CHANGES IN PLASMA LEVELS OF NON-ESTERIFIED FATTY ACIDS (NEFA), GLUCOSE AND L(+)-LACTATE IN: I. BEEF CATTLE UNDER FEEDLOT CONDITIONS AND BACKGROUND-AGE CALVES WITH RESPIRATORY DISEASE II. DAIRY COWS FROM LATE PREGNANCY INTO EARLY LACTATION

by

DANIEL B. OLUMEYAN

D. V. M., Ahmadu Bello University, Zaria, Nigeria, 1970

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Department of Surgery and Medicine

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Approved by:

Russell A. Frey

Major Professor

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To my dear wife and children

GENERAL INTRODUCTION

Criteria presently used for establishing optimum nutrient intake for ruminants are not adequate. In pregnant animals, changes in body weight have been used for measuring nutrient adequacy. However, the change in the weight of the fetus confuses the interpretation of the change in weight of the female. In nutritional studies where short-term effects are of interest, the use of body weight, milk or wool production is often not satisfactory as a measure of response. In order to demonstrate a response in these situations it is necessary to establish criteria that can be accurately measured and are closely associated with the metabolic processes within the body. Most physiological processes involve transportation of metabolites by the blood. An index of changes in types or rates of biochemical processes, as influenced by nutrition or disease, may be obtained by measuring blood concentrations of various metabolites.

Levels of nutrient intake are associated with variation in fat metabolism, especially mobilization of depot fat during periods of low intake. Related to this process are changes in blood levels of non-esterified fatty acids (NEFA), ketone bodies and glucose. NEFA are released into plasma when adipose tissue is mobilized to supply metabolic needs for energy. Although the quantity of NEFA in ruminant blood is small, the turnover rate is high and therefore, important in caloric hemeostasis. Physiological regulation of the release of NEFA from adipose tissue has not been well defined in ruminants.

Studies in sheep have indicated the possibility of using blood levels of NEFA and ketone bodies as indicators for defining nutrient requirements for pregnant ewes. Information on NEFA levels in cattle has been collected primarily in conjunction with establishing relationships of NEFA, ketone

bodies and glucose in the blood of ketotic cows or those on diets resulting in the production of low-fat milk. Very little information is available on these blood constituents in beef cattle. We are not aware of any published reports on the plasma NEFA or blood ketones for defining nutritional status in pregnant or lactating cattle.

We are also not aware of published reports on studies relating blood levels of NEFA or ketones to the nutritional status of growing sheep or cattle. A few extensive literature reviews of NEFA and ketone bodies in the blood as indicators of nutritional status in ruminants suggested that more detailed information on the nutrient content of diets, including energy levels, protein, mineral and vitamin contents will be required. It was further suggested that the relationship of these constituents to blood glucose should be considered.

Lactate metabolism in ruminants is complicated by the endogenous production and that variable and undetermined amounts are absorbed from the digestive tract. Endogenous lactate produced can be metabolized to glucose in the liver and kidney via the Cori cycle. This does not, however, result in a net increase in glucose production for the body as endogenous lactate is derived from glucose. Unlike lactate involved in the Cori cycle, absorbed lactate will furnish new glucose.

There has been some work done on the metabolic interrelationships of glucose and lactate in the blood of sheep and goats. There does not appear to be any published attempts to correlate levels of blood lactate to those of glucose, NEFA and ketone bodies in cattle under common feeding practices.

The objectives of this study, therefore, were:

(I) to determine plasma levels of glucose, (L(+)-lactate, and NEFA in:

(a) feedlot cattle - from start to finish,

(b) dairy cattle - from late pregnancy into early lactation,

(c) background-age calves* with respiratory disease;

(II) to relate blood levels of these metabolites to one another and to correlate them with energy intake and any known physiological and/or pathological conditions.

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*Background-age calves are weaned calves in their growing and preparatory phase for feedlot fattening.

CHANGES IN PLASMA LEVELS OF NON-ESTERIFIED FATTY ACIDS (NEFA), GLUCOSE AND L(+)-LACTATE:

PAPER I. IN BEEF CATTLE UNDER FEEDLOT CONDITIONS AND BACKGROUND-AGE CALVES WITH RESPIRATORY DISEASE

SUMMARY

Determinations of NEFA, glucose and L(+)-lactate on plasma samples collected from 24 steers randomly selected from 180 animals on arrival into the Kansas State University Feedlot, and when put on finishing ration trials, which lasted for seven months, indicated that the effect of transportation stress and excitement significantly elevated plasma NEFA and glucose concentrations.

Ration composition and switches had little or no effect on these plasma constituents within animal ration groups. When put on increasingly carbohydrate-rich rations, however, gradual elevation of plasma glucose, decline in plasma NEFA and L(+)-lactate levels with time, became highly significant ($P < 0.001$). Correlation of glucose and NEFA was 0.973, and that of glucose and L(+)-lactate, 0.915, for the means for all 24 steers.

The determinations of the same plasma constituents for 17 background-age calves arriving into a backgrounding feedlot with subsequent treatment for respiratory disease showed that a fast-response group had significantly higher plasma glucose levels ($P = 0.05$) than a slow-response group. Plasma NEFA levels were consistently higher and L(+)-lactate lower in slow- than in fast-response group.

INTRODUCTION

The metabolic importance of plasma non-esterified fatty acids (NEFA) was first recognized by Dole[8] and Gordon and Cherkes[12] who showed that on fasting, plasma NEFA concentrations rose, but oral administration of glucose depressed NEFA levels. This observation suggested a relationship between the nutritional state of human subjects and plasma NEFA levels. Since that time many

workers have directed their attention to a possible use of plasma NEFA levels as an indicator of the nutritional status of ruminants.

In sheep, plasma NEFA levels decreased after feeding, after glucose administration and transiently after insulin was given.[1] A 40% reduction of the feed intake of fat non-pregnant sheep greatly elevated the plasma NEFA level within one day, yet had no significant effect on blood glucose and ketone bodies. Thus NEFA levels seemed to be a sensitive indicator of fat mobilization during undernourishment in sheep.[26] Russel et al.[27] suggested that the usefulness of plasma NEFA and blood ketones as indicators of nutritional status may vary with the degree of undernourishment.

The rise of NEFA concentration before feeding or fasting and the fall after feeding have been investigated by several workers.[20,23,26] When fed once a day, ruminants showed a characteristic diurnal fluctuation in NEFA concentration due to alterations in fat mobilization reciprocal to absorption of nutrients. Plasma concentrations of glucose and NEFA have been found to be inversely proportional to each other.[26] Annison,[1] however, found that in fasting ewes, the blood glucose levels declined more slowly than NEFA increased and he concluded that increase in NEFA was not directly due to hypoglycemia, but may be the result of a shortage of short-chain fatty acids.

The type of ration and level of intake may markedly influence levels of blood constituents. Carbohydrate content of the ration, when high, decreased plasma NEFA.[1,26] In Hereford cattle, plasma NEFA levels varied with digestible energy intake and fell after a meal.[17] Plasma NEFA levels in cattle on a high-grain ration did not increase with reduction in digestible energy intake as they did on rations with less than 75% grain.[17] Blood glucose also increased with high-grain intake.[17]

Although plasma NEFA concentration has been regarded as a sensitive indicator of fat mobilization during energy insufficiency, striking variations in concentration unrelated to energy intake have been found. Excitement elevated plasma NEFA levels in sheep[21,23,30] and in cattle.[17] Stressful conditions[30] and the administration of several hormones[4] have been noted to elevate plasma NEFA levels.

All studies have been concerned with establishing relationships between NEFA, ketone bodies and glucose in ovine and bovine ketosis, and recently[17] in grazing cattle. There does not appear to be any information available on these blood constituents in beef cattle under feedlot conditions, where the ultimate goal is to fatten these animals for slaughter in the shortest possible time. The high concentrate rations for finishing feedlot animals may result in an increased rate of lactic acid production in the rumen[25,32] and this may result in lowering the rumen pH thus enhancing lactate absorption.[29]

This study was conducted to follow the trends of plasma NEFA, glucose and L(+)-lactate in feedlot cattle from time of entering until finish, and in background-age calves with some respiratory disease.

MATERIALS AND METHODS

Animals:

(i) Twenty-four yearling steers of various beef breeds, were randomly selected from a population of 180 entering the Kansas State University feedlot. The first blood sample was collected within twenty-four hours of their arrival into the feedlot. Subsequent blood samples were taken as the steers were put on rations which gradually changed in composition from high-roughage-low-concentrate to low-roughage-high-concentrate from entry to finishing time. Blood samples were collected from July, 1973 to March, 1974.

Feeding:

From arrival until commencement of feeding trials, the steers were fed hay supplemented with silage and grain.

Six groups of steers were put on rations which varied in composition throughout the feeding trials. The animal in the same ration group, ration number and composition, and the feeding schedule with ration switches are presented in tables 1, 2, and 3.

Steers in groups 1, 2, and 3 together (I) were on (a) and 4, 5, and 6 (II) on (b) of rations 1-4 respectively, until a switch to ration 5 was made, when the animals were rearranged into new groups as shown in table 3 and remained so until the end of the trial.

Until December 6, 1973, the average daily feed intake per head (D.M. basis) was 8.46 kg and 8.63 kg for groups I and II respectively. From December 6th until the end of the study the average daily intake for both groups was 9.98 kg per head.

TABLE 1

ANIMAL RATION GROUPS

	RATION GROUPS	ANIMAL NUMBER					
	1	00	103	335	343	--	--
I	2	111	119	--	--	--	--
	3	127	143	303	311	319	327
	4	219	227	--	--	--	--
II	5	203	235	243	403	411	--
	6	211	419	427	435	443	--

TABLE 2

RATION COMPOSITION

RATION NUMBERS	1a	1b	2a	2b	3a	3b	4a	4b	5a	5b
INGREDIENTS:	PER CENT COMPOSITION (D.M. BASIS)									
Sorghum silage	60	60	--	--	55	55	30	30	20	20
Haylage	--	--	56.2	56.2	--	--	--	--	--	--
Milo (sorghum grain)	35.5	29.6	43.8	37.3	20	16	--	--	--	--
Corn (maize)	--	--	--	--	15	14	28	32	37.5	34.5
Corn flakes	--	--	--	--	--	--	31.5	35.5	40	37
Cotton seed meal	--	5.9	--	6.5	7.5	12.5	8	--	--	--
Soybean meal	--	--	--	--	--	--	--	--	--	6
Supplements (+ additives)*	4.5	4.5	--	--	2.5	2.5	2.5	2.5	2.5	2.5
Crude protein	11	13	11	13	11.6	13.2	11.2	8.7	9	11
Gross energy (mcal/g)	NC	NC	NC	NC	NC	NC	NC	NC	4.313	4.313

NC = not calculated

*Supplement (+ additives) consisted of:

Rolled milo -- 69.4%	Limestone --- 12.3%	Trace mineral -- 1.5%
Dicalcium P -- 4.8%	Salt -------- 7.2%	Soybean meal --- 1.0%
Dyna K ------- 2.5%	Aureomycin -- 1.4%	

TABLE 3

RATION SCHEDULE AND RATION SWITCHES

PERIOD FED	DATE OF BLOOD SAMPLING	SAMPLING NUMBER*	RATION FED	ANIMAL GROUPS ON RATION
8/23/73-9/10/73	8/23/73	2	1a	1, 2, and 3
			1b	4, 5, and 6
9/10/73-10/5/73	10/4/73	3	2a	1, 2, and 3
			2b	4, 5, and 6
10/5/73-12/8/73	11/15/73	4	3a	1, 2, and 3
			3b	4, 5, and 6
12/8/73-1/3/74	--	--	4a	1, 2, and 3
			4b	4, 5, and 6
1/3/74-1/24/74	1/24/74	5	5a	1 and 4
			5b	2, 3, 5, and 6
1/24/74-3/7/74	3/7/74	6	5a	1, 2, 4, and 5**
			5b	3 and 6*

*First sampling collected July 23 and 30, 1973.

**Steer no. 235 was added to those on 5b ration.

(ii) Background-age calves entering the Caley Cattle Company feedlot and either showing clinical signs of respiratory disease or elevated body temperatures were isolated and kept indoors and treated daily with antibiotics, sulfonamides and supportive therapy. Rumen stimulants and supporting gruels were administered by rumen infusion to calves not eating or ruminating well. Treatments were continued until the body temperature had stabilized at normal for two days and calves had started to eat properly, at which time they were turned out into the feedlot.

Calves were fed 1.36 kg - 1.81 kg prairie hay per head daily

1.36 kg alfalfa hay per head daily

and 0.45 kg of concentrate initially and gradually increased to 1.36 kg per head daily until the end of the study.

Blood samples were collected daily from animals undergoing treatment between December 20, 1973, and January 6, 1974.

Blood preservation:

Twenty-five ml of blood was collected from each animal by jugular venepuncture, into a 50 ml plastic centrifuge tube containing about 0.04 gm of a mixture of etylene-diamine-tetra-acetic acid (EDTA) and sodium fluoride in a ratio of 9:1 (as anticoagulant and preservative, respectively). Each tube was covered with a piece of Parafilm* and the blood and anticoagulant mixed. Plasma samples were obtained by centrifuging the blood at 4000 x g at 4°C for fifteen minutes in a Sorvall centrifuge.** Centrifugation was completed within one to two hours after collection. Plasma was pipetted off into polyethane tubes and was immediately frozen at -20°C until analyzed.

Analytical Methods:

All determinations were made using plasma. Whole blood glucose levels are subject to many variations. Ruminant erythrocytes do not contain an appreciable amount of glucose and the number in circulation can be rapidly altered by their release from the spleen caused by mild excitement or stress. This causes fluctuations in blood glucose levels. Plasma was used for the determination of glucose levels in this study in order to minimize these fluctuations.

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*Parafilm "M", American Can Company, Marathon Products, Neenah, Wisconsin.

**Ivan Sorvall Inc., Norwalk, Connecticut.

(i) Glucose was determined by the glucose oxidase (Sigma* G-6125 type III), peroxidase (Sigma P-8125 type I), and o-dianisidine method as described by Krebs et al.[19] and modified by Chapman;[6]

(ii) Non-esterified fatty acids (NEFA) were determined by the method described by Falholt et al.[10] and

(iii) L(+)-lactate was measured using lactic dehydrogenase (LDH) (Sigma L-2625 Type III), by the method of Hohorst[16] as modified by Chapman.[6]

RESULTS

(i) Feedlot Cattle:

The means for plasma glucose, L(+)-lactate and NEFA levels for animal ration groups on ration changes are presented in tables 4 to 6.

Statistical comparisons of these means indicated no significant difference in plasma glucose levels for all animal groups due to the effect of different ration compositions and changes throughout the feeding trials. A switch to ration 5 (see tables 2 and 3), however, caused a significant difference ($P < 0.05$) in the means of plasma L(+)-lactate concentrations within animal groups on (a) and (b) of ration 5. The fifth sampling (see table 3) was taken 3 weeks after the ration switch. It is interesting to note that plasma L(+)-lactate levels were high from the beginning of the feeding of concentrate diet up until a switch to ration 5, when they decreased and remained low until the end of the study. There was little variation in the mean levels within groups on the 5th sampling.

Plasma NEFA levels were generally high for all arrivals on the first sampling, possibly due to the effect of stress of transportation. There were

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*Sigma Chemical Company, P.O. Box 14508, St. Louis, Missouri 63178.

TABLE 4

MEAN (±1 SD) FOR PLASMA GLUCOSE (MG%) FOR ANIMALS IN RATION GROUPS

RATION GROUP	SAMPLE SIZE	SAMPLING NUMBER					
		1	2	3	4	5	6
1	4	77.4±8.5	48.7±6.3	64.3±8.6	73.1±6.3	92.8±6.2	85.4±6.0
2	2	66.3±14.8	48.5±10.9	67.4±14.8	78.2±11.0	92.3±10.7	84.7±10.5
3	6	58.6±6.6	44.3±4.9	47.7±6.6	70.7±4.9	88.1±4.8	78.9±4.7
4	2	63.5±14.8	41.0±10.9	52.9±14.8	68.5±11.0	97.2±10.7	82.3±10.5
5	5	61.3±7.4	42.4±5.4	54.6±7.4	73.3±5.5	83.3±5.3	75.9±5.2
6	5	70.3±7.4	41.0±5.4	53.8±7.4	78.8±5.5	90.4±5.3	91.4±5.2

TABLE 5

MEAN (±1 SD) FOR PLASMA L(+)-LACTATE (MG%) FOR ANIMALS IN RATION GROUPS

RATION GROUP	SAMPLE SIZE	SAMPLING NUMBER					
		1	2	3	4	5*	6
1	4	24.5±6.0	44.1±11.5	61.7±11.0	34.5±10.3	21.9±3.5	25.9±10.2
2	2	23.4±10.5	66.2±20.0	56.6±19.2	45.5±17.9	26.9±6.0	39.3±17.7
3	6	26.5±4.6	45.0±8.9	48.6±6.6	43.0±8.0	21.0±2.7	18.3±7.3
4	2	25.3±10.5	63.8±20.0	39.1±19.2	31.9±17.9	19.4±6.0	25.2±17.7
5	5	22.3±5.2	54.0±10.0	38.4±9.6	38.0±9.0	21.5±3.0	18.6±8.9
6	5	27.6±5.2	62.9±10.0	50.6±9.6	44.0±9.0	32.9±3.0	35.6±8.9

*Difference in means significant at $P < 0.05$ level.

