	4.7 ± .82[b]
Alkaline Phosphatase	mU/ml	103 ± 4.5[a]	80 ± 4.5[b]	64 ± 11[c]
GPT	mU/ml	22.1 ± 3.2[a]	30.3 ± 3.2[a]	19.5 ± 1.6[b]
Total Protein	g/dl	6.9 ± .1[a]	7.6 ± .1[b]	7.6 ± .1[b]
Albumin	g/dl	3.7 ± .1	3.6 ± .1	3.6 ± .1
Calcium	mg/dl	9.6 ± .1[a]	9.2 ± .1[b]	9.75 ± .17[c]
Inorganic Phosphate	mg/dl	6.7 ± .2[a]	7.7 ± .2[b]	5.7 ± .3[c]
Glucose	mg/dl	83 ± 2[a]	63 ± 2[b]	106.5 ± 9.4[c]
Creatinine	mg/dl	1.18 ± .03[a]	.83 ± .03[b]	1.22 ± .04[c]
Cortisol	ng/ml	26.5 ± 2.1	26.1 ± 2.1	26.2 ± 3.0

*Normal values for 10 healthy feedlot steers 37 days after arrival in the feedlot.

[a,b,c]Values for each parameter with different superscripts are significantly different ($P < .05$).

TABLE 4

Biochemical Values for 10 Feedlot Steers
During and 2 Weeks After Respiratory Illness

	Units	Illness Mean ± S.D.	2 Weeks After Recovery Mean ± S.D.	Normal Values* Mean ± S.D.
Total Lipids	mg/dl	338 ± 90	339 ± 65	279 ± 14.2
Triglycerides	mg/dl	30.3 ± 3.2	26.7 ± 3.2	30 ± 1.8
Cholesterol	mg/dl	103 ± 10	89 ± 13	84 ± 6.5
Phospholipids	mg/dl	38.1 ± 9.2[a]	135.4 ± 9.2[b]	104 ± 9.7[c]
Plasma FFA	µEq/L	695 ± 29[a]	527 ± 39[b]	378.4 ± 28.7[c]
D (-) Lactate	mg/dl	0	.2 ± .2	0
L (+) Lactate	mg/dl	42.3 ± 4.5[a]	46.2 ± 6.4[a]	28.4 ± 4.2[b]
Sodium	mEq/L	138 ± 1[a]	138 ± 1[a]	148.0 ± 1.2[b]
Potassium	mEq/L	3.9 ± .1[a]	4.7 ± .2[b]	5.3 ± .1[c]
Total CO_2	mEq/l	24.8 ± 2.0[a]	25.5 ± 2.7[a]	30.9 ± 1.0[b]
Urea Nitrogen	mg/dl	7.7 ± 1.0[a]	12.0 ± 1.4[b]	4.7 ± .82[c]
Alkaline Phosphatase	mU/ml	65 ± 6[a]	108 ± 8[b]	64 ± 11[a]
GPT	mU/ml	32 ± 4[a]	39 ± 5[a]	19.5 ± 1.6[b]
Total Protein	g/dl	6.5 ± .1[a]	7.4 ± .2[b]	7.6 ± .1[b]
Albumin	g/dl	3.1 ± .1[a]	3.5 ± .1[b]	3.6 ± .1[b]
Calcium	mg/dl	7.8 ± .1[a]	8.2 ± .2[b]	9.8 ± .2[c]
Inorganic Phosphate	mg/dl	6.0 ± .2[a]	7.2 ± .3[b]	5.7 ± .3[a]
Glucose	mg/dl	91 ± 3[a]	74 ± 4[b]	106.5 ± 9.4[c]
Creatinine	mg/dl	1.3 ± .04[a]	1.0 ± .06[b]	1.22 ± .04[a]
Cortisol	ng/ml	64 ± 3[a]	14 ± 3[b]	26.2 ± 3.0[c]

*Normal values for 10 healthy feedlot steers 37 days after arrival in the feedlot.

[a,b,c] Values for each parameter with different superscripts are significantly different ($P < .05$).

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APPENDIX I

LITERATURE REVIEW

The blood serves as a transport system precursors and products which in many cases reflect metabolic events occurring in the body tissues. The interrelationship of carboyhydrate, protein and fat metabolism, hormones, electrolytes, enzymes and blood cells will be discussed in relationship to normal ruminant metabolism, metabolism in the unfed state and metabolism during stress. Special emphasis will be placed on lipid metabolism.

Volatile Fatty Acid Metabolism

Foodstuffs presented to the ruminant stomach are mainly carboyhydrate with lesser amounts of proteins and fats. On a roughage diet these carbohydrates are mainly in the form of cellulose, pentosans and pectins[125] while in a grain diet starch predominates. Approximately 70% of dry matter in the rumen is digested by bacteria and protozoa[60]. The digestion of complex carboyhydrates results in the production of volatile fatty acids mainly propionic, acetic and butyric. These short chain acids have been found to account for 45-60% of the digestible energy and 60-80% of the total energy expenditure in sheep on a roughage diet[9,19]. They are absorbed directly across the rumun epithelium at a rate that varies inversely with the rumen pH. This suggests that undissociated acids penetrate the rumen epithelium more readily than do ionized acids[125,132]. Propionate levels in the portal vein suggest that a considerable amount (50-75%) is metabolized by the rumen epithelium[16]. Butyrate is largely converted to beta-hydroxybutyrate resulting in very low levels of butyrate in the portal blood[132,9]. Interconversion of acetate and butyrate also occurs in the rumen epithelium[9].

Volatile fatty acids enter the liver via the portal vein. Butyrate is converted to acetyl CoA in the liver and thus is not glucogenic. Acetate is not metabolized in the liver but is readily oxidized to acetyl CoA by skeletal muscle, heart and kidney. Lindsay et al.[87] found that 15 to 20% of the absorbed acetate is oxidized to carbon dioxide and this accounts for at least 20% of the total CO_2 production. Acetate also represents the main precussor for synthesis of fatty acids in adipose tissue[125]. Propionate is the only volatile fatty acid that is glucogenic and is rapidly converted to glucose by the liver[16,82]. Bergman et al.[16] infused labeled propionate into the rumen vein of sheep and found that 50% of the propionate was converted to glucose and 40% to other compounds by the liver. This represented 20-40% of the total glucose turnover on a roughage diet and could be increased to 67% by infusion of high levels of propionate. Grain diets markedly elevate rumen propionate[24].

Glucose Metabolism

Glucose metabolism has been extensively studied in the sheep. Bergman et al.[14,8] infused ^{14}C glucose into the portal vein of sheep and found that hepatic gluconeogenesis accounted for 85% of the glucose turnover. About 30% of the glucose was oxidized and accounted for 10-20% of the total CO_2 production.

Since very little glucose is absorbed directly from the gastrointestinal tract glucose production depends on gluconeogenesis. Substrates for gluconeogenesis include propionate, amino acids, glycerol and lactate[8,4,14,82]. Amino acids are converted to glucose in the liver. On the basis of ^{14}C amino acid infusion, it was found that 17-26% of alanine, aspartate and glutamate and 5-7% glycine and serine were incorporated into glucose[162].

They also reported that up to 30% of the glucose turnover could be derived from amino acids. Glycerol contribution to glucose turnover was found to be less than 5% in the fed animal but this could be increased to 40% in severe hypoglycemia[17]. Lactate contributes no more than 4-10% of the total glucose turnover[4]. Propionate therefore must contribute approximately 50% of the total glucose turnover.

Lactate Metabolism

Rumen micro-organisms produce both D(-) and L(+) lactate. The rumen epithelium does not metabolize either of these substances but does synthesize L(+) lactate from propionate. Leng, et al. suggest that up to 70% of the glucose derived from propionate is first converted to lactate however others report this to be less than 5%[151]. L(+) lactate is readily converted to glucose in the liver but D(-) lactate cannot be metabolized and is excreted in the urine. Silage and grain feeding resulted in high rumen levels of lactate and an increase in the percentage of D(-) lactate[120,148].

Ketone Metabolism

The main sites of ketogenesis are the liver and rumen. Alimentary ketone bodies are produced from butyrate by the rumen epithelium. In addition a few amino acids are ketogenic. Hepatic ketones are produced in the liver primarily from non-esterified fatty acids which are mobilized from body fat. These are converted to acetoacetyl CoA which is reversibly converted to acetoacetate and beta-hydroxybutyric (BOHB) and irreversibly to acetone. BOHB accounts for 75% of the blood ketone level. In the fed state alimentary ketogenesis accounts for nearly all the ketone production while in fasting hepatic ketogenesis predominates.

Most body tissues utilize acetoacetate and BOHB by converting them to acetyl CoA with subsequent oxidation through the Krebs cycle but utilization of acetone is very limited. The rate of utilization is directly proportional to the blood level up to 20 mg/100 ml after which increased blood level do not increase tissue utilization[81]. Bergman et al.[15] infused ^{14}C acetoacetate into sheep and determined that it accounted for 2% of the total CO_2 production. BOHB is utilized by the mammary gland for lipogenesis and to a lesser extent by adipose tissue[17]. Excess ketones are cleared from the body in urine and milk.

