NOVEMBER, 1951 RESEARCH BULLETIN 488

UNIVERSITY OF MISSOURI COLLEGE OF AGRICULTURE

AGRICULTURAL EXPERIMENT STATION

J. H. LONGWELL, *Director*

Environmental Physiology

With Special Reference to Domestic Animals

XVII. The Influence of Temperature on Blood Composition of Cattle

CLIFTON BLINCOE AND SAMUEL BRODY

IN COLLABORATION WITH

GLORIA BURGE, H. G. TURNER, DOROTHY WORSTELL, AND J. R. ELLIOTT

(Publication authorized October 26, 1951)

COLUMBIA, MISSOURI

TABLE OF CONTENTS

	Page
Introduction	3
The Overall Picture of the Data	8
Peculiarities of the Experimental Procedure	8
Interpretation of the Blood Composition Changes with Rising Environmental Temperature	14
Discussion and Summary	27
Appendix	28
Abstract	43
Bulletins in the Environmental Physiology Series	43

ACKNOWLEDGMENT

This is a part of a broad cooperative investigation between the Departments of Dairy Husbandry and Agricultural Engineering of the Missouri Agricultural Experiment Station, University of Missouri, and the Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Department of Agriculture. The Bureau of Dairy Industry, and the Bureau of Animal Industry, Agricultural Research Administration, U. S. Department of Agriculture, advised with the Bureau of Plant Industry, Soils, and Agricultural Engineering on various aspects of this work.

Special acknowledgments are made to A. C. Ragsdale, Department of Dairy Husbandry, University of Missouri, for selecting and managing the animals, and for counsel; and to Harold J. Thompson, Resident Agricultural Engineer, BPISAE, USDA, for managing the climatic chambers.

The work on the protein-bound iodine and radioiodine in cattle as function of temperature was done under contract with the Atomic Energy Commission (Division of Biology and Medicine); the other blood analyses were done under contract with the Office of Naval Research (Ecology Section). We are deeply grateful for the encouragement and financial support of the ONR and AEC.

Acknowledgments are made to J. V. Gates, Poteet, Texas, for the Brahman cattle, and to H. L. Dannen, St. Joseph, Missouri, for the Brown Swiss cattle.

Environmental Physiology

With Special Reference to Domestic Animals

XVII. The Influence of Temperature on Blood Composition of Cattle

CLIFTON BLINCOE AND SAMUEL BRODY
IN COLLABORATION* WITH
GLORIA BURGE, H. G. TURNER, DOROTHY WORSTELL, AND J. R. ELLIOTT

1. INTRODUCTION

This is a continuation of Missouri Research Bulletin 433 which was concerned with the effect of *rising temperature*, 50° to 100°F, on the blood composition of Holstein and Jersey cows during the *first experimental period* (Table 1 and Figure 1), during the summer of 1948.

This progress report is concerned with similar material obtained during *four subsequent experimental periods*—October 1948 to May 1950—including the effects of *declining temperature*, 50° down to about 0°F, on Holstein and Jersey (European-evolved), and on Brahman or Zebu (Indian-evolved) cattle; and the effects of *rising temperature*, 40° up to 105°F on Jersey, Holstein, Brown Swiss, and Brahman cows, and on Brown Swiss and Brahman heifers.

Data on the blood carotene and vitamin A were reported by O. T. Stallcup and H. A. Herman in Missouri Agricultural Experiment Station Research Bulletin 457, 1950.

Tables 1 and 2 and Figure 1 are summaries of the experimental designs, including chronologies, of all five periods.

The changes in respiration rate; feed consumption and milk production; evaporative cooling and body temperature; heat production, respiratory quo-

*H. G. Turner (present address: Animal Health Laboratory, Parkville, N. 2, Victoria, Australia) initiated the measurements for the protein-bound iodine in the blood and obtained and wrote up the preliminary data on the effect of temperature on protein-bound iodine in rats and cows as explained in the text. Gloria Burge then took over Mr. Turner's blood protein-bound iodine analyses on cows. Mrs. Burge, with the assistance of Mrs. Newcom, also analyzed the blood for creatinine, creatine, glucose, calcium, and did most of the hematology. Mr. Elliott did the analysis for ketone bodies. Clifton Blincoe managed the chemical laboratory and did the other analyses, including radioiodine. Dorothy Worstell, resident statistician of the BPISAE, USDA, assisted with the editorial work, particularly in arranging the data and preparation of tables and charts.

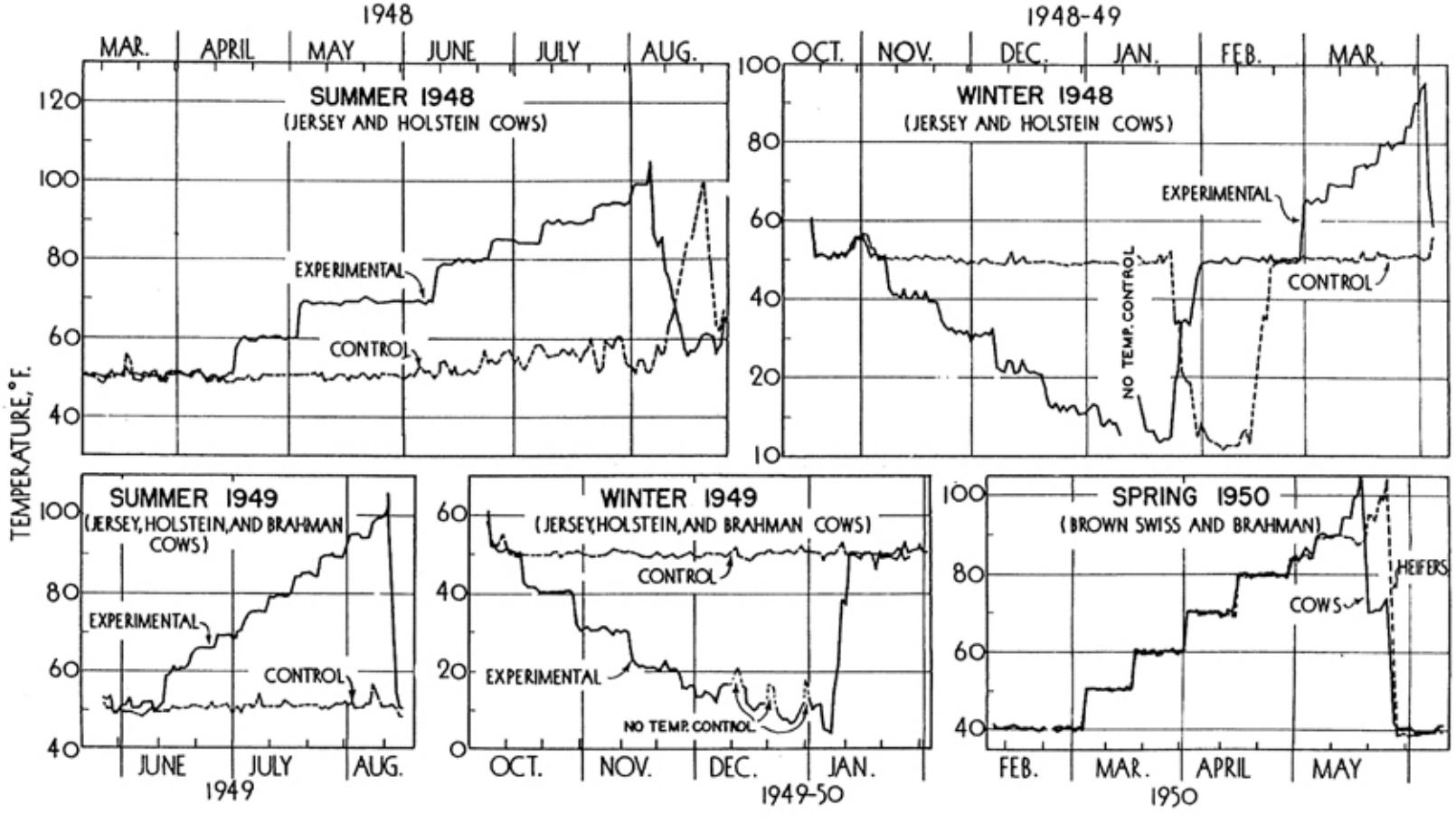


Fig. 1—Time-temperature schedule for the five experimental periods listed in Table 1.

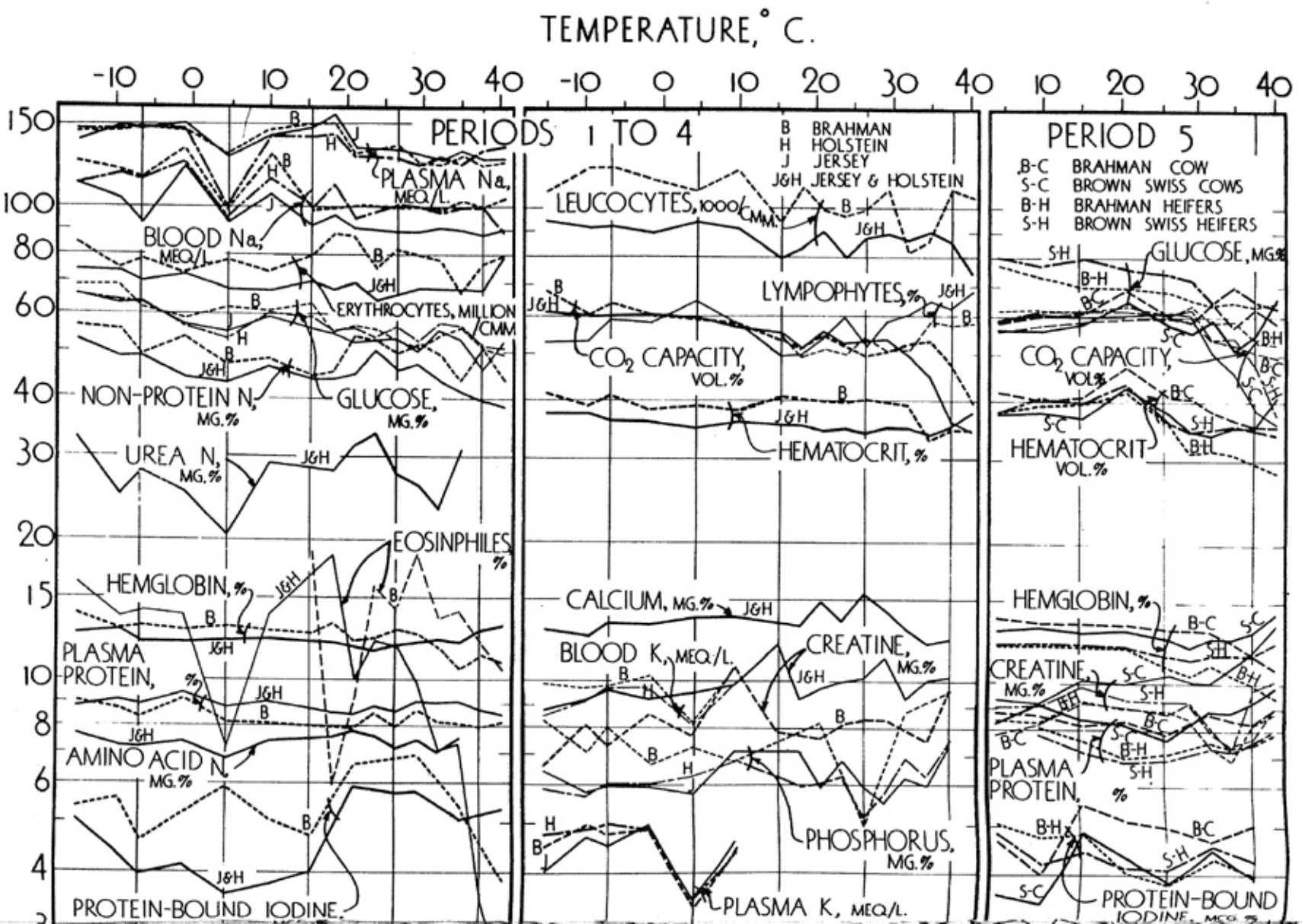
tient and pulse rate have been previously reported (see Appendix, page 43, for list of research bulletins in the "Environmental Physiology" series). The above physiological changes are closely correlated above 65° F with changes in alkali reserve, creatinine, cholesterol, and ascorbic acid in blood.

The numerical data for all the *Control* animals (kept at 50°F with other conditions similar to those for the Experimental animals) are listed in Table 3. The data for the *Experimental* animals are listed in Tables 4 to 8 (in the appendix), graphically summarized in Figure 2, and discussed in detail in Figures 3 and 4 in the text and Figures 5 to 10 in the appendix.

TABLE 1.--ANIMALS, VITAL STATISTICS
(Mature cows except the 6 yearling heifers in Period 5)

	At Beginning of Experiment				At Beginning of Experiment		
Cow No.	Age Years	Body Wt. Lbs.	Milk Yield* Lbs./day	Cow No.	Age Years	Body Wt. Lbs.	Milk Yield* Lbs./day
Period 1--April 16 to August 5, 1948, Rising Temperature 50° to 105°F.							
Experimental				Control			
Jersey 212	3 1/4	895	31.6	Jersey 205	3 1/2	917	30.7
Jersey 202	3 2/3	850	29.0	Jersey 204	3 1/2	805	35.5
Jersey 994	4 1/2	728	25.8	Jersey 504	3 1/2	747	26.7
Holstein 83	5 1/2	1260	46.2	Holstein 100	4 3/4	1290	52.7
Holstein 118	4 1/3	1125	38.4	Holstein 132	3 1/2	1150	38.5
Holstein 106	4 2/3	1085	dry	Holstein 90	5 1/4	1070	dry
Period 2--October 25, 1948, to April 4, 1949, Declining Temperature 50° to 0°F, and Rising Temperature 50° to 95°F.							
Experimental				Control			
Jersey 502	4	820	16.6	Jersey 957	8 3/4	870	15.7
Jersey 508	3 3/4	850	14.0	Jersey 979	4 2/3	840	15.9
Jersey 933	9 3/4	840	18.8	Jersey 977	6 3/4	910	17.2
Holstein 136	4	1220	33.6	Holstein 125	4 1/3	1230	40.2
Holstein 109	5	1200	36.0	Holstein 95	5 1/2	1170	29.4
Holstein 14	9 3/4	1450	dry	Holstein 5	9	1400	dry
Period 3--May 23 to August 15, 1949, Rising Temperature 50° to 105°F.							
Experimental				Control			
Jersey 994	5 2/3	770	32.5	Jersey 504	4 3/4	840	30.0
Jersey 212	4 1/2	990	34.0	Jersey 205	4 2/3	950	38.2
Brahman 190	2 1/2	750	†	Brahman 196	2 1/4	880	†
Brahman 209	2	710	†	Brahman 189	2 1/3	710	†
Holstein 109	5 3/4	1250	36.3	Holstein 147	4	1180	36.9
Holstein 7	10	1270	47.7	Holstein 146	4	1010	47.7
Period 4--October 4, 1949, to February 1, 1950, Declining Temperature 50° to 0°F.							
Experimental				Control			
Jersey 957	8 3/4	840	23.6	Jersey 979	6 2/3	870	19.6
Jersey 977	6 3/4	920	19.8	Jersey 508	4 3/4	890	22.1
Brahman 190	3	850	dry	Brahman 196	2 1/2	940	dry
Brahman 209	2 1/3	820	dry	Brahman 189	2 2/3	800	dry
Holstein 118	5 3/4	1200	38.8	Holstein 132	5	1190	41.4
Holstein 154	3 3/4	1200	39.1	Holstein 149	4	1130	38.8
Period 5--February 6 to June 9, 1950, Rising Temperature 40° to 105°F.							
Cows, Experimental				Heifers, Experimental			
Brown Swiss 16	6	1350	48.7	Brown Swiss 1	2/3	410	
Brown Swiss 20	6 1/2	1270	49.7	Brown Swiss 2	2/3	340	
Brown Swiss 24	6 1/4	1410	42.6	Brown Swiss 3	1/2	370	
Brahman 189	3	940	dry	Brahman 1	3/4	500	
Brahman 190	3	980	after 1st	Brahman 2	3/4	410	
Brahman 209	2 3/4	930	lactation	Brahman 3	3/4	410	

* Milk yield is average for month before start of experiment.
† Not milked before placed in laboratory.