TABLE 6

MEAN (±1 SD) FOR NEFA (μEq/L) FOR ANIMALS IN RATION GROUPS

RATION GROUP	SAMPLE SIZE	SAMPLING NUMBER					
		1*	2	3	4*	5	6
1	4	309.9±120.6	155.4±57.0	188.2±43.5	76.8±31.3	51.7±26.5	33.3±18.2
2	2	398.2±208.9	120.7±98.7	255.7±75.5	107.9±54.3	88.2±45.9	57.4±31.5
3	6	483.7±93.4	277.9±44.1	156.1±33.7	101.2±24.3	95.7±20.5	65.9±14.1
4	2	964.0±208.9	274.6±98.7	234.7±75.5	235.9±54.3	115.1±45.9	46.8±31.5
5	5	278.7±104.4	166.9±49.4	156.5±27.1	158.9±27.1	125.8±22.9	61.4±15.7
6	5	484.7±104.4	165.4±49.4	169.3±37.7	183.4±27.1	98.6±22.9	67.7±15.7

*Difference in means significant at $P < 0.05$ level.

TABLE 7

MEAN FOR PLASMA GLUCOSE, L(+)-LACTATE, AND NEFA FOR ANIMAL GROUPS WITHIN GROUP

ANIMAL RATION GROUP	SAMPLE SIZE	SAMPLING NUMBER					
		1	2	3	4	5	6
		(a). GLUCOSE (MG%)					
I	12	66.17	44.85	52.54	74.71	88.71	83.12
II	12	65.41	43.19	57.96	72.80	90.32	82.67
		(b). L(+)-LACTATE (MG%)					
I	12	25.29	49.70	45.32	38.79	22.51	27.05
II	12	25.01	57.91	52.60	41.30	25.69	24.08
		(c). NEFA (μEq/L)					
I	12	411.49	153.79	166.55	122.69	113.12	57.80
II	12	466.33	241.30*	199.68	153.46	79.39	57.42

*Difference in means significant at $P < 0.05$ level.

significant differences ($P < 0.05$) in the NEFA levels on the first and fourth samplings. The significant difference on the first sampling might be due to a much higher level for the two steers in group 4 that happened by chance to be grouped together. It is also difficult to interpret whether the difference in sampling 4 was due to the effect of the ration or a carry-over of the initially high NEFA levels for group 4 animals.

Animal-ration groups 1, 2, and 3 (I) fed on (a) and groups 4, 5, and 6 (II) fed on (b) of rations 1 to 4 were compared as groups I and II (see table 7). There was no significant difference in plasma glucose and L(+)-lactate levels for the two groups due to the effect of ration composition and changes.

The means for plasma NEFA levels for the two groups were significantly different ($P < 0.05$) during the second sampling, as the two groups went on rations 2a and 2b, respectively.

The changes in the mean levels for all measured plasma constituents for the 24 steers were examined with time. The results are presented in table 8. A characteristic pattern can be observed from table 8. There were much higher plasma glucose and NEFA levels and a lower plasma L(+)-lactate level in the first compared to the second sampling. This corresponded to commencement of the ration trials. The higher glucose and NEFA levels were probably due to hormonal effects associated with stress.

From the second through the fifth sampling there was a gradual increase in plasma glucose with a corresponding decrease in L(+)-lactate levels. NEFA level decreased throughout the study period. The changes in levels of plasma glucose, L(+)-lactate, NEFA and weight gains with time were highly significant ($P < 0.001$).

TABLE 8

CHANGES IN MEAN VALUES FOR PLASMA GLUCOSE, L(+)-LACTATE AND NEFA LEVELS AND WEIGHT GAINS FOR ALL ANIMALS WITH TIME

SAMPLING NUMBER	SAMPLE SIZE	PLASMA CONSTITUENTS			WEIGHT KG
		GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)	
1	24	65.79	25.15	438.91	--
2	24	44.02	53.80	197.53	227.13
3	24	55.25	48.96	179.12	272.03
4	24	73.76	40.05	138.07	324.04
5	24	89.47	24.10	96.24	376.99
6	24	82.90	25.56	57.62	413.98

Considering the means for glucose, L(+)-lactate, and NEFA levels for all animals, there were inverse and linear relationships between glucose and NEFA ($r = 0.973$) and glucose and L(+)-lactate ($r = 0.915$). These are shown in figure 1. There were diverse variations, however, from animal to animal for these plasma constituents (see table 1, appendix II).

(ii) Background-age Calves:

Treated calves were classified into two groups according to their response to treatment:

(a) The fast-response group were those treated and discharged within four days.

(b) The slow-response group were those calves which received treatment for five days or longer.

Table 9 shows the means for plasma glucose, L(+)-lactate and NEFA levels for the two groups.

THIS BOOK CONTAINS NUMEROUS PAGES WITH DIAGRAMS THAT ARE CROOKED COMPARED TO THE REST OF THE INFORMATION ON THE PAGE.

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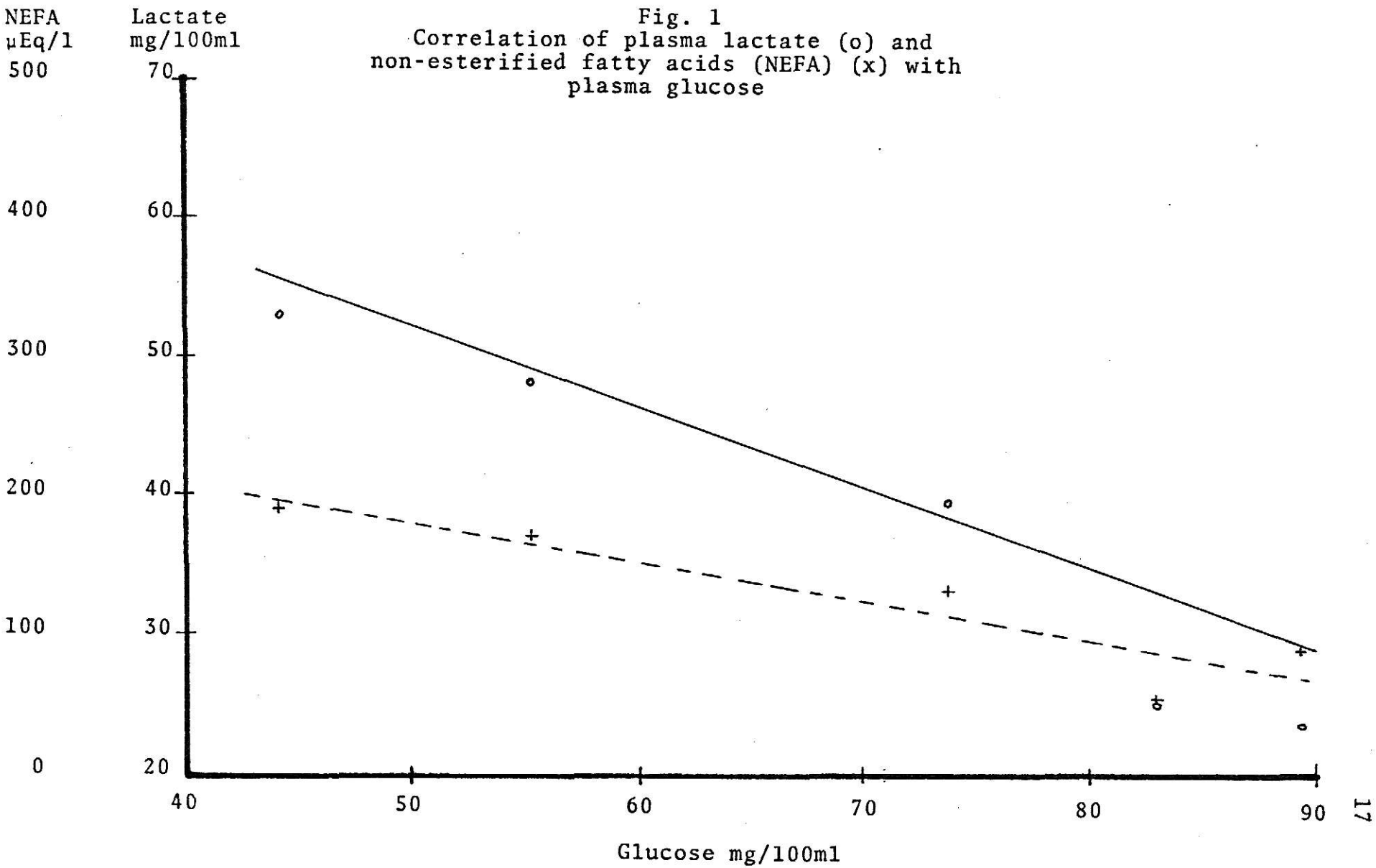

Fig. 1
Correlation of plasma lactate (o) and non-esterified fatty acids (NEFA) (x) with plasma glucose

TABLE 9

MEANS FOR PLASMA GLUCOSE, L(+)-LACTATE, NEFA LEVELS AND RECTAL TEMPERATURE

TREATMENT DAY	NUMBER OF CALVES	TEMP. (°F)	GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
		FAST-RESPONSE GROUP			
1	7	104.9	69.83	21.34	178.77
2	7	101.7	74.99	20.98	217.63
3	10	102.0	76.06	20.86	237.03
4	4	101.6	79.10	13.53	173.77
		SLOW-RESPONSE GROUP			
1	3	105.4	69.25	11.16	488.86
2	5	103.2	65.55	14.98	200.97
3	6	102.6	61.84	9.54	387.45
4	7	102.5	63.63	10.50	327.61
5	7	102.98	63.58	13.26	374.99
6	6	102.8	65.36	11.28	339.87
7	6	102.6	61.16	10.97	332.23
8	6	101.9	57.82	17.10	323.20
9	4	102.5	66.18	12.63	206.06
10	4	101.8	62.19	12.35	204.99
11	4	102.6	61.07	8.81	147.86
12	4	103.1	63.30	11.32	219.48
13	4	101.3	62.99	8.79	62.43
14	3	101.4	64.20	5.95	111.62
15	2	100.4	58.20	12.33	42.06
16	2*	101.9	61.07	9.08	42.21

*One calf was commatose and later died.

Comparison of the first four days of treatment for both fast- and slow-response groups indicated that the fast-response group had a significantly higher plasma glucose level ($P = 0.05$). Although statistical analysis revealed no significant difference in the levels of L(+)-lactate and NEFA between the two groups, plasma lactate levels appeared lower and plasma NEFA levels were higher in the slow- than fast-response groups. Plasma NEFA levels were persistently higher than 300 μEq/L for a week before declining (except day 2).

Individual animal data (see table 2, Appendix II) varied. Calf no. 15 was a good example of slow-response animal. The plasma NEFA level on day 1 was very high (1027.43 μEq/L) with daily fluctuations but rarely fell below 350 μEq/L for the 16 treatment days. The last day the NEFA level suddenly fell to 47.50 μEq/L, and the calf was comatose and died later that day. Calf no. 10 was another example of a slow-response animal which later recovered. NEFA levels fluctuated between 500-800 μEq/L for several days during the period it was anorectic and exhibited weak ruminal motility. Ruminal infusion resulted in stimulation of appetite of the calf after which the NEFA level declined until discharged.

Fast-response calves, e.g. numbers 3, 7, 9, and 28 maintained plasma NEFA levels below 250 μEq/L during the four days of treatment. Changes in the plasma glucose and L(+)-lactate in these calves were not consistent.

DISCUSSION

(i) Feedlot Cattle:

Analysis of the plasma from first sampling for the 24 steers, taken within 24 hours after arrival to the feedlot, revealed higher plasma NEFA and glucose levels than found in the second sampling, collected about a month later (see table 8). These steers were hauled from a collecting point to the

feedlot and were en route for a period of about ten hours. It is quite possible that the initial elevation of plasma NEFA and glucose levels was due to excitement and stress of transportation.

Mobilization of the depot fat is increased by epinephrine, growth hormone, adrenocortical hormones, and sympathetic nervous system.[13,36] Excitement,[23] exercise[7] and other forms of stress[30] also increase plasma NEFA levels. All these factors were undoubtedly involved during handling and transportation and probably were responsible for the elevated plasma NEFA levels. Furthermore, the long-term effect of growth hormone (i.e., 3 hours after injection) has been described as anti-insulin-like, with a reduction in the peripheral utilization of glucose, an increase in gluconeogenesis with an induced hyperglycemia.[11,34] Synthesis of long-chain fatty acids is depressed and adipose tissue lipolysis is increased with a resulting increase in plasma NEFA.[11,34] Head et al.,[14] however, found in dairy calves, an increased plasma NEFA level but no significant change in glucose utilization after treatment with growth hormone. Further work on the effect of growth hormone and insulin on glucose utilization in feedlot cattle could help to explain the interaction of growth hormone and insulin.

As these steers were put on an increasingly higher percent grain ration, there was a gradual increase in plasma glucose and a corresponding decline in plasma NEFA levels. This was in agreement with Holmes and Lambourne[17] who showed that Hereford cattle on a 75% grain ration had lower plasma NEFA levels than those with less than 75% grain ration. These authors also found that a considerable quantity of grain escaped ruminal fermentation and therefore starch was available for hydrolysis and absorption from the intestine. Wright et al.[37] showed that a large amount of starch passed through the abomasum and underwent rapid intestinal digestion, resulting in a significant increase in

blood glucose in sheep fed 64% cracked corn. Corn starch escaping fermentation may contribute up to 8.6 gm glucose/$kg^{3/4}$ to the ruminant per day.[33] In sheep, increasing the level of flaked corn in the ration increased the concentration of propionic acid.[22] An increase in blood glucose could thus be the consequence of an increase in production of this glucogenic volatile fatty acid.[24]

The plasma L(+)-lactate levels also seemed to show a pattern in these animals. It is interesting to note the generally higher lactate levels in feedlot cattle than in dairy cattle (see Paper II). The L(+)-lactate levels on first sampling were much lower than on the second sampling. Comparatively low lactate levels at the first sampling were probably due to the endogenous production of L(+)-lactate as food intake was limited during transportation to the feedlot.

In ruminants fed a normal diet, lactic acid has been found in the rumen liquor in small quantities shortly after feeding.[5,31] Dunlop and Hammond[9] and Ryan[28] reported that diets high in grain increased rumen lactic acid concentration. Furthermore, Whanger and Matrone[35] reported that rumen microorganisms were capable of forming both the D(-) and L(+) isomers of lactic acid, the latter being commonly found in biological systems. Ryan[29] found that initially, L(+) lactic acid was predominantly formed in the rumen, but as grain feeding increased the D(-) form became more evident until it predominated. Decreased rumen pH values increased rumen and blood lactate concentrations.[28] Huster et al.[18] showed that closing the rumen with soluble lactates led to the appearance of high lactate concentrations in the peripheral blood, suggesting its rapid absorption from the rumen.

It would seem logical that introduction of the feedlot steers in this study to a relatively higher grain diet about the time of the second sampling

caused an increased production and absorption of lactate from the rumen and this was reflected by the high levels of plasma L(+)-lactate. There was a gradual decrease in L(+)-lactate levels until they approximated pre-ration trial levels. Since the readily fermentable carbohydrate content of the diet was gradually increased, steers tolerated the increase in dietary carbohydrate without further lactate accumulation in the rumen as measured by plasma levels. This tolerance was probably due to the low rumen pH which discouraged growth of organisms that usually metabolize glucose to lactate,[29] and encouraged proliferation of organisms which convert lactate into propionate.[3]

It may be concluded then, that the increasing plasma glucose levels with decreasing plasma NEFA levels during these feeding trials favored lipogenesis. Hinkson et al.[15] showed that fasted sheep liver slices readily utilized L(+)-lactate, and it is a favored substrate for gluconeogenesis.[2,6] It follows then that the high plasma L(+)-lactate levels in these animals contributed to the available substrates for lipogenesis. This was manifested in the deposition of fat in the tissues that produced the desired carcass marbling and by a rapid weight gain (table 8).