Fasting Ruminant Metabolism

Adipose tissue provides a large readily available energy reserve in ruminants but glycogen stores are very low[64,124]. Increased utilization of FFA and ketones for energy production occurs and glucose turnover is reduced. Glycerol mobilized from adipose tissue becomes an important glucogenic precursor providing up to 40% of the glucose turnover in severely hypoglycemic sheep[10,17]. Plasma FFA mobilized from adipose tissue can readily be converted to acetyl CoA by the liver. When acetyl CoA production exceeds the capacity of the Krebs cycle it is either used to synthesize fatty acids and cholesterol or ketone bodies. Plasma FFA are also oxidized by peripheral tissues. In the post-absorptive state glucose and acetate levels decline while plasma FFA and ketone levels increase[112,19,115].

Lipid Metabolism

Lipid metabolism with the exception of FFA has not been extensively studied in the ruminant. Least[77] found very low levels of total lipids in newborn lambs which rose rapidly to adult levels in the first 20 days of

life. Breed differences in plasma lipids were found to be quite small[138]. Moore[98] published normal values for total lipids, cholesterol and triglycerides for 39 feedlot steers. Total lipids were reported to be 244 ± 98 mg/dl, while total cholesterol was 113 ± 48 mg/dl and triglycerides were 20 ± 11 mg/dl. He also found that total lipids increased at the end of the feeding period but cholesterol and triglycerides did not. Another report[119] lists mean values for plasma triglycerides (22 mg/dl, free cholesterol (27 mg/dl), cholesterol esters (170 mg/dl) and phospholipids (110 mg/dl) in 8 herford and angus steers. A diurnal variation in plasma FFA and triglycerides was reported but no diurnal change was found in total lipids, phospholipids, free cholesterol or cholesterol esters.

Dietary triglycerides undergo extensive lipolysis and hydrogenation by rumen microorganisms and thus primarily free fatty acids are presented to the small intestine[66]. Long chain fatty acids which are absorbed and incorporated into triglycerides and to a lesser extent cholesterol by the intestinal mucosa form chylomicrons and enter the peripheral blood by way of the thorasic duct[66]. Short chain and perhaps medium chain fatty acids and glycerol are absorbed directly into the portal circulation.

Lipids in plasma circulate in the form of spherical lipoprotein complexes. They are composed of a nonpolar lipid core consisting of triglyceride and cholesterol esters covered by a layer of phospholipid, cholesterol and apoproteins[118]. These are classified according to sedimentation coefficient and electrophoretic mobility as high density lipoproteins (HDL) with alpha migration, low density lipoproteins (LDL) with beta migration, very low density lipoproteins (VLDL) with pre-beta migration and chylomicrons which remain at the origin[118].

Protein moieties vary in their terminal residues, amino acid content and immunochemical behavior. A-apoproteins refer to two protein structures on the surface of high density lipoproteins. They have a helical structure and have great affinity for lipid. The protein is an aggregate of 2-6 identical subunits with a molecular weight of 23,000 to 36,000 with an amino terminal aspartic acid and a carboxyterminal threonine. B-apoproteins represent the major proteins of low density lipoproteins. C-apoproteins are low molecular weight proteins present in both VLDL and HDL. These apoproteins appear to represent binding sites for cellular and enzume interactions which control lipid transport[70].

In man chylomicrons contain primarily tricylyceride (85%) with the rest made up of cholesterol esters, phospholipid, cholesterol, and free fatty acids[70]. The primary apoprotein is A but B-apoprotein is present. Plasma chylomicron triglyceride is removed by the liver and adipose tissue while chylomicron cholesterol is primarily removed by the liver[13,70]. In sheep 10% of the ^{3}H or ^{14}C labeled chylomicrons was taken up by the liver while in the dog, 22% was removed by the liver[13]. In dogs about 40% of the triglyceride entering the liver is converted to plasma FFA while in the sheep only 10% of the FFA from triglyceride is recirculated[13]. In both sheep and dogs about 1/2 of the chylomicron triglyceride hydrolyzed in extrahepatic tissue is recycled as plama FFA[13]. The rest is oxidized or incorporated into adipose tissue.

VLDL are synthesized in the endoplasmic reticulum of liver hepatocytes[70,118,54]. The incorporation of B-apoprotein into the outer layer appears to be essential for secretion[70]. Secretion involves passage through secretory vesicles of the Golgi apparatus and the microtubular system into hepatic sinusoids[118]. Once secreted both VLDL and chylomicrons

are modified by acquiring C-apoproteins from the surface of HDL[70]. This appears to increase the affinity of the VLDL particle for lipoprotein lipase. In addition surface cholesterol increases and surface phospholipid decreases[70].

VLDL and chylomicrons are rapidly degraded by lipoprotein lipase[118,54,70]. This interaction appears to occur at the capillary endothelium[118]. Seventy to 90% of the triglyceride is removed while cholesterol esters are retained[118,70]. The particle surface retains its B-apoprotein and cholesterol content but loses much of its phospholipd and C-apoprotein. Each time the particle and lipoprotein lipase interact more triglyceride is lost from the central core[70]. When the triglyceride content is reduced to a critical level the surface phospholipid and C-apoprotein are transfered to HDL spontaneously[70]. The C-apoprotein has been found to be reused repeatedly in the formation of VLDL.

Low density lipoproteins (the major cholesterol-carrying lipoprotein) are either the direct result of VLDL and cylomicron metabolism or are produced by the liver from these particles[118,70,13,131]. LDL are rapidly taken up by the liver in the rat but a stable LDL is present in man[63]. In swine hepatectomy caused an increased rate of removal of LDL from plasma indicating that the liver is not the major site of LDL removal[131]. Cultured human fibroblasts have been shown to bind (^{125}I) LDL with high affinity and specificity[21].

Beta-lipoproteins have been found to be almost devoid of lecithin and sphingomyelin in stressed dairy cattle with depressed milk fat levels and in ketosis[96]. Treatment with methionine caused the LDL fraction to return to normal. Havel[70] suggests that methionine, which is necessary for phospholipid production, is deficient in these animals.

High density lipoproteins are synthesized in the liver and intestinal mucosa. They consist almost totally of polar lipids and contain much protein. Lecithincholesterol acyl transferase (LCAT) is produced by the liver and appears to alter HDL from disc shape to a spherical shape[118,70]. The enzyme esterifies cholesterol to cholesterol esters and converts lecithin to lysollcithin[118]. The surface composition of HDL is not fixed but depends upon the lipids available to it[70]. HDL apparently picks up lipid from other lipoproteins and also from cell membranes[70].

Deposition and mobilization of triglycerides in adipose tissue is mediated by blood levels of lipoproteins, several hormones and the autononic nervous system. Glucose uptake by adipose tissue is controlled by insulin levels[125]. Glucose is metabolized to produce fatty acids and a glycerol phosphate necessary for glycerol production. In the ruminant acetate replaces glucose to a great extent as a precursor for lipogenesis[125]. The rate of lipogenesis depends on the availability of a glycerol phosphate[95]. NADPH produced by the hexose-monophosphate shunt is required as a cofactor for the production of long chain fatty acids from glucose. Fatty acids for lipogenesis are produced by the action of lipoprotein lipase on the triglycerides of chylomicrons and VLDL[125,118]. These fatty acids are taken up by the cell and esterified with a glycerol phosphate and stored as triglycerides.

Mobilization of fatty acids from adipose tissue is hormonally mediated. Hormonally activated adenylate cyclase catalyzes the production of 3'5' AMP from ATP[70,125]. 3'5' AMP activates a protein kinase which phosphorylates the lipase enzyme converting it from a relatively inactive to an active form[125]. Lipase hydrolyzes triglycerides to free fatty acids and glycerol.

3'5' AMP is rapidly metabolized to 5' AMP by the action of a phosphodiesterase. Phosphodiesterase thus acts to modulate hormone stimulated lipogenesis. However, ATP levels and not 3'5' AMP levels appear to be the rate limiting factor in the intensity and duration of lipolysis[70].

Hormones which stimulate lipogenesis include glucagon, cathecholamines, growth hormone and glucocorticoids[125]. Antilipolytic substances include insulin, prostaglandin E-1, beta-adrenergic blockers, and nicotenic acid[125]. The effects of insulin injection have been evaluated in sheep[91]. Plasma FFA were initially depressed but then underwent a rebound elevation perhaps due to growth hormone (GH) secretion in response to hypoglycemia. In the same experiment ACTH, TSH and prolactin injection had no effect on plasma FFA while oxytocin injection caused an initial increase and then a gradual decrease in plasma FFA levels. Growth hormone produced a slight but large increase in plasma FFA. Estradiol injection caused an initial decrease in plasma FFA followed by a long lasting increase while progesterone had no effect[93]. Luthman et al.[89] found that when lipolysis was stimulated by epinephrine injection significant uptake of plasma calcium by adipose tissue occurred. This resulted in hypocalcemia which was not calcitonin dependent. The calcium content of adipose tissue increased 600 fold following epinephrine injection. These was a significant inverse correlation between plasma calcium and plasma FFA levels.

The liver plays a central role in the regulation of fat metabolism[125]. The liver is involved in the synthesis of plasma phospholipids, cholesterol, VLDL and HDL. It also synthesizes fatty acids from carboyhydrates and amino acids. Degradation of plasma FFA, and phospolipids for energy production, lipid synthesis and ketone production also occurs

in the liver. The liver normally contains about 5% lipid by weight of which 75% is phospholipid and 25% is triglyceride[125]. Lipid accumulation within hepatocytes has been associated with many disease processes[125]. The pathogenesis of fatty livers in bovine ketosis has been related to excessive mobilization of FFA from adipose tissue[89,125]. Other possible cuases of fatty livers include excessive dietary fat intake, oversynthesis of fatty acids from carbohydrate or animal acids and defective lipid transport from the liver.