TEMPERATURE, ° C.
-10 0 10 20 30 40
CONSTITUENT
PERIODS 1 TO 4
B BRAHMAN
H HOLSTEIN
J JERSEY
J&H JERSEY & HOLSTEIN
PERIOD 5
B-C BRAHMAN COW
S-C BROWN SWISS COWS
B-H BRAHMAN HEIFERS
S-H BROWN SWISS HEIFERS
PLASMA Na, MEQ/L.
BLOOD Na, MEQ/L.
ERYTHROCYTES, MILLION/CMM.
NON-PROTEIN N, MG.%
GLUCOSE, MG.%
UREA N, MG.%
EOSINPHILES %
HEMGLOBIN, %
PLASMA PROTEIN, %
AMINO ACID N, MG.%
PROTEIN-BOUND IODINE
LEUCOCYTES, 1000/CMM.
LYMPHOPHYTES, %
CO2 CAPACITY, VOL.%
HEMATOCRIT, %
CALCIUM, MG.%
BLOOD K, MEQ/L.
CREATINE, MG.%
PHOSPHORUS, MG.%
PLASMA K, MEQ/L.
GLUCOSE, MG.%
CO2 CAPACITY, VOL.%
HEMATOCRIT VOL.%
HEMGLOBIN, %
CREATINE, MG.%
PLASMA PROTEIN, %
PROTEIN-BOUND IODINE

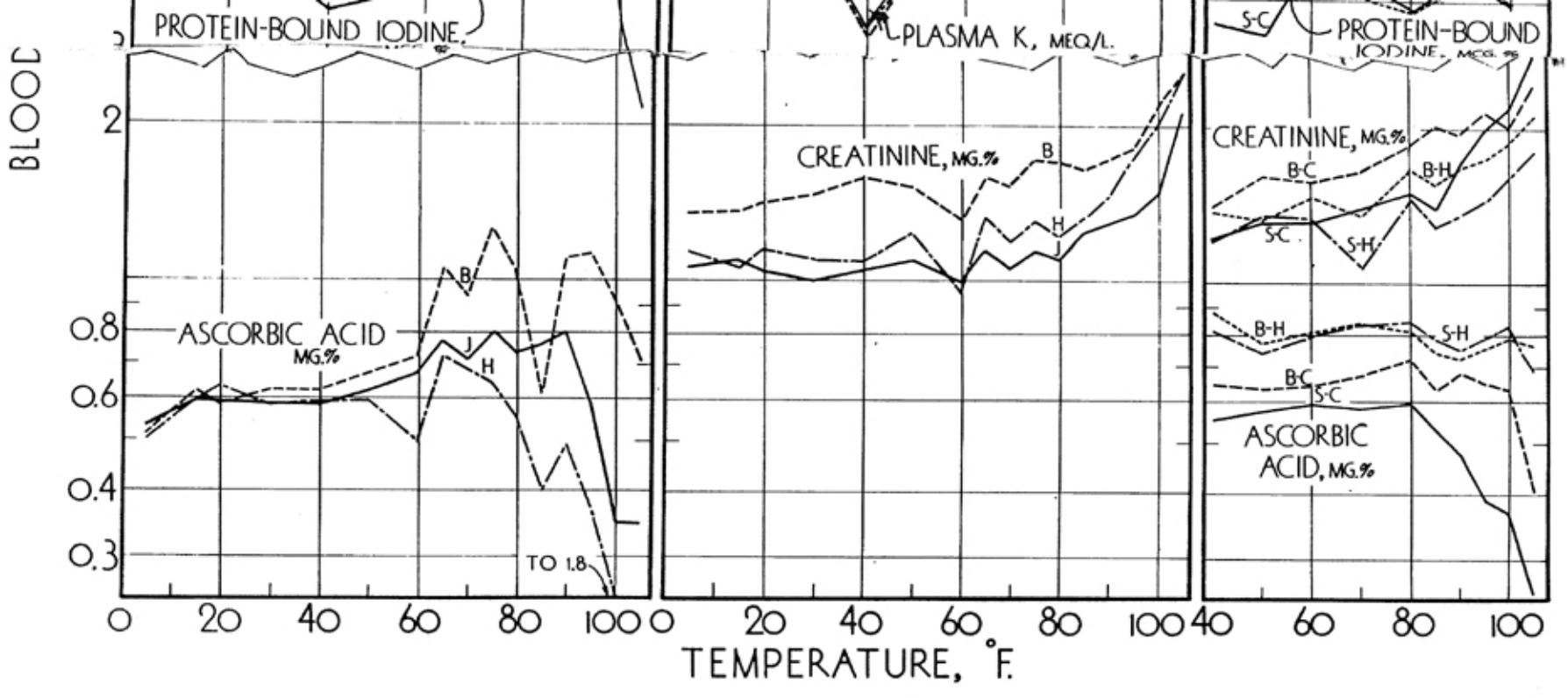


Fig. 2—The overall picture on an *arithlog grid* of the several blood constituents as functions of environmental temperature, 0° to 105°F, plotted from Tables 4 to 8. The left and middle sections represent averages of the data obtained during Periods I through 4 (Period 1 previously reported in Res. Bul. 433) on Jersey, Holstein, and Brahman cattle. The right-hand section gives the data obtained during Period 5 on Brown Swiss and Brahman cows and heifers. The following charts and those in the appendix give the detailed features of each individual curve for each temperature period on *arithmetic* grids.

2. THE OVERALL PICTURE OF THE DATA

Figure 2 gives an overall picture of the distribution of the data as function of environmental temperature as seen on an *arithlog* (semi-log) grid.

> In arithlog grids the vertical axis is divided logarithmically; the horizontal axis arithmetically. It is here useful because: (a) it enables plotting data of very wide range, in Figure 2 from 0.3 to 150, or 2 in 1000; (b) equal slopes on this paper represent equal *relative*, or *percentage* changes, thus giving a direct comparison of percentage rate changes in all the blood constituents regardless of their absolute values. The other charts are on arithmetic paper.
>
> The reaction of each animal is simply plotted against the corresponding time and temperature. No attempt was made to compute the statistical parameters that characterize populations because of the fewness of the animals, which were not selected at random, and which were subjected to acclimatizing, or deteriorating, influences by the changing temperature. The Gauss Law of Errors, on which computation of such parameters are based is not generally applicable to this type of rate-of-progress data. This may, however, become possible in the future when sufficient data will have been accumulated to determine its applicability.

Inspection of the curves of the overall picture in Figure 2, taken along with Figures 3a and 3b representing data for Period 5, and Figures 4a and 4b representing the ratios of Experimental to Control values, indicate the following three categories of *change with rising temperature above thermoneutrality:*

(a) *Steep change:* over 100 per cent rise in the creatinine level; over 50 per cent decline in carbon dioxide-combining capacity, presumably associated with rise in metabolic acids; over 50 per cent decline in ascorbic acid in some *cows* (not in the yearling heifers), and in cholesterol, and to a less extent in fatty acids.

(b) *Variable change:* in glucose, eosinophils and lymphocytes; protein-bound iodine and radioiodine.

(c) *No appreciable change:* total solids and electrolytes. There was no significant change with *declining* temperature from 65°F down to about 5°F, with exception of a possible increase in blood sugar.

3. PECULIARITIES OF THE EXPERIMENTAL PROCEDURE

The primary object of this project was to obtain data needed by the building and dairy industries. Hence, the design of this research differs from the heat-stress and cold-stress type of experiment usually reported in the physiological literature.

For instance, a typical physiological experiment[1] on the effect of heat (or cold) stress on blood or tissue composition consists in placing a homogeneous

[1]Long, C. N. H., Federation Proceedings, *6*, 461, 1947; also Long, Recent Progress in Hormone Research, *1*, 99, 1947; Sayers, G., and M. A., Id., *2*, 81, 1948, and Ann. N. Y. Ac. Sc., *50*, 522, 1949. Sayers, et al., Yale J. Biol. Med., *16*, 361, 1949. Sayers, G., Physiol. Rev., *30*, 241, 1950. Selye, H., et al., Endocr., *46*, 27, 1950.

group of rats for an hour in a high (or low) temperature medium, then at appropriate intervals after the exposure analyze the blood and tissues of a sample of the exposed group. The time changes are plotted, an interpretative theory is formulated, which is tested by "critical experiments" until proved or disproved.

In the present experimental design a few valuable cows—which could not be sacrificed or operated on—were initially placed at what was believed to be the most comfortable temperature (to cows) of 40° to 50° F (4° to 10° C):

TABLE 2.--ANALYSIS
FOR THE FOLLOWING BLOOD CONSTITUENTS WERE MADE DURING THE PERIODS INDICATED

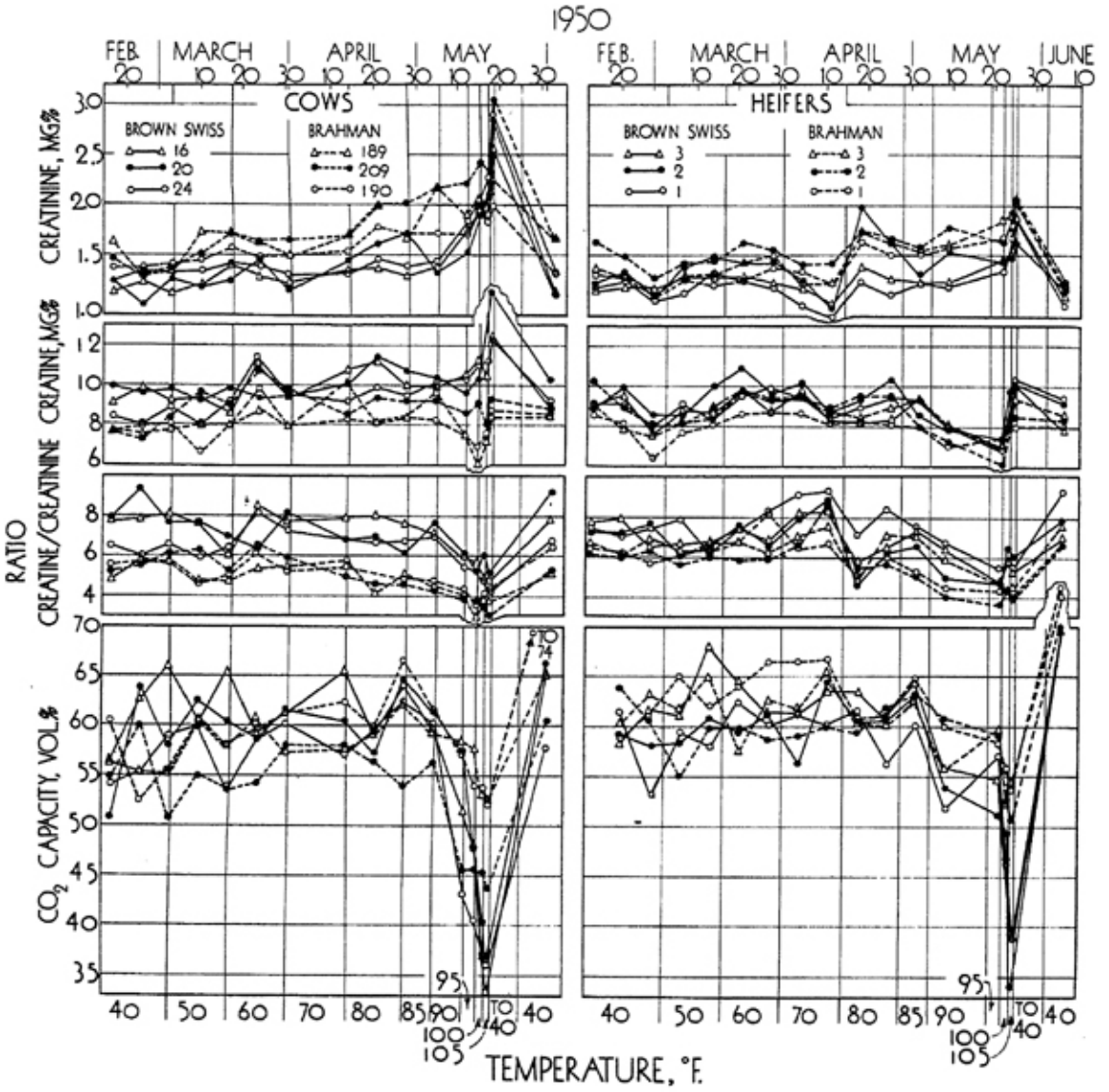
	PERIOD				
Constituent	1	2	3	4	5
Amino Acid Nitrogen		XXXX			
Ascorbic Acid			XXXX	XXXX	XXXX
Calcium	XXXX	XXXX			
Carbon Dioxide Capacity	XXXX	XXXX	XXXX	XXXX	XXXX
Catalase	XXXX				
Cholesterol	XXXX				
Creatine		XXXX	XXXX	XXXX	XXXX
Creatinine	XXXX	XXXX	XXXX	XXXX	XXXX
Fatty Acids	XXXX	XXXX			
Glucose	XXXX	XXXX	XXXX	XXXX	XXXX
Hematology:					
Differential Counts	XXXX	XXXX	XXXX		
Erthrocytes	XXXX	XXXX	XXXX	XXXX	
Leucocytes	XXXX	XXXX	XXXX	XXXX	
Hematocrit	XXXX	XXXX	XXXX	XXXX	XXXX
Hemoglobin	XXXX	XXXX	XXXX	XXXX	XXXX
Iodine, Protein Bound		XXXX	XXXX	XXXX	XXXX
Magnesium*	XXXX				
Nitrogen, Non-Protein	XXXX	XXXX	XXXX	XXXX	
Phosphorus, Inorganic	XXXX	XXXX	XXXX	XXXX	
Plasma Protein	XXXX	XXXX	XXXX	XXXX	XXXX
Potassium				XXXX	
Sodium			XXXX	XXXX	
Urea Nitrogen		XXXX			
Uric Acid Nitrogen		XXXX			
Vitamin A and Carotene†	XXXX	XXXX	XXXX	XXXX	

* Reported in Missouri Research Bulletin 433.
† Reported in Missouri Research Bulletin 457.

accustomed to the environment for 2 to 3 weeks; and the various measurements taken; then the temperature was lowered or raised, by 5° to 10°F increments, at weekly or biweekly intervals, to the limiting temperatures of 0°F (–18°C), or 105°F (41°C), repeating the measurements at each temperature level.

This type of experimental design allows time for acclimatization, or deterioration if the temperature is outside the acclimatization limits.

The region of thermoneutrality in these animals (producing 500 to 1000 kilo-calories, or 2000 to 4000 Btu, heat an hour) was between near-freezing



Figs. 3a (above) and 3b—Details of temperature, and also time (upper axis), trends of individual blood constituent data on high-milking Brown Swiss cows, dry Brahman cows, yearling Brown Swiss and Brahman heifers observed in Period 5 during exposure to increasing temperature, 40° to 105°F, and return down to 40°F. Note the breed and age differences and temperatures at which the declines begin in carbon dioxide-combining capacity and the rise in creatinine. Note that the heifers, but not the cows, show decline in *creatine* above 80°F, and that the cows show a much higher rise in *creatinine* than the heifers. See Fig. 3b on p. 11.

temperature and 65°F (18°C)—with the apparent "comfort zone" nearer the freezing temperature. The precise "comfort" or "optimal" temperature, of course, depends on the productive level, size and specific surface area, thickness of subcutaneous fat, and insulating properties of the hair which differ in individuals and breeds.