(ii) Background-age calves:

The calves in the fast-response group had significantly higher plasma glucose level than those in the slow-response group as they continued to eat and therefore had a constant supply of precursors for gluconeogenesis. It would be anticipated that plasma NEFA levels would be lower in the fast-response group. This was true (table 9), although statistically there was no significant difference in levels in the two groups. Similarly plasma L(+)-lactate was higher in the fast-response group, as the animals were fed a diet containing some grain.

Many workers[1,17,23,26] have noted an increase in plasma NEFA and a decrease in blood glucose levels in fasting ruminants, a trend that was reversed when the animals were fed. Therefore, it was not surprising, that plasma NEFA levels in anorectic calves were persistently high, and that these levels declined soon after the calves started eating. Annison[1] and Patterson[23] suggested that plasma NEFA levels might be an indicator of the nutritional status of ruminants. Reid and Hinks[26] concluded that plasma NEFA levels were more sensitive indicators of undernourishment in pregnant ewes than blood glucose and ketones. Since this information was obtained from experimentally fasted healthy animals, it may be informative to look more closely into changes in plasma NEFA levels of sick ruminants. Especially fruitful for investigation would be those that cause anorectic conditions, to see whether plasma NEFA levels could give an indication of the progress of the disease.

THIS BOOK CONTAINS NUMEROUS PAGES WITH MULTIPLE PENCIL AND/OR PEN MARKS THROUGHOUT THE TEXT.

THIS IS THE BEST IMAGE AVAILABLE.

REFERENCES

1. ANNISON, E.F. 1960. Plasma non-esterified fatty acids in sheep. Aust. J. Agri. 11: 58-64.

2. ANNISON, E.F.; D.B. LINDSAY and R.R. WHITE. 1963. Metabolic relationships of glucose and lactate in sheep. Biochem. J. 88: 243-248.

3. ANNISON, E.F. 1965. Absorption from the ruminant stomach. In: Physiology of Digestion in Ruminant. Second International Symposium. Edited by R.W. DOUGHERTY, Ames, Iowa, 1964. Butterworth Inc., Washington.

4. BAILE, C.A. and H.F. MARTIN. 1971. Hormones and amino acids as possible factors in the control of hunger and satiety in sheep. J. Dairy Sci. 54: 897-905.

5. BRUNO, C.F. and W.E. MOORE. 1962. Fate of lactic acid in rumen ingesta. J. Dairy Sci. 45: 109.

6. CHAPMAN, T.E. 1969. The metabolic role of L(+)-lactic acid at resting and elevated concentrations of plasma in lactating goats. Ph.D. Dissertation in Physiol. Grad. Sch. Univ. of Cal. Davis.

7. COSTILL, D.L. 1970. Metabolic response during distant running. J. Appl. Physiol. 28: 251-255.

8. DOLE, V.P. 1956. A relation between non-esterified fatty acids in plasma and the metabolism of glucose. J. Clin. Invest. 35: 150.

9. DUNLOP, R.H. and P.B. HAMMOND. 1965. D-lactic acidosis of ruminants. Ann. N.Y. Acad. Sci. 119: 1109-1132.

10. FALHOLT, K.; B. LUND and W. FALHOLT. 1973. An easy Calorimetric Micromethod for routine determination of free fatty acids in plasma. Clin. Chim. Acta. 46: 105-111.

11. GOODMAN, H.M. 1968. Growth hormone and the metabolism of carbohydrate and lipid in adipose tissue. Ann. N.Y. Acad. Sci. 148: 419.

12. GORDON, R.S. and A. CHERKES. 1956. Unesterified fatty acid in human blood plasma. J. Clin. Invest. 35: 206.

13. HAVEL, R.J. 1967. Autonomic nervous system and adipose tissues; In: Handbook of Physiology. Adipose tissue. Washington, D.C. Am. Physiol. Soc. Sect. 5, p. 575-582.

14. HEAD, H.H.; M. VENTURA; D.W. WEBB and C.J. WILCOX. 1970. Effect of growth hormone on glucose, non-esterified fatty acids and insulin levels and on glucose utilization in dairy calves. J. Dairy Sci. 53: 1496-1501.

15. HINKSON, R.S., JR.; W.H. HOOVER and B.R. POULTON. 1967. Metabolism of lactate isomers by rat and sheep liver and rumen epithelial tissue. J. Anim. Sci. 26: 799-803.

16. HOHORST, H.J. 1963. Determination of L(+)-lactate with lactic dehydrogenase and DPN. In: Method of Enzymatic Analysis by H.V. BERGMEYER Ed. Acad. Press Inc., N.Y., p. 266-270.

17. HOLMES, J.H.G. and L.J. LAMBOURNE. 1970. The relationship between plasma free fatty acid concentrations and the digestible energy intake of cattle. Res. Vet. Sci. 11: 27-36.

18. HUNTER, F.G.; J.C. SHAW and R.N. DOETSCH. 1956. Absorption and dissimilation of lactates added to bovine rumen and resulting effect on blood glucose. J. Dairy Sci. 39: 1430.

19. KREBS, H.A.; C. DIERKS and T. GASCOYNE. 1964. Carbohydrate synthesis from lactate in pigeon-liver homogenate. Biochem. J. 93: 112.

20. KRONFELD, D.S. 1965. Plasma non-esterified fatty acid concentration in the dairy cow: responses to nutritional and hormonal stimuli, and significance in ketosis. Vet. Rec. 77: 30-35.

21. MIKULEC, J.C. and A. TAYLOR. 1965. Serum albumin-bound fatty acid values in wethers. N.Z.J. Agr. RES. 8: 889-892.

22. NICHOLSON, J.W.G. and J.D. SUTTON. 1969. The effect of diet composition and level of feeding on digestion in the stomach and intestine of sheep. Brit. J. Nutri. 35: 585.

23. PATTERSON, D.S.P. 1963. Some observations on the estimation of non-esterified fatty acid concentration in cow and sheep plasma. Res. Vet. Sci. 4: 230-237.

24. PATTERSON, D.S.P. 1964. The effect of intravenous glucose on depot fat mobilization in the sheep. Res. Vet. Sci. 5: 286-293.

25. PHILLIPSON, A.T. and R.A. McANALLY. 1942. Studies on the fate of carbohydrates in the rumen of sheep. J. Exptl. Biol. 19: 199-214.

26. REID, R.L. and N.T. HINKS. 1962. Studies on the carbohydrate metabolism in sheep. XVII. Feed requirements and voluntary feed intakes in late pregnancy, with particular reference to prevention of hypoglycemia and hyperketonemia. Aust. J. Agr. Res. 13: 1092-1111. XVIII. The metabolism of glucose, free fatty acids, ketone bodies and amino acids in late pregnancy and lactation, i.b.d. 1112-1123. XIX. The metabolism of glucose, free fatty acids and ketones after feeding and fasting or undernourishment of non-pregnant, pregnant and lactating ewes. i.b.d. 1124-1136.

27. RUSSEL, A.L.F.; J.M. DONEY and R.L. REID. 1967. The use of biochemical parameters in controlling nutritional state in pregnant ewes, and the effect of undernourishment during pregnancy on lamb birth-weight. J. Agri. Sci. Camb. 68: 351-358.

28. RYAN, R.K. 1964. Concentrations of glucose and low-molecular-weight acids in the rumen of sheep following the addition of large amounts of wheat to the rumen. Am. J. Vet. Res. 25: 646-652.

29. RYAN, R.K. 1964. Concentrations of glucose and low-molecular-weight acids in the rumen of sheep changed gradually from a hay to a hay-grain diet. Am. J. Vet. Res. 25: 653-658.

30. SLEE, J. and R. HALLIDAY. 1968. Some effect of cold exposure, nutrition and experimental handling on serum free fatty acid levels in sheep. Ani. Pro. 10: 67-76.

31. WALDO, D.R. and L.H. SCHULTZ. 1955. Lactic acid production in the rumen. J. Dairy Sci. 38: 605.

32. WALDO, D.R. and L.H. SCHULTZ. 1956. Lactic acid production in the rumen. J. Dairy Sci. 39: 1453-1460.

33. WALDO, D.R. 1973. Extent and partition of cereal grain starch digestion in ruminants. J. Ani. Sci. 37: 1062-1074.

34. WEIL, R. 1965. Pituitary growth hormone and intermediary metabolism. I. The hormonal effect on the metabolism of fat and carbohydrate. Acta. Endocrinol. Suppl. 98.

35. WHANGER, P.D. and G. MATRONE. 1965. Production and absorption of lactate. Fed. Proc. 24: 244.

36. WINEGRAD, A.L. 1962. Endocrine effects on adipose tissue metabolism. Vitamins and Hormones 20: 141-197.

37. WRIGHT, P.L.; R.B. GRAINGER and G.J. MARCO. 1966. Post-ruminal degradation and absorption of carbohydrate by the mature ruminant. J. Nutri. 89: 241-246.

CHANGES IN PLASMA LEVELS OF NON-ESTERIFIED FATTY ACIDS (NEFA), GLUCOSE AND L(+)-LACTATE:

PAPER II. IN DAIRY COWS FROM LATE PREGNANCY INTO EARLY LACTATION

SUMMARY

Plasma non-esterified fatty acids (NEFA), glucose and L(+)-lactate were measured in 10 Holstein cows from late pregnancy into early lactation. Glucose levels were highest and NEFA levels were significantly elevated a day before parturition. L(+)-lactate levels were elevated around parturition time. Plasma glucose concentrations were lowest by the fourth day, but started rising gradually to the pre-partum levels by the eleventh day following calving. There was no consistent increase in plasma NEFA levels after parturition and by the 39th day had returned to the range of pre-partum levels.

Two cows showed elevated post-partum plasma NEFA levels in the range reported for clinical ketosis without showing any clinical signs. A cow with displaced abomasum had the highest NEFA and beta-hydroxybutyric acid levels comparable to the ranges reported in literature. A treated case of metritis showed a very high and very low plasma NEFA and glucose levels respectively.

INTRODUCTION

Since Annison[2] observed that levels of plasma non-esterified fatty acids (NEFA), blood glucose and ketone bodies are usually closely related in ruminants, many studies have been carried out to determine the relationships between these blood metabolites under various physiological and clinical conditions.

Increased energy requirements of pregnancy and lactation usually produce an increase in plasma NEFA levels. In ewes on equal feed intake, Reid and Hinks[11] found that levels of plasma NEFA in late pregnancy were highly correlated with the total fetal weight per unit of maternal weight. They

also found that NEFA levels of ewes on a constant feed intake increased more than those receiving increased feed intake two weeks before parturition. The relationship between plasma NEFA, blood glucose and ketone bodies were similar in pregnant and lactating ewes, although plasma NEFA levels were consistently higher during lactation at all blood glucose levels.[11] Karihaloo et al.[8] observed that in the last six weeks of pregnancy NEFA and ketone body levels increased but glucose levels did not change with ewes fed ad libitum.

Radloff et al.[10] found in cows that levels of plasma NEFA and blood glucose was highest at calving time. They also noted that NEFA levels varied during lactation with the highest values at the peak of milk production. These authors also found that blood glucose was lowest and ketone bodies highest two weeks after parturition before returning to normal levels. Vermann and Schultz[12] found the highest levels of plasma NEFA, blood glucose and ketone bodies in cows during early lactation, with a highly positive arteriovenous differences in NEFA levels.

Adler et al.[1] and Yamdagni[15] observed an increase in plasma NEFA levels in cows with clinical ketosis. Kronfeld[7] also observed an increase in plasma NEFA levels but found that the elevation in spontaneous ketosis was less consistent than in cows with ketosis associated with abomasal displacement or fasting.

The present study was conducted to examine changes in plasma components of glucose, NEFA and L(+)-lactate in dairy cows under common feeding and management practices from late pregnancy into early lactation.

MATERIALS AND METHODS

Animals:

Ten Holstein cows between the ages of three and four years and approaching their second and third lactations were selected from a large herd of high-producing animals belonging to the Dairy Science Department, Kansas State University. All cows were 6 to 8 months pregnant at the beginning of the experiment.

Blood samples were collected from all animals once a week until a few days pre- and post-partum, during which time they were placed in the maternity barn. Daily blood samples were collected as long as the cows remained in the barn. This procedure was followed until all the cows calved, then weekly sampling continued for three weeks after the last calving. All samples were collected between December, 1973 and March, 1974.

Feeding:

The cows were fed on grass hay until two weeks before parturition, when they were placed on alfalfa hay (9.07 kg per head daily) and concentrate (gradually increasing amounts until receiving 9.07 kg per head daily at the time of parturition). After parturition, the cows were adjusted to 9.98 kg alfalfa hay and 18.14 kg of the concentrate diet per head daily. The cows were fed twice daily. Free choice (loose mix) trace mineral supplements containing 24% calcium and 21% phosphorous were allowed at all times. Analysis of the alfalfa hay gave 17% crude protein, 1.4% calcium, 0.28% phosphorous and a net energy content of 40 Mcal/Cwt. The concentrate ration contained 96.82% partially ground sorghum grains, 1.4% urea supplement, 0.5% trace minerals, 0.52% calcium, 0.76% phosphorous and 45,000 I.U. vitamins A

and D/kg of concentrate. Crude protein was 14% and the net energy content was 76 Mcal/Cwt.

Preservation of Blood:

The method of obtaining blood and its preservation were the same as described in Paper I.

Analytical Methods:

Analyses of plasma glucose, NEFA and L(+)-lactate were performed as described in Paper I.

Beta-hydroxybutyric acid was determined by the method described by Williamson et al.[13] using beta-hydroxybutyrate dehydrogenase (Sigma H-6126, Type II), except that 8% perchloric acid was used for deproteinization of plasma instead of 30% (w/v) used for whole blood, and acid neutralization was found unnecessary.

RESULTS AND DISCUSSION

Mean values of the changes in plasma levels of the various components examined, along with the weekly body weights and average daily milk production are presented in table 1.

Plasma glucose reached the highest level and there was a sharp rise in plasma NEFA level a day prior to calving. These findings are in general agreement with Radloff et al.[10] who examined a weekly blood sampling, two weeks before and up to ten weeks after calving and indicated that blood sugars and plasma free fatty acid concentrations were highest around calving time. They thought this was probably due to the hormonal changes and stress of parturition.

TABLE 1

MEANS OF CHANGES IN PLASMA COMPONENTS, WEEKLY WEIGHT AND DAILY MILK PRODUCTION IN DAIRY CATTLE

DAYS AFTER CALVING	SAMPLE SIZE	GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)	BODY WEIGHT (KG)	MILK PRODUCTION (KG)
- 38	5	59.8±6.0	3.9±1.2	142.5±121.6	683.4±43.9	-
- 31	7	58.9±5.4	6.0±3.9	73.7±42.0	687.6±43.5	-
- 24	7	61.0±5.0	4.8±1.0	159.3±109.8	690.6±41.3	-
- 17	10	60.0±4.0	4.1±1.6	147.3±123.9	690.0±47.5	-
- 10	10	63.4±10	7.4±4.6	193.7±131.0	686.7±50.5	-
- 3	4	59.8±4.9	4.0±1.7	110.4±35.4	-	-
- 2	5	63.0±7.8	5.0±2.1	163.4±37.6	-	-
- 1	7	69.3±12.9	7.6±3.0	387.9±339.4	-	-
0	9	68.6±12.2	8.0±2.6	242.5±144.4	-	-
1	10	55.7±4.5	5.7±1.9	360.1±250.6	-	15.9±6.9
2	10	50.6±6.1	4.6±1.7	317.7±255.5	-	21.4±5.1
3	9	51.3±10	4.6±1.7	432.0±493.4	-	23.1±8.8
4	9	44.5±5.0	4.0±1.2	362.2±335.9	-	25.2±8.9
11	10	57.8±5.8	3.3±1.3	404.9±209.7	599.0±52.8	27.3±4.8
18	10	56.6±6.9	4.5±3.4	241.3±104.5	586.1±47.5	31.2±4.6
25	10	58.4±7.4	3.8±2.1	248.6±201.3	575.2±43.1	31.7±4.1
32	9	51.8±10	5.1±3.1	439.1±411.9	560.2±19.0	30.9±3.1
39	9	59.5±11	7.9±7.1	268.3±372.3	555.9±17.9	31.1±4.7
46	8	56.5±5.3	4.9±2.2	148.8±130.7	554.9±26.9	29.9±5.6
53	8	55.4±5.9	4.6±1.7	181.1±101.9	551.6±25.8	29.3±4.5
60	5	54.2±12.7	5.2±2.1	159.0±188.8	545.0±17.6	29.4±2.2

There was an increase in plasma L(+)-lactate level around calving time. There were no significant changes in L(+)-lactate levels throughout the study period.