Metabolism in Stress

Seyle[124] defines stress as "the nonspecific response of the body to any demand." In this theory stressors elicit specific responses affecting one specific area or organ in the body and nonspecific responses which are the same regardless of the stressor involved. He also suggests a triphasic response to stress which involves both a general adaptation syndrome and a local adaptation syndrome. The first phase involves alarm, tissue catabolism, weight loss, thymico-lymphatic involution, adrenal hyperactivity, acute gastrointestinal erosions, eosinopenia, lymphopenia and neutrophilia. Phase II is a period of resistance in which the body shows no outward signs of disease. Phase III is represented by exhaustion and recurrance of the alarm reaction.

Table 1

trauma	drugs
hormones	diet
physical agents	micro-organisms
immunity	hypoxia
hemorrhage	muscular exercise
restraint	athletics
neuropsychologic stimuli	climate
environment	biorhythms
occupation	physiological states
genetics	constitution
race	combined effects of various agents
tumors	

Transport Stress

In reviewing the list of stressors proposed by Seyle[124] (Table 1) several appear to be involved in bovine transports. Trauma from bruising and musuclar exercise is suggested by elevated levels of glutamic-oxalacetic transaminase (GOT) and creatine phosphokinase (CPK)[47]. Drugs and biologicals administered before, during or after transport may be stressful. Dietary changes or fasting either due to inanition or failure to provide feed are commonly encountered in transport[119]. Physical agents encountered in transport include noise, vibration, air blasts, smoke and dust. Muscular exercise, restraint and physiologic stresses such as estrus, pregnancy and lactation also appear involved[142,147]. Environmental stresses include crowding, captivity, relocation, accidents, fighting, wind, cold, heat and rain. Neuropsychologic stimuli are difficult to evaluate but would appear to include pain, anxiety, fear, and sleep deprivation.

Biochemical alterations related to transportation include increased GOT (7-38%), GPT (29-83%), LDH (14-42%) and CPK (30-308%) levels[47]. On the basis of 5000 blood analysis before and after transport Wohler[160].

reported a decrease in plasma total protein and an increase in serum sodium with no change in serum calcium, phosphorus and chloride. Early in transport calves showed a rapid rise in serum cortisol and corticosterone levels[141]. In another report serum 17 hydroxcorticosteroid levels evaluated in calves after transport showed either elevated or depressed levels[129]. Decreased levels usually remained so during the first week post-transport and then increased to above normal values before returning to normal. Both sexes responded similarly. In sheep plasma cortisol levels returned to near normal levels within two hours after transport.

Other changes associated with transport include an increased number of short estrus cycles and reduced resistance to experimental salmonella infection[142,147,46].

Heat Stress

Summer heat among Holstein-Freisian cows in Arizona was associated with significantly reduced fertility[97,133]. Estrus interval, ovulation time and corpus luteal time were not affected but the length of estrus was shortened[97]. Conception rate was significantly reduced at a controlled temperature of 32.2°C[31]. Short-term heat stress resulted in a 15% decrease in total body solids apparently related to loss of body fat stores[71].

Laboratory evaluation of heat stressed cattle revealed no change in total red cell count, total leukocyte count, hemoglobin or packed cell volume (PCV) but did result in a decreased mean corpuscular hemoglobin (MCH) and mean corpuscular volume (MCV). Cortisol levels in steers increased within the first 20 minutes of exposure to heat stress and continued to rise for 2 hrs[26]. This increased level was maintained for

7-10 weeks after which values fell to subnormal levels[25]. Cortisol levels did not return to normal until 9 weeks after termination of the heat exposure[26]. In another study cortisol levels were decreased in 8 chronically heat-stressed hereford bulls.

Cold Stress

Evaluation of sheep exposed to winter cold in Canada showed that cold weather had no effect on food intake, plasma FFA or ketone levels. However, blood glucose levels did increase[67]. In the same study cold associated with starvation markedly elevated plasma FFA and ketones and depressed glucose levels. In another study more severe winter conditions markedly elevated plasma FFA levels (> 2000 μEg/1) but mild cold exposure resulted in no significant change[115]. Cold exposure at 3°C with wetting of sheep resulted in elevated cortisol levels and fatty infiltration of the liver and adrenal cortex[51].

Holliday et al.[51] in evaluating the cold adaptation of sheep found elevated protein bound iodine (PBI), plasma FFA, glucose and acetone levels. Near the end of cold exposure glucose levels fell to subnormal levels. Urinary excretion of norepinephrine and epinephrine was increased during cold exposure in sheep sheep but, injection of additional epinephrine had no direct thermogenetic effect[149].

Environmental Stress

Stress factors other than temperature extremes are crowding, confinement, limited exercise, insects and high intensity production practices. These factors are difficult to evaluate because of many variables which are difficult to control. Confinement of cattle has been associated with

a marked increased incidence of hepatic absessation, laminitis, bloat and lactic acidosis[36]. Increased herd size with decreased management time per animal has been shown to increase disease incidence and morbidity in beef cattle.

Mediators of the Stress Response

Catecholamines

Increased plasma and/or urinary catecholamine levels are one of the most reliable indications of acute stress in man[124]. Few references could be found to their measurement in ruminants. Urinary excretion of epinephrine and norepinephrine were found to increase significantly following exposure to cold environmental temperatures in sheep[149]. Injection of cathecholamines or dopamine was associated with increased plasma FFA levels in sheep[113,72,52].

Research on the control of catecholamine synthesis has centered around two enzymes, tyrosine hydroxylose and phenylethanolamine N-methyltransferase (PNMT) in man. Tyrosine hydroxylase has been identified as the rate limiting enzyme[74]. The level of tyrosine hydroxylase in the adrenal gland appears to be regulated by the splanchnic nerve supply to the adrenal whereas PNMT levels are controlled by adrenal glucocorticoid concentrations[74]. Normal catcholamine biosynthesis requires the presence of ACTH[5]. Thus stimulation of the autonomic nervous system and production of adrenal conticoids stimulate production of catecholamines. Both tyrosine hydroxylase and PNMT have been found to increase in immobilization stress in rats and in cold stress in mice[72,27].

Metabolic consequences of elevated catecholamine levels include hyperglycemia, increased plasma FFA, selective vasoconstriction,

splenic contraction and smooth muscle stimulation[125]. Hyperglycemia is related to increased hepatic glucogenesis due to the activation of phosphorylase through the mediator 3'5' AMP[125]. Increased plama FFA are related to stimulation of lipolysis in adipose tissue by elevated plasma catecholamine levels or stimulation of the sympathetic nervous system[125].

Glucocorticoids

Adrenal corticoids are secreted in response to the circulating level of pituitary ACTH. Neural responses to environmental stimuli effect ACTH secretion through the neuroendocrine pathway. This is mediated by release of conticotrophin releasing factor from the hypothalamus[125,154].

Hydrocortisone (cortisol) and corticosterone are the principle functional corticoids in cattle[104,34,140]. Administration of ACTH more than doubled the plasma concentration of cortisol in one hour while plasma corticosterone levels decreased[140]. This suggests that cortisol is of primary importance in response to stress. Willett et al.[154] in a review of literature reported that plasma corticosteroids increased due to low environmental temperatures, elevated environmental temperatures, trauma, manipulation, psychologic stress, administration of ether, sodium pentobarbitol or morphine and successive venipuncture. Cattle accustomed to handling had lower cortisol values than previously unhandled range cattle. They also found as much as a 5-fold increase in plasma cortisol over a 10 minute period when animals were stressed by anticipation of feeding or manipulation of the head. 17-hydroxy corticosteroids (17-OH-CSO levels in plasma did not vary significantly among lactating, pregnant, or dry dairy cattle[126]. Estrus and milking were associated with increased corticoid levels[126,105]. Disease stressed dairy cattle were found to have elevated cortisol levels[140].

Hematologicic changes associated with increased corticoid levels have been termed the "stress response"[123]. The total white blood cell count (WBC) is elevated due to redistribution of body leukocyte pools. Mature neutrophilia is associated with the redistribution of the bone marrow storage pool and marginated pool. Lymphopenia is due to redistribution, direct toxic effect and suppression of production of lymphocytes[123]. Monocytosis is not characteristic of acute stress in cattle but may be seen in chronic stress[123]. Eosinopenia is perhaps due to redistribution of eosinophils and blockage of release from bone marrow[123]. An elevated packed cell volume (PCV) has been found in dairy cattle injected with corticosteroids[104].

Polyuria, seen with both exogenous and endogenous cortiocoids, is due to an enhanced free water clearance[125]. Elevated plasma enzyme levels are related to enzyme induction and include SGPT, SGOT, and alkaline phosphatase[66]. Electrolyte alterations are not commonly associated with elevated glucocorticoid levels.

Increased susceptibility to infection has been documented during transport stress in cattle[46]. It apparently relates to several factors including stabilization of neutrophil lysosomal granules and suppression of cell mediated and perhaps humonal immunity[125,123].