The cows adjusted themselves perfectly to declining temperature down to 0°F (as low as the experiments went). On lowering the temperature below

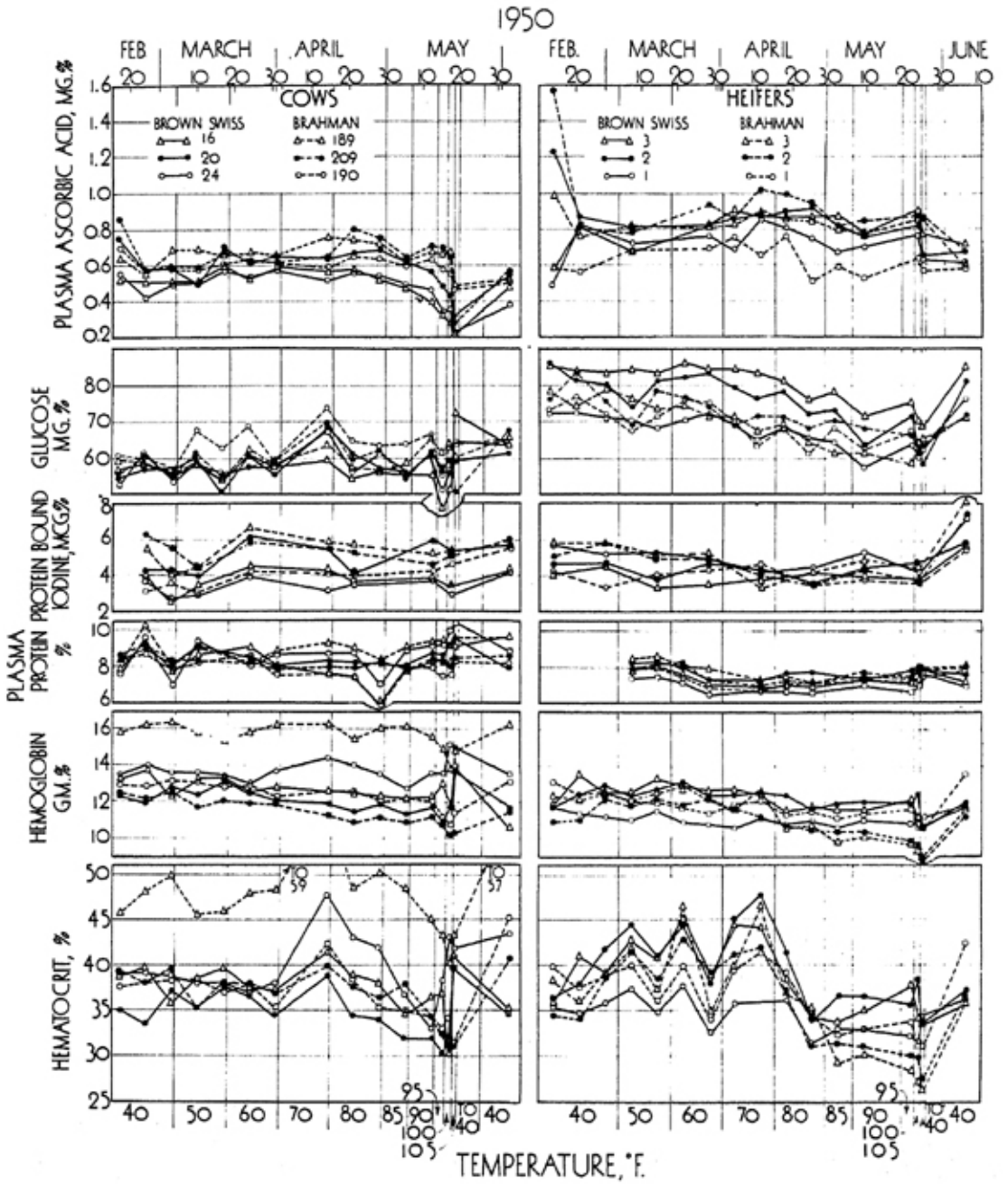
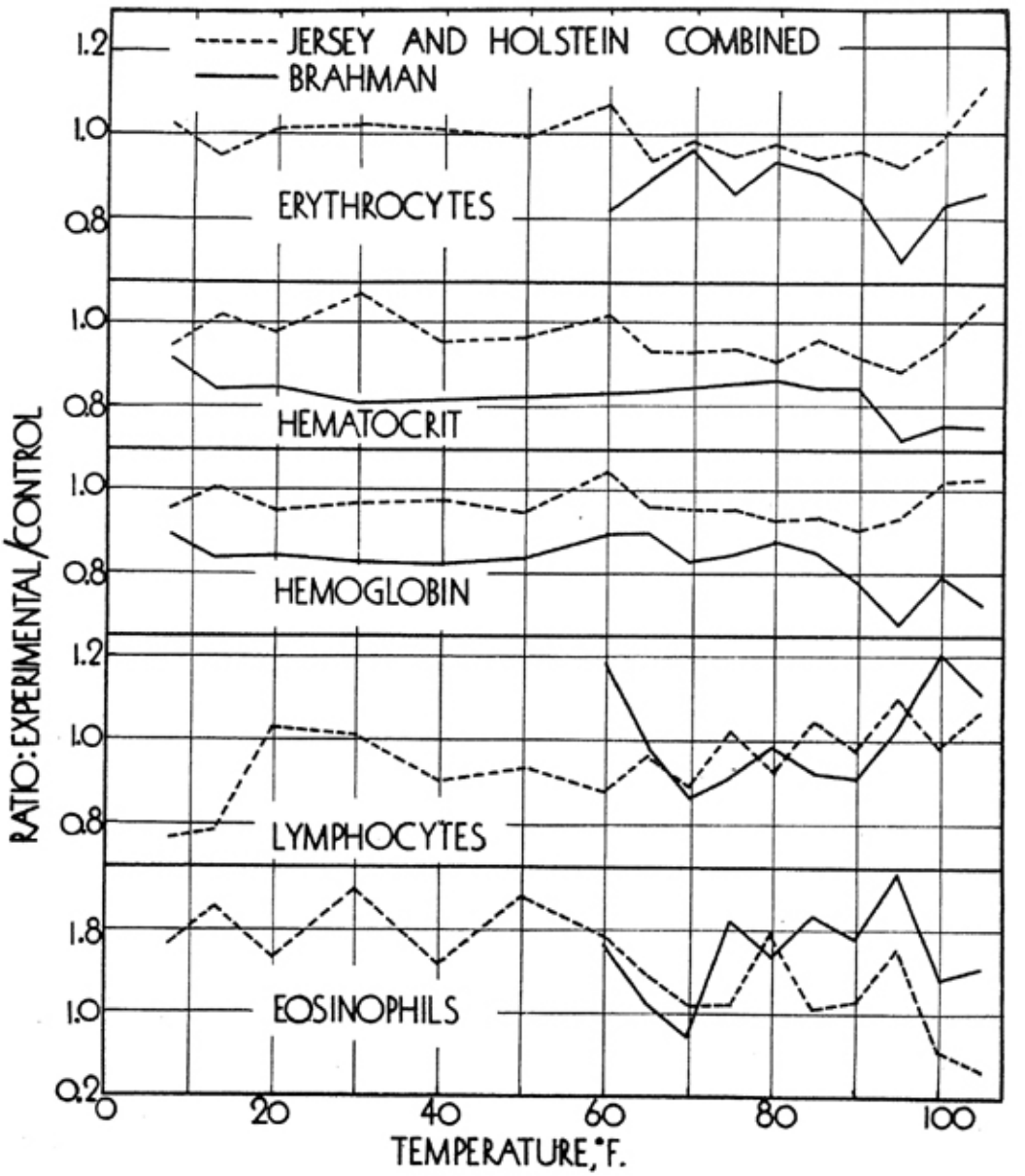


Fig. 3b.—For explanation, see page 10.

freezing, their hair increased in length; their heat production and feed consumption increased (more in the small, Indian-evolved, low-producing, than in the large, European-evolved, high-producing cows). On raising the temperature above 50°F, the evaporative cooling and respiration rate took a steep upturn at 65°F (18°C), reaching a maximum at about 80°F (27°C). The steep rise in evaporation rate at about 65°F in quietly standing cattle was presumably analogous to the breaking out in sweat at about 80°F in man. But whereas



Figs. 4a (above) and 4b—Summary chart for Period 1 through 4 in the form of ratios of experimental to Control values (environmental temperature, 50°F as function of environmental temperature. The changes in slope appear to begin at about 65°F. The ratio curves show the expected rise for creatinine, carbon dioxide-combining capacity and ascorbic acid; the rise for eosinophiles and lymphocytes, and decline for glucose are less certain. See Fig. 4b, p. 13.

in man the evaporative cooling increases exponentially with increasing environmental temperature, in our cows it did not increase with increasing environmental temperature above about 80°F with the result that at about 80°F there was a sudden rise in rectal temperature, decline in food consumption and milk production, and so on, as previously described (see Appendix page 28).

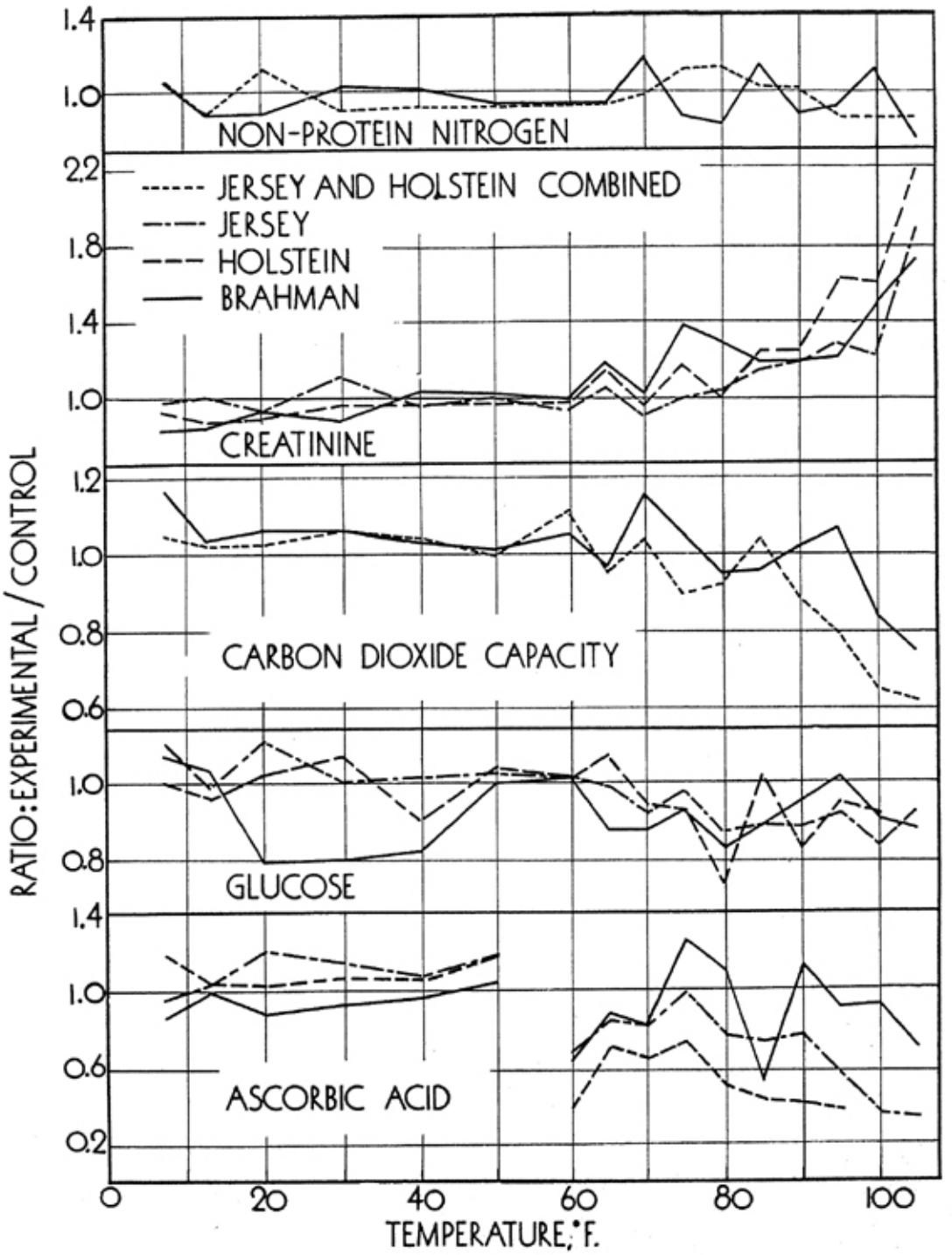


Fig. 4b.—For explanation, see page 12.

This report is, then, concerned mostly with following the changes in the levels of several blood constituents with increasing temperature above about 65°F. The levels of some constituents were quite stable, hardly affected by changing temperature; others changed dramatically, especially on raising the temperature above 80°F (27°C). Why?

TABLE 3.--MEAN VALUES OF BLOOD CONSTITUENTS FOR CONTROL COWS
(Maintained at 50°F)

Blood Constituent	Jersey			Holstein			Brahman		
	N	Mean	Standard Error	N	Mean	Standard Error	N	Mean	Standard Error
Amino Acid Nitrogen, mg. %	42	7.57	.095	42	7.56	.090			
†Ascorbic Acid , mg. %									
Period III	14	.96	.030	14	1.02	.032	14	1.13	.036
Period IV	22	.52	.012	22	.52	.014	22	.63	.010
Calcium, mg. %	68	13.5	.26	69	13.5	.20			
Carbon Dioxide Capacity, vol. %	101	55.5	.36	103	56.8	.46	39	55.8	.74
*Catalase , activity units	8	2588	328	9	1530	222			
*Cholesterol (lactating), mg. %	27	174	3.1	17	149	3.8			
Creatine, mg. %	81	9.49	.129	82	9.28	.134	40	7.26	.207
Creatinine, mg. %	106	1.09	.012	105	1.19	.016	40	1.53	.028
†Fatty Acids (lactating), mg. %	39	185	11.2	24	196	10.3			
Glucose, mg. %	109	57.4	.56	108	60.8	.75	39	63.4	1.42
Hematology:									
Eosinophiles, %	80	8.3	.62	85	8.1	.59	16	9.1	.67
Lymphocytes, %	82	61.7	1.03	86	58.9	1.26	16	51.4	2.27
Erythrocytes, millions/cmm.	111	6.69	.071	111	7.19	.091	40	9.22	.132
Leucocytes, thousands/cmm.	111	9.0	.17	112	8.4	.16	40	10.6	.30
Hematocrit, vol. %	109	36.8	.28	109	37.5	.36	40	46.2	.33
Hemoglobin, %	100	12.8	.08	100	12.6	.12	40	15.1	.12
Iodine, Protein Bound, mcg. %	20	3.27	.188	20	4.26	.210	20	5.49	.312
*Magnesium , mg. %	23	3.82	.141	22	3.87	.137			
Nitrogen, Non-Protein, mg. %	105	47.6	.50	106	47.4	.67	37	53.8	1.33
Phosphorus, Inorganic, mg. %	100	5.73	.101	101	6.11	.106	29	7.48	.129
Plasma Protein, %	105	8.80	.080	107	8.97	.070	34	9.12	.121
Potassium, meq./liter:									
Plasma				16	4.78	.098	16	4.86	.082
Blood				16	10.4	.271	16	9.50	.338
Sodium, meq./liter:									
Plasma	37	141	1.8	38	139	1.7	38	143	1.9
Blood	38	102	2.5	38	110	2.1	38	109	2.1
Urea Nitrogen, mg. %	42	23.7	.88	42	25.3	.76			
†Uric Acid Nitrogen , mg. %	42	1.87	.136	41	2.25	.173			
†Vitamin A and Carotene									

* From Research Bulletin 433.

† Declines with advancing stage of lactation, with time, or hay consumption, so a stable value cannot be given. See tables giving values for time intervals. For vitamin A and carotene, see Research Bulletin 457.

4. INTERPRETATION OF THE BLOOD COMPOSITION CHANGES WITH RISING ENVIRONMENTAL TEMPERATURE ABOVE THERMONEUTRALITY WITH SPECIAL REFERENCE TO ENDOCRINE MECHANISMS

On raising the environmental temperature above thermoneutrality, the animals began to suffer from heat stress and loss of appetite.

Starvation is very stressful to cattle (particularly for the highly lactating, highly metabolizing type) because on onset of fast, the typically herbivorous fat-poor and carbohydrate-rich metabolic mix is changed to the typically carnivorous fat-rich and carbohydrate-poor mix. Such a shift was demonstrated objectively (Mo. Res. Bul. 435) by the decline in the respiratory quotient with

rising temperature from over 1.0 at 50°F to about 0.7 at 100°F. This situation affected the level of some blood constituents but not of others, and the problem is how to interpret the presence or absence of change.