The plasma glucose fell sharply, reaching the lowest level four days after calving. The level on the fourth day was significantly lower than the levels on the day before and the day of calving, and 1st, 2nd, 3rd and 11th day after calving ($P < 0.001$). By the eleventh day plasma glucose level had risen approaching the pre-partum level. Lactation poses a great demand for glucose upon the animal since milk alone contains about 90 times as much total sugars (lactose) as does blood.[4] In terms of daily lactose production alone this could easily amount to 1000 g of glucose per day for a high-producing cow.[5] This would therefore explain the sharp drop in plasma glucose within the first four days of calving and by the eleventh day the cows had adjusted to the glucose demand for milk production.

There was no consistent increase in plasma NEFA levels after parturition and by the 39th day the levels had approached the pre-partum levels. There was a significant drop in plasma NEFA levels by the 18th to 25th day post-partum, during which period the plasma glucose level was still gradually rising. This was probably due to the increase in grain consumption following parturition, so that the cows were adjusting their intake to the energy demand of milk production. Furthermore, it has been shown in sheep[14] and cattle[6] that with increase in grain concentrate ration, a considerable amount of grain escapes fermentation in the rumen and undergoes rapid enzymatic hydrolysis in the intestine. This results in a significant increase in blood sugar concentrations. Increase in grain ration will also increase the production of propionic acid,[9] which is highly glucogenic. Holmes and Lambourne[6] have also noted that there was a significant drop in plasma NEFA levels in

cattle fed high grain ration. This was to be expected since Patterson[9] has shown that there was a depression of NEFA level in sheep following intravenous injection of glucose.

It would appear then that the gradual increase in plasma glucose, and the decrease in plasma NEFA levels after the increase in concentrate ration in these dairy cows favored an increased utilization of glucose by the adipose tissues. This caused a depression of plasma NEFA levels and promoted the rate of fatty acid and neutral fat synthesis,[3] which helped reduce rapid weight loss in these cows.

Clinical Cases:

Four out of ten cows studied exhibited elevated plasma NEFA concentrations far above the mean values at one time or another after parturition. Two of these cows (036 and 095) had elevated plasma NEFA levels without showing any clinical manifestation of disease. The other cows were treated for abomasal displacement (086) and metritis (C133).

Tables 2 and 3 show the post-partum plasma glucose and NEFA concentrations for cows 036 and 095 up to 32 days following calving, respectively.

Although cows 036 and 095 manifested no clinical signs after parturition, they both had elevated plasma NEFA levels on the second or third day, with decreasing plasma glucose levels after calving (tables 2 and 3). Adler et al.[1] gave a range of 266 to 1,310 μEq/1 as plasma NEFA concentrations of 9 clinical ketotic cows, and 6 of them with levels over or near 1000 μEq/1. Kronfeld[7] gave a range of 356 to 1,805 μEq/1 for 18 cows with spontaneous ketosis. Adler's ketotic cows also had very low blood glucose concentrations. It was quite possible that cows 036 and 095 developed or were on the verge of developing clinical ketosis and probably spontaneously recovered. This would not

TABLE 2

POST-PARTUM PLASMA GLUCOSE AND NEFA CONCENTRATIONS FOR COW 036

DAYS AFTER CALVING	GLUCOSE (MG%)	NEFA (μEq/L)
1	55.97	240.09
2	50.05	375.33
3	48.44	1481.84
4	37.10	301.64
5	46.84	547.47
11	49.66	700.15
18	40.20	264.46
25	44.53	685.23
32	53.58	570.55

TABLE 3

POST-PARTUM PLASMA GLUCOSE AND NEFA CONCENTRATIONS FOR COW 095

DAYS AFTER CALVING	GLUCOSE (MG%)	NEFA (μEq/L)
1	55.59	372.79
2	40.49	926.02
3	43.99	922.59
4	46.44	672.22
5	34.54	215.94
11	47.62	425.83
18	53.62	240.84
25	51.61	282.02
32	42.32	250.28

be surprising, as these cows were producing over 30 kg/day of milk within a week post-partum.

Tables 4 and 5 show the post-partum plasma glucose and NEFA concentrations for cows 086 and C133, respectively.

A left displaced abomasum was diagnosed in cow 086 four days after parturition and she was sent to Dykstra veterinary hospital for surgical correction. She was ketotic as evidenced by plasma B-OHB levels and her milk production was very poor at this time. Kronfeld[7] reported a range of 670 to 1,198 μEq/l as plasma concentrations of NEFA for 8 ketotic cows with abomasal displacement. He also stated that only one case had a plasma glucose concentration below 50 mg% and one with ketones less than 10 mg%. It is interesting

TABLE 4

POST-PARTUM PLASMA GLUCOSE, NEFA AND BETA-HYDROXYBUTYRIC ACID CONCENTRATIONS FOR COW 086

DAYS AFTER CALVING	GLUCOSE (MG%)	NEFA (μEq/L)	B-OHB (MG%)
1	55.33	370.95	-
2	54.47	422.73	-
3	72.72	267.55	6.41
4*	41.99	944.54	29.82
5**	46.84	547.47	41.78
6	48.62	349.81	25.35
11	56.20	253.80	-
18	54.54	226.81	-
25	56.14	141.27	-
32	54.15	93.90	-

*Left Abomasal Displacement (LDA) diagnosed.

**Surgical correction of LDA.

TABLE 5

POST-PARTUM PLASMA GLUCOSE AND NEFA CONCENTRATIONS FOR COW C133

DAYS AFTER CALVING	GLUCOSE (MG%)	NEFA (μEq/L)
1	54.62	209.09
2	51.34	314.12
3	50.49	97.22
4	51.22	234.65
5	47.93	352.55
11	64.49	511.91
18	63.52	417.80
25	56.32	500.31
32	34.61	1370.73
39	40.36	1248.17
46	48.67	451.82

to note the high concentration of plasma NEFA for cow 086 on the day LDA was diagnosed. After surgery the level declined. The plasma concentrations of beta-hydroxybutyric acid (B-OHB) was highest on the day of surgery. The cow made a rapid recovery and was producing an average of over 30 kg/day of milk by the fifth week of surgery.

Cow C133 was treated for about two weeks for metritis a month after calving. During this period, she had plasma NEFA concentrations of over 1000 μEq/l (table 5), a very low plasma glucose concentration and her milk

production dropped from an average of over 30 kg/day to about 20 kg/day. By the third week of treatment, her plasma NEFA level dropped and glucose level started rising and her milk production once again rose over 30 kg/day.

REFERENCES

1. ALDER, J.H.; E. WERHEIMER; U. BARTANA and J. FLESH. 1963. Free fatty acids and the origin of ketone bodies in cows. Vet. Rec. 75: 304-307.

2. ANNISON, E.F. 1960. Plasma non-esterified fatty acids in sheep. Aust. J. Agr. 11: 58-64.

3. BALLY, P.R.; G.F. CAHILL; B. LeBOEVE and A.E. RENOLD. 1960. Studies on rat adipose tissue in vitro. V. Effects of glucose and insulin on the metabolism of palmitate-1-C^{14}. J. Biol. Chem. 253: 333.

4. BERGMAN, E.N. 1970. Disorders of carbohydrate and fat metabolism. In: Duke's Physiology of Domestic Animals by M.J. SWENSON. Ithaca, N.Y. Cornell Univ. Press, p. 595-618.

5. BERGMAN, E.N. 1973. Glucose metabolism in ruminants as related to hypoglycemia and ketosis. Cornell Vet. 63: 341-382.

6. HOLMES, J.H.G. and L.J. LAMBOURNE. 1970. The relationship between plasma free fatty acid concentrations and the digestible energy intake of cattle. Res. Vet. Sci. 11: 27-36.

7. KRONFELD, D.S. 1965. Plasma non-esterified fatty acid concentration in the dairy cows: response to nutritional and hormonal stimuli and significance in ketosis. Vet. Rec. 77: 30-35.

8. KARIHALOO, A.K.; A.J.F. WEBSTER and N. COMBS. 1970. Effect of cold, acute starvation and pregnancy on some indices of energy metabolism in Lincoln and Southdown sheep. Can. J. Ani. Sci. 50: 191-198.

9. PATTERSON, D.S.P. 1964. The effect of intravenous glucose on depot fat mobilization in the sheep. Res. Vet. Sci. 5: 286-293.

10. RADLOFF, H.D.; L.H. SCHULTZ and W.G. HOEKSTRA. 1966. Relationship of plasma free fatty acid to other blood components in ruminants under physiological conditions. J. Dairy Sci. 49: 179-182.

11. REID, R.L. and N.T. HINKS. 1962. Studies on the carbohydrate metabolism in sheep. XVII. Feed requirements and voluntary feed intakes in late pregnancy, with particular reference to prevention of hypoglycemia and hyperketonemia. Aust. J. Agr. Res. 13: 1092-1111. XVIII. The metabolism of glucose, free fatty acids, ketone bodies and amino acids in late pregnancy and lactation. i.b.d. 1112-1123. XIX. The metabolism of glucose, free fatty acids and ketones after feeding and fasting or undernourishment of nonpregnant, pregnant and lactating ewes. i.b.d. 1124-1136.

12. VERMANN, P.N. and L.H. SCHULTZ. 1968. Blood lipids of cows at different stages of lactation. J. Dairy Sci. 51: 1971-1974.

13. WILLIAMSON, D.H.; J. MELLAMBY and H.A. KREBS. 1962. Enzymic determination of D(-) beta-hydroxybutyric acid and acetoacetic acid in blood. Biochem. J. 82: 90-96.

14. WRIGHT, P.L.; R.B. GRAINGER and G.J. MARCO. 1966. Post-ruminal degradation and absorption of carbohydrate by the ruminant. J. Nutri. 89: 241-246.

15. YAMDAGNI, S. and L.H. SCHULTZ. 1970. Free fatty acid composition of blood plasma of normal and ketotic cows. J. Dairy Sci. 53: 1046-1050.

APPENDIX I

LITERATURE REVIEW

The metabolic interrelationships of glucose, NEFA, ketone bodies and lactate have been shown to be correlated to their levels in blood. These levels are markedly influenced by the level of energy intake and the metabolism of each constituent. A discussion of the metabolism of each metabolite in the body will, therefore, help to elucidate their interrelationships.

Glucose Metabolism

Most of the carbohydrates in the ruminant's feed are fermented in the rumen to volatile fatty acids (VFA), propionic, acetic and butyric acids, so that only a negligible amount of glucose is absorbed along the alimentary tract.[13,48,84] These VFA furnish approximately 70% of the animal's total caloric requirements.[9,84]

The blood glucose concentration in the adult ruminant is reported to be 40-60 mg/100 ml and 80-100 mg/100 ml in calves up to six weeks of age.[16] The ruminant, therefore, appears to maintain its glucose requirements from gluconeogenesis. Hypoglycemia and ketosis frequently occur in animals, especially in sheep during late pregnancy, if the intake of energy is insufficient[40,67] or in cattle during heavy lactation.[27] A supply of glucose precursors and/or the functional status of the organs that synthesize glucose could, therefore, become limiting factors for the animal's overall productivity and survival.

The glucose turnover rate is reduced to about one-third in ewes when fasted and also during lactation and increases during the last month of pregnancy and is highest at the peak of lactation.[6,11,14] The latter workers found that the glucose turnover time was nearly two hours in nonpregnant

animals, but averaged 78 and 43 minutes during pregnancy and lactation respectively. The actual quantity of glucose would be about 100, 180 and 320 mg per day for normal nonpregnant, pregnant and lactating sheep respectively. Comparative figures for cattle were about 500 mg per day during the dry period and 1700 mg per day during heavy lactation.[11,38,45] It is estimated that lactose production alone uses as much as 60-80% of the glucose turnover in goats,[5] in sheep[11] and in cattle.[45]

In lactating animals glucose utilization is reduced as the blood concentration falls and lactation could cease if the glucose concentration fell significantly.[6,11,14] Glucose requirements of ruminants are, therefore, variable and they are especially high during pregnancy and lactation.

The sites of glucose production in all mammals are considered to be the gut (by absorption), the liver and the kidneys. Little or no glucose is absorbed from the digestive tract of animals fed only hay and roughage diet. In sheep and cattle fed barley and oats, the small amount of starch escaping fermentation indicates that these feeds are readily fermented in the rumen.[56,79] Corn, on the other hand, when fed in larger than maintenance amounts, may have one-third of its starch escaping into the intestinal tract.[34,54,80] Fine grinding of grain may also result in faster rate of passage through the rumen and thus enable more glucose to be absorbed in the gut.[75]

Hepatic production of glucose in sheep under a variety of conditions of feeding, fasting and pregnancy accounts for about 85% of the total glucose produced.[11] In the same study, pregnant sheep had the highest rate of hepatic glucose production and also the highest turnover rate, while nonpregnant animals had the lowest rate, and fasting reduced these values even further.

The kidneys account for about 8-10% of the body's glucose turnover in sheep and for about 15% during fasting.[42]

Substrates for gluconeogenesis are derived from four main metabolites; propionate, glycerol, amino acids and lactate.[11,24,43,88]

About 90% of the propionate absorbed into the portal blood is converted into glucose entirely in the liver, so that very small amounts reach the general circulation,[15,21] but only 25-55% of the propionate produced in the rumen reaches the portal blood.[12,37,49] Much of the rumenally produced propionate is metabolized by the rumen epithelium during absorption.[63]

The contribution of glycerol to gluconeogenesis in the well-fed animal is small, probably less than 5%, but may become an important glucose precursor during periods of undernutrition.[12]

Wolf et al.[91,92] assessed the contribution of amino acids to glucose production in sheep fed alfalfa hay. From their work hepatic uptake showed that 30% of the glucose could have been derived from all the amino acids. Of these, alanine and glutamine were the most glucogenic, contributing at least 40% of the glucose produced from all amino acids.

The contribution of lactate and pyruvate to glucose production in ruminants is difficult to assess because of the variable and undetermined amounts absorbed from the digestive tract. Endogenous glucose production via the Cori cycle, results in no net increase in glucose supply to the body. Unlike that from the Cori cycle, absorbed lactate will contribute towards glucose production in the animal's body.[49] In fed sheep, lactate is thought to contribute about 4-10% of the body's glucose turnover.[4,11]

In ruminants, glucose is released into the blood at all times.[11,41] In addition, gluconeogenesis reaches maximum levels during digestion and becomes reduced during starvation. Propionate and amino acids are the principal

glucose precursors in ruminants and large amounts would be available only in fed animals. It follows that adequately fed ruminants are unlikely to be short of glucose precursors. In an animal not eating, however, or if large amounts of glucose are needed for high productivity, an excessive drain of glucose precursors occur and results in hypoglycemia.[16]

Metabolism of Non-esterified Fatty Acids

The albumin-bound long-chain fatty acids of plasma are usually referred to as unesterified or non-esterified fatty acids (NEFA), to distinguish them from the long-chain fatty acids present in the esterified form in neutral fats and phospholipids. In the case of ruminants' plasma it is also important to distinguish these "free fatty acids" from the small molecular weight, steam volatile fatty acids (VFA) of dietary origin which are found in the free states as the carboxylate ions.

Changes in levels of NEFA in plasma, being the transport form of adipose tissue fat, generally reflect changes in the rate of depot fat mobilization.[25]

Numerous factors, both nutritional and non-nutritional, are known to affect the release of NEFA from adipose tissues. Excitement increases NEFA levels in the blood of sheep[59,61,73] and cattle.[35] Injection of epinephrine increases plasma NEFA levels, its effect being similar to that of excitement in cattle.[44] Patterson[61] considered a difference of 40 μEq/liter of NEFA between pairs of closely spaced analyses from the same animal to be a significant indication of the effect of excitement.

The increased energy requirements of pregnant and lactating animals usually produce an increase in plasma NEFA levels. In ewes on equal feed intake, levels of NEFA in late pregnancy were highly elevated with the total

weight per unit of the maternal weight.[66] Radloff et al.[65] found in cows that levels of blood NEFA increased at parturition. They noted that NEFA levels varied during lactation, with the highest values usually at the peak of milk production. Plasma NEFA levels were higher in lactating Hereford cows than in non-lactating heifers with equivalent digestible energy intake.[35] This difference probably reflects the influence of lactation on NEFA levels.