Normal values vary among papers reviewed[143,145,107,86,140,32]. Colorometric determination of 17 OHCS in dairy cattle resulted in normal values with means near 40 ng/ml and acute stress concentrations greater than 100 ng/ml. Plasma cortisol determined by competetive protein binding resulted in normal values between 5 and 15 ng/ml. The lower values were among animals sampled without handling via indwelling caiheter. Venasesha et al.[140] measured cortisol by a fluorometric procedure in dairy cattle and found a

value of 26 ± ng/ml. No values could be found for cortisol determined by radioimmunoassay. These values are quite low compared to man, however only 39 ± 5% of plasma cortisol was found to be protein bound compared to greater than 90% in man[140].

Growth Hormone

Elevated growth hormone (GH) levels in stress have been well documented in man[125] and rat[73] and also in cattle[125]. Seyle[4] states that stress directly stimulates GH secretion and that GH levels parallel ACTH and glucocorticoid levels. The control of GH secretion is related to blood glucose and perhaps catecholamine concentrations[124]. Hypoglycemia is a potent stimulus for GH secretion[124].

The metabolic effects of GH on growth and development will not be reviewed here except as they relate to stress. GH has a hyperglycemic effect perhaps related to direct stimulation of alpha cells of the pancreatic islets resulting in increased glucogon secretion[125]. Increased plasma FFA concentration is related to direct stimulation of lipolysis in adipose tissue[70,155]. In addition, GH limits glucose uptake by adipose tissue. Acetate turnover is also increased by elevated GH levels[70].

BIOCHEMICAL AND CELLULAR PROFILING IN RUMINANTS

Metabolic profiling in the ruminant has been suggested as a means of determining the general health of populations, assessing nutritional adequacy of rations, screening of individuals for overt or occult disease and as a predictor of milk production and weight gains.

Diurnal and seasonal variations in the various chemistrys of blood profiles have been established by several authors. Coggins et al.[176] found a diurnal variation in glucose, plasma FFA, ketones, urea nitrogen, albumin, calcium and magnesium among lactating beef cows on a roughage diet. Urea nitrogen was highest 8 hours after feeding. Glucose and plasma FFA decreased after feeding while ketones increased[176,212]. During a 7-day fast, both plasma FFA and ketones increased and there was an inverse relationship between plasma FFA and blood glucose levels[212]. Among Angus and Hereford heifers fasted for 48 hours, plasma FFA increased within the first hour and continued to increase throughout the fast[172]. Blood glucose did not change in the first hour but was increased at 24 and 48 hours. Ketones were decreased at 48 hours while packed cell volume increased progressively beginning at 1 hour. Angus heifers had a higher blood glucose and PCV than herefords.

The effect of season of the year was evaluated in 24 dairy herds in lactating and non-lactating cattle[218]. Seasonal patterns were evident for PCV, urea nitrogen, and hemoglobin, all of which were higher during the summer. PCV, hemoglobin and serum iron were higher in non-lactating cows than in lactating cows while magnesium values were lower. Herd differences in calcium, serum globulin, serum iron and urea nitrogen were noted.

Payne et al.[208] evaluated biochemical tests in 191 herds of dairy cattle in Great Britian using a statistical tool, the Compton Metabolic Profile Test, which identifies abnormalities within herds based on deviation from a population mean. Mean values were determined with respect to stage of lactation and season of the year. Serum sodium was found to be lower in summer than in winter while the reverse was true for hemoglobin, albumin, urea nitrogen, magnesium, and copper. Blood glucose levels increased during January. Increased milk yield was associated with decreased hemoglobin, calcium, phosphate and potassium while dry cows had lower serum magnesium and copper. Albumin was found to decrease with reduced feed quality (decreased non-fat solids) but glucose and hemoglobin showed no relationship with this parameter.

Blowey[170] suggests a modified profiling approach to assess the adequacy of energy and protein intake in dairy cattle herds. Six cows from each herd were sampled 4-8 weeks post-calving and glucose, urea and albumin determined. This data was compared with expected normals. An increase in urea nitrogen associated with a decrease in albumin was interpreted as an inefficient utilization of nitrogen by rumen microorganisms. The conversion of ammonia to protein by rumen microorganisms is energy dependent and in the absence of adequate energy intake more ammonia escapes the rumen and is converted to urea by the liver resulting in an elevated plasma urea nitrogen concentration. High energy diets results in a more acid rumen environment decreasing ammonia absorption.

Harrison[144] found that urea nitrogen was directly proportional to dietary protein intake and inversely proportional to energy intake. Urea nitrogen was found to increase in feedlot cattle with time on feed.

Harrison also suggests that a urea nitrogen greater than 10 mg/dl in feedlot cattle indicates an adequate protein intake but a urea nitrogen of 7-9 mg/dl indicates inadequate protein intake.

Increased carbohydrate content in the diet resulted in a decreased plasma FFA levels suggesting that plasma FFA concentration is the most sensitive indicator of nutritional stress[171]. Holmes et al.[192] measured plasma FFA, glucose and PCV in hereford cattle six times a day for 30 days and found that glucose increased after feeding and plasma FFA and PCV decreased with decreased digestible energy in the ration. Plasma FFA was increased within 10 minutes after acute stress.

Kitchenham et al.[196] evaluated dairy calves reared under conventional and rapid-growth systems. Rapid growth calves showed increased glucose, urea nitrogen, inorganic phosphate and serum globulins. Growth rate was closely correlated with serum inorganic phosphate among conventionally reared calves but not amont the rapid-growth calves.

Selection of stock based on heritability of blood constituents was evaluated by Rowlands et al.[219]. They found serum concentrations of glucose, hemoglobin and potassium to be related to weight gains. There was a significant positive correlation between growth rate of calves from 1-12 weeks of age and glucose, hemoglobin, potassium, sodium and inorganic phosphorous. Sodium and albumin correlations persisted at 9 months of age. The average difference in weight gain during the 9 month period was 55 kg.

Metabolic profiling in the diagnosis of occult herd disease has been reviewed[144]. Hypoglycemia has been associated with infertility, ketoses and milk fever. Hypophosphatemia was related to infertility, bone disease and milk fever. Hyperglobulinemia was associated with chronic inflammation such as mastitis.

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APPENDIX II

Table 1

Individual Animal Data on Feedlot Cattle

Animal No.	Determination	Units	Date of Collection 1/24/76	4/22/76	6/3/76	7/4/76	8/24/76
1	Leukocytes	µℓ		11,770	9,000	6,600	7,300
	Band	µℓ		0	0	0	0
	Neutrophil	µℓ		3,413	2,250	660	1,095
	Lumphocyte	µℓ		6,591	5,760	4,752	5,183
	Monocyte	µℓ		1,412	630	924	584
	Eosinophil	µℓ		235	360	198	438
	Basophil	µℓ		118	90	66	0
	PCV	Vol %		38	38	41	37
	Total Lipids	mg/dℓ	280	408	313	425	398
	Triglyceride	mg/dℓ	35	27	14	9	31
	Cholesterol	mg/dℓ	80	113	82	113	124
	Phospholipid	mg/dℓ	116	153	94	68	196
	Plasma FFA	µeg/ℓ	545	325	283	250	87.6
	Cortisol	ng/mℓ	18	33	35	32	34
	L(+) Lactate	mg/dℓ	28.4	84.7	57.3	28.3	32.5
	D(-) Lactate	mg/dℓ	0	2.5	2.5	2.5	3.2
	Sodium	meg/ℓ	149	155	143	143	144
	Potassium	meg/ℓ	6.2	5.0	5.2	4.2	4.2
	Total CO_2	meg/ℓ	31	23	19	27	29
	Urea Nitrogen	mg/dℓ	5	25	11	13	13
	Alk. Phos.	mµ/mℓ	40	90	70	80	20
	GPT	mµ/mℓ	20	35	25	15	20
	Total Protein	g/dℓ	6.9	7.7	7.4	7.6	7.2
	Albumin	g/dℓ	3.2	3.7	3.6	3.7	3.5
	Calcium	mg/dℓ	9.6	9.8	9.0	8.9	9.1
	Phosphate	mg/dℓ	7.8	8.6	7.7	8.8	7.3
	Glucose	mg/dℓ	105	115	85	90	90
	Creatinine	mg/dℓ	1.2	1.3	1.4	1.6	1.7

Table 1 (Cont'd)

Animal No.	Determination	Units	Date of Collection 1/24/76	4/22/76	6/3/76	7/4/76	8/24/76
2	Leukocytes	µℓ	6,675	12,300	16,200	9,900	11,600
	Band	µℓ	0	0	0	0	0
	Neutrophil	µℓ	1,535	3,444	6,642	2,772	3,944
	Lumphocyte	µℓ	4,406	7,503	3,428	4,952	5,916
	Monocyte	µℓ	534	1,353	372	1,683	1,392
	Eosinophil	µℓ	134	0	162	495	348
	Basophil	µℓ	67	0	0	0	0
	PCV	Vol %	38	39	41	42	40
	Total Lipids	mg/dℓ	266	408	386	475	434
	Triglyceride	mg/dℓ	37	24	22	38	49
	Cholesterol	mg/dℓ	84	125	102	132	121
	Phospholipid	mg/dℓ	118	118	118	104	161
	Plasma FFA	µeg/ℓ	325	118	178	74	53
	Cortisol	ng/mℓ	32	28	28	18	25
	L(+) Lactate	mg/dℓ	38.7	24.0	50.5	24.1	24.2
	D(-) Lactate	mg/dℓ	0	2.5	3.2	2.5	2.5
	Sodium	meg/ℓ	148	154	145	142	144
	Potassium	meg/ℓ	5.9	6.2	4.8	4.1	4.0
	Total CO_2	meg/ℓ	27	31	22	24	12
	Urea Nitrogen	mg/dℓ	1	22	7	8	16
	Alk. Phos.	mµ/mℓ	65	285	270	205	160
	GPT	mµ/mℓ	35	35	30	15	20
	Total Protein	g/dℓ	7	7.8	7.3	7.3	7.2
	Albumin	g/dℓ	2.9	3.9	3.7	3.7	3.3
	Calcium	mg/dℓ	8.9	10.6	9.2	9.0	9.1
	Phosphate	mg/dℓ	5.8	8.4	6.7	8.0	7.8
	Glucose	mg/dℓ	110	120	60	115	245
	Creatinine	mg/dℓ	1.5	1.3	1.3	1.4	1.8