A. Creatinine: Figures 2 to 4 show consistent rise in blood creatinine with rising temperature associated with declining food consumption. Since the rate of creatinine production is the best index of endogenous protein catabolism[2], and since there must be an accelerated rise in endogenous catabolism on declining feed consumption below maintenance, it is not surprising to find a rise in blood creatinine. The surprising aspect is that there was no corresponding rise in creatine, from which the creatinine must have been derived. It will not be possible to interpret this peculiarity until data for urinary creatinine and creatine have been obtained. It is known that there is a reduced creatinine clearance in Addison's disease (adrenal insufficiency)[3], and it is possible that heat stress raised the renal threshold value for creatinine.

The rise in blood creatinine is, then, tentatively interpreted as being due to rise in endogenous nitrogen catabolism with decline in feed consumption, perhaps reinforced by *heat stress* with associated acceleration of adrenal hormone secretion which stimulates protein deamination, or possibly rise in renal threshold for creatinine. The literature on this subject, which contradicts some of these statements (as Beard's book), is listed in Mo. Res. Bul. 433.

B. Carbon Dioxide-Combining Capacity: The bottom segment of Figure 3a shows that the CO_2-combining capacity, or alkali reserve, of blood plasma remains roughly constant (about 60 vol %) between temperatures 40° and 85°F, and decreases sharply thereafter with rise in temperature to 105°F (to about 35 vol %). This sharp decline in CO_2-combining capacity was first thought to be due to blowing off of excess CO_2 as result of increasing panting with increasing temperatures, with consequent loss of $BHCO_3$.

One objection was that there was a great lag in time and temperature between the beginning of panting (hyperventilation) at 70°F and the beginning of decline in CO_2-combining capacity of blood, weeks afterwards, at 90°F (see charts on pp. 13, 15, 17, 31 and 32, Mo. Res. Bul. 435; p. 4, Mo. Res. Bul. 471 and p. 10, Mo. Res. Bul. 473); why did the decline in the CO_2-combining capacity begin at 90°F instead of at 80°F when the ventilation rate reached the maximal level?

A second objection was that the decline is more of a verbal than factual kind, namely, that while both decline in CO_2-capacity and hyperventilation tended to occur in the cattle at the same time in a certain temperature region, yet textbooks[4] state that hyperventilation causes *alkalosis*, and decline in CO_2-

[2]Folin, O., "A theory of protein metabolism." Am. J. Physiol., *13*, 66, 1905.
[3]Talbot, J. H., et al., J. Clin. Inv., *21*, 107, 1942. Lockett, M. F., J. Physiol., *109*, 250, 1949.
[4]Peters, J. P., and Van Slyke, D. D., Quantitative Clinical Chemistry, Vol. I, 1931, ch. XVIII, especially p. 917. Bard, P., et al., "Macleod's Physiology in Modern Medicine," 1941. Wiggers, C. J., "Physiology in Health and Disease," 1949. Soskine, S., and Levine, R., "Carbohydrate Metabolism," University of Chicago Press, 1946, and Physiol. Rev. *25*, 395, 1945.

combining capacity measures increasing *acidosis*. Were the animals suffering from alkalosis or acidosis? or possibly respiratory alkalosis and fixed-base acidosis simultaneously?

The generally accepted Van Slyke theory[4] that the CO_2-combining capacity of blood measures the alkali reserve is not universally accepted. Henderson reported[5], and Shock and Hastings[6] apparently confirmed (p. 260) that when excess CO_2 is blown off in hyperventilation, the alkali ($BHCO_3$) migrates to the tissue fluids rather than excretes in the urine; and Sykes[7] suggested that while the hyperventilation in a hot environment causes *respiratory alkalosis* (reduction of the CO_2 to $BHCO_3$ ratio), it also causes a compensatory increase in $BHCO_3$ excretion and therefore leads to a lowered CO_2-combining capacity. Determination of urinary (as well as blood) changes in the major cations (Na, K, Ca, Mg) as well as anions (phosphate, sulfate, chloride, carbonate, lactate, as well as ketone bodies), together with the pH of the blood, should clarify some of these problems, especially whether or not bicarbonate and pH levels move in the same or in opposite directions in this particular case.

A second possible explanation of the decline in CO_2-combining capacity of blood at high temperatures, is that the animals refused their feed with rising temperature above 90°F, with consequent dietary carbohydrate deprivation, accelerated body fat oxidation, and therefore increased production of incompletely oxidized fragments of fat catabolism, namely acetoacetic ($CH_3.CO.CH_2.COOH$) and β-hydroxybutyric ($CH_3.CHOH.CH_2.COOH$) acids[4]. A preliminary survey of the ketone-body levels in the blood of our high-temperature and control cows, however, showed no substantial difference in ketone bodies as shown in the following table. This needs confirmation, indeed, intensive methodological study.[11]

ACETONE AND KETONE BODY DETERMINATIONS

	Acetone, mg. %		Total Ketone, mg. %	
	100°F	65°F	100°F	65°F
Brown Swiss	.72	.76	5.4	4.6
Holstein	.71	.73	5.3	4.5
Jersey	.66	.64	5.6	4.7
Brahman	.59	.52	5.4	4.6

This very complex problem is discussed in the older literature.[4,6,8] A new literature is also developing on the endocrine aspects of this problem.

The normal pH of blood is 7.4; it is below 7.4 in acidosis, and above 7.4 in alkalosis. To maintain the pH at 7.4 in the face of excess production or

[5]Henderson, Y., Physiol. Rev. *5*, 131, 1925; "Adventures in Respiration," Baltimore, 1938.
[6]Shock, N. W., and Hastings, A. B., J. Biol. Chem. *112*, 239, 1935-6.
[7]Sykes, J. F., Personal Communication, Oct. 3, 1951; also J. Dairy Sc., *24*, 193, 1941.

administration of acids or alkalies, the blood has many buffer systems[8], the most sensitive and important of which is the CO_2-$BHCO_3$ system. Hence, the $BHCO_3$ content in blood serum or plasma, measured by its capacity to combine with CO_2, is the usual measure of the degree of pH stability or alkali reserve of the blood. The acid-base balance in blood is currently[4,6] expressed in terms of three variables, CO_2 tension, $BHCO_3$ concentration, and pH. The simultaneous determination of two variables fixes the third (Henderson-Hasselbach equation). The determination of only one of these variables—as in the present case only CO_2-combining capacity, or $BHCO_3$ level—is not, therefore, sufficient for drawing conclusions about the status of the acid-base balance; we cannot explain the decline in the CO_2-combining capacity at high temperatures without further study of the pH as well as the composition of blood and urine because there may be an accelerated formation of sulfuric, phosphoric, hydrochloric, lactic and related acids associated with shift from exogenous (hay and grain) to endogenous (body protein-fat) catabolism, following the refusal of the animals to eat at high temperatures; and/or as a result of heat stress, with perhaps impairment of renal function with reduced acid or increased base elimination[9], and with other complications.

There are circumstances under which there is excessive base loss without acid production, as occurs in certain types of adrenal insufficiency[10]. It is generally known that NH_4Cl and $CaCl_2$ administration leads to acidosis by elimination of the cations and retention of HCl.

The decline in alkaline reserve could also be explained on an endocrine basis as reflecting *heat stress*, as it is known—or is assumed—to occur in *cold stress*[11], *exercise stress*[11], *dietary stress*[11], and many other forms of stress or shock. Several stresses operating simultaneously tend to reinforce each other. Starvation stress, for example, reinforces cold stress.

Stress accelerates the anterior pituitary secretion of adrenocorticotrophic hormone—presumably ACTH. The ACTH, in turn, accelerates the secretion by the adrenal cortex of its steriod hormones—one of which is presumably cortisone—thus supplying the peripheral tissues under stress with the increased needs of adrenal hormones, which control the metabolism of carbohydrate, protein, and fat, including ketone bodies[1,12]. That this stimulation is by way of the pituitary is demonstrated by injection of ACTH which accelerates the adrenal secretion in the absence of stress, as does stress itself; and by the failure of the

[8]See Henderson, L. J., "Blood, A Study in General Physiology," New Haven, 1928; "The Excretion of Acid in Health and Disease," Harvey Lectures, 1914-15, pp. 132-53, and Physiol. Rev., *5*, 131, 1925.

[9]Pitts, R. F., J. Clin. Inv., *28*, 35, 1949.

[10]Harrop, O. A., et al., J. Exp. Med., *58*, 1, and 17, 1933.

[11]Sargent, F., and Consolazio, C. F., "Stress and ketone body metabolism," Science, *113*, 631, 1951; also in: "Biologic observations during Arctic winter ration trials, 1950"; "Stress and ketone body metabolism," Report 82, Medical Nutritional Laboratory, United States Army, Feb. 1951; also Personal conference with Dr. Sargent, Oct. 10 and 11, 1951.

[12]Long, C. N. H., Cold Spring Harbor Symposia on Quantitative Biology, *5*, 344, 1937. Russell, J. A., Physiol. Rev., *18*, 1, 1938. Mirsky, I. A., Am. J. Physiol., *115*, 424, and *116*, 322, 1936.

adrenals to respond with accelerated secretion to stress after removal of the pituitary[1].

In addition to ACTH, the accelerated pituitary secretion contains other trophic hormones, and pituitary growth hormones, which, under certain conditions, are ketogenic[13]. One could, therefore, assume that the decline in alkaline reserve was produced by the increased fat catabolism as result of progressive starvation with rising temperature and by the accelerated pituitary secretions (containing ketogenic agents) in response to the heat stress.

The severe decline in alkaline reserve in our cows, developed under the *laboratory* stress of heat and starvation, was not accompanied by serious decline in blood sugar. Figures 2, 3b, 5, and 8 show that the blood-sugar level of the cows at 100°F was about 50 mg% as compared to about 60 mg% at 40° to 50°F. This contrasts sharply with *clinical* cases of ketosis described by Shaw[14], in which the blood sugar level was down to 23 mg% with blood ketone bodies up to 65 mg%. Shaw[14] reported that intramuscular injection of 1.5 and 1.0 grams of cortisone eight hours apart elevated the blood-sugar level to 53 mg% and reduced the ketone-body level to 2 mg%. This is good proof—provided there were adequate controls of similar cows similarly injected with saline in place of cortisone—of the importance of the pituitary-adrenal system in this syndrom.

Administration of cortisone apparently: (a) elevated the blood sugar (gluconeogensis is accelerated by injection of adrenal cortex extracts); (b) depressed the pituitary ketogenic hormone production (adrenal hormones depresses ACTH production).

Summarizing, the 50 per cent decline in the carbon dioxide-combining capacity of the blood of our cows at high temperatures may be due in part to accumulation of ketone bodies following severe decline in feed consumption due to loss in appetite[15]; and in part to accelerated pituitary secretion (containing ketogenic agents) in response to heat stress. It could, however, also be due to direct loss of bicarbonate associated with adrenal insufficiency in the late stages of the stress[10].

C. Cholesterol and Ascorbic Acid: A striking feature of the adrenals is their richness in cholesterol (mostly in ester form) and ascorbic acid (4 and 0.4 gm per 100 gm gland, respectively) and their rapid decline under stress[1]. The decline may be explained by the fact that cholesterol is definitely an adrenal-hormone precursor, and ascorbic acid a possible precursor, or factor, involved in adrenal-hormone synthesis. It was suggested[16] that there is an ascorbic

[13]Bennett, L. L., et al., Am. J. Physiol., *152*, 20, and *155*, 18, 1948, and J. Clin. Endocr., *9*, 675, 1949. White, A., Recent Progress in Hormone Research, *4*, 153, 1949. Hartman, F. A., et al., Endocr., *41*, 213, 1947.

[14]Shaw, J. C., Hatzialos, B. C., and Chung, A. C., J. Dairy Sc., *34*, 498, (June) 1951.

[15]Brobeck, J. R., "Food intake as mechanism of temperature regulation." Yale J. Biol. & Med., *20*, 545, 1948; Ann. Rev. Physiol., *10*, 315, 1948. Johnson, R. E., and Marks, R. M., Science, *105*, 378, 1947.

[16]Lowenstein, B., and Zwemer, R. L., Endocrinology, *39*, 63, 1946.

acid conjugated compound with adrenocortical activity, but this had not been confirmed[1, 17].

The decline, by over 50 per cent, in blood ascorbic acid (Figure 2) and in cholesterol (p. 34, Mo. Res. Bul. 433) with rising temperature above thermoneutrality may be logically explained in terms of the above facts. Stress accelerates adrenal cortex secretion; with corresponding depletion of the secretion precursors, cholesterol and ascorbic acid. To test this explanation, it seems necessary to compare the steroid and ascorbic acid excretions[18] with those in blood. The irregularities in the ascorbic acid curves (Figure 2) may perhaps be interpreted in terms of Selye's concept of "alarm reaction" and the complex sequence in the "general adaptation syndrom"[19]. Incidentally, the Selye concept is an extension, on the adrenal cortex level, of Cannon's "emergency theory," and on the adrenal medulla level, of "homeostasis"[20]; and the supposed physiological dividing line between the adrenal cortex and medulla seem to be disappearing[1, 21].

D. Lymphocytes and Eosinophils: A considerable literature[21] indicates that stress, or ACTH injection, which increases adrenal cortex secretion (particularly sugar-active 11-oxygenated corticosteroids), tends to atrophy the lymphatic organs (thymus, lymph glands) with consequent decline in the lymphocyte level in the blood.

Accordingly, counts were made of lymphocytes, eosinophils, and leucocytes. The trends of the *absolute* counts—Figures 2, 4a, 7, and 9—show no very decisive decline. The complicated sequence in Selye's[19] "general adaptation syndrom" may perhaps explain the failure in this case of slow temperature rise to develop the expected decline in eosinophils. A similar situation was observed in the complex sequence in the ascorbic acid curve.

Summarizing, it appears (Figure 4a) that the eosinophils tend to decrease with rising temperature above 60°F, and the lymphocytes to increase, but not significantly. These trends should properly be compared with excretion trends of nitrogen, ketosteroids, and ascorbic acid.

E. Protein-Bound Iodine (PBI): The thyroid gland is the major metabolic regulator as shown by the near halving of "basal" heat production following thyroid removal, and the near doubling it following suitable thyroxine

[17]Vogt, M., J. Physiol., *104*, 60, 1945.

[18]For measuring urinary ketosteroids, see, among others, Pincus, G., Recent Progress in Hormone Research, *1*, 123, 1947. Liberman, S., and Dobriner, K., Id., *3*, 71, 1948. Dougherty, W. H., et al., J. Clin. Endocr., *8*, 166, 1948; Hioco, D., Id., *10*, 1570, 1950; Lloyd, C. W., Id., p. 1559. Mason, H. L., Id., *11*, 743, 1951.

[19]Selye, H., "The alarm reaction." J. Canadian Med. Assn., *34*, 706, 1936; "The general adaptation syndrom and the disease of adaptation." J. Clin. Endocr., *6*, 117, 1946; also in his book on "Stress," Montreal, 1950. White, I. G., J. Endocr., *7*, 143, 1951.

[20]Cannon, W. B., Shohl, A. T., and Wright, W. S., Am. J. Physiol., *29*, 280, 1911. Cannon and Britton, S. W., Id., *79*, 433, 1926. Cannon, "Bodily Changes in Pain, Hunger, Fear, and Rage," Appleton, 1929, and "The Wisdom of the Body," 1932.

[21]Dougherty, T. F., and White, G., Endocrinology, *35*, 1, 1944, and *39*, 370, 1946; also J. Lab. Clin. Med., *32*, 584, 1947. Forsham, P. H., et al., J. Clin. Endocr., *8*, 15, 1948. Thorn, J. A., and Forsham, P. H., Recent Progress in Hormone Research, *4*, 275, 1949.

administration. How is thyroid activity affected by changing temperature?

A considerable literature[22] indicates adaptative functional changes of the thyroid to *cold* (temperatures below thermoneutrality), although "basal metabolism," generally used as index of thyroid function, could reflect increased neuro-muscular tension, appetite[15], and adrenal activity[11, 23], as well as thyroid activity.