Age does not appear to have any consistent effect on blood NEFA levels in ruminants. Lambs had a level of 700 μEq/liter at 45 days of age, declining to adult levels at weaning,[47] but no variation was found in the blood of dairy calves up to 100 days of age.[85]

The effect of cold on levels of NEFA varied with the amount and rapidity of temperature change, energy intake and acclimatization of the animal used.[28,39,73]

Breed differences in the levels of NEFA in the blood appear to exist in sheep,[28,70] but no significant differences were found among breeds of cattle.[60,90]

Administration of growth hormone to cattle[31,90] or sheep[22] generally increases blood levels of NEFA, but variations in degree and duration of effect have yet to be elucidated. Injection of insulin and glucose into sheep reduced the levels of NEFA in the blood[3] and in cattle.[44] Intraruminal administration of coffee or caffeine into cows[30] or intravenous infusion of dopamine into sheep[77] and also oral administration of fenfluoramine into Holstein male calves[19] all increased levels of blood NEFA.

A number of nutritional factors have been found to influence the levels of NEFA in the blood of ruminants. In sheep, plasma NEFA levels were lowest between 2-4 hours after feeding[3,66,73] and rose gradually from three to five times, 24 hours after feeding.[69] In cows and goats, NEFA levels in the plasma

decreased 4-6 hours after feeding and these changes were more marked in high producing cows.[65] Plasma NEFA levels vary with feeding time,[29,44] and fall after every meal in Hereford heifers fed six times per day.[35] Reid and Hinks[66] suggested that variations in plasma NEFA and blood ketone levels in relation to feeding time is dependent more on the rate rather than total level of feed consumption.

The type of ration may affect the plasma NEFA levels. Annison[3] and Reid and Hinks[66] found that in sheep, the decrease in plasma NEFA level after feeding was greatest with rations having the highest content of carbohydrate. When the ratio of grain to alfalfa hay was increased sufficiently to depress the fat content of milk from cows, Varmann and Schultz[82] observed that plasma levels were decreased. Holmes and Lambourne[35] found, with a high-grain ration that plasma NEFA levels of Hereford cattle did not increase with reduction in digestible energy intake as they did on a ration with less than 75% grain. Adding safflower or cod-liver oil to a normal ration for dairy cows reduced the fat content of milk produced and increased the levels of NEFA and ketones in the blood.[82]

The level of feed intake can markedly influence the level of blood constituents. Fasting has been shown to increase NEFA levels in ewes.[36,39,61,62,69] Patterson[61] concluded that in both sheep and cattle, a concentration of 1000 μEq/liter or more of plasma NEFA indicated increased fat depot mobilization. Russel et al.[69] found that with prolonged fasting, levels of plasma NEFA could increase to a maximum of 1500 to 2500 μEq/liter.

The effect of restricted feeding on NEFA levels in the blood is increased by pregnancy,[3,39,66] lactation[65,66] and exposure to cold.[73] Russel and Doney[70] suggested that the relationship between plasma NEFA levels and feed intake in sheep was not linear and that a suitable transformation of the data

might improve their usefulness. They indicated the need for further information to determine the nature of the response of NEFA concentration to the change in the level of feed intake.

Two recent studies[58,78] indicated that levels of NEFA in the blood of sheep after 7-15 hours of fasting may be closely related to voluntary feed intake. A limited amount of information has been obtained on the process of NEFA formation during fasting in ruminants.[36,46,89]

Ketone Body Metabolism

The compounds commonly referred to as ketone bodies are: acetoacetate (AcAc), beta-hydroxybutyrate (BOHB) and acetone. A fourth compound isopropanol, has been detected in the rumen contents, but its significance is not known at the present time.[76] Ketone bodies arise from acetoacetyl CoA, which is a normal intermediate in fatty acid oxidation, but which also is rapidly formed from acetyl CoA. Acetoacetate is the parent ketone body and acetone and BOHB are its products. Most of the AcAc is reduced to BOHB by the action of the enzyme beta-hydroxybutyrate dehydrogenase. The reaction is reversible and the two compounds are interconvertible.[7] Acetoacetate by itself is an unstable compound and forms acetone and carbon dioxide irreversibly and non-enzymatically at the rate of about 5% per hour at body temperature.[7] Acetone is poorly utilized in the body and is of little importance in a normal animal. It is volatile and its characteristic odor arises from the breath of ketotic animals.

The principal precursors of ketone bodies are the non-esterified free fatty acids (NEFA).[25,50,57] These are mainly non-esterified free fatty acids of 16 to 18 carbon chain length, mobilized from the fat stores of the body. Dietary short-chain fatty acids with even number of carbon atoms are also

precursors. Butyrate is the principal fatty acid in this group. A few amino acids are also ketogenic but the majority are glucogenic.

In ruminants, the main sites of ketogenesis are the liver and rumen (omasal) epithelium. These can be termed hepatic and alimentary ketogenesis respectively. Alimentary ketogenesis occurs both in isolated tissues from cattle and sheep[33,74] and in the living animal.[2,41,68] Butyrate is the principal dietary fatty acid involved in this synthesis.[94] Since ketone bodies are formed during the process of absorption into the blood, this alimentary ketogenesis entirely ceases if the animal stops eating.[41,68]

Hepatic ketogenesis accounts for the bulk of ketone body production in the body particularly if the animal has a reduced feed intake. The major source of these ketone bodies are the NEFA, mobilized from the body's fat stores. In adequately fed sheep, alimentary ketogenesis accounted for virtually all the ketone body formation but this gave way to a much greater hepatic ketogenesis during fasting ketosis.[8,68] Katz et al.[41] found that the blood concentrations of total ketone bodies increased in all pregnant sheep and most nonpregnant sheep when fasted for three days. The mean concentration after fasting was higher in pregnant than nonpregnant sheep, but more variation was seen among individual animals.

The most prominent ketone body in the blood of all species is BOHB because it is much more stable than AcAc. The latter readily decomposes to acetone and carbon dioxide. Katz et al.[41] found that BOHB was the main ketone body produced by the liver in fed sheep but part of the production was offset by a small hepatic AcAc uptake, which probably represents its conversion to BOHB. In fasted sheep, however, acetoacetate accounted for 20-30% of the total hepatic ketone body production, so that the ratio of acetoacetate to the

total ketone body concentration in the blood increased during fasting and ketosis.

Most tissues can utilize ketone bodies and this is done by converting AcAc to acetoacetyl CoA. The latter can then form acetyl CoA and undergo oxidation in the Kreb's cycle, or be used for fat synthesis, particularly by the mammary gland, or the formation of other compounds. Free acetone, however, is glucogenic by forming pyruvate but it is of little importance.[54,55]

Isotopically labelled ketone bodies have shown that the rate of ketone body turnover and their complete oxidation to carbon dioxide (in the case of AcAc) are proportional to their plasma concentrations and this is true of a total ketone body concentration of 20 mg/100 ml of blood.[7,8,48] It was concluded that ketone body concentrations up to about 20 mg/100 ml are merely a reflection of the rate of ketone body production and that utilization in turn is regulated by blood ketone body concentrations. Maximal ketone body utilization evidently occurs at about 20 mg/100 ml and after reaching this level only small increases in production could cause large increases in blood concentrations.

Two main avenues of ketone body excretion are available in cows and these are by the way of the urine and milk. The kidney[86] and particularly the mammary gland also utilize ketone bodies for metabolism, the latter using BOHB mainly for lipogenesis.[17,53] A third avenue of excretion is that of acetone by the way of breath. Schultz[72] states that ketones in milk are roughly one-half of the blood values but urine concentrations are more variable and usually about four times the blood values.

Lactate Metabolism

Lactate is often produced at a fast rate in the rumen soon after the start of feeding,[64,83] but the total amount reaching the portal blood, at least under normal conditions of rumen fermentation, is small.[68] High grain diets, however, will increase both lactate[23] and propionate[51,84] production and absorption. The lactic acid in the rumen of sheep and cattle overfed on grain was found to be both the D(-) and L(+) forms, the ratio of these two forms varying. When sheep were put on increasing daily grain intake, initially the L(+) lactic acid was predominantly formed, but as grain feeding increased, the D(-) form became more evident until it predominated.[71] Increased ruminal production of lactic acid decreases the rumen pH and enhances its absorption.

Roe et al.[68] found that the portal concentrations of lactate and pyruvate were increased over that of the arterial blood of sheep after feeding. These workers felt that these two metabolites possibly may meet some of the ruminant's requirements as glucose precursors, especially in the case of pregnant fed sheep, where the increase in portal concentration was not offset by glucose utilization. They were unable to determine in their study, however, the precise origin of the portal lactate and pyruvate. Weigand et al.[87] also noticed that the concentrations of propionate and lactate were always higher in the portal blood than that of the arterial blood of calves fed on a diet containing 65% corn, 21% oats, 12% soybean meal and alfalfa hay ad libitum.

It is difficult to determine the extent of the contribution of propionate to glucose production in the whole ruminant animal. In sheep, where glucose synthesis was measured using ^{14}C-propionate, only about one-half of the

propionate carbon was actually converted to glucose.[10] It seems, therefore, that some of the ruminally produced propionate is metabolized by the rumen epithelium during absorption. Pennington and Sutherland[63] using ^{14}C-propionate as a metabolite for sheep rumen epithelium, found the greater part of the isotope appeared in carbon dioxide, but most or all of the remainder was found in the carboxyl group of the lactate formed. These authors concluded that when propionate was metabolized, carbon dioxide was fixed into lactate.

Leng et al.[49] found the incorporation into lactate and glucose of the isotope from (1-^{14}C) propionate to that from (2-^{14}C) or (3-^{14}C) propionate was much less than 50% and indicated that there was probably extensive incorporation of propionate carbon into lactate before conversion to glucose. In two out of three experiments the specific radioactivity of blood lactate was higher than that of glucose during (2-^{14}C) or (3-^{14}C) propionate infusion into the rumen. This supports the suggested extensive conversion of propionate into lactate. Since the conversion of propionate into lactate may occur in the rumen epithelium during absorption,[63] it is permissible to use the conversion of C_2 or C_3 of propionate into glucose or lactate as an indication of glucose or lactate synthesis from propionate and approximately 54% of both the glucose and lactate entry rate was apparently arising from propionate produced in the rumen. Leng et al.[49] concluded that a maximum of 70% of the propionate converted into glucose was first converted into lactate.

In a recent study on the propionate metabolism during absorption from the bovine ruminoreticulum, Weigand et al.[87] found that although the concentrations of propionate and lactate were higher in the portal blood than the arterial blood at the time of sampling, the direct conversion of propionate into lactate by the ruminoreticulum epithelium was quite small, probably less than 5%. These workers reported that the radioactivity of circulating

glucose was higher than that of the arterial lactate and concluded that the majority of propionate reaching the liver was synthesized into glucose before it was converted into lactate. They argued that because Leng et al.[49] analyzed blood from the jugular vein, they failed to provide proof for their proposal that the rumen epithelium was the site of propionate metabolism into lactate.

Annison et al.[4] showed a linear relationship between the rate of entry of infused L(+)-lactate and the blood concentration of lactate. The radioactivity of blood lactate and plasma glucose during the infusion of ^{14}C-lactate showed that a minimum of about 15% of the glucose pool was derived from lactate and about 40% of the lactate pool was derived from glucose when ^{14}C-glucose was infused. Evidence for the inhibition of lactate entry rate from sources other than glucose was obtained by infusing ^{14}C-glucose to raise the concentration of blood sugar. Comparison of the radioactivities of blood lactate and plasma glucose during the infusion of ^{14}C-glucose or ^{14}C-lactate indicated that about 6% of glucose was recycled through the glucose-lactate interconversion. Evidence from the relocation of ^{14}C in glucose during the infusion of (6-^{14}C) glucose indicated about 10% recycling. There was negligible incorporation of glucose or lactate into liver or muscle glycogen.

Chapman[20] reported that increasing the lactate concentration in the plasma by ten-fold caused the glucose flux to approximately double in lactating goats. During the intravenous infusion of sodium lactate, the lactate flux increased about five-fold and the rate of conversion of lactate into glucose and carbon dioxide increased by about three-fold. At "normal" levels of lactate, the rate of conversion of lactate into carbon dioxide was nearly twice the rate of its conversion into glucose. Lactate, however, was quantitatively more important as a source of carbon for glucose synthesis than

it was as an oxidative substrate leading to carbon dioxide production. Increasing the plasma level led to an increase in utilization of lactate for gluconeogenesis. Infusion of sodium lactate increased carbon dioxide fixation into plasma glucose. It appears that at elevated levels, lactate is a preferred substrate for gluconeogenesis and that it displaces other metabolites which may serve as glucogenic precursors.

The quantitative significance of lactate metabolism in ruminants is still difficult to evaluate, since some variable and unknown amounts of lactate are absorbed from the digestive tract. Annison et al.[4] and Bergman et al.[14] suggested that lactate contributes no more than 4-10% of the total glucose turnover in the body of fed sheep.

Metabolic Interrelationships Between Glucose, Non-esterified Fatty Acids, Ketone Bodies and Lactate in Relation to Blood Levels

Annison[3] first noticed that levels of non-esterified fatty acids (NEFA), ketone bodies and glucose in the blood are usually closely related in ruminants, with NEFA and ketones increasing while glucose decreases. In fasting ewes, the blood glucose levels declined more slowly than NEFA levels increased. He concluded that increase in NEFA was not directly due to hypoglycemia but may be the result of a shortage of short-chain fatty acids.

Reid and Hinks[66] found that levels of ketones or glucose in pregnant ewes carrying a single lamb and receiving increasing feed intake two weeks before parturition were not significant from those in ewes on a constant feed intake, but the NEFA levels increased in those with constant feed intake. Ewes on ad libitum feed maintained lower NEFA and higher glucose levels than ewes on restricted intake in late pregnancy. In nonpregnant sheep, a reduction of 40-70% in the ration did not affect blood glucose or ketone body

levels but elevated plasma NEFA levels. Karihaloo et al.[39] observed that in the last six weeks of pregnancy, NEFA and ketone body levels increased but glucose levels did not change, with ewes fed ad libitum.

Reid and Hinks[66] noted that the relationships between plasma NEFA, blood glucose and ketone bodies were similar in pregnant and lactating ewes, although plasma NEFA levels were consistently higher during lactation at all blood glucose levels. Ketone body levels were linearly related to plasma NEFA levels when blood glucose values exceeded 25-30 mg/100 ml of blood. Below this level of glucose there was no further increase in NEFA, but ketone body level increased markedly and could assume almost any magnitude. In lactating ewes, serum NEFA levels decreased as blood ketones increased with blood glucose above 25 mg/100 ml. These ketones may not originate from depot fat via serum NEFA, but probably participate in caloric hemostasis and are associated with a mechanism that spares glucose and suppresses NEFA.[1]

Patterson et al.[62] observed that undernutrition in ewes resulted in higher NEFA, lower glucose and slow-developing hyperketonemia. Hyperketonemia was associated with plasma NEFA levels greater than 900 μEq/liter. They found that glucose values were distributed normally, but suggested that NEFA values had a positively skewed distribution requiring a logarithemic transformation.

In dairy cows, Adler et al.[1] found that plasma NEFA levels were elevated in cows with clinical ketosis. They demonstrated that ketogenesis in normal cows was independent of plasma NEFA levels. They suggested that in normal cows ketones are derived from butyric and acetic acids originating from the rumen, whereas in clinical ketosis, ketones may have a dual origin; from ketogenic volatile fatty acids absorbed from the rumen and NEFA released from depot fat. In normal cows, the levels of blood glucose decreased as plasma NEFA levels increased. Radloff et al.[65] observed in cows and goats that blood

levels of NEFA decreased, ketones increased and glucose decreased after feeding. When the animals fasted, NEFA increased, blood ketones increased and blood glucose decreased. Changes in ketone and glucose levels were much greater due to fasting than feeding. Correlation between glucose, ketones and NEFA in the blood suggested that glucose was an important factor in ketogenesis under both fed and fasted conditions but that plasma NEFA was a primary source of ketones only under fasting conditions. In the fed animal, ketones in the blood arise from absorption of ketogenic volatile fatty acids from the rumen. Kronfeld[44] and Yamdagni et al.[94] also observed high NEFA levels in ketotic cows. Varmann et al.[81] found increased NEFA and increased ketones in the blood of cows fed safflower or cod-liver oil. They suggested that the high ketone levels were a result of high NEFA and increased butyric acid absorption from the rumen into the blood. Gardner,[26] with dairy cows in early lactation, observed an inverse relationship between blood ketones and blood glucose levels. Holmes and Lambourne[35] in beef heifers and Webb et al.[85] in male dairy calves found that plasma NEFA levels decreased while blood sugar increased.