Table 1 (Cont'd)

Animal No.	Determination	Units	Date of Collection 1/24/76	4/22/76	6/3/76	7/4/76	8/24/76
3	Leukocytes	µℓ	9,000	11,800	11,700	12,900	14,000
	Band	µℓ		0	0	0	0
	Neutrophil	µℓ	1,260	3,776	4,329	2,839	2,660
	Lumphocyte	µℓ	6,480	5,726	5,382	5,837	8,820
	Monocyte	µℓ	990	1,298	1,755	2,451	1,820
	Eosinophil	µℓ	180	0	234	645	420
	Basophil	µℓ	90	0	0	129	0
	PCV	Vol %	37	37	35	41	35
	Total Lipids	mg/dℓ	239	294	304	357	270
	Triglyceride	mg/dℓ	33	26	9	24	25
	Cholesterol	mg/dℓ	73	144	80	93	91
	Phospholipid	mg/dℓ	103	97	30	113	107
	Plasma FFA	µeg/ℓ	400	260	151	168	83
	Cortisol	ng/mℓ	22	16	18	9	22
	L(+) Lactate	mg/dℓ	27.8	35.0	17.0	30.2	31.0
	D(-) Lactate	mg/dℓ	0	6.3	3.2	2.5	1.5
	Sodium	meg/ℓ	151	154	149	143	142
	Potassium	meg/ℓ	4.8	4.8	4.3	4.0	4.1
	Total CO_2	meg/ℓ	31	31	26	26	30
	Urea Nitrogen	mg/dℓ	7	20	9	7	11
	Alk. Phos.	mµ/mℓ	60	160	165	155	160
	GPT	mµ/mℓ	20	25	25	20	15
	Total Protein	g/dℓ	7.1	7.6	7.7	7.5	6.9
	Albumin	g/dℓ	3.8	4.0	3.8	3.8	3.6
	Calcium	mg/dℓ	10.-	10.3	9.2	9.0	9.4
	Phosphate	mg/dℓ	5.6	7.7	6.5	7.5	8.3
	Glucose	mg/dℓ	100	95	110	85	90
	Creatinine	mg/dℓ	1.3	1.2	1.1	1.3	1.5

Table 1 (Cont'd)

Animal No.	Determination	Units	Date of Collection 1/24/76	4/22/76	6/3/76	7/4/76	8/24/76
4	Leukocytes	µℓ	8,420	7,200	8,400	8,300	9,850
	Band	µℓ	0	0	0	0	0
	Neutrophil	µℓ	2,694	3,240	2,268	1,411	3,152
	Lumphocyte	µℓ	4,968	2,880	5,124	6,976	5,024
	Monocyte	µℓ	574	936	504	913	1,279
	Eosinophil	µℓ	84	0	336	0	296
	Basophil	µℓ	0	144	168	0	0
	PCV	Vol %	32	35	32	30.5	32
	Total Lipids	mg/dℓ	337	359	326	385	368
	Triglyceride	mg/dℓ	31	17	13	22	33
	Cholesterol	mg/dℓ	104	126	81	109	126
	Phospholipid	mg/dℓ	115	112	49	55	180
	Plasma FFA	µeg/ℓ	400	260	151	168	82.6
	Cortisol	ng/mℓ	43	40	36	38	32
	L(+) Lactate	mg/dℓ	12.5	75.5	20.8	38.2	23.3
	D(-) Lactate	mg/dℓ	1	2.5	2.5	3.2	1.5
	Sodium	meg/ℓ	146	155	153	141	140
	Potassium	meg/ℓ	4.6	4.9	4.7	4.2	4.3
	Total CO_2	meg/ℓ	28	32	26	28	26
	Urea Nitrogen	mg/dℓ	4	25	16	5.0	10
	Alk. Phos.	mµ/mℓ	85	190	120	90	110
	GPT	mµ/mℓ	25	10	20	15	20
	Total Protein	g/dℓ	8.4	7.4	7.0	7.3	7.3
	Albumin	g/dℓ	3.7	3.8	3.7	3.5	3.3
	Calcium	mg/dℓ	10.9	10.2	8.4	9.2	9.2
	Phosphate	mg/dℓ	6.0	7.1	10.8	8.2	7.6
	Glucose	mg/dℓ	120	85	95	80	70
	Creatinine	mg/dℓ	1.3	1.1	1.3	.9	1.3

Table 1 (Cont'd)

Animal No.	Determination	Units	Date of Collection 1/24/76	4/22/76	6/3/76	7/4/76	8/24/76
5	Leukocytes	µℓ	14,500	11,700	14,100	12,500	16,500
	Band	µℓ	0	117	0	0	0
	Neutrophil	µℓ	7,830	3,510	5,217	4,125	5,280
	Lumphocyte	µℓ	6,525	6,669	6,627	7,500	9,075
	Monocyte	µℓ	145	1,404	2,256	750	2,145
	Eosinophil	µℓ	0	0	0	0	0
	Basophil	µℓ	0	0	0	125	0
	PCV	Vol %	36	37	35	38	42
	Total Lipids	mg/dℓ	204	217	296	315	-
	Triglyceride	mg/dℓ	26	14	12	39	33
	Cholesterol	mg/dℓ	57	61	76	89	111
	Phospholipid	mg/dℓ	72	78	47	150	134
	Plasma FFA	µeg/ℓ	82	138	114	85	66.8
	Cortisol	ng/mℓ	25	20	22	32	28
	L(+) Lactate	mg/dℓ	27.8	50.6	21.3	24.4	39.3
	D(-) Lactate	mg/dℓ	0	4.0	4.0	2.5	2.5
	Sodium	meg/ℓ	144	>160	146	145	143
	Potassium	meg/ℓ	5.6	6.1	5.4	4.4	4.6
	Total CO_2	meg/ℓ	29	21	24	24	26
	Urea Nitrogen	mg/dℓ	6	20	7	5	8
	Alk. Phos.	mµ/mℓ	50	110	165	120	152
	GPT	mµ/mℓ	25	30	20	20	20
	Total Protein	g/dℓ	6.7	6.7	6.9	7.5	7.0
	Albumin	g/dℓ	3.4	3.4	3.6	3.7	3.7
	Calcium	mg/dℓ	9.0	8.6	9.5	9.2	10.0
	Phosphate	mg/dℓ	4.5	7.2	8.3	8.2	6.8
	Glucose	mg/dℓ	100	125	105	85	90
	Creatinine	mg/dℓ	1.2	1.3	1.1	1.3	1.5

Table 1 (Cont'd)

Animal No.	Determination	Units	Date of Collection 1/24/76	4/22/76	6/3/76	7/4/76	8/24/76
6	Leukocytes	µℓ	8,700	12,000	13,700	12,300	11,800
	Band	µℓ	87	0	137	0	0
	Neutrophil	µℓ	2,697	4,800	4,932	2,952	3,894
	Lumphocyte	µℓ	5,307	5,880	7,535	7,995	7,080
	Monocyte	µℓ	609	1,320	1,096	861	826
	Eosinophil	µℓ	0	0	0	369	0
	Basophil	µℓ	0	0	0	123	0
	PCV	Vol %	37	38	34	41	38
	Total Lipids	mg/dℓ	348	245	429	511	439
	Triglyceride	mg/dℓ	21	20	27	32	34
	Cholesterol	mg/dℓ	121	123	120	148	153
	Phospholipid	mg/dℓ	134	117	80	108	156
	Plasma FFA	µeg/ℓ	758	518	256	272	118
	Cortisol	ng/mℓ	19	14	35	42	35
	L(+) Lactate	mg/dℓ	31.5	26.5	50.4	17.5	20.5
	D(-) Lactate	mg/dℓ	0	4	3.2	2.5	2.5
	Sodium	meg/ℓ	144	160	150	142	144
	Potassium	meg/ℓ	5.6	6.0	5.1	4.3	4.1
	Total CO_2	meg/ℓ	34	32	22	26	27
	Urea Nitrogen	mg/dℓ	2	27	10	8	12
	Alk. Phos.	mµ/mℓ	50	180	165	14.5	125
	GPT	mµ/mℓ	10	30	25	15	20
	Total Protein	g/dℓ	7.0	6.8	7.0	7.5	7.4
	Albumin	g/dℓ	2.5	3.5	3.6	3.7	3.8
	Calcium	mg/dℓ	9.1	9.4	9.3	9.2	9.5
	Phosphate	mg/dℓ	4.5	7.0	7.7	9.0	8.5
	Glucose	mg/dℓ	80	85	165	120	85
	Creatinine	mg/dℓ	.9	1.3	1.2	1.4	1.6