The evidence of adaptive changes of thyroid function to *heat*[24] (temperatures above thermoneutrality), as measured by "basal metabolism," is equally weak because changes in basal metabolism with increasing temperature may reflect homeostatic changes in nutritional condition,[15] neuro-muscular tension, adrenal activity[11], as well as in thyroid function. For instance, Mason's[25] observation of a 5 per cent lower basal metabolism in a group of women in tropical India than in temperate Europe may reflect reduced appetite and reduced neuro-muscular tension and exercise associated with tropical climate. Besides, a 5 per cent change in basal metabolism is within the rights of experimental error of such data.

The "basal metabolism" of our cows was not measured. The "resting heat production," however, was measured (Mo. Res. Buls. 435, 450, 464, 473), and, surprisingly, it declined not only with rising environmental temperature above 75°F, but also with rising rectal temperature, from the normal of 101°F to high fever temperature of 105° to 107°F, which, according to the van't Hoff rule and the observations by Du Bois[26] on man, should have greatly increased the heat production. The decline in heat production in our cows with slow increasing temperature above thermoneutrality was attributed to (a) decline in feed consumption which eliminated its heat increment (SDA); (b) decline in milk production which eliminated its heat increment; (c) possible decline in thyroid activity—as *time was available for acclimatization*—a decline here under question.

How shall one measure changing thyroid function in cattle with slowly increasing temperature? Heat production—"metabolism"—cannot be taken as index of thyroid function, since the observed decline in metabolism with increasing rectal temperature is the question under consideration. It is generally believed that the cholesterol level increases with decreasing thyroid activity, whereas here (p. 34, Mo. Res. Bul. 433) it decreased with rising temperature. Moreover, cholesterol cannot be taken as index of metabolism associated with thyroid function, because as explained above (section 4C), decline in cholesterol is apparently associated with increased adrenal activity due to increasing heat

[22]Ring, G. C., Am. J. Physiol., *116*, 129, 1936; *125*, 244, 1939; *137*, 582, 1942. Saller, E. A., and You, S. S., Id., *163*, 81, 1950.

[23]Cramer, W., "Fever, Heat Regulation, Climate, and Adrenal Apparatus." Longmans, Green & Co., London, 1928.

[24]MacGregor, R. G. S., and Loh, G. L., J. Physiol., *99*, 496, 1941.

[25]Mason, E. D., J. Nutr., *8*, 695, 1936.

[26]Du Bois, E. F., "Fever and the Regulation of Body Temperature." C. C. Thomas, 1948, pp. 46 and 53.

stress. The thyroxine-thiouracil method of Dempsey and Astwood[27] cannot be used because it involves sacrificing the animal and weighing the thyroids. Moreover, the users of the thyroxine-thiouracil method for estimating the rate of thyroxine secretion[27, 28] make the unverified assumption that the thyroxine required to block the characteristic goiter development following thiouracil ingestion equals the normal rate of thyroxine production. For instance, Dempsey and Astwood[27] observed that at 35°C (95°F) it required 1.7 mcg* of daily thyroxine administration to prevent the development of the goiter in their 100-gram rats receiving 0.1 per cent thiouracil in the drinking water; at 25°C (77°F), it required 5.2 mcg and at 1°C (34°F), it required 9.5 mcg thyroxine to block the goiter formation. Dempsey and Astwood, therefore, assumed that these values were quantitatively equivalent to the daily rates of thyroxine production by the normal thyroid gland at 35°, 25° and 1°C. Can this assumption be accepted without independent verification?

This attempt to estimate the PBI in blood plasma is based on reports[29] that most of the thyroxine released from the thyroid is in the circulating blood plasma in the form of protein complexes, therefore precipitated by protein-precipitating reagents, such as zinc hydroxide, tungstic acid, and trichloroacetic acid. Little of the organic iodine compounds is excreted in the urine[30]. At any rate plasma PBI level is being widely used as index of thyroid activity in man[31], and we thought that we should try it on cows.

Before proceeding with the assay of PBI in cow blood, trial assays were made on three groups of 240-gram female rats, kept for two weeks at 34°, 25° and 7-10°C. Some animals at the high temperature died and insufficient blood was obtained from some animals to make satisfactory analyses. The following determinations of PBI, in mcg per 100 ml were obtained:

Temperature, °C	PBI
7-10	10.0, 7.8, 9.0, 2.9
25	2.6, 3.8, 3.3, 7.0, 2.9
34	1.4, 17.9

[27]Dempsey, E. W., and Astwood, E. B., Endocrinology, *32*, 509, 1943.

[28]Monroe, R. A., and Turner, C. W., Univ. Missouri Agric. Exp. Sta. Res. Bul. 403, 1946; Hurst, V., and Turner, C. W., Id., Res. Bul. 417, 1948; Mixner, J. P., Reineke, E. P., and Turner, C. W., Endocrinology, *34*, 168, 1944. Hoffman, N. E., and Shaffner, C. W., Poultry Science, *29*, 365, 1950.

*Mcg = microgram = μ g = γ = gamma = .001 mg.

[29]Perlman, I., Morton, M. E., and Chaikoff, I. L., J. Biol. Chem., *139*, 449, 1941. Taurog, A., and Chaikoff, I. L., Id., *176*, 639, 1948; *184*, 99, 1950; and Ann. N. Y. Ac. Sc., *50*, 377, 1949. Gross, J., Leblond, C. P., Franklin, A. E., and Quastel, J. H., Science, *111*, 605, 1950. Chaikoff, in: "Symposium on the Use of Isotopes in Biology and Medicine." Univ. Wis. Press, 1948. Johnson, H. W., and Albert, A., Endocrinology, *48*, 669, 1951. Dougherty, J., Gross, I., and Leblond, C. P., Id., pp. 700 and 714. Robbins, J., et al., J. Clin. Endocr., *11*, 759, 1951.

[30]Rall, J. E., J. Clin. Endocr., *10*, 996, 1950.

[31]Salter, W. T., et al., Am. J. Med. Sc., *202*, 527, 1941. Bassett, A. M., et al., Id., *202*, 516, 1941. Man, E. B., et al., J. Clin. Inv., *21*, 773, 1942, and *25*, 404, 1946. Riggs, D. S., et. al., Id., *20*, 345, 1941, and *24*, 722, 1945. Lowenstein, B. E., et al., J. Clin. Endocr., *4*, 268, 1944. Talbot, N. B., et al., J. Biol. Chem., *153*, 479, 1944. Swenson, R. E., and Curtis, G. M., J. Clin. Endocr., *8*, 934, 1948. Perry, W. F., Canad. M. A. J., *60*, 602, 1949. Soisalo, P., Acta med. Scandinav., *133*, 186, 1949. Brody, E. B., and Man, E. B., Am. J. Psychiatry, *107*, 357, 1950.

Some values are erratic but the mean for 25°C agrees well with Barker's[32] normal value for rat plasma. There is a fairly clear contrast between the groups at 7-10° and 25°C; the means being 7.4 and 3.9 mcg respectively. This relationship may be compared with the estimates of thyroxine secretion rate per 100 gm per day by Dempsey and Astwood[27] which were 9.5 and 5.2 mcg d,l-thyroxine at 1°C and 25°C. Little account can be taken of the scanty, erratic values at 34°C.

The following data were obtained on cows during the course of rising temperature in Period 2. Each value is the mean of either duplicate or triplicate determinations.

Microgram Protein-Bound Iodine per 100 ml Plasma

Cow	50°F (10°C)	75°F (24°C)	95°F (35°C)
Jersey:			
502	4.1	3.4	3.3
508	3.7	3.4	2.7
933	3.5	2.7	2.7
Holstein:			
136	4.1	2.8	3.0
109	3.6	3.5	3.3
14	5.6	4.3	4.1

On the whole the values declined fairly consistently with increasing temperature from 50°, through 75°, to 95°F. Analysis of variance showed that the differences between temperatures, and also the differences between cows, were significant, both *F* values being more than twice the values needed for P equals 0.01.

Determinations of plasma of a cow receiving 15 gm of iodinated protein ("thyroprotein") a day, gave 19.0 and 19.6 mcg per cent PBI. These interesting values cannot, of course, be compared with those of normal cows without experimental calibration.

Employing the same technique, Mrs. Burge took over the analyses of cow's blood after Mr. Turner left. The Burge data are plotted in Figures 2, 3b, and 8. All one can say at this time is that the normal PBI level in cows is of the same order as in normal man[31], 3 to 8 mcg per cent (in hyperthyroid man and in thyrotoxicosis it is 6 to 34 mcg %, and in hypothyroid and myxedema, 0.3 to 2 mcg %).

Barker[32] gives the following average values in mcg %:

Normal man, 7
Normal rat, 4
Normal dog, 2.5
Thyroxine-treated rat, 18
Thiouracil-treated rat, 1

[32]Barker, S. B., J. Biol. Chem., *173*, 715, 1948; Thomas, J. W., Shinn, L. A., Wiseman, H. G., and Moore, L. A., Analytical Chemistry, *22*, 726, 1950.

Summarizing, the range in protein-bound iodine in these cows was between 3 and 7 mcg %. The temperature trend in PBI was relatively slight; in yearling heifers it declined from 5 mcg % at 40°F to 4 mcg % at 100°F, then rose to 7 mcg % on sudden decline in temperature to 40°F (Figure 3b). There was no significant difference between the Brown Swiss and Brahman heifers. The temperature change in plasma PBI in the cows was much less (Figures 2, 3b, and 8); it fluctuated between 3 and 7 mcg %; some animals showed temperature trends, others not. The temperature trend in Mr. Turner's values, from 4.1 mcg % at 50° through 3.35 at 75°F and down to 3.2 at 95°F, are consistent but slight by comparison with the temperature trends by Dempsey and Astwood for rats[27].

F. Radioiodine*: As the PBI level did not change decisively with changing temperature, an attempt is being made to determine whether the rate of iodine turn-over, labelled with radioiodine, I^{131}, would show significant changes with changing temperature.

The use of radioiodine as diagnostic index of thyroid activity in man began[33] almost immediately after the Hiroshima event when it was announced in 1946 that radioisotopes produced by the chain-reaction pile-production method[34] would become available for biological use.

The radioiodine diagnostic method for thyroid function is considered by some[35] to be the most reliable for man and may turn out to be so for livestock. In addition to our immediate objective of measuring thyroid activity as function of temperature, it would be instructive to measure thyroid activity as function of milk production because, largely as result of the enthusiastic researches of C. W. Turner and W. R. Graham, Jr.[36], the degree of thyroid activity is believed to be functionally related to the level of milk yield. If this be so, then a measure of thyroid activity is also a partial measure of lactational capacity, and this method—administering I^{131} by mouth and monitoring the thyroid—may turn out to be the long-sought method for estimating lactational potentiality.

[33]Hertz, S., Roberts, A., and Evans, R. D., Proc. Soc. Exp. Biol. & Med., *38*, 510, 1938; also J. Am. Med. Assoc., *131*, 81, 1946. Hamilton, J. G., Am. J. Physiol., *124*, 667, 1938. Hamilton, and Soley, M. H., Id., *127*, 557, 1939, and J. Dis. Child., *66*, 495, 1943.

*The I^{131} here used was obtained from the Oak Ridge National Laboratories on allocation by the U. S. Atomic Energy Commission.

[34]Fermi, Enrico, "Theory of chain-reaction pile." Nature, *133*, 747, 1934, and Science, *105*, 27, 1947.

[35]Jaffe, H. L., and Ottoman, R. E., J. Am. Med. Assn., *143*, 515, 1950, give a lucid exposition of the clinical methods on man. Seidlin, S. M., Recent Progress in Hormone Research, *4*, 483, 1949, gives instructive time curves of radioiodine uptake in normal, hypo- and hyperthyroid man.

[36]For the literature, see "Iodinated Protein Bibliography," Cerophyl Laboratories, 2438 Broadway, Kansas City, Missouri, 1951.

Radioiodine is used clinically for diagnosing thyroid activity in *man* in three ways:

(1) Measuring the rate of radioiodine uptake by the thyroid with an externally-placed Geiger-Mueller (G-M) tube after administering a given dose, about 100 microcurie, of I^{131}; correcting for background count; and comparing the corrected reading with the count obtained over a 100 microcurie "standard dose" of I^{131} placed in the same geometrical position and distance from the G-M tube as the thyroid[35, 37]. The percentage of the administered dose taken up by the thyroid is taken as index of the rate of thyroid activity. Normally, somewhat less than a third of the administered radioiodine dose is taken up by the thyroid, the remaining is excreted, mostly in the urine.

(2) Measuring the urinary excretion of I^{131}. Hamilton and Soley[33] used this as a supplementary method to (1) above, as did others[38].

(3) Measuring the rate of release from the thyroid into the blood of the administered radioiodine converted to protein-bound iodine in the form of organic compounds, the PBI discussed in the preceding section.

It would, naturally, be best to follow the fate of the radioiodine by all of the above three methods[39].

Because of the involved difficulties, including dosage determination for cattle, construction of G-M tube suitable for cattle, arrangement of the laboratory schedule for different purposes than for radioiodine or thyroid studies, this exploratory work with I^{131}, now in its second year, has been developing too slowly to enable presentation of definite data at this time. Since, however, these bulletins are meant to be only progress reports it seems appropriate to present the following preliminary data on the effect of ambient temperature on rabbits and cattle.

Twenty-four hours after injection of a tracer dose of NaI^{131} two categories of I^{131} were measured: (1) the amount in the thyroid and (2) the amount in blood plasma. The I^{131} in blood plasma was divided into protein-bound I^{131} (PBI^{131}), and total I^{131}. The absolute values as well as the ratios of PBI^{131} to total I^{131} in the plasma are given in the following table. As this "conversion ratio" is significantly lower at the higher temperatures in both rabbits and cows, it is tentatively concluded that increasing temperature reduces significantly thyroid activity.

[37]Seley, M. H., and Miller, E. R., Med. Clin. N. America, *3*, 17, 1948.

[38]Keating, F. R., Jr., et al., J. Clin. Inv., *26*, 1138, 1947. Berkson, J., et al., J. App. Physiol., *2*, 522, 1950. Gordon, E. S., and Albright, E. C., J. Am. Med. Assn., *143*, 1129, 1950. McArthur, J. W., Rawson, R. W., and Means, J. H., Ann. Internal. Med., *29*, 229, 1948.

[39]Keating, F. R., Jr., and Albert, A., Recent Progress in Hormone Research, *4*, 429, 1949.

RADIOIODINE DETERMINATIONS
24 HOURS AFTER INJECTION OF TRACER DOSE OF I^{131}

1. Thyroid I^{131}

G-M Counts per Minute Over Thyroid

White Rabbits			Cows*		
Animal	46-50°F	88-95°F	Animal	65°F	95-100°F
1[h]	2300	1400	Jersey		
2[h]	2000	2500	549	----	370
3[c]	3100	3100	559	----	480
4[c]	3000	2600	999	----	400
			Holstein		
			317	1500	710
			314	2900	1200

2. Blood Plasma I^{131}

White Rabbits

	dps/ml plasma				Ratio	
	PBI^{131}		Total I^{131}		PBI^{131}/Total I^{131}	
Animal	46-50°F	88-95°F	46-50°F	88-95°F	46-50°F	88-95°F
1[h]	6	35	25	760	0.24	0.05
2[h]	16	14	59	540	0.27	0.03
3[c]	15	16	213	340	0.07	0.05
4[c]	14	29	160	510	0.09	0.06

Cows

	dps/ml plasma/rd. injected				Ratio	
	PBI^{131}		Total I^{131}		PBI^{131}/Total I^{131}	
Animal	65°F	95-100°F	64°F	95-100°F	65°F	95-100°F
Jersey						
549	32.7	2.16	34.9	157	0.94	0.01
559	34.7	5.38	40.9	---	1.18	----
999	----	5.39	33.0	167	----	0.03
Holstein						
317	7.9	16.0	11.3	131	0.70	0.12
314	30.0	14.7	47.6	407	0.63	0.04

[h] Subjected first to high temperature.

[c] Subjected first to low temperature.

* Corrected for variation in dosage and instrumental sensitivity.