Isotopically labelled carbon lactate infused intravenously into sheep[4] and lactating goats[20] showed that there is some metabolic interrelationships between the blood concentration of lactate and plasma glucose concentration. A minimum of 15% of the glucose pool was derived from the infusion of ^{14}C-lactate into sheep and 40% of lactate pool was derived from the infusion of ^{14}C-glucose.[4] Increasing the lactate concentration in the plasma ten-fold caused the glucose influx to be approximately doubled.[20] Hinkson et al.[32] incubated liver slices and epithelia dices from the rumen of sheep fasted for 60 hours, with L(+) and D(-) lactate and noticed that the ketone body content of the liver slice-media appeared to be unrelated to the presence of the two

forms of lactate. Neither lactate isomers were utilized by the rumen epithelia dices but some ketone bodies were synthesized. There has not been any attempt to relate blood concentrations of lactate to those of glucose, NEFA and ketone bodies in ruminants under common feeding practices.

Non-esterified Fatty Acids and Ketone Bodies as Indicators of Nutritional Status

Annison[3] was the first to suggest that the levels of plasma NEFA could be a useful indicator of nutritional status of ruminants, particularly as to carbohydrate utilization. Results of studies with pregnant ewes[66,69] supported this suggestion.

Reid and Hinks[66] concluded that plasma NEFA levels were more sensitive indicators of undernutrition during pregnancy in ewes than blood glucose or ketones. They observed that blood glucose and ketone body levels underestimated the additional feed requirements during late pregnancy. Apparently, a fat pregnant ewe, when undernourished can better maintain its blood glucose and ketone body levels in the normal range than a ewe in medium condition.

Russel et al.[69] suggested that the usefulness of plasma NEFA and blood ketone levels as indicators of nutritional status may vary with the degree of undernourishment. They found blood ketone body levels to be less sensitive as plasma NEFA levels approach or have reached their maximum. The results of their studies indicated the possibility for using biochemical indicators of nutritional status to define more accurately the response to nutrition in the pregnant animal, where body weight changes are confounded with weight changes of the fetus.

REFERENCES

1. ADLER, J.H. and E. LOTAN. 1967. The relationship of circulating glucose, ketone and free fatty acids to milk production in Awasi ewes. J. Agr. Sci. Camb. 69: 349-354.

2. ANNISON, E.F.; K.J. HILL and D. LEWIS. 1957. Studies on the portal blood of sheep. Biochem. J. 66: 592.

3. ANNISON, E.F. 1960. Plasma non-esterified fatty acids in sheep. Aust. J. Agr. 11: 58-64.

4. ANNISON, E.F.; D.B. LINDSAY and R.R. WHITE. 1963. Metabolic interrelationships of glucose and lactate in sheep. Biochem. J. 88: 243-248.

5. ANNISON, E.F. and J.L. LINZELL. 1964. Oxidation and utilization of glucose and acetate by the mammary gland of the goat in relation to their overall metabolism and to milk formation. J. Physiol. (Lond.) 175: 372-385.

6. BERGMAN, E.N. 1963. Quantitative aspects of glucose metabolism in pregnant sheep. Am. J. Physiol. 204: 147-152.

7. BERGMAN, E.N.; K. KON and M.L. KATZ. 1963. Quantitative measurements of acetoacetate metabolism and oxidation in sheep. Am. J. Physiol. 205: 449.

8. BERGMAN, E.N. and K. KON. 1964. Acetoacetate turnover and oxidation rates in ovine pregnancy ketosis. Am. J. Physiol. 206: 449.

9. BERGMAN, E.N.; R.S. REID; M.G. MURRAY; J.M. BROCKWAY and F.G. WHITELOW. 1965. Interconversions and production of volatile fatty acids in sheep rumen. Biochem. J. 97: 53-58.

10. BERGMAN, E.N.; W.E. ROE and K. KON. 1966. Quantitative aspects of propionate metabolism and gluconeogenesis in sheep. Am. J. Physiol. 211: 793-799.

11. BERGMAN, E.N. and D.E. HOGUE. 1967. Glucose turnover and oxidation rate in lactating sheep. Am. J. Physiol. 213: 1378-1384.

12. BERGMAN, E.N.; D.J. STARR and S.S. REULEN. 1966. Glycerol metabolism and gluconeogenesis in the normal and hypoglycemia ketotic sheep. Am. J. Physiol. 215: 874-880.

13. BERGMAN, E.N. 1970. Disorders of carbohydrate and fat metabolism. In: Duke's Physiology of Domestic Animals, by M.J. SWENSON. Ithaca, N.Y. Cornell Univ. Press, p. 595-618.

14. BERGMAN, E.N.; M.L. KATZ and C.F. KAUFMAN. 1970. Quantitative aspects of hepatic and portal glucose metabolism and turnover in sheep. Am. J. Physiol. 219: 785-793.

15. BERGMAN, E.N. and J.E. WOLF. 1971. Metabolism of volatile fatty acids by liver and portal-drained viscera in sheep. Am. J. Physiol. 221: 586-592.

16. BERGMAN, E.N. 1973. Glucose metabolism in ruminants as related to hypoglycemia and ketosis. Cornell Vet. 63: 341-382.

17. BLACK, A.L. and J.R. LUICK. 1965. The metabolism of ketone bodies in normal and ketotic cows and their utilization for milk synthesis. Radioisotopes in animal nutrition and physiol. Vienna: Int. Atomic Energy Agency, p. 71.

18. BOWDEN, D.M. 1971. Non-esterified fatty acids and ketone bodies in blood as indicators of nutritional status in ruminants: a review. Can. J. Ani. Sci. 51: 1-13.

19. CHANDLER, P.T.; W.N. DANNEBURG; C.E. POLAN and N.R. THOMPSON. 1970. Effect of fenfluoramine on appetite and lipid metabolism of the young ruminant. J. Dairy Sci. 53: 1747-1756.

20. CHAPMAN, T.E. 1969. The metabolic role of L(+)-lactic acid at resting and elevated concentrations of plasma lactate in lactating goats. Ph.D. Dissertation in Physiol. Grad. Sch. Univ. of Cal., Davis.

21. COOK, R.M. and L.D. MILLER. 1965. Utilization of volatile fatty acids in ruminants. I. Removal from portal blood by the liver. J. Dairy Sci. 48: 1339-1345.

22. DAVIS, S.L.; U.S. GARRIGUS and F.C. HINDS. 1970. Metabolic effects of growth hormone and diethylstilbesterol in lambs. I & II. J. Ani. Sci. 30: 229-240.

23. DUNLOP, R.H. and P.B. HAMMOND. 1965. D-lactic acidosis of ruminants. Ann. N.Y. Acad. Sci. 119: 1109-1132.

24. EXTON, J.H. 1972. Gluconeogenesis. Metabolism. 21: 945-990.

25. FRITZ, I.B. 1961. Factors influencing the rates of long-chain fatty acid oxidation and synthesis in mammalian systems. Physiol. Rev. 41: 52.

26. GARDNER, R.W. 1969. Interaction of energy levels offered to Holstein cows pre-partum and post-partum. I. Production responses and blood composition changes. J. Dairy Sci. 52: 1973-1984.

27. HIBBITT, K.G. and G.D. BAIRD. 1967. An induced ketosis. Vet. Record. 81: 511-517.

28. HALLIDAY, R.; A.R. SKYKES; J. SLEE; A.C. FIELD and A.J.F. RUSSEL. 1969. Cold exposure of Southdown and Welsh mountain sheep. 4. Changes in concentrations of free fatty acids, glucose acetone, protein-bound iodine, protein and antibody in the blood. Ani. Prod. 11: 479-491.

29. HARTMANN, P.E. and A.K. LASCELLES. 1965. Variation in the concentration of lipids and some other constituents in the blood plasma of cows at various stages of lactation. Aust. J. Biol. Sci. 18: 114-123.

30. HAWKINS, G.E. and W.E. DAVIS. 1970. Changes in plasma free fatty acids and triglycerides in dairy cattle after dosing with coffee or caffeine. J. Dairy Sci. 53: 52-55.

31. HEAD, H.H.; M. VENTURA; D.W. WEBB and C.J. WILCOX. 1970. Effect of growth hormone on glucose, nonestrified fatty acids and insulin levels and on glucose utilization in dairy calves. J. Dairy Sci. 53: 1496-1501.

32. HINKSON, R.S., JR.; W.H. HOOVER and B.R. POULTON. 1967. Metabolism of lactate isomers by rat and sheep liver and rumen epithelial tissue. J. Ani. Sci. 26: 799-803.

33. HIRD, F.J.R. and R.J. SYMONS. 1961. The mode of formation of ketone bodies from butyrate by tissue from the rumen and omasum of sheep. Biochim. Biophys. Acta. 46: 457.

34. HOGUE, D.E.; J.M. ELLIOT; E.F. WALKER and H. VIDAL. 1968. The effect of hay-grain ration on the composition of the gut contents of lambs. Proc. Cornell Nutr. Conf. 34: 57-60.

35. HOLMES, J.H.G. and L.J. LAMBOURNE. 1970. The relationship between plasma free fatty acid concentration and the digestible energy intake of cattle. Res. Vet. Sci. 11: 27-36.

36. JACKSON, H.D. and V.W. WINKER. 1970. Effect of starvation on the fatty acid composition of adipose tissue and plasma lipids of sheep. J. Nutr. 100: 201-207.

37. JUDSON, G.J. and R.A. LENG. 1968. Effect of diet on glucose synthesis in sheep. Proc. Aust. Soc. Ani. Prod. 7: 354-358.

38. KANEKO, J.J. and C.E. CORNELIUS. 1970. Clinical Biochemistry of Domestic Animals. Vol. I. 2nd ed. N.Y. Acad. Press, p. 40-52.

39. KARIHALOO, A.K.; A.J.F. WEBSTER and N. COMBS. 1970. Effects of cold, acute starvation and pregnancy on some indices of energy metabolism in Lincoln and Southdown sheep. Can. J. Ani. Sci. 50: 191-198.

40. KATZ, M.L. and E.N. BERGMAN. 1966. Acid-base and electrolyte equilibrium in ovin pregnancy ketosis. Am. J. Vet. Res. 27: 1285-1292.

41. KATZ, M.L. and E.N. BERGMAN. 1969. Hepatic and portal metabolism of glucose, free fatty acid and ketone bodies in sheep. Am. J. Physiol. 216: 953-960.

42. KAUFMAN, C.F. and E.N. BERGMAN. 1971. Renal glucose, free fatty acids ketone body metabolism in the unanesthetized sheep. Am. J. Physiol. 221: 967-972.

43. KREBS, H.A.; R. HENS; J.J. WEIDEMAN and R.N. SPEAKE. 1966. The fate of isotope carbon in kidney synthesizing glucose from lactate. Biochem. J. 101: 242-249.

44. KRONFELD, D.S. 1965. Plasma non-esterified fatty acid concentrations in the dairy cows: response to nutritional and hormonal stimuli and significance in ketosis. Vet. Rec. 77: 30-35.

45. KRONFELD, D.S. 1971. Hypoglycemia in ketotic cows. J. Dairy Sci. 54: 949-961.

46. LEAT, W.M.F. and E.J.H. FORD. 1966. Utilization of free fatty acids by starved and pregnant sheep. Biochem. J. 101: 317-322.

47. LEAT, W.M.F. 1967. Plasma lipids of newborn and adult ruminants and of lambs from birth to weaning. J. Agr. Sci. Camb. 69: 241-246.

48. LENG, R.A. and G.J. LEONARD. 1965. Measurement of the rates of production of acetic, propionic acids in the rumen of sheep. Brit. J. 19: 469-484.

49. LENG, R.A.; J.W. STEEL and J.R. LUICK. 1967. Contribution of propionate to glucose synthesis in sheep. Biochem. J. 103: 785-790.

50. LENG, R.A. and C.E. WEST. 1969. Contribution of acetate, butyrate, palmitate stearate and oleate to ketone body synthesis in sheep. Res. Vet. Sci. 10: 57.

51. LINDSAY, D.B. 1959. The significance of carbohydrate in ruminant metabolism. Vet. Rev. Annot. 5: 103-128.

52. LINDSAY, D.B. and R.E. BROWN. 1966. Acetate metabolism in sheep. Biochem. J. 100: 59.

53. LINZELL, J.L.; E.F. ANNISON; S. FAZAKERLEY and R.A. LENG. 1967. The incorporation of acetate, stearate and beta-hydroxyburate into milk fat by the isolated mammary gland of the goat. Biochem. J. 104: 34.

54. LITTLE, C.O.; G.E. MITCHELL and C.M. REITNOUR. 1968. Postruminal digestion of corn starch in steers. J. Ani. Sci. 27: 790-792.

55. LUICK, J.R.; A.L. BLACK; M.G. SIMESEN; M. KAMETAKA and D.S. KRONFELD. 1967. Acetone metabolism in normal and ketotic cows. J. Dairy Sci. 50: 544.

56. McRAE, J.C. and D.G. ARMSTRONG. 1966. Investigations of the passage of alpha-linked glucose polymers into the duodenum of sheep. Proc. Nutr. Soc. 25: 33-34.

57. MAYES, P.A. and J.M. FELTS. 1967. Regulation of fat metabolism in the liver. Nature. 215: 716.

58. MEARS, G.J. and V.E. MENDEL. 1970. Glucose, NEFA and insulin relations to intake. J. Ani. Sci. 30: 1038 (Abstr.).

59. MIKULEC, J.C. and A. TAYLOR. 1965. Serum albumin-bound fatty acid values in wethers. N.Z. J. Agr. Res. 8: 889-892.

60. O'KELLY, J.C. 1968. Comparative studies of lipid metabolism in Zebu and British cattle in a tropical environment. II. Blood lipid levels of cattle on different diets. Aust. J. Biol. Sci. 21: 1025-1032.

61. PATTERSON, D.S.P. 1963. Some observations on the estimation of non-esterified fatty acid concentrations in cows and sheep plasma. Res. Vet. Sci. 4: 230-237.

62. PATTERSON, D.S.P.; K.N. BURNS; N.F. CONNINGHAM; C.N. HEBERT and N. SABA. 1964. Plasma concentrations of glucose, non-esterified fatty acids in the pregnant and lactating ewe and the effect of dietary restriction. J. Agr. Sci. Camb. 62: 253-262.

63. PENNINGTON, R.J. and T.M. SUTHERLAND. 1956. The metabolism of short-chain fatty acids in sheep. 4. The pathway of propionate metabolism in rumen epithelium tissue. Biochem. J. 63: 618-628.

64. PHILLIPSON, A.T. and R.A. McANALLY. 1942. Studies on the fate of carbohydrates in the rumen of sheep. J. Exptl. Biol. 19: 199-214.

65. RADLOFF, H.D.; L.H. SCHULTZ and W.G. HOEKSTRA. 1966. Relationship of plasma free fatty acids to other blood components in ruminants under physiological conditions. J. Dairy Sci. 49: 179-182.

66. REID, R.L. and N.T. HINKS. 1962. Studies on the carbohydrate metabolism of sheep. XVII. Feed requirements and voluntary feed intake in late pregnancy, with particular reference to prevention of hypoglycemia and hyperketonemia. Aust. J. Agr. Res. 13: 1092-1111. XVIII. The metabolism of glucose, free fatty acids, ketones and amino acids in late pregnancy and lactation. i.b.d. 1112-1123. XIX. The metabolism of glucose, free fatty acids and ketones after feeding and fasting or undernourishment of nonpregnant, pregnant and lactating ewes. i.b.d. 1124-1136.

67. REID, R.L. 1968. The physiology of undernourishment in pregnant sheep, with particular reference to pregnancy toxemia. Adv. Vet. Sci. 12: 163-238.

68. ROE, W.E.; E.N. BERGMAN and K. KON. 1966. Absorption of ketone bodies and other metabolites via the portal blood of sheep. Am. J. Vet. Res. 27: 729.

69. RUSSEL, A.J.F.; J.M. DONEY and R.L. REID. 1967. The use of biochemical parameters in controlling nutritional state in pregnant ewes and the effect of undernourishment during pregnancy on lamb birth-weight. J. Agr. Sci. Camb. 68: 351-358. i.b.d. Energy requirement of pregnant ewes, p. 359-363.

70. RUSSEL, A.J.F. and J.M. DONEY. 1969. Observation on the use of plasma free fatty acid concentrations in the determination of maintenance requirements of sheep. J. Agr. Sci. Camb. 72: 59-63.

71. RYAN, R.K. 1964. Concentrations of glucose and low-molecular-weight acids in the rumen of sheep changed gradually from a hay to a hay-grain diet. Am. J. Vet. Res. 25: 653-658.

72. SCHULTZ, L.H. 1968. Ketosis in dairy cattle. J. Dairy Sci. 51: 1133.

73. SLEE, J. and R. HALLIDAY. 1968. Some effects of cold exposure, nutrition and experimental handling on serum free fatty acid levels in sheep. Ani. Pro. 10: 67-76.