Table 1 (Cont'd)

Animal No.	Determination	Units	Date of Collection 1/24/76	4/22/76	6/3/76	7/4/76	8/24/76
7	Leukocytes	µℓ	13,500	14,200	12,800	13,300	8,800
	Band	µℓ	135	0	0	0	0
	Neutrophil	µℓ	3,105	6,958	3,328	2,962	2,024
	Lumphocyte	µℓ	8,775	6,390	8,448	8,911	5,896
	Monocyte	µℓ	1,350	710	896	1,197	880
	Eosinophil	µℓ	0	0	128	266	0
	Basophil	µℓ	0	142	0	0	0
	PCV	Vol %	38	40	40	39	37
	Total Lipids	mg/dℓ	245	397	380	445	-
	Triglyceride	mg/dℓ	40	24	14	37	53
	Cholesterol	mg/dℓ	77	210	107	115	82
	Phospholipid	mg/dℓ	96	99	57	90	96
	Plasma FFA	µeg/ℓ	115	117.5	98.5	62.5	73.8
	Cortisol	ng/mℓ	36	30	21	33	32
	L(+) Lactate	mg/dℓ	26.9	82.6	37.0	22.8	32.1
	D(-) Lactate	mg/dℓ	0	4	2.5	3.2	2.5
	Sodium	meg/ℓ	144	150	147	144	137
	Potassium	meg/ℓ	4.2	5.3	5.5	4.5	4.4
	Total CO_2	meg/ℓ	33	28	24	24	30
	Urea Nitrogen	mg/dℓ	9	28	13	14	8
	Alk. Phos.	mµ/mℓ	50	170	135	105	85
	GPT	mµ/mℓ	25	35	30	20	25
	Total Protein	g/dℓ	7.9	8.4	7.3	7.5	6.7
	Albumin	g/dℓ	4.0	4.6	3.9	3.9	3.5
	Calcium	mg/dℓ	9.4	10.8	9.2	9.4	9.3
	Phosphate	mg/dℓ	4.4	8.7	8.2	8.2	6.5
	Glucose	mg/dℓ	90	100	105	95	65
	Creatinine	mg/dℓ	1.1	1.2	1.2	1.3	1.5

Table 1 (Cont'd)

Animal No.	Determination	Units	Date of Collection 1/24/76	4/22/76	6/3/76	7/4/76	8/24/76
8	Leukocytes	µℓ	9,560	15,400		10,800	9,500
	Band	µℓ	0	0		0	0
	Neutrophil	µℓ	3,155	6,160	clot	1,188	2,660
	Lumphocyte	µℓ	5,067	7,854		7,668	5,700
	Monocyte	µℓ	1,338	1,028		1,404	950
	Eosinophil	µℓ	0	154		540	190
	Basophil	µℓ	0	0		0	0
	PCV	Vol %	37	39		43	40
	Total Lipids	mg/dℓ	361	386	457	553	452
	Triglyceride	mg/dℓ	26	18	18	28	32
	Cholesterol	mg/dℓ	105	117	130	164	153
	Phospholipid	mg/dℓ	107	57	117	81	190
	Plasma FFA	µeg/ℓ	363	182.6	134.7	93.8	92.8
	Cortisol	ng/mℓ	16	24	21	33	32
	L(+) Lactate	mg/dℓ	42.2	62.4	54.2	24.3	28.4
	D(-) Lactate	mg/dℓ	0	6.3	2.5	2.5	1.8
	Sodium	meg/ℓ	150	149	148	143	139
	Potassium	meg/ℓ	5.7	5.0	5.2	4.1	3.7
	Total CO_2	meg/ℓ	29	25	20	22	23
	Urea Nitrogen	mg/dℓ	5	20	13	14	11
	Alk. Phos.	mµ/mℓ	80	120	170	120	95
	GPT	mµ/mℓ	5	30	25	15	15
	Total Protein	g/dℓ	9	8.5	8.0	8.0	7.5
	Albumin	g/dℓ	4.5	4.1	4.0	3.9	3.8
	Calcium	mg/dℓ	11.0	10.0	9.8	9.7	9.2
	Phosphate	mg/dℓ	5.6	7.2	7.8	7.9	6.8
	Glucose	mg/dℓ	155	105	150	100	105
	Creatinine	mg/dℓ	1.3	1.3	1.2	1.4	1.5

Table 1 (Cont'd)

Animal No.	Determination	Units	Date of Collection 1/24/76	4/22/76	6/3/76	7/4/76	8/24/76
9	Leukocytes	µℓ	8,890	8,200	11,200	9,300	9,500
	Band	µℓ	0	0	0	0	0
	Neutrophil	µℓ	2,756	3,280	3,920	2,697	1,805
	Lumphocyte	µℓ	5,512	4,100	6,495	5,766	6,270
	Monocyte	µℓ	267	410	672	744	1,425
	Eosinophil	µℓ	89	82	0	93	0
	Basophil	µℓ	267	0	112	0	0
	PCV	Vol %	34	33	35	42	40
	Total Lipids	mg/dℓ	340	326	359	434	422
	Triglyceride	mg/dℓ	28	23	16	21	45
	Cholesterol	mg/dℓ	82	101	85	127	140
	Phospholipid	mg/dℓ	125	60	65	68	205
	Plasma FFA	µeg/ℓ	278	292	163	206	82
	Cortisol	ng/mℓ	14	16	16	18	22
	L(+) Lactate	mg/dℓ	25.2	55.6	15.2	19.6	22.9
	D(-) Lactate	mg/dℓ	0	4.0	3.2	2.5	2.5
	Sodium	meg/ℓ	147	147	153	141	138
	Potassium	meg/ℓ	5.3	5.1	5.0	4.3	4.3
	Total CO_2	meg/ℓ	33	26	27	25	28
	Urea Nitrogen	mg/dℓ	4	20	13	12	12
	Alk. Phos.	mµ/mℓ	85	120	180	180	160
	GPT	mµ/mℓ	10	20	20	10	10
	Total Protein	g/dℓ	7.5	7.2	7.2	6.9	7.0
	Albumin	g/dℓ	4.0	3.7	3.6	3.7	3.8
	Calcium	mg/dℓ	10.0	8.6	9.1	9.0	9.0
	Phosphate	mg/dℓ	7.2	5.7	8.8	8.0	7.8
	Glucose	mg/dℓ	100	95	90	90	75
	Creatinine	mg/dℓ	1.3	1.7	1.4	1.3	1.6

Table 1 (Cont'd)

Animal No.	Determination	Units	Date of Collection 1/24/76	4/22/76	6/3/76	7/4/76	8/24/76
10	Leukocytes	µℓ	4,760	8,020		10,900	7,400
	Band	µℓ	0	80		0	0
	Neutrophil	µℓ	950	1,925		4,905	1,702
	Lumphocyte	µℓ	2,832	4,972	clot	5,123	4,736
	Monocyte	µℓ	470	642		654	740
	Eosinophil	µℓ	0	401		109	222
	Basophil	µℓ	0	0		109	0
	PCV	Vol %	38	40		38.5	35
	Total Lipids	mg/dℓ	169	326	283	264	-
	Triglyceride	mg/dℓ	23	17	20	26	27
	Cholesterol	mg/dℓ	58	88	67	65	101
	Phospholipid	mg/dℓ	55	76	13	125	134
	Plasma FFA	µeg/ℓ	736	442	418	313	98
	Cortisol	ng/mℓ	20	24	21	22	23
	L(+) Lactate	mg/dℓ	23.3	48.5	31.9	22.3	18.9
	D(-) Lactate	mg/dℓ	0	4	2.5	3.2	2.5
	Sodium	meg/ℓ	>160	154	145	142	139
	Potassium	meg/ℓ	5.4	5.2	4.7	4.0	3.7
	Total CO_2	meg/ℓ	34	29	22	23	28
	Urea Nitrogen	mg/dℓ	4	19	12	12	14
	Alk. Phos.	mµ/mℓ	75	130	145	110	110
	GPT	mµ/mℓ	20	25	25	15	15
	Total Protein	g/dℓ	8.8	8.0	7.7	7.4	7.5
	Albumin	g/dℓ	3.9	3.9	3.5	3.6	3.6
	Calcium	mg/dℓ	9.6	9.2	8.8	8.0	8.6
	Phosphate	mg/dℓ	5.4	8.7	6.5	6.2	7.5
	Glucose	mg/dℓ	105	100	115	100	85
	Creatinine	mg/dℓ	1.1	1.0	1.2	1.0	1.4