G. Water Balance, Plasma Protein, Electrolytes, Glucose, Erythrocytes: The water and, therefore, the solids concentration—composed largely of plasma protein, electrolytes, erythrocytes—in blood with rising temperature above

thermoneutrality appear to be dependent on two factors: (a) the moisture loss from the body surface by sweating and related processes in comparison to moisture gain; (b) the activity of the pituitary-adrenal system under the influence of changing heat stress.

As the sweating rate in man increases exponentially[40] with increasing environmental temperature above 80°F, the water absorption tends to fall behind the water loss from the body surface, with resulting tendency to rise in concentration of the blood constituents[41]. The evaporation curve of cattle, on the other hand, appears to flatten out above 80°F (Res. Buls. 451, 461, and 479), and so does not show the tendency for hemoconcentration.

At any rate, the major colloid, electrolyte, and non-electrolyte regulators of osmotic pressure and water balance were not seriously affected by temperature change; hence, the tentative conclusion that, unlike in man[41], the water balance in cattle is not affected by changing temperature, 0° to 100°F (−18° to 38°C), and is not a factor in their heat stress as it appears to be in man.

In addition to sweating, the concentration of the aforementioned constituents—and many others—are under the influence of the endocrine system, which are in turn under the influence of many categories of stress—including (to cite a dramatic example) the apparently harmless emotional "stress" of watching a football game (emotional glucosuria)[26]. The sugar level in blood is affected not only by available insulin, but also by[42, 20, 12] most adrenal hormones, particularly C-11-oxygenated steroids.

Somewhat similar statements could be made about the effect of other endocrines. For instance[43], fasting may stimulate the pituitary to secrete its many hormones, including thyrotrophic, adrenotrophic, and growth (hormone). The growth hormone may lead to growth of some organs at the expense of others, as illustrated by the dramatic growth of the gonads of the salmon at the expense of his muscles during his stressful pilgrimage on an empty stomach to the spawning waters. Under such rapid use of body protein for anabolic (growth) purposes, and in the absence of external food, the energy is necessarily obtained from body fat. While, therefore, as aforenoted, starvation stress reduces lymphoid tissue, yet following the first 48 hours of fast the increase in pituitary growth hormone secretion may actually increase the growth of lymphatic tissues and so increase the lymphocyte count. The intensity of nitrogen metabolism in stress under endocrine influence is determined by the effect of the stress on the endocrines—on the pituitary which determines the rates of secretion of these various hormones, particularly adrenal-cortical steroid, and thyroid.

[40]See chart on page 278 in Brody's "Bioenergetics and Growth," Reinhold, 1945.
[41]Adolph, E. F., and associates, "Physiology of Man in the Desert," New York, 1947.
[42]Cf., Ingle, D. J., Recent Progress in Hormone Research, *2*, 229, 1948.
[43]Cf., White, A., Id., *4*, 153, 1949.

Or, consider the fabulous effects of the adrenal cortex[44] on blood composition. By way of its desoxy-compounds (especially the "sodium retaining", 11-desoxy-corticosterone), it regulates the metabolism of electrolytes—especially sodium, potassium, and therefore, water. By way of its 11-oxysteroids (especially 11, 17-oxysteroids and to a less extent 11-oxysteroids), it regulates the metabolism of carbohydrate, protein (gluconeogenesis), and fat; by its effect on the thymus, affects the eosinophil count. By way of its androgens and estrogens, it regulates sex activity and sex characteristics, including sex differences in metabolic rates.

Stresses may also affect the blood composition by way of the coenzymes and enzymes of the metabolic systems, including, as far as known, cocarboxylase, diphosphopyridine, adenine dinucleolides[45]. Since enzymes contain vitamins, minerals, and proteins, the fasting or malnutrition associated with stress may reduce the levels of essential enzymes; and, on the other hand, heavy administration of vitamins, excellent proteins, and essential minerals, may help maintain the enzyme level and so ameliorate the effects of stress.

Summarizing, unlike man, cattle maintain a fairly normal water, colloid, electrolyte, and sugar balance at high temperatures. The species difference between man and cattle in sweating rate with increasing temperature seems the major factor in the species difference in water balance; although many other complicated interrelations, including hormones, enzymes, and change in the nature of the metabolic mix, are involved in maintaining this shifting balance with changing environmental conditions.

5. DISCUSSION AND SUMMARY

The unique feature of the design of this research is that the environmental temperature remained constant one to two weeks at each temperature level before changing to another temperature, thus simulating somewhat seasonal temperature changes and allowing time for acclimatization (or deterioration). Under these conditions no obvious changes in blood composition occurred between 0° and 65°F (–18° and 18°C), except possibly increase in glucose level at the lower temperatures.

On raising the temperature above 65°F, changes in some blood constituents appeared; the temperature of change varied with the productive level, size (specific surface area), and breed.

The most dramatic changes on raising the temperature from 65° to 100°F occurred in creatinine (increased over 100 per cent); carbon dioxide-combining capacity, ascorbic acid, cholesterol (all reduced to less than half the level at 50°F). These changes were interpreted as reflecting reduction, or refusal, of food with increasing temperature, and reaction of the pituitary-adrenal system to heat and hunger stress.

[44]Cf., Thorne, G. W., and Forsham, P. H., Id., *4*, 229, 1949.

[45]Beecher, H. K., J. Am. Med. Assn., *145*, 193, 1951; Nutrition Reviews, *9*, 204, 1951. Green, H. N., and Stoner, H. B., "Biological Actions of the Adenine Nucleotides." London, H. K. Lewis, 1950.

Unlike in man, there were no apparent disturbances in water, electrolyte, and colloid concentration on increasing environmental temperature from 65° to 100°F. This species difference in hemoconcentration is attributed to species differences in evaporative losses which did not increase in cattle above about 80°F, whereas in man they increase exponentially, almost indefinitely, with increasing environmental temperature so that the moisture absorption cannot keep pace with evaporative moisture loss.

The trends in plasma protein-bound iodine with changing temperature were too uncertain to permit interpretations in their bearing on thyroid activity.

The data on radioiodine in their bearing on the effect of temperature on thyroid activity are as yet too few for discussion.

The blood ascorbic acid and glucose levels were significantly higher in yearling heifers than in cows (Figure 3b). The blood creatine declined with increasing temperature above 80°F in the heifers, but not in the cows. While the creatinine increased over 100 per cent with rising temperature above 80°F in cows, it increased slightly in the yearling heifers. There were no striking blood composition differences between the Indian-evolved (Brahman) and European-evolved cows in their response to changing temperature. The hematocrit, erythrocyte, and hemoglobin values, however, tended to be higher in the Brahman cows.

The work here reported will be of value chiefly as orientation for future, more precise, investigations on the effect of slowly changing seasonal changes from 0° to 105°F on the blood composition of cattle and related slightly sweating species.

6. APPENDIX

This section contains outlines on analytic methods, literature references, tables, and charts.

A. Analytic Methods and Literature References: Missouri Research Bulletin 433 presented data on calcium, carbon dioxide-combining capacity, catalase, cholesterol, creatinine, fatty acids, glucose, hematology (erythrocytes, leucocytes, hematocrit, hemoglobin, lymphocytes, eosinophils), magnesium, non-protein nitrogen (NPN), inorganic phosphorus, and plasma protein, as function of rising temperature, 50° to 100°F, with a list of references on methods and interpretations.

In addition to these constituents, this bulletin presents data on protein-bound iodine, radioiodine, ascorbic acid, sodium and potassium, amino acid, uric acid, and ketone bodies. Notes on the analytic methods with literature references are here given for the data not presented in Research Bulletin 433.

*Protein-bound Iodine** (see footnote on p. 3): In the main, Barker's pro-

*Grateful acknowledgments are made to J. W. Thomas who, at the suggestion of J. F. Sykes (both of the Bureau of Dairy Industry, U. S. Dept. of Agric.), sent us in August 1948 the diagram of his distillation apparatus and for helpful suggestions; also to Drs. Evelyn B. Mann, A. L. Chaney, and I. L. Chaikoff for helpful suggestions on this problem and for reprints.

cedure[32] was followed but with the reflux distillation apparatus and technique by J. W. Thomas[32], in turn based on preceding methods[46]. Somogyi's $Zn(OH)_2$ protein-precipitation method was used[47]. This precipitate is said to contain virtually all the hormonal and very little of the non-hormonal iodine.

The procedure, in brief, was: (1) precipitation and washing plasma protein; (2) digesting precipitate with chromic-sulfuric acids; (3) reducing iodide with phosphorus acid and distilling; (4) colorimetric determination of iodine by catalysis of iodine on the reduction of ceric sulfate by arsenious acid. In triplicate determinations, variations were usually in the order of 10 per cent but sometimes as high as 20 per cent. This error was contributed to by a high blank value and the difficulty of completely avoiding contamination, as Nessler's reagent was prepared and used on the same floor. The mean of recoveries of 0.1 mcg added iodine was 85 per cent. Such details of procedure as were developed are not given as new methods are now being developed. For instance, a greatly simplified new method[48] was just published. It does not involve distillation, and only 0.5 ml serum is used. It yielded 5.3 mcg PBI per 100 ml pooled human blood serum, with the usual range of 4 to 8 mcg.

Radioactive Iodine: The radioactivity measurements on the blood plasma and its derivatives were made with a 1-3 mg/cm^2 mica and window Geiger-Muller tube and conventional scaler. The measurements on the animals were made with the counters described below and an integrating rate meter. All calibrations were made with I^{131} preparations standardized against National Bureau of Standards I^{131} beta-ray standards*. The isotonic NaI^{131} solution was adjusted to pH8 with acetate buffer.

Rabbits: 0.05-0.3 rd† I^{131} per kilogram body weight was injected into the marginal vein of one ear, and blood samples taken from the marginal vein of the other ear. The protein from one ml. of plasma was precipitated with 10 per cent trichloroacetic acid; the precipitate washed three times with trichloroacetic acid, dissolved in 0.5 ml of 2N sodium hydroxide, and radioactivity determined to give the PBI^{131}. The total plasma I^{131} was determined by counting one ml of plasma. For I^{131} in the thyroid (of the living animal) counts were made over the thyroid gland using an end window G-M counter shielded by one-half inch of lead for columnation and a 1/16-inch aluminum beta-ray shield over the window. This was held in contact with the rabbit's neck and positioned so as to give the maximum count rate.

[46]Chaney, A. L., Ind. Eng. Chem. Anal. Ed., *12*, 179, 1940. Taurog and Chaikoff, J. Biol. Chem., *163*, 313, 1946. Connor, A. C., et al., Surgery, *25*, 510, 1949.

[47]Somogyi, M., J. Biol. Chem., *86*, 655, 1930.

[48]Salter, W. T., and Rosenblum, I., J. Endocr., 7, 180, June 1951.

*Acknowledgments are made to the Radioactivity Section, National Bureau of Standards, for these standards.

†The "rd" stands for *rutherford*, a unit of disintegration rate of radio-isotopes. The other unit is the *curie*. A rutherford represents 1 million (10^6) and a curie 37 million (3.7×10^{10}) disintegrations per second. Therefore, one mc (millicurie) equals to 37 rd.

Cows: 1-4 rd I^{131} was injected into one jugular vein, and blood samples were taken from the other jugular vein 24 and 96 hours after injection. Total plasma I^{131} was determined as for rabbits. The PBI^{131} was determined as for PBI^{131} by the Thomas method previously discussed, except that the distillate was dried in a defined area of a copper plate for I^{131} measurement. The I^{131} in the thyroid of the living cow was estimated by measuring the count rate with a 1 x 12 inch copper wall G-M counter 10 inches from the skin over the thyroid gland.

Sodium and Potassium: These constituents were determined in whole blood and plasma by direct flame spectrophotometry. The blood or plasma was diluted with glass-distilled water to give approximately a 1 mg % solution of the ion in question (1:250 for sodium, 1:10 for plasma potassium and 1:100 for whole blood potassium). These solutions were then analyzed with the Beckman Flame Spectrophotometer, sodium at 5890 Å (27 Å band width) and potassium at 7665 Å (48 Å band width). Using this technique the mean recovery of added sodium was 99.6 ±1.4% and the mean difference between duplicate readings was 1.9 ± 0.53%. Iron, calcium or magnesium did not interfere when present in amounts up to 100 times those normally present in blood as was the case with sodium or potassium in the determination of the other.

Uric Acid was determined by the method of Newton as given by Hawk[49]. Interfering materials were removed by acid silver chloride precipitation and the uric acid determined through its reaction with a lithium arsenotungstate reagent in the presence of cyanide.

Amino Acids were determined by the reaction of the alpha-amino group with beta-naphtholsulfonic acid in alkaline solution[49].

Creatine was converted to creatinine by autoclaving an acid solution of whole blood filtrate, the creatinine was then determined as described in Research Bulletin 433.

Urea was determined by the reaction with diacetyl monoxime as described by Barker[50].

Ascorbic Acid was determined as its 2,4-dinitrophenylhydrazone[51].

Acetone and Total Ketone Bodies were determined through their reaction with 2,4-dinitrophenylhydrazine as described by Greenberg and Lister[52].

[49]Hawk, P. B., Oser, B. L., and Summerson, W. H., "Practical Physiological Chemistry," Philadelphia, Blakiston, 1947.

[50]Barker, S. B., J. Biol. Chem., *152*, 453, 1943.

[51]Johnson, B. C., "Methods of Vitamin Determination," Minneapolis, Burgess Pub. Co., 1948.

[52]Greenberg, L. A., and Lister, D., J. Biol. Chem., *154*, 177, 1944.

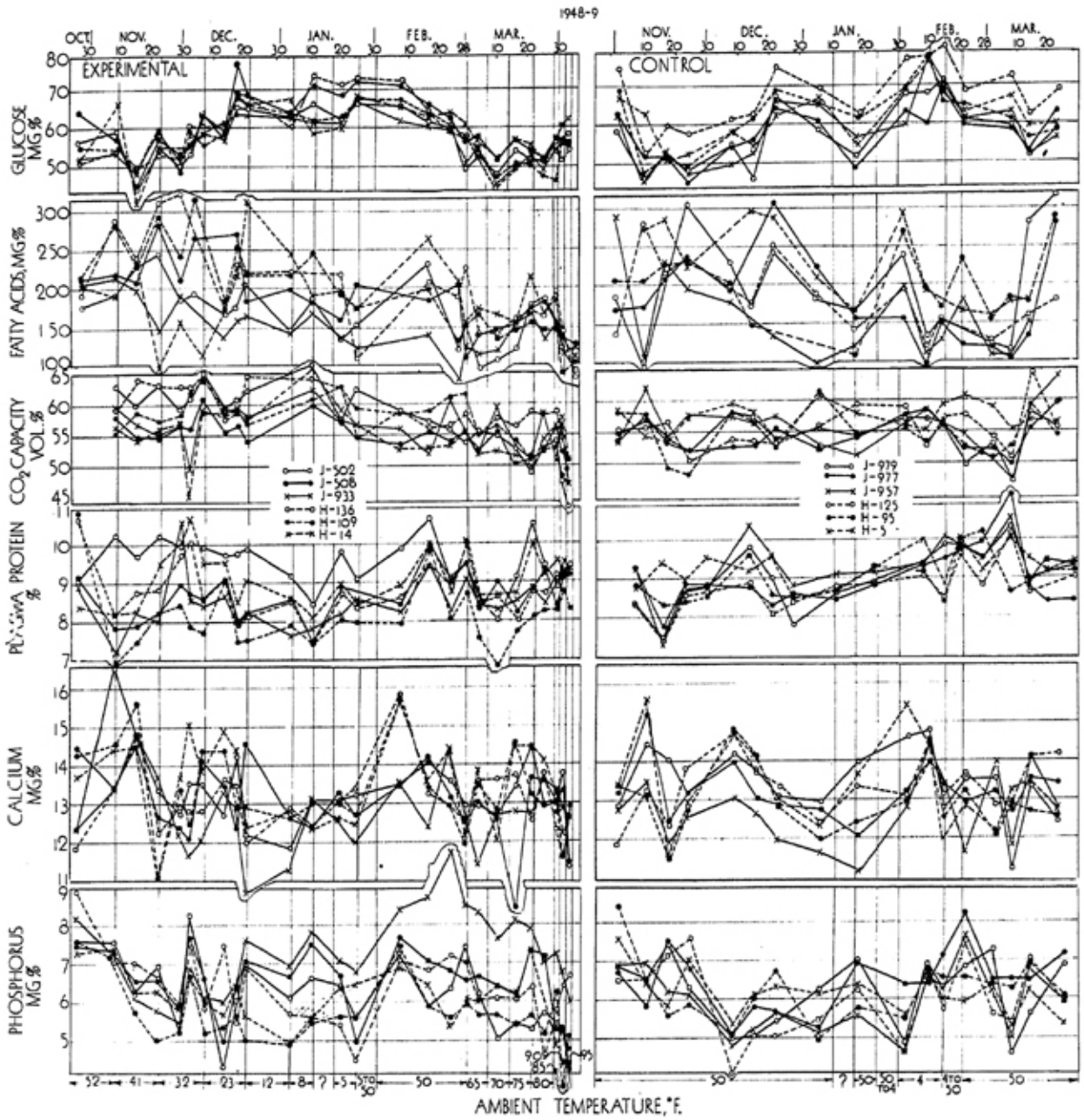


Fig. 5—Left side shows effect of declining, 50° to 8°F, and rising, 50° to 95°F, temperature on blood constituents for Jersey (J) and Holstein (H) cows during Period 2; right side shows effect of time on Control cows maintained at a temperature of 50°F throughout.