74. STEVENS, C.E. and B.K. STETTLER. 1966. Factors affecting the transport of volatile fatty acids across rumen epithelium. Am. J. Physiol. 210: 365.

75. SUTTON, J.D. and J.W.G. NICKOLSON. 1968. The digestion of energy and starch along the gastro-intestinal tract of sheep. Proc. Nutr. Soc. 27: 49-50[A].

76. THIN, C.; H. PAVER and A. ROBERTSON. 1959. The metabolism of ketone bodies in the ruminants. J. Comp. Path. Tharap. 69: 45.

77. THOMPSON, G.E. and D.P. CLOUGH. 1970. The effect of intravenous infusion of dopamine on the unesterified fatty acids of sheep plasma. Res. Vet. Sci. 11: 428-430.

78. THYE, F.W.; R.G. WARNER and P.D. MILLER. 1970. Relationship of various blood metabolites to voluntary feed intake in lactating ewes. J. Nutr. 100: 565-572.

79. TOPPS, J.H.; N.B. KAY; E.D. GOODALL; F.G. WHITELAW and R.S. REID. 1968. Digestion of concentrate and hay diets in the stomach and intestines of ruminants. 2. Young steers. Brit. J. Nutr. 22: 281-290.

80. TUCKER, R.E.; G.E. MITCHELL and C.O. LITTLE. 1968. Ruminal and post-ruminal starch digestion in sheep. J. Ani. Sci. 27: 824-826.

81. VARMANN, P.N. and L.H. SCHULTZ. 1968. Blood lipid changes in cows of different breeds fed ration depressing milk-fat test. J. Dairy Sci. 51: 1597-1605.

82. VARMANN, P.N. and L.H. SCHULTZ. 1968. Blood lipids of cows at different lactation. J. Dairy Sci. 51: 1971-1974.

83. WALDO, D.R. and L.H. SCHULTZ. 1956. Lactic acid production in the rumen. J. Dairy Sci. 39: 1453-1460.

84. WARNER, A.C.I. 1964. Production of volatile fatty acids in the rumen: methods of measurement. Nutr. Abstr. Rev. 34: 339-346.

85. WEBB, D.W.; H.H. HEAD and C.J. WILCOX. 1969. Effect of age and diet on fasting blood and plasma glucose levels, plasma non-esterified fatty acid levels and glucose tolerance in dairy calves. J. Dairy Sci. 52: 2007-2013.

86. WEIDEMAN, E.J. and H.A. KREBS. 1969. The fuel of respiration of rat kidney cortex. Biochem. J. 112: 149.

87. WEIGAND, E.J.; N. YOUNG and A.D. McGILLIARD. 1972. Extent of propionate metabolism during absorption from bovine ruminoreticulum. Biochem. J. 126: 201-209.

88. WEISS, L. and G. LOFFLER. 1970. Interrelationship between adipose tissue and liver: gluconeogenesis and ketogenesis. Hormone and Metabolic Res. Suppl. 2. N.Y. Acad. Press, p. 196-203.

89. WEST, C.E. and E.F. ANNISON. 1964. Metabolism of palmitate in sheep. Biochem. J. 92: 573-578.

90. WILLIAMS, W.F.; S.D. LEE; H.H. HEAD and J. LYNCH. 1963. Growth hormone effects on bovine blood plasma fatty acid concentration and metabolism. J. Dairy Sci. 46: 1405-1408.

91. WOLF, J.E. and E.N. BERGMAN. 1972. Gluconeogenesis from plasma amino acids in fed sheep. Am. J. Physiol. 223: 455-460.

92. WOLF, J.E.; E.N. BERGMAN and H.H. WILLIAMS. 1972. Net metabolism of plasma amino acids by liver and portal-drained viscera of fed sheep. Am. J. Physiol. 233: 438-446.

93. YAMDAGNI, S.; L.H. SCHULTZ and H.D. RADLOFF. 1968. Effect of carbon-chain length on fatty acids on ketogenesis in ruminants. J. Dairy Sci. 51: 1094.

94. YAMDAGNI, S. and L.H. SCHULTZ. 1970. Fatty acid composition of blood plasma lipids of normal and ketotic cows. J. Dairy Sci. 53: 1046-1050.

APPENDIX II

TABLE 1

K.S.U. FEEDLOT CATTLE - BLOOD METABOLITES DATA

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
103	7/23/73	-	78.11	20.08	241.20
	8/23/73	-	42.81	46.07	137.72
	10/4/73	-	51.04	58.46	250.24
	11/15/73	-	93.13	39.00	64.71
	1/24/74	37.5	92.64	19.89	45.61
	3/7/74	39.5	76.92	17.10	46.62
203	7/23/73	-	67.67	30.65	183.80
	8/23/73	-	54.35	53.52	169.75
	10/4/73	-	63.73	16.81	134.86
	11/15/73	-	76.26	41.17	210.42
	1/24/74	44	81.14	21.23	186.82
	3/7/74	41.5	78.62	17.10	41.47
303	7/23/73	-	49.67	27.33	245.52
	8/23/73	-	59.22	27.96	173.63
	10/4/73	-	51.04	26.09	105.01
	11/15/73	-	69.58	25.80	72.62
	1/24/74	37.5	90.81	11.34	69.85
	3/7/74	39	66.18	10.81	20.15
403	7/23/73	-	53.44	37.62	77.00
	8/23/73	-	21.41	51.74	140.42
	10/4/73	-	30.19	53.42	204.62
	11/15/73	-	74.90	22.67	116.74
	1/24/74	38	68.20	17.91	82.73
	3/7/74	39	64.87	18.02	44.56

TABLE 1 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
111	7/23/73	-	69.00	17.10	525.40
	8/23/73	-	42.95	81.52	117.33
	10/4/73	-	57.60	66.49	291.93
	11/15/73	-	86.45	63.56	116.22
	1/24/74	40	106.33	31.22	103.03
	3/7/74	43.5	91.73	51.11	49.71
211	7/23/73	-	78.56	30.03	834.00
	8/23/73	-	45.43	79.84	131.99
	10/4/73	-	35.73	57.12	113.71
	11/15/73	-	92.66	75.86	151.29
	1/24/74	45	96.92	41.94	79.39
	3/7/74	48	138.51	96.27	41.47
311	7/23/73	-	69.00	34.78	574.40
	8/23/73	-	39.72	52.65	119.69
	10/4/73	-	43.46	54.38	107.86
	11/15/73	-	72.28	44.49	97.91
	1/24/74	40	87.51	24.21	101.82
	3/7/74	40.5	80.98	15.13	31.32
411	7/23/73	-	62.89	20.80	356.20
	8/23/73	-	48.52	32.72	181.72
	10/4/73	-	62.37	21.33	131.56
	11/15/73	-	54.80	26.52	82.99
	1/24/74	42	77.98	15.26	156.06
	3/7/74	39.5	72.73	16.38	86.74

TABLE 1 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
119	7/23/73	-	63.67	29.69	271.00
	8/23/73	-	53.99	50.87	124.07
	10/24/73	-	77.14	46.74	219.55
	11/15/73	-	69.93	27.38	99.62
	1/24/74	40	78.22	22.67	73.33
	3/7/74	37.5	77.58	27.53	65.15
219	7/23/73	-	65.11	25.46	974.40
	8/23/73	-	45.43	56.96	131.15
	10/4/73	-	48.42	43.81	184.97
	11/15/73	-	67.12	36.85	227.33
	1/24/74	45.5	98.68	22.38	183.64
	3/7/74	39.5	87.01	22.53	54.85
319	7/23/73	-	71.11	25.51	300.20
	8/23/73	-	37.34	49.34	293.65
	10/4/73	-	46.08	54.86	155.27
	11/15/73	-	67.18	25.65	111.88
	1/24/74	40.5	82.74	17.68	207.88
	3/7/74	36.5	82.56	14.32	78.09
419	7/23/73	-	74.33	6.10	376.00
	8/23/73	-	47.09	13.26	124.41
	10/4/73	-	63.73	44.29	99.01
	11/15/73	-	72.22	36.56	120.33
	1/24/74	42.5	102.30	24.41	67.27
	3/7/74	44	79.81	18.26	133.53

TABLE 1 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
127	7/30/73	-	30.52	23.16	490.74
	8/23/73	-	45.53	59.04	234.48
	10/4/73	-	65.19	48.95	338.26
	11/15/73	-	69.58	46.55	104.43
	1/24/74	48.5	92.03	28.06	53.94
	3/7/74	48.5	69.19	21.57	45.88
227	7/30/73	-	61.84	25.17	753.62
	8/23/73	-	36.63	70.62	418.06
	10/4/73	-	57.46	34.40	284.43
	11/15/73	-	69.82	26.95	244.55
	1/24/74	-	95.58	16.48	46.52
	3/7/74	39	81.64	27.82	38.82
327	7/30/73	-	60.45	25.85	956.58
	8/23/73	-	57.68	41.94	440.98
	10/4/73	-	54.85	49.00	116.56
	11/15/73	-	77.19	68.81	96.21
	1/24/74	44.5	86.17	28.15	61.52
	3/7/74	42	85.44	31.13	135.44
427	7/30/73	-	61.95	37.23	693.71
	8/23/73	-	47.69	71.00	104.35
	10/4/73	-	50.02	50.20	277.08
	11/15/73	-	77.20	6.63	216.47
	1/24/74	40.5	86.61	30.07	111.52
	3/7/74	38	86.62	27.33	66.76

TABLE 1 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
00	7/30/73	-	63.22	11.63	258.84
	8/23/73	-	53.16	45.69	155.76
	10/4/73	-	90.85	110.01	296.09
	11/15/73	-	74.62	32.04	96.05
	1/24/74	40	85.43	19.50	82.27
	3/7/74	35.5	93.17	40.35	47.35
235	7/30/73	-	32.58	11.86	448.50
	8/23/73	-	46.50	35.89	225.05
	10/4/73	-	59.35	34.35	163.52
	11/15/73	-	70.87	33.34	140.59
	1/24/74	40.5	82.26	24.36	108.18
	3/7/74	35.25	75.87	16.81	51.76
335	7/30/73	-	77.50	26.90	324.64
	8/23/73	-	47.09	54.29	128.11
	10/4/73	-	74.67	45.83	99.01
	11/15/73	-	59.61	34.68	60.67
	1/24/74	45	110.24	24.08	38.79
	3/7/74	43	84.39	29.54	23.82
435	7/30/73	-	55.62	21.86	388.93
	8/23/73	-	44.48	78.93	115.13
	10/4/73	-	54.98	35.84	170.86
	11/15/73	-	61.16	50.57	103.35
	1/24/74	41.5	81.23	39.73	104.85
	3/7/74	39.9	75.48	15.61	56.76

TABLE 1 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
143	7/30/73	-	71.05	22.10	334.90
	8/23/73	-	26.52	39.30	405.24
	10/4/73	-	25.81	58.22	113.41
	11/15/73	-	68.41	46.79	123.98
	1/24/74	39	89.47	16.33	79.39
	3/7/74	37	89.11	16.96	84.60
243	7/30/73	-	90.16	10.52	328.85
	8/23/73	-	41.03	96.13	117.49
	10/4/73	-	57.31	65.96	147.85
	11/15/73	-	89.50	66.30	243.93
	1/24/74	47	106.94	28.92	95.45
	3/7/74	43.5	87.28	24.60	82.50
343	7/30/73	-	90.74	39.34	414.51
	8/23/73	-	51.61	30.51	199.92
	10/4/73	-	40.69	32.67	107.41
	11/15/73	-	65.01	32.43	85.97
	1/24/74	44	87.87	24.26	40.15
	3/7/74	34	87.14	16.72	15.44
443	7/30/73	-	80.84	42.85	180.92
	8/23/73	-	20.34	71.53	351.13
	10/4/73	-	64.46	65.72	185.72
	11/15/73	-	90.65	50.49	325.34
	1/24/74	47	85.07	28.30	130.15
	3/7/74	39	76.66	20.47	40.00

TABLE 1 (concl.)

MEAN VALUES FOR ALL ANIMALS

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
Mean	7/23/73 & 7/30/73		65.79	25.15	438.90
	8/23/73		44.42	53.59	192.86
	10/4/73		55.26	48.96	179.12
	11/15/73		73.76	40.05	138.01
	1/24/74		89.48	24.14	96.26
	3/7/74		82.90	25.56	52.62

TABLE 2

H.K. CALEY BACKGROUND-AGE CALVES - BLOOD METABOLITES DATA

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
3	12/28/73	-	72.99	17.54	192.45
	12/29/73	-	69.45	19.74	283.58
	12/31/73	-	78.16	24.31	97.94
	1/1/74	-	81.12	20.75	111.91
5	12/21/73	-	64.12	7.54	203.75
	12/22/73	-	56.78	9.61	88.58
	12/23/73	-	61.75	10.28	191.25
	12/24/73	-	64.69	6.34	201.00
	12/25/73	-	57.00	19.22	172.25
	12/26/73	-	57.40	9.27	237.25
	12/27/73	-	59.49	14.36	312.59
	12/28/73	-	57.29	12.25	185.31
7	12/26/73	-	68.80	11.87	161.07
	12/27/73	-	61.07	9.70	363.18
	12/28/73	-	61.59	6.29	247.36
9	12/27/73	-	69.72	12.15	173.42
	12/28/73	-	61.16	8.79	153.35
	12/29/73	-	61.20	7.45	146.04
	12/31/73	-	72.73	16.48	127.06
	1/1/74	-	75.88	7.21	187.79
10	12/20/73	-	55.29	11.34	851.00
	12/21/73	-	56.54	8.07	812.82
	12/22/73	-	62.13	10.14	892.51
	12/23/73	-	64.36	10.86	587.10
	12/24/73	-	62.38	12.20	526.25
	12/25/73	-	70.90	26.33	229.50

TABLE 2 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
10 (cont.)	12/26/73	-	67.09	19.55	209.79
	12/27/73	-	58.84	10.71	147.86
	12/28/73	-	56.09	8.65	125.13
	12/29/73	-	59.49	9.80	330.67
	12/31/73	-	51.76	4.95	115.44
	1/1/74	-	58.45	5.38	145.59
	1/4/74	28	55.96	14.80	21.47
	1/6/74	27	64.87	12.11	38.82
15*	12/20/73	-	73.93	13.79	1027.43
	12/21/73	-	75.92	17.92	241.70
	12/22/73	-	70.20	8.21	396.03
	12/23/73	-	66.10	12.73	361.19
	12/24/73	-	69.99	22.15	274.75
	12/25/73	-	65.62	9.99	427.03
	12/26/73	-	71.42	12.68	601.67
	12/27/73	-	58.71	15.23	842.25
	12/28/73	-	62.64	9.37	430.96
	12/29/73	-	66.18	12.15	361.95
	12/31/73	-	66.44	7.64	269.26
	1/1/74	-	57.27	16.14	460.88
	1/4/74	27	69.45	21.14	47.50
19	12/20/73	-	64.86	9.80	713.86
	12/21/73	-	73.31	10.90	135.39
	12/22/73	-	78.65	9.66	290.77
	12/23/73	-	69.96	14.17	244.81

*Calf died.