Table 2. Individual Animal Data on Transport Stressed Cattle

Animal No.		1		2		3		4	
Determination	Units	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76
Leukocytes	µℓ	9,250	16,500	6,875	9,600	9,350	17,500	11,300	11,500
Band	µℓ	0	330	0	675	0	175	0	230
Neutrophil	µℓ	1,110	4,620	2,063	4,224	1,965	5,650	2,710	4,715
Lumphocyte	µℓ	6,660	10,056	4,263	4,032	5,330	9,100	5,311	4,300
Monocyte	µℓ	1,110	1,485	413	384	1,124	1,400	2,034	920
Eosinophil	µℓ	0	0	0	96	655	0	791	230
Basophil	µℓ	185	0	138	193	187	175	0	0
PCV	Vol %	45	32	40	40	32	31	34	32
Total Lipids	mg/dℓ	362	382	273	247	335	382	355	442
Triglyceride	mg/dℓ	19	20	23	38	29	38	22	36
Cholesterol	mg/dℓ	114	138	86	90	90	129	108	153
Phospholipid	mg/dℓ	51	176	68	146	104	167	44	191
Plasma FFA	µeg/ℓ	480	325	600	425	660	375	525	627
Cortisol	ng/mℓ	45	32	22	19	36	35	22	24
L(+) Lactate	mg/dℓ	81.8	50.4	78.9	56.4	17.4	39.2	39.2	24.1
D(-) Lactate	mg/dℓ	0	0	0	0	0	1.5	0	1.5
Sodium	meg/ℓ	142	131	138	131	141	137	139	133
Potassium	meg/ℓ	4.4	5.1	5.4	5.9	4.5	4.4	3.8	4.3
Total CO_2	meg/ℓ	16	22	21	15	35	21	33	26
Urea Nitrogen	mg/dℓ	9	8	13	5	6	8	6	12
Alk. Phos	mµ/mℓ	90	115	50	60	105	80	90	70
GPT	mµ/mℓ	15	30	10	25	25	40	25	10
Total Protein	g/dℓ	6.4	6.8	7.7	8.1	6.5	7.3	6.4	7.6
Albumin	g/dℓ	3.7	3.5	4.2	3.3	3.5	8.6	3.2	3.4
Calcium	mg/dℓ	8.9	9.4	9.8	9.8	10	3.5	9.6	8.6
Phosphate	mg/dℓ	8.2	8.6	5.1	6.2	7.8	8.4	6.1	7.8
Glucose	mg/dℓ	95	60	100	70	80	65	90	55
Creatinine	mg/dℓ	1.6	1.1	1.5	.8	1.0	.7	1.1	.8

Table 2 (Cont'd)

Animal No.		5		6		7		8	
Determination	Units	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76
Leukocytes	µℓ	10,450	8,000	7,685	7,600	9,300	9,200	12,100	9,600
Band	µℓ	0	80	0	152	0	92	0	0
Neutrophil	µℓ	3,746	2,480	1,153	3,192	1,860	920	2,904	2,784
Lumphocyte	µℓ	5,723	4,400	5,075	3,340	6,231	7,084	7,018	5,376
Monocyte	µℓ	415	640	154	760	372	1,104	847	1,056
Eosinophil	µℓ	208	160	1,306	152	279	0	726	430
Basophil	µℓ	312	240	0	0	93	0	121	0
PCV	Vol %	35	35	38	38	41	40	40	39
Total Lipids	mg/dℓ	444	426	352	369	466	282	343	406
Triglyceride	mg/dℓ	20	36	18	59	30	36	23	25
Cholesterol	mg/dℓ	142	143	117	127	137	83	120	132
Phospholipid	mg/dℓ	68	180	43	172	72	107	45	155
Plasma FFA	µeg/ℓ	162	213	421	193	587	418	315	287
Cortisol	ng/mℓ	29	21	12	19	41	28	18	23
L(+) Lactate	mg/dℓ	35.9	33.7	21.1	21.3	69.3	32.7	39.4	37.6
D(-) Lactate	mg/dℓ	0	0	0	0	0	0	0	0
Sodium	meg/ℓ	140	138	138	141	144	138	142	130
Potassium	meg/ℓ	4.0	4.1	3.8	4.2	4.2	4.6	5.0	4.0
Total CO_2	meg/ℓ	31	9	26	22	20	25	32	29
Urea Nitrogen	mg/dℓ	5	10	6	10	7	14	3	15
Alk. Phos	mµ/mℓ	125	85	105	70	115	130	70	50
GPT	mµ/mℓ	25	30	15	30	80	30	5	35
Total Protein	g/dℓ	7.8	8.6	7.7	8.9	6.5	6.7	6.5	7.9
Albumin	g/dℓ	4.0	3.8	3.8	3.7	4.1	3.9	3.4	3.7
Calcium	mg/dℓ	9.8	9.5	9.1	9.1	9.4	8.9	10.6	9.3
Phosphate	mg/dℓ	6.9	8.1	5.9	7.9	8.3	7.3	6.7	8.3
Glucose	mg/dℓ	95	70	75	60	90	50	75	60
Creatinine	mg/dℓ	1.0	.8	1.0	.9	.9	.8	1.1	.8

Table 2 (Cont'd)

Animal No.		9		10		11		12	
Determination	Units	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76
Leukocytes	µℓ	9,400	11,850	6,800	10,500	5,700	7,810	8,880	9,000
Band	µℓ	0	474	272	0	0	0	178	0
Neutrophil	µℓ	1,686	4,977	1,768	2,100	912	3,202	2,045	720
Lumphocyte	µℓ	5,358	5,214	3,876	7,560	3,306	3,202	5,417	7,560
Monocyte	µℓ	752	593	816	525	513	1,250	977	630
Eosinophil	µℓ	1,316	593	68	315	786	156	88	90
Basophil	µℓ	0	0	0	0	0	0	0	0
PCV	Vol %	35	36	41	33	40	38	33	23
Total Lipids	mg/dℓ	368	320	351	347	407	344	436	377
Triglyceride	mg/dℓ	22	26	21	23	20	34	25	17
Cholesterol	mg/dℓ	155	113	103	123	135	121	188	63
Phospholipid	mg/dℓ	37	136	51	155	30	157	61	82
Plasma FFA	µeg/ℓ	421	373	222	228	824	558	761	487
Cortisol	ng/mℓ	16	27	24	24	33	32	38	35
L(+) Lactate	mg/dℓ	22.5	21.9	91.7	22.2	67.4	39.8	27.3	40.4
D(-) Lactate	mg/dℓ	0	0	0	0	0	0	0	0
Sodium	meg/ℓ	140	139	134	133	148	139	137	137
Potassium	meg/ℓ	4.2	4.2	3.5	4.4	5.5	4.9	4.1	4.4
Total CO_2	meg/ℓ	27	19	10	24	25	23	29	23
Urea Nitrogen	mg/dℓ	8	8	3	8	5	11	8	9
Alk. Phos	mµ/mℓ	125	60	60	85	120	70	145	110
GPT	mµ/mℓ	17	30	11	35	20	35	18	35
Total Protein	g/dℓ	6.9	7.7	6.1	8.1	6.9	7.6	6.6	6.4
Albumin	g/dℓ	3.9	3.8	2.9	3.5	3.7	3.7	3.8	3.7
Calcium	mg/dℓ	9.4	10.0	8.4	8.7	9.4	9.3	8.6	8.6
Phosphate	mg/dℓ	6.8	6.3	5.3	6.8	7.7	7.9	8.4	8.7
Glucose	mg/dℓ	65	50	90	60	85	65	75	85
Creatinine	mg/dℓ	1.0	.7	1.1	.7	1.1	.7	1.5	1.0

Table 2 (Cont'd)

Animal No.		13		14		15			
Determination	Units	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76	Date of Collection 7/4/76	8/22/76
Leukocytes	µℓ	6,550	11,600	8,150		12,000	13,800		
Band	µℓ	0	0	0		0	690		
Neutrophil	µℓ	786	812	2,200		3,840	5,796		
Lumphocyte	µℓ	4,716	9,744	4,972	clot	5,160	6,201		
Monocyte	µℓ	917	812	408		1,680	1,104		
Eosinophil	µℓ	0	116	326		840	0		
Basophil	µℓ	0	116	163		360	0		
PCV	Vol %	42	38	41		36	36		
Total Lipids	mg/dℓ	307	372	328	341	408	320		
Triglyceride	mg/dℓ	18	16	23	33	27	30		
Cholesterol	mg/dℓ	85	91	98	110	134	111		
Phospholipid	mg/dℓ	81	119	54	141	94	161		
Plasma FFA	µeg/ℓ	385	325	675	598	392	331		
Cortisol	ng/mℓ	22	23	28	29	12	21		
L(+) Lactate	mg/dℓ	24.3	39.9	32.6	24.9	48.1	24.9		
D(-) Lactate	mg/dℓ	0	0	0	0	0	0		
Sodium	meg/ℓ	136	137	132	139	138	136		
Potassium	meg/ℓ	4.3	4.7	4.1	4.6	5.0	4.2		
Total CO_2	meg/ℓ	25	26	34	26	28	17		
Urea Nitrogen	mg/dℓ	5	8	9	7	7	9		
Alk. Phos	mµ/mℓ	120	70	125	85	100	65		
GPT	mµ/mℓ	16	30	22	30	27	30		
Total Protein	g/dℓ	6.9	6.9	6.4	7.4	8.3	8.6		
Albumin	g/dℓ	4.0	3.4	3.3	3.5	3.6	3.8		
Calcium	mg/dℓ	9.7	9.0	10.0	8.9	11.2	9.6		
Phosphate	mg/dℓ	5.7	7.8	5.2	7.6	5.8	7.9		
Glucose	mg/dℓ	70	65	80	65	75	65		
Creatinine	mg/dℓ	1.5	1.1	1.2	.9	1.1	.7		

Table 3. Individual Animal Data on Disease Stressed Cattle.