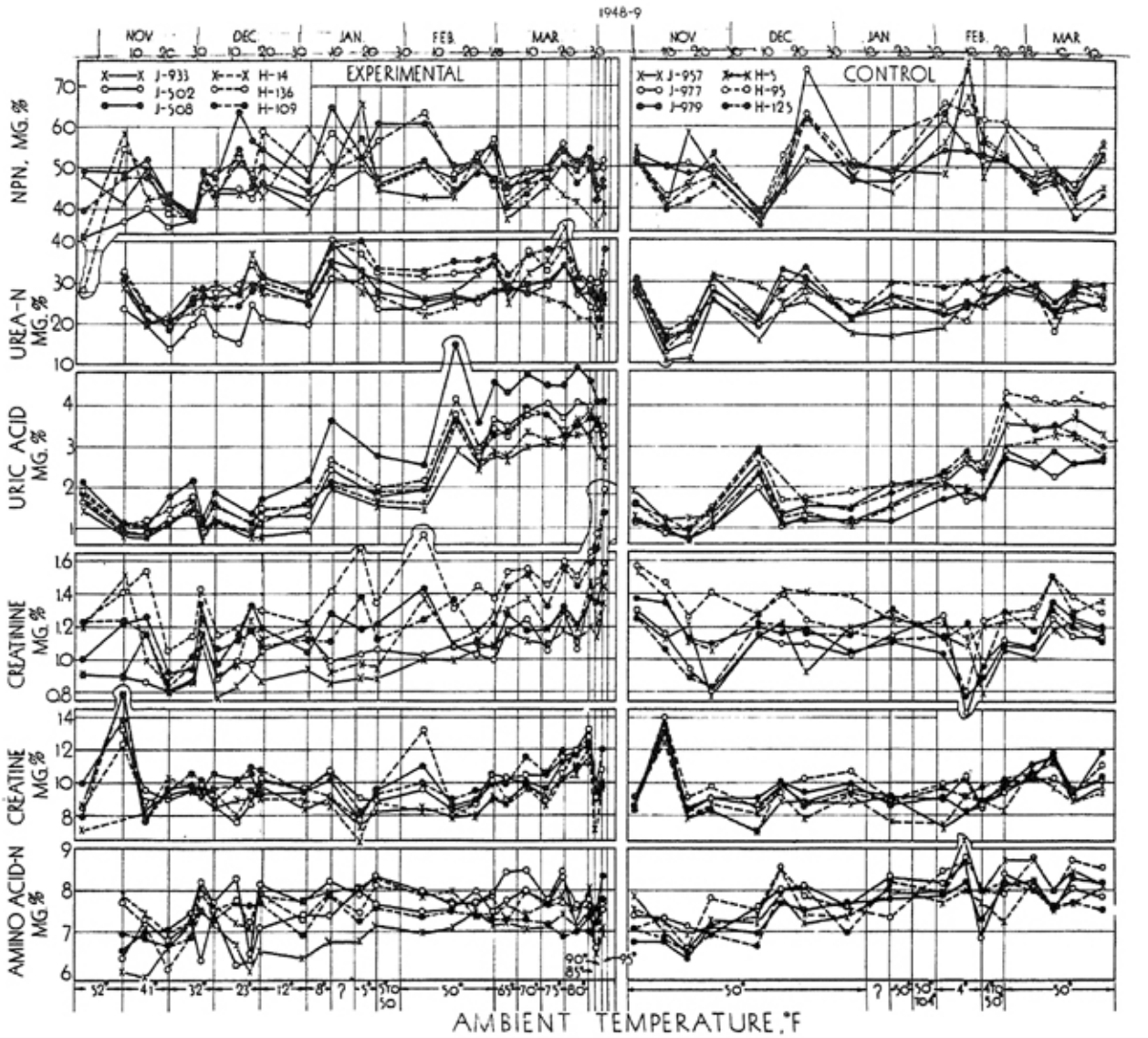


Fig. 6—Further constituents during Period 2.

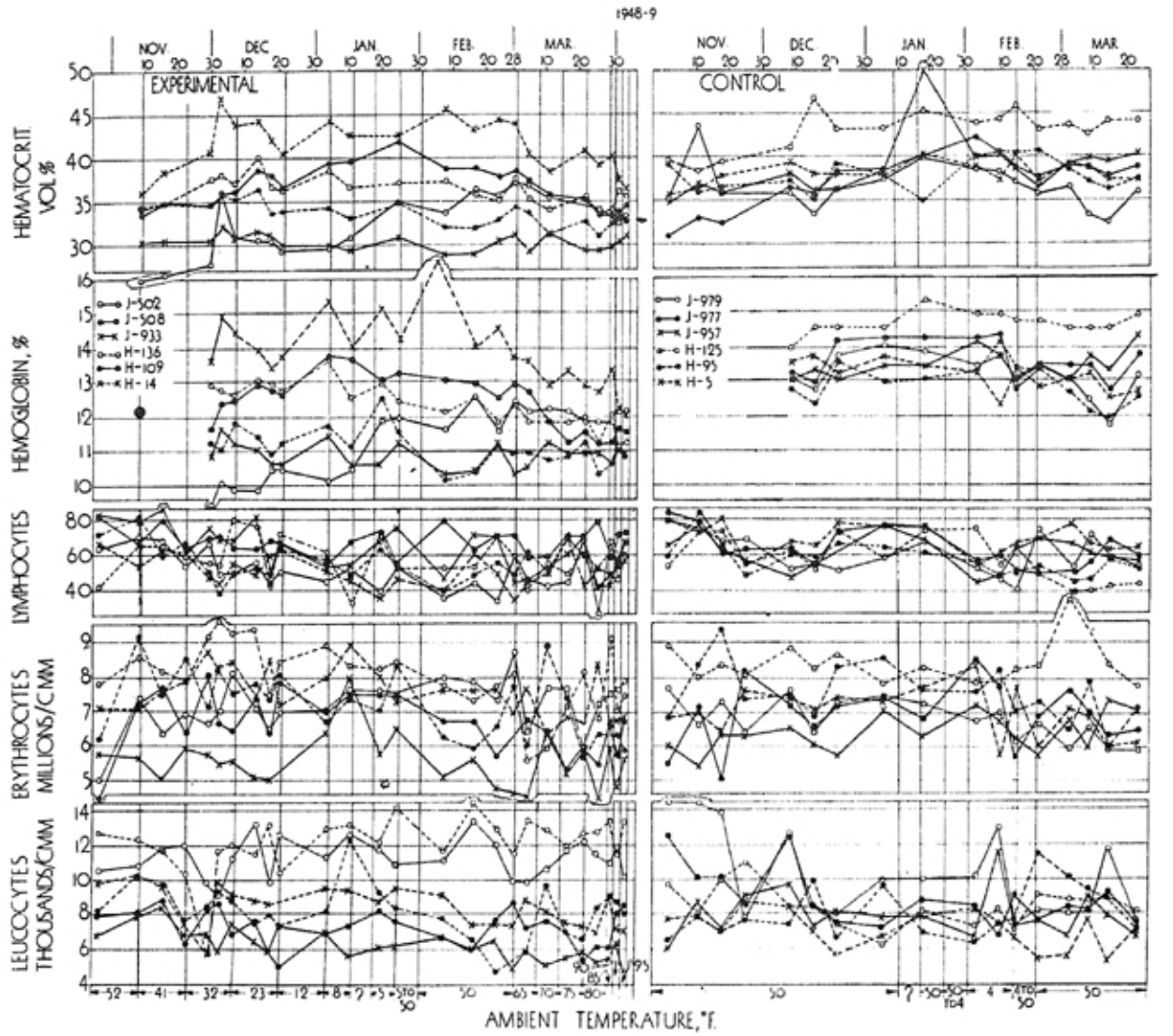


Fig. 7—Hematology during Period 2 for Experimental and Control Jersey (J) and Holstein (H) cows.

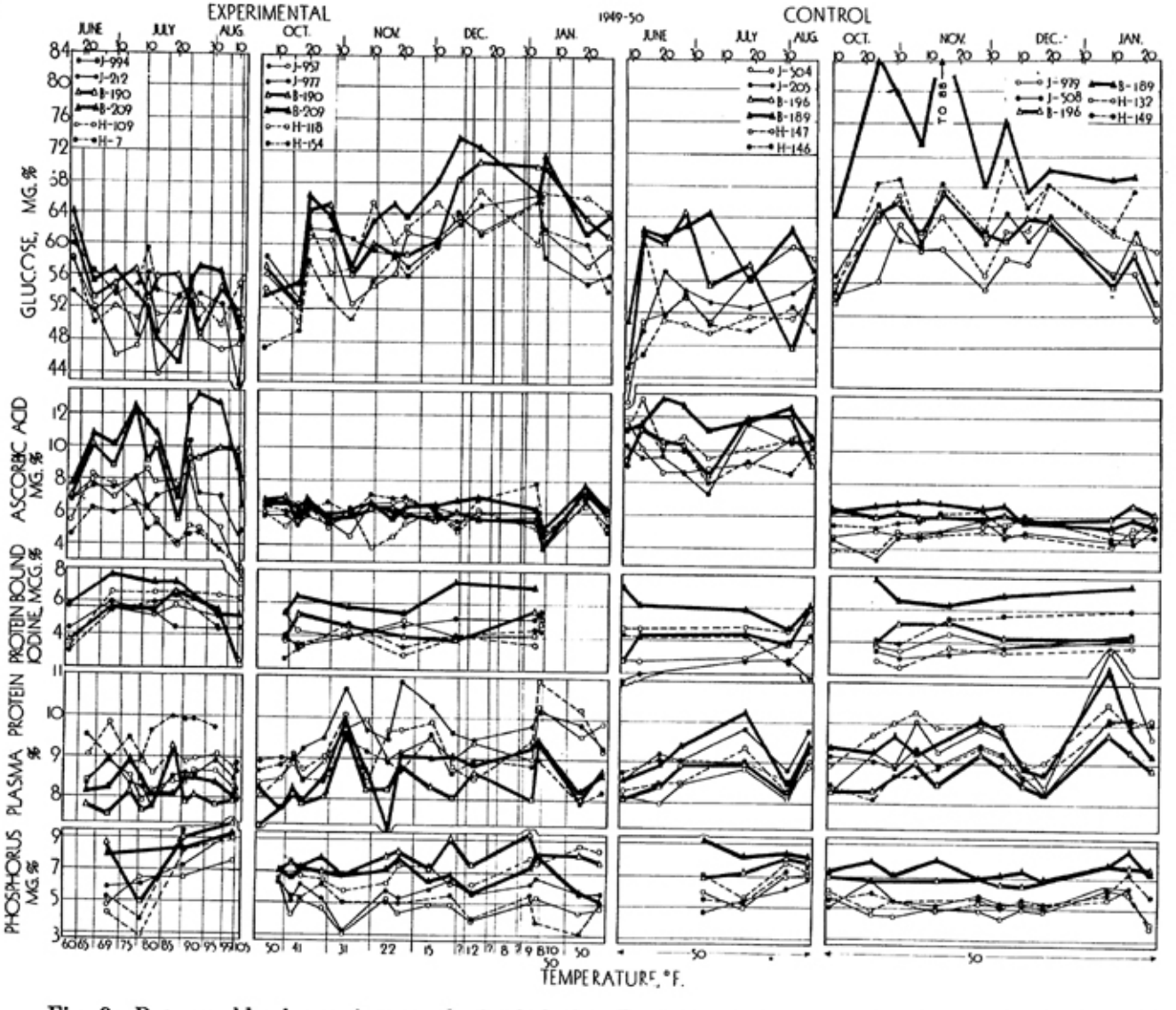


Fig. 8—Data on blood constituents obtained during Period 3 (increasing temperature, 50° to 105°F) and Period 4 (declining temperature, 50° to 8°F) on Jersey (J), Holstein (H), and Brahman (B) cattle, and their "paired" Control cows maintained at a temperature of 50°F throughout. There appears to be a substantial rise in blood glucose with declining temperature.

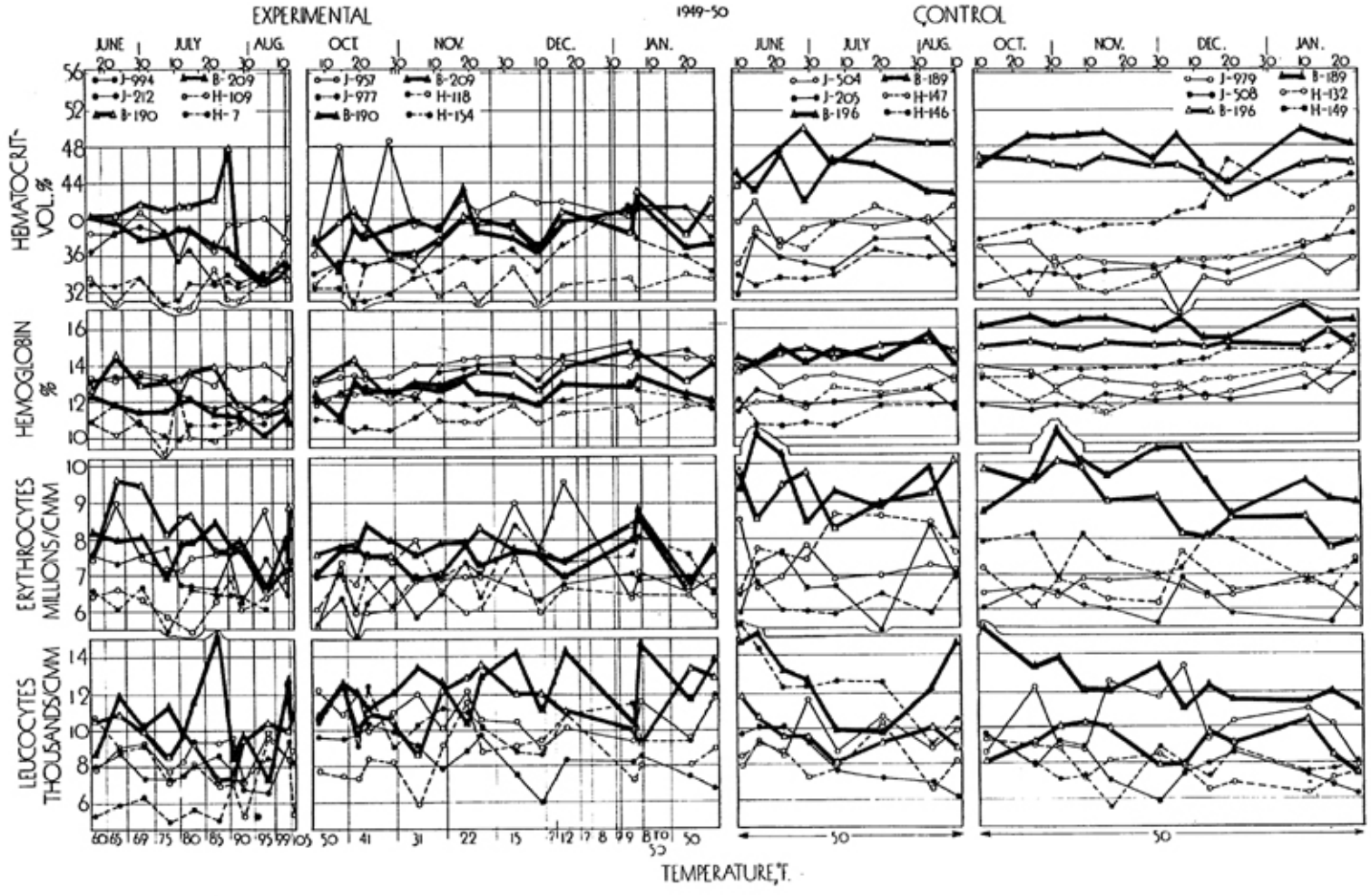


Fig. 9—Hematology data obtained during Period 3 and 4.