TABLE 2 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
28	12/27/73	-	67.62	14.65	150.02
	12/28/73	-	61.85	8.74	99.29
	12/29/73	-	59.64	8.84	71.74
44	12/27/73	-	79.41	15.61	129.38
	12/28/73	-	78.10	29.11	150.63
	12/29/73	-	80.33	17.63	104.73
47	12/21/73	-	64.74	8.41	184.36
	12/22/73	-	59.02	8.98	339.69
	12/23/73	-	61.26	16.62	330.80
	12/24/73	-	58.45	7.64	120.25
	12/25/73	-	43.24	21.23	201.25
	12/26/73	-	66.19	10.67	76.00
	12/27/73	-	64.87	11.09	223.72
	12/28/73	-	59.89	12.20	96.91
	12/29/73	-	70.37	10.09	50.15
	12/31/73	-	66.57	3.41	42.94
	1/1/74	-	65.51	6.49	59.71
51	12/28/73	-	75.23	44.82	123.60
	12/29/73	-	91.86	27.91	375.38
	12/31/73	-	88.98	25.73	124.41
	1/1/74	-	82.69	12.73	244.56
56	12/26/73	-	68.68	23.35	137.39
	12/27/73	-	69.04	39.87	137.30
57	12/20/73	-	90.71	22.05	493.70
	12/21/73	-	83.87	10.62	169.57
	12/22/73	-	67.72	7.49	386.58

TABLE 2 (concl.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (µEq/L)
59	12/26/73	-	62.12	31.61	332.21
	12/27/73	-	94.61	38.53	131.78
	12/28/73	-	85.31	17.58	230.53
63	12/20/73	-	52.19	11.24	318.76
	12/21/73	-	63.12	10.91	258.63
	12/22/73	-	60.64	19.31	184.19
	12/23/73	-	50.57	7.69	92.85
	12/24/73	-	61.07	7.06	103.50
	12/25/73	-	62.64	15.66	151.75
	12/26/73	-	68.80	10.91	107.47
	12/27/73	-	58.84	15.42	86.41
	12/28/73	-	61.85	6.73	100.14
	12/29/73	-	66.05	9.22	36.21
	12/31/73	-	64.08	5.62	43.82
	1/1/74	-	68.67	5.96	129.56
	1/4/74	31	60.15	9.85	62.65
	1/6/73	33	57.27	6.05	45.59
69	12/28/73	-	56.89	13.31	162.70
	12/29/73	-	68.01	13.16	119.86
	12/31/73	-	82.03	36.51	148.82
	1/1/74	-	79.28	9.75	203.23
70	12/20/73	-	65.23	15.56	383.83
	12/21/73	-	70.45	8.74	421.62
	12/22/73	-	57.53	10.57	348.22
	12/23/73	-	60.39	5.81	573.24
	12/26/73	-	92.91	13.21	364.14
	12/27/73	-	54.12	11.87	329.09
	12/28/73	-	48.09	10.52	409.72

TABLE 3

K.S.U. DAIRY CATTLE - BLOOD METABOLITES DATA

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
020	12/28/73	-	55.81	7.06	118.33
	1/5/74	41	53.22	4.85	143.74
	1/11/74	36.5	65.92	5.43	55.14
	1/18/74	36.5	58.15	7.06	70.35
	1/25/74	35	52.15	2.93	330.73
	2/1/74	37	53.85	2.59	123.12
	2/8/74	37	60.83	3.75	253.88
	2/15/74	37	57.59	3.56	278.43
	2/22/74	34	59.35	8.22	234.53
	3/1/74	33.5	56.46	3.03	164.18
	3/2/74	36	51.44	4.90	69.51
	3/3/74	36.5	43.34	6.73	94.66
	3/4/74	37	52.93	2.64	104.42
	3/5/74	37	50.35	2.35	152.90
	3/6/74	39.5	61.21	8.94	747.56
	3/7/74*	37.5	61.35	7.16	274.24
	3/8/74	35	54.97	8.70	240.09
	3/9/74	43	52.12	4.56	140.24
	3/10/74	42	45.20	7.06	88.87
	3/11/74	40.5	40.31	6.01	44.21
	3/15/74	33	60.15	5.24	195.43
	3/22/74	30.5	54.22	4.71	199.94
	3/29/74	31.5	57.19	2.35	242.52
036	12/28/73	-	58.87	2.93	72.00
	1/4/74	37.5	63.24	18.06	83.43
	1/5/74	37	69.82	12.08	148.48

*Cow calved.

TABLE 3 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
036 (cont.)	1/6/74	33	60.90	9.08	163.41
	1/7/74	36	62.05	9.03	143.74
	1/8/74	-	61.46	6.44	65.19
	1/9/74	35	65.82	7.83	83.86
	1/10/74	-	70.06	11.91	61.60
	1/11/74*	36.5	61.95	7.78	507.46
	1/12/74	34	55.89	8.46	775.02
	1/13/74	36.5	50.05	3.31	375.52
	1/14/74	34.5	48.44	4.71	1481.84
	1/15/74	36	37.10	5.96	301.64
	1/18/74	32.5	31.58	4.13	590.13
	1/25/74	30.5	49.66	1.83	700.15
	2/1/74	30	40.20	8.60	264.46
	2/8/74	33	44.53	3.33	685.23
	2/15/74	30	53.58	1.92	570.55
	2/22/74	30	53.26	1.58	115.90
	3/1/74	30.5	52.12	2.50	136.28
	3/8/74	28	45.74	5.14	322.86
	3/15/74	30	50.06	5.67	29.57
	3/22/74	30.5	52.52	4.13	73.06
	3/29/74	30.5	54.60	3.46	106.90
050	12/28/73	-	60.87	6.44	131.17
	1/5/74	37.5	60.87	4.04	147.90
	1/11/74	31.75	66.85	5.43	86.27
	1/18/74	38	56.43	4.95	48.36
	1/25/74	36	60.47	5.04	293.60
	2/1/74	35	60.79	4.26	100.96
	2/8/74	38.5	61.80	6.01	124.41

*Cow calved.

TABLE 3 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
050 (cont.)	2/15/74	34	63.04	3.17	132.49
	2/21/74*	33.5	63.84	4.76	201.78
	2/22/74	35	61.92	6.63	202.29
	2/23/74	35	53.07	2.79	125.76
	2/25/74	34.5	50.35	4.37	175.15
	3/1/74	33.5	55.37	3.07	747.26
	3/8/74	33.5	56.32	5.67	332.16
	3/15/74	32.5	67.62	7.64	106.86
	3/22/74	29	62.06	3.03	706.42
	3/29/74	29.5	67.18	6.87	229.54
072	12/28/73	-	59.70	8.98	48.00
	1/5/74	35.25	63.23	13.74	142.45
	1/11/74	34.5	63.47	4.56	58.17
	1/18/74	36.75	59.98	6.34	81.91
	1/25/74	36	57.78	2.93	400.91
	2/1/74	39	60.55	3.07	122.07
	2/2/74	37.5	66.75	4.37	137.14
	2/3/74	35.5	88.99	9.90	240.20
	2/4/74*	41	99.26	12.25	369.74
	2/5/74	36.5	58.68	5.38	673.13
	2/6/74	35.5	51.61	5.14	260.39
	2/7/74	35.5	55.84	5.57	664.29
	2/8/74	34.5	39.90	3.22	740.27
	2/9/74	-	42.26	4.37	308.00
	2/10/74	35	50.37	1.83	217.39
	2/11/74	33.5	47.32	3.17	300.11
	2/15/74	31.5	57.91	2.16	150.32

*Cows calved.

TABLE 3 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
072 (cont.)	2/22/74	31	63.36	2.07	267.96
	3/1/74	37	69.22	4.47	137.96
	3/8/74	33	53.47	10.62	139.02
	3/15/74	34	71.81	25.56	43.90
	3/22/74	32	57.75	3.56	155.90
	3/29/74	34	62.90	4.23	159.11
074	12/28/73	-	60.64	3.12	30.50
	1/5/74	41	66.41	3.51	72.66
	1/11/74	32	67.09	5.67	67.28
	1/18/74	34.5	90.71	4.18	106.99
	1/25/74	37	63.17	6.25	330.40
	1/27/74	37.5	64.39	3.80	149.81
	1/28/74	34.5	61.57	4.04	196.38
	1/29/74	36	47.49	6.69	467.68
	1/30/74*	34.5	66.23	5.72	232.98
	1/31/74	34	45.29	3.46	615.44
	2/1/74	33	39.21	7.97	449.45
	2/2/74	32	37.75	3.12	175.43
	2/3/74	35.5	45.04	1.49	103.84
	2/8/74	33.5	60.10	5.09	432.67
	2/15/74	29.5	58.39	1.49	281.71
	2/22/74	31	61.76	3.22	216.01
	3/1/74	31.5	53.47	1.92	409.76
	3/8/74	29	53.47	4.47	225.46
	3/15/74	30.5	64.21	5.38	58.38
	3/22/74	30	56.31	3.41	98.15
	3/29/74	30.5	64.59	2.64	113.31

*Cow calved.

TABLE 3 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
086	12/28/73	-	50.28	4.71	54.33
	1/5/74	37	62.52	5.96	280.44
	1/11/74	35	59.39	4.47	168.06
	1/18/74	34.5	56.43	5.91	166.91
	1/25/74	37.5	69.41	4.56	932.09
	1/26/74*		NO SAMPLING		
	1/27/74	35	55.33	4.28	370.95
	1/28/74	40	54.47	6.77	422.73
	1/29/74	35	72.72	4.18	267.55
	1/30/74**	39.5	41.99	5.72	944.54
	1/31/74†	37.5	36.72	2.79	679.43
	1/31/74††	39	56.95	5.33	415.51
	2/1/74	37	48.14	6.29	349.81
	2/2/74	36.5	46.53	7.45	591.84
	2/3/74	36	55.96	2.02	249.38
	2/4/74	37	60.79	12.25	369.74
	2/5/74	33	54.84	6.58	260.13
	2/6/74	33.5	50.00	5.62	128.75
	2/7/74	33.5	53.89	3.70	75.48
	2/8/74	35.5	56.20	3.60	253.38
	2/9/74	35	68.61	3.56	93.80
	2/10/74	33.5	91.36	6.63	97.70
	2/11/74	33.5	71.65	6.01	86.63
	2/12/74	32	61.07	5.43	74.58

*Cow calved.

**Abomasal displacement diagnosed.

†Blood sample collected just before surgical correction.

††Blood sample collected after surgery.

TABLE 3 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
086 (cont.)	2/13/74	32	54.50	3.27	186.94
	2/14/74	-	52.54	11.34	171.96
	2/15/74	32.5	54.54	11.34	226.81
	2/18/74	31	48.93	3.60	186.22
	2/22/74	31	56.14	1.73	141.27
	3/1/74	30.5	54.15	7.06	93.90
	3/8/74	28.5	54.02	7.64	123.93
	3/15/74	29	55.82	5.67	29.57
	3/22/74	29	57.88	6.15	326.09
	3/29/74	31.5	58.89	3.51	141.02
087	12/28/73	-	61.46	5.38	116.67
	1/5/74	31	61.46	12.63	77.69
	1/11/74	31.25	68.84	9.32	41.55
	1/15/74*	33	59.27	9.03	111.86
	1/16/74	32.25	60.20	4.42	75.98
	1/17/74	33	57.75	4.47	45.25
	1/18/74	31.5	56.92	4.61	115.43
	1/25/74	30	64.51	4.08	190.69
	2/1/74	28.5	60.17	5.38	24.65
	2/8/74	29.5	63.14	7.45	8.63
	2/15/74	28.5	67.21	6.01	53.81
	2/22/74	29.5	70.58	10.86	102.69
	3/1/74	29	61.75	6.29	92.53
	3/8/74	29	62.70	7.73	95.27
	3/15/74	27.5	63.29	7.78	26.52
	3/22/74	28	61.67	4.04	34.12
	3/29/74	29	63.01	5.43	36.46

*Cow calved.

TABLE 3 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
088	12/28/73	-	55.40	4.80	27.33
	1/5/74	37.5	55.10	4.28	197.30
	1/11/74	-	63.28	8.26	64.37
	1/18/74	35.75	50.56	4.47	71.32
	1/25/74	37.5	51.17	1.05	443.57
	2/1/74	38	55.83	6.01	397.53
	2/7/74*	38	67.39	5.38	105.97
	2/8/74	37	54.02	6.82	65.80
	2/9/74	37.5	55.72	4.08	117.80
	2/10/74	36	50.37	5.81	74.91
	2/11/74	36.5	48.29	6.20	43.15
	2/15/74	35	62.39	3.23	441.11
	2/22/74	33.5	62.08	3.75	156.52
	3/1/74	36.5	56.73	1.92	165.24
	3/8/74	33.5	40.31	8.21	357.62
	3/15/74	34	53.99	5.76	139.63
	3/22/74	33	53.70	6.00	103.54
	3/29/74	33	54.08	4.28	92.46
095	12/28/73	-	61.34	2.40	40.32
	1/5/74	38.75	64.29	4.52	52.56
	1/8/74	43	70.65	6.34	246.55
	1/9/74	40.25	70.65	3.51	153.50
	1/10/74*	-	73.00	8.21	323.81
	1/11/74	38.75	55.89	4.32	372.79
	1/12/74	39	40.49	3.31	926.02
	1/13/74	39.5	43.99	1.10	922.59
	1/14/74	39.5	46.44	5.33	672.22

*Cows calved.

TABLE 3 (cont.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
095 (cont.)	1/15/74	33.5	34.54	5.62	215.94
	1/18/74	36	47.62	3.70	425.83
	1/25/74	32.5	53.62	0.43	240.84
	2/1/74	32	51.61	2.45	282.02
	2/8/74	33.75	47.32	2.02	250.28
	2/15/74	34.5	70.58	2.83	185.20
	2/22/74	31.5	61.28	1.54	162.87
	3/1/74	31.5	53.61	2.35	79.27
	3/8/74	31	33.93	6.53	484.45
	3/15/74	32.5	51.63	4.56	107.47
	3/22/74	34	52.78	11.96	67.96
	3/29/74	30	56.54	7.21	72.63
C133	12/28/73	-	55.95	8.79	50.00
	1/5/74	34	60.76	11.10	195.86
	1/11/74	37	62.77	6.10	85.75
	1/18/74	37.5	62.19	4.95	103.73
	1/25/74	36	63.17	5.28	292.46
	2/1/74	36.5	58.31	3.41	263.01
	2/5/74	40	77.29	7.54	112.49
	2/6/74*	39	65.38	11.39	54.28
	2/7/74	36.5	54.62	4.28	209.09
	2/8/74	37.5	51.34	3.36	314.12
	2/9/74	-	50.49	5.48	97.22
	2/10/74	38	51.22	3.03	234.65
	2/11/74	35	47.93	4.18	352.55
	2/15/74	33	64.49	1.39	511.91
	2/22/74	32.5	63.52	1.73	417.80

*Cow calved.

TABLE 3 (concl.)

ANIMAL NO.	DATE OF COLLECTION	PCV	PLASMA CONCENTRATIONS		
			GLUCOSE (MG%)	L(+)-LACTATE (MG%)	NEFA (μEq/L)
C133 (cont.)	3/1/74	33	56.32	4.13	500.31
	3/8/74	32	34.61	5.38	1370.73
	3/15/74	34	40.36	5.57	1248.17
	3/22/74	35	48.67	8.18	451.50
	3/29/74	31	49.67	3.75	275.63

CHANGES IN PLASMA LEVELS OF NON-ESTERIFIED FATTY ACIDS (NEFA), GLUCOSE AND L(+)-LACTATE IN:
I. BEEF CATTLE UNDER FEEDLOT CONDITIONS AND BACKGROUND-AGE CALVES WITH RESPIRATORY DISEASE
II. DAIRY COWS FROM LATE PREGNANCY INTO EARLY LACTATION

by

DANIEL B. OLUMEYAN

D. V. M., Ahmadu Bello University, Zaria, Nigeria, 1970

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I. Plasma non-esterified fatty acids (NEFA), glucose and (L(+)-lactate were measured in 24 randomly selected steers arriving into the Kansas State University Feedlot. First bleeding was within 24 hours of their arrival. They were distributed into six ration groups for finishing trials that lasted for 7 months. Five more bleedings were done, each corresponding to a ration switch.

The effects of transportation stress and excitement were revealed in first sampling by elevated plasma glucose and NEFA levels. Ration composition and switches appeared to have little or no effect on plasma glucose, NEFA and L(+)-lactate levels among the ration groups. A gradual increase in plasma glucose and a decline in NEFA and L(+)-lactate levels were highly significant ($P < 0.001$) with time, as the steers were fed increasingly high carbohydrate-rich rations. Correlation of glucose and NEFA was 0.973 and glucose and L(+)-lactate 0.915 for the means for all 24 steers.

In a separate study on background-age calves arriving into a backgrounding feedlot, the same plasma constituents were measured in 17 such calves undergoing treatments for respiratory disease. Fast-response calves had significantly higher glucose levels ($P = 0.05$) than slow-response calves. Plasma NEFA levels were consistently higher and L(+)-lactate lower in slow- than in fast-response calves.

II. Blood samples were collected from ten Holstein cows from late pregnancy into early lactation. Samples were collected weekly except during the immediate parturient period when daily samples were collected.

Determination of plasma concentrations of NEFA, glucose and L(+)-lactate showed that glucose levels were highest and highly significant ($P < 0.001$) and NEFA levels were significantly elevated a day before parturition. L(+)-lactate

levels were elevated only around the parturient period. Plasma glucose concentrations were lowest on the fourth day post-partum but had approached the pre-partum levels by the eleventh day following calving. Plasma NEFA levels remained inconsistently elevated until the 39th day after calving when they approached pre-partum levels.

Two cows had elevated post-partum plasma NEFA levels in the range reported for clinical ketosis, but showed no clinical signs. A cow with a left displaced abomasum had the highest plasma NEFA and beta-hydroxybutyric acid levels. A treated case of metritis showed a very high plasma NEFA and very low glucose levels.