Animal No.		1		2		3		4	
Determination	Units	Date of Collection 7/8/76	8/22/76	Date of Collection 7/8/76	8/22/76	Date of Collection 7/8/76	8/22/76	Date of Collection 7/8/76	8/22/76
Leukocytes	µℓ	8,900	11,000	11,400	16,500	9,900	12,500	12,400	10,800
Band	µℓ	0	0	0	0	0	0	0	0
Neutrophil	µℓ	1,513	1.750	2,622	4,620	2,178	4,125	5,456	2,268
Lumphocyte	µℓ	5,963	7,590	7,866	10,065	6,930	7,125	5,704	7,128
Monocyte	µℓ	1,068	1,540	798	1,485	693	1,250	496	756
Eosinophil	µℓ	356	0	0	0	99	0	620	540
Basophil	µℓ	0	110	0	0	0	0	124	0
PCV	Vol %	41	36	40	32	35	32	36	32
Total Lipids	mg/dℓ	455	310	336	382	374	424	314	248
Triglyceride	mg/dℓ	33	32	28	20	27	28	36	30
Cholesterol	mg/dℓ	153	89	105	138	109	131	110	79
Phospholipid	mg/dℓ	31	132	71	176	27	164	38	108
Plasma FFA	µeg/ℓ	990	663	630	325	785	692	832	677
Cortisol	ng/mℓ	58	32	73	19	59	8	83	9
L(+) Lactate	mg/dℓ	-	67.3	-	50.4	-	44	-	31.7
D(-) Lactate	mg/dℓ	0	0	0	0	0	1.5	0	0
Sodium	meg/ℓ	138	138	138	137	136	138	135	134
Potassium	meg/ℓ	4.1	4.3	4.6	5.1	4.0	4.7	4.0	5.3
Total CO_2	meg/ℓ	28	12	18	22	25	22	26	27
Urea Nitrogen	mg/dℓ	8	6	6	8	4	13	2	9
Alk. Phos	mµ/mℓ	50	115	60	115	45	85	65	110
GPT	mµ/mℓ	45	30	20	30	45	45	25	45
Total Protein	g/dℓ	6.4	7.7	6.2	6.8	6.5	7.3	6.6	7.7
Albumin	g/dℓ	3.5	3.3	3.2	3.5	3.1	3.4	3.1	3.7
Calcium	mg/dℓ	9.2	9.7	8.6	9.4	8.6	9.2	8.9	9.6
Phosphate	mg/dℓ	6.9	7.1	8.5	8.6	5.4	8.1	6.3	7.5
Glucose	mg/dℓ	80	75	75	60	80	60	85	65
Creatinine	mg/dℓ	1.2	.7	1.7	1.1	.9	.6	.9	.9

Table 3 (Cont'd)

Animal No.		5		6		7		8	
Determination	Units	Date of Collection 7/8/76	8/22/76	Date of Collection 7/8/76	8/22/76	Date of Collection 7/8/76	8/22/76	Date of Collection 7/8/76	8/22/76
Leukocytes	µℓ	8,900	6,250	12,800	9,000	6,800	10,500	13,700	
Band	µℓ	0	0	0	0	272	0	685	
Neutrophil	µℓ	2,047	625	1,152	720	1,768	2,100	8,354	
Lumphocyte	µℓ	6,230	4,688	9,088	7,560	3,876	7,564	3,288	
Monocyte	µℓ	534	813	2,048	630	816	525	1,370	
Eosinophil	µℓ	0	125	512	90	68	313	0	
Basophil	µℓ	0	0	0	0	0	0	0	
PCV	Vol %	37	34	31	23	41	33	35	
Total Lipids	mg/dℓ	314	286	451	377	351	347	171	
Triglyceride	mg/dℓ	22	37	45	17	21	23	28	
Cholesterol	mg/dℓ	97	97	140	63	103	123	46	
Phospholipid	mg/dℓ	29	134	20	82	51	155	53	
Plasma FFA	µeg/ℓ	663	652	782	487	222	228	377	
Cortisol	ng/mℓ	82	11	84	14	98	12	36	
L(+) Lactate	mg/dℓ	-	57.7	-	50.4	91.7	22.2	57	
D(-) Lactate	mg/dℓ	0	0	0	0	0	0	0	
Sodium	meg/ℓ	136	139	139	137	134	133	145	
Potassium	meg/ℓ	3.7	5.1	3.9	4.4	3.5	4.4	3.9	
Total CO_2	meg/ℓ	21	26	23	23	10	24	31	
Urea Nitrogen	mg/dℓ	2	10	8	9	3	8	17	
Alk. Phos	mµ/mℓ	45	85	75	110	60	85	60	
GPT	mµ/mℓ	30	35	35	35	11	35	15	
Total Protein	g/dℓ	7.0	7.4	6.3	6.4	6.1	8.1	8.0	
Albumin	g/dℓ	3.1	3.8	3.4	3.7	2.9	3.5	3.3	
Calcium	mg/dℓ	9.0	9.3	8.7	8.6	8.4	8.7	8.2	
Phosphate	mg/dℓ	5.5	7.6	7.6	8.7	5.3	6.8	7.3	
Glucose	mg/dℓ	90	60	85	85	90	60	135	
Creatinine	mg/dℓ	1.1	1.1	1.4	1.0	1.1	.7	1.7	

Table 3 (Cont'd)

Animal No.		9		10					
Determination	Units	Date of Collection 7/8/76	8/22/76	Date of Collection 7/8/76	8/22/76	Date of Collection 7/8/76	8/22/76	Date of Collection 7/8/76	8/22/76
Leukocytes	µℓ	8,900		6,800					
Band	µℓ	445		680					
Neutrophil	µℓ	2,670		4,488					
Lumphocyte	µℓ	4,450		1,360					
Monocyte	µℓ	445		272					
Eosinophil	µℓ	445		0					
Basophil	µℓ	0		0					
PCV	Vol %	30		16					
Total Lipids	mg/dℓ	270		350					
Triglyceride	mg/dℓ	28		18					
Cholesterol	mg/dℓ	84		84					
Phospholipid	mg/dℓ	63		72					
Plasma FFA	µeg/ℓ	850		820					
Cortisol	ng/mℓ	29		42					
L(+) Lactate	mg/dℓ	-		-					
D(-) Lactate	mg/dℓ	0		0					
Sodium	meg/ℓ	149		132					
Potassium	meg/ℓ	2.4		5.2					
Total CO_2	meg/ℓ	36		30					
Urea Nitrogen	mg/dℓ	19		8					
Alk. Phos	mµ/mℓ	90		95					
GPT	mµ/mℓ	112		-					
Total Protein	g/dℓ	7.0		5.3					
Albumin	g/dℓ	3.5		2.4					
Calcium	mg/dℓ	8.2		8.6					
Phosphate	mg/dℓ	6.8		5.8					
Glucose	mg/dℓ	100		92					
Creatinine	mg/dℓ	-		1.2					

BIOCHEMICAL AND CELLULAR PROFILES IN FEEDLOT CATTLE DURING NORMAL FEEDING TRIALS FOLLOWING TRANSPORT AND DURING RESPIRATORY DISEASE

by

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DVM: University of Minnesota, 1971

AN ABSTRACT OF A MASTER'S THESIS

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ABSTRACT

I. Ten feedlot steers were sampled at five intervals during feeding trials at the K.S.U. Beef Research Unit and hematologic and biochemical parameters compared with time on feed. The PCV increased significantly during the feeding trial while total leukocytes and leukocyte differential showed a significant change. Total serum lipids increased throughout the trial but the summation of component lipids was much less than total lipids as measured by the phosphovanillin reaction. Cholesterol, triglycerides and phospholipids decreased until the middle of the trial and increased thereafter. Plasma FFA were high initially and decreased throughout the feeding trial varying inversely with the level of concentrate feeding.

L(+) and D(-) lactate peaked at day 125 and decreased thereafter suggesting that high concentrate rations produce an increased rumen lactate absorption but that feedlot animals adapt perhaps by changes in body metabolism.

Serum sodium, potassium and calcium decreased throughout the trial. Serum urea nitrogen increased with time on feed with a five fold increase at day 125 which cannot presently be explained. Mean serum total protein decreased slightly during the feeding trial ($P = .1$) with no change in albumin values. Mean serum cortisol values increased throughout the trial but significant changes were not noted.

II. Fifteen feedlot steers were sampled within 24 hours post-transport and biochemical and hematologic profiles indicated an increased PCV,

sodium and creatinine, and plasma FFA and a decreased total protein, inorganic phosphate and phospholipids compared to values for the same group 30 days later. The presence of hemoconcentration due to transport may explain the elevated PCV, sodium and creatinine. Stress-related hormonal release may explain the elevated plasma FFA levels post-transport. The increased total protein and inorganic phosphate at the second sampling may be related to the switch from a roughage to a roughage plus concentrate ration which was relatively high in protein and phosphate. Phospholipids were very low post-transport.

III. Ten feedlot steers were sampled two days after developing an acute respiratory disease and biochemical and hematologic profiles indicated an increased PCV, plasma FFA, potassium, cholesterol, glucose, creatinine, and cortisol and a decreased phospholipid, urea nitrogen, alkaline phosphatase, total protein, albumin, calcium, and inorganic phosphate compared to values for the same group two weeks after recovery. Most changes were similar to the transport group suggesting that they may be part of a nonspecific stress response. Serum cortisol levels were increased almost three-fold during illness and decreased to subnormal levels at the second sampling suggesting that cortisol is a good indicator of acute stress in feedlot cattle.