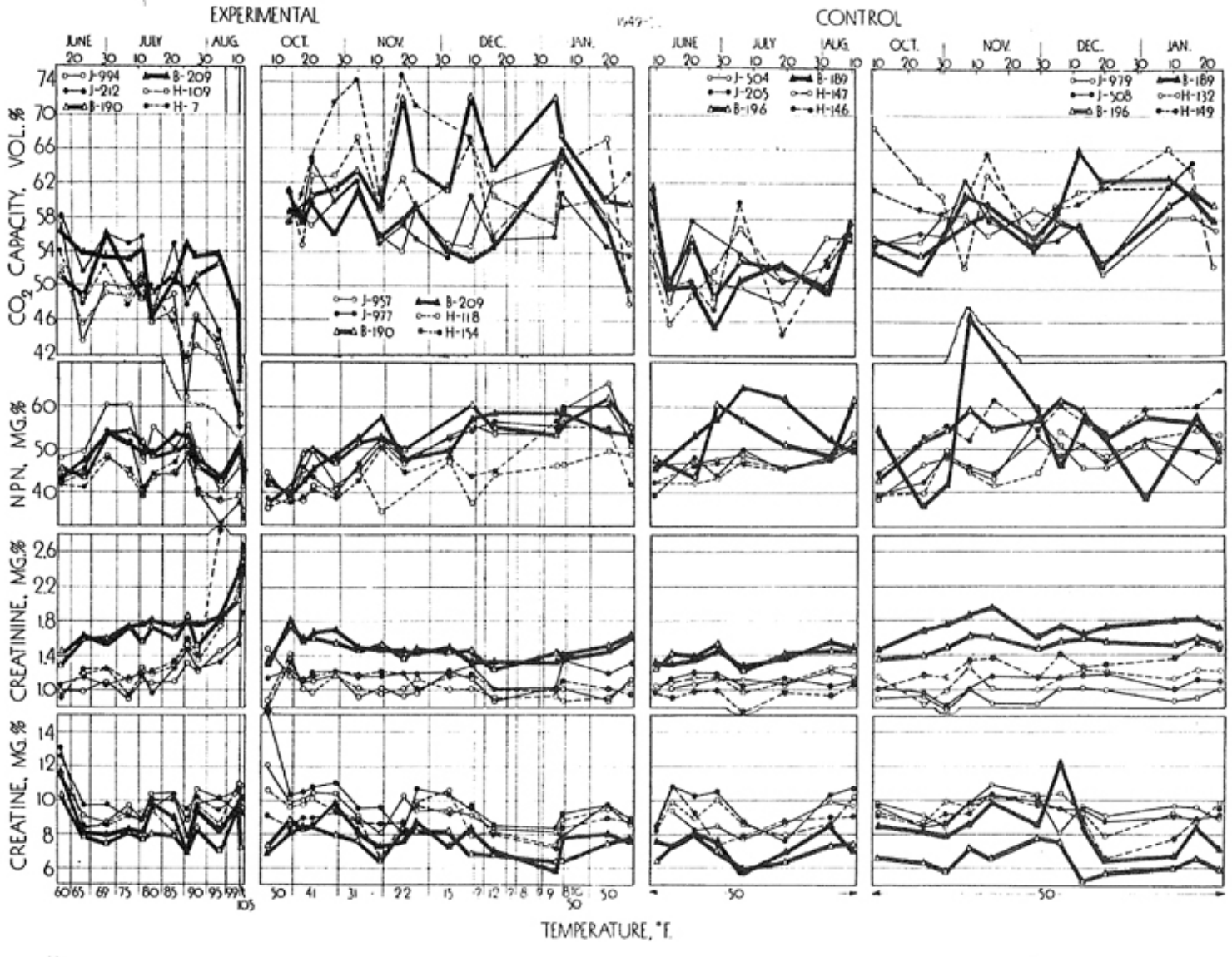


Fig. 10—Further blood constituent data obtained during Periods 3 and 4. Note that the second segment represents the effects of declining temperature.

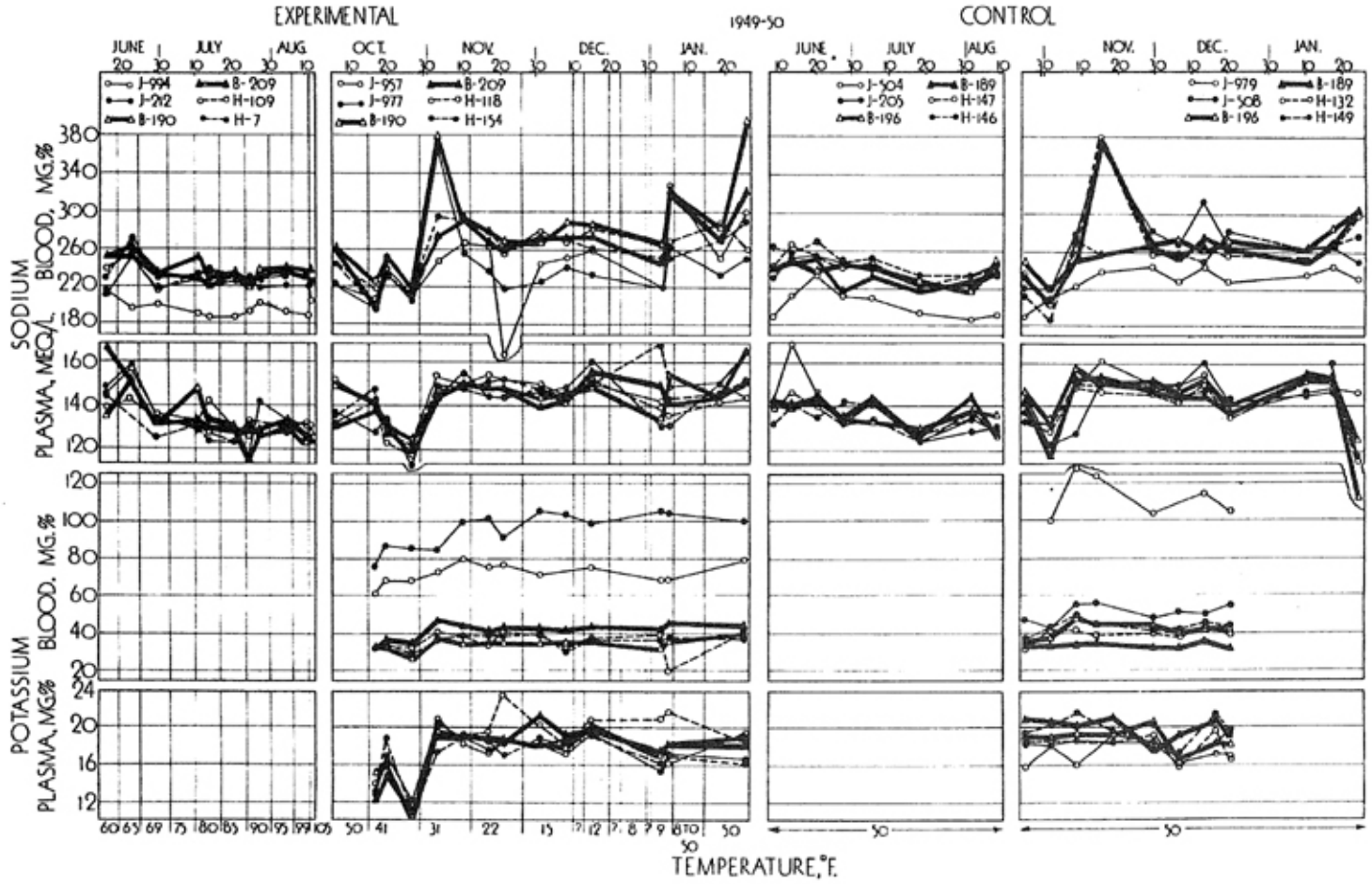


Fig. 11—Blood and plasma sodium obtained during Periods 3 and 4 and blood and potassium data obtained during Period 4. The second segment represents the effects of declining temperature whereas the first segment, the effects of increasing temperature.

TABLE 4.--BLOOD COMPOSITION OF BROWN SWISS AND BRAHMAN COWS AND HEIFERS
(Period 5 -- February 6 to June 9, 1950)

Temp. Level °F	No. of Samples	Hematocrit %		Hemoglobin gm. %		Plasma Protein, %		Protein Bound Iodine, mcg. %		Glucose mg. %		Ascorbic Acid, mg. %		CO_2 Capacity vol. %		Creatine mg. %		Creatinine mg. %	
		Brah-man	Brown Swiss	Brah-man	Brown Swiss	Brah-man	Brown Swiss	Brah-man	Brown Swiss	Brah-man	Brown Swiss	Brah-man	Brown Swiss	Brah-man	Brown Swiss	Brah-man	Brown Swiss	Brah-man	Brown Swiss
							COWS												
40	9	41.6	37.3	13.8	13.0	8.67	8.27	4.70*	3.62*	58.1	56.3	0.64	0.55	55.6	58.5	7.81	9.27	1.40	1.22
50	6	40.1	37.8	13.5	13.2	8.57*	9.07*	4.03*	3.45*	60.2	56.1	0.63	0.57	57.6	60.5	8.21	9.06	1.61	1.31
60	6	40.8	36.8	13.5	12.8	8.21	8.41	5.54*	4.90*	61.0	58.1	0.64	0.59	58.5	60.0	9.16	10.17	1.57	1.31
70	3	47.2	42.7	13.4	13.0	8.34	8.23	5.16	4.37	68.9	65.1	0.67	0.58	59.4	60.5	8.94	10.08	1.65	1.39
80	6	40.8	37.8	13.1	12.6	7.82	7.60	5.01*	3.84*	60.8	58.0	0.72	0.59	59.7	61.4	8.65	10.50	1.86	1.49
85	3	40.6	34.5	13.1	12.1	8.27	8.24	----	----	59.0	55.8	0.63	0.53	59.1	60.2	9.05	10.19	2.03	1.39
90	3	38.0	33.9	13.0	12.5	8.59	8.76	4.69	4.52	64.6	59.2	0.68	0.48	53.7	50.8	7.94	10.16	1.95	1.72
95	3	36.4	35.2	12.3	12.5	8.41	8.71	----	----	57.3	51.6	0.65	0.39	52.4	45.7	7.37	10.90	2.15	2.00
100	3	35.4	35.2	11.6	12.9	8.25	9.12	5.06	3.92	62.4	55.6	0.63	0.37	50.8	38.4	7.62	11.59	2.03	2.19
105	3	35.4	41.0	12.2	14.2	8.79	9.69	----	----	58.5	65.4	0.41	0.26	49.5	35.6	8.84	13.28	2.44	2.78
40†	3	48.1	37.8	13.7	12.0	8.85	8.80	5.73	4.76	66.0	62.9	0.54	0.47	66.7	63.3	8.67	9.54	1.56	1.20
							HEIFERS												
40	9	37.4	37.6	12.1	12.1	----	----	5.08*	4.84*	76.8	79.7	0.88*	0.81*	61.4*	58.8*	8.33	8.94	1.37	1.20
50	6	39.2	40.2	12.1	12.2	8.10	7.80	4.78*	4.20*	73.0	77.2	0.77*	0.74*	61.4	61.1	8.26	8.99	1.33	1.34
60	6	39.6	39.5	12.0	12.1	7.62	7.23	4.83*	4.43*	74.7	80.0	0.81*	0.80*	61.5	61.7	9.24	9.85	1.47	1.33
70	6	41.8	43.5	11.9	12.0	6.97*	6.82*	4.04*	4.17*	69.2	76.3	0.84	0.84	63.3	61.3	8.88	9.23	1.35	1.07
80	6	35.8	36.0	10.9	11.5	7.12	6.87	3.80*	4.14*	67.2	73.9	0.82	0.85	60.5	60.7	8.79	9.18	1.66	1.45
85	3	31.0	34.5	10.4	11.3	----	----	----	----	66.0	72.0	0.74	0.79	63.6	62.7	8.47	9.19	1.55	1.28
90	3	31.4	34.9	10.6	11.5	7.51	7.31	4.35	4.60	64.3	64.4	0.72	0.75	58.7	53.9	7.43	8.09	1.67	1.35
95	3	30.8	35.3	10.4	11.6	7.34	7.17	----	----	64.5	70.0	----	----	59.1	54.5	6.86	7.22	1.74	1.44
100	3	29.4	34.8	9.9	11.5	7.39	7.43	3.99	4.31	63.0	74.9	0.79	0.83	53.6	47.7	8.18	9.72	1.87	1.60
105	3	28.2	33.9	9.4	10.8	7.73	7.75	----	----	63.9	63.0	0.77	0.69	53.0	37.5	8.63	10.22	2.10	1.81
40†	3	38.3	36.5	12.1	11.9	7.96	7.21	7.08	6.30	74.1	81.3	0.62	0.67	72.6	69.9	8.36	8.95	1.21	1.11

* Indicates 3 samples less than number given in second column.
† At end of experiment.

TABLE 5.--NON-PROTEIN NITROGEN CONSTITUENTS FOR JERSEY, HOLSTEIN, AND BRAHMAN COWS

Approx. Temp. Level* °F	TOTAL NON-PROTEIN NITROGEN, Mg. %								CREATININE, Mg. %												CREATINE, Mg. %							
	Jersey & Holstein				Brahman				Jersey				Holstein				Brahman				Jersey & Holstein				Brahman			
	Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control	
	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean
5	26	53.0	20	50.2	4	56.6	4	50.3	13	1.07	10	1.09	13	1.14	10	1.23	4	1.36	4	1.65	26	8.67	20	8.85	4	6.56	4	6.17
15	18	48.4	18	55.2	6	55.4	6	56.6	9	1.09	9	1.08	9	1.06	9	1.22	6	1.36	6	1.60	18	9.13	18	9.34	6	7.30	6	8.26
20	22	48.8	16	43.6	2	48.6	1	54.5	13	1.04	8	1.11	13	1.15	8	1.27	4	1.42	2	1.77	26	9.51	16	9.18	4	8.13	2	8.16
30	26	44.2	14	48.8	4	53.5	4	59.0	13	1.00	7	0.90	13	1.10	7	1.14	4	1.47	4	1.68	26	9.23	14	9.10	4	7.30	4	7.32
40	24	42.8	16	45.8	6	46.9	2	44.4	12	1.05	8	1.09	12	1.09	8	1.13	6	1.59	2	1.53	24	9.44	16	10.23	6	8.46	2	7.23
50	58	46.5	28	49.7	8	47.9	8	50.7	25	1.09	13	1.09	26	1.24	14	1.27	8	1.53	8	1.50	50	9.85	27	9.06	8	7.68	8	7.09
60	4	43.5	0	----	2	44.4	0	----	2	1.00	2	1.06	2	0.96	2	0.99	2	1.32	2	1.34	4	12.12	4	10.17	2	10.86	2	7.25
65	10	43.4	10	46.6	2	45.2	2	48.3	5	1.14	5	1.08	5	1.33	5	1.16	2	1.59	2	1.35	10	9.25	10	9.91	2	7.83	2	8.07
70	28	44.6	19	47.4	2	53.9	2	58.3	12	1.06	7	1.16	13	1.19	6	1.23	2	1.53	2	1.49	10	9.72	10	10.19	2	7.63	2	7.23
75	16	49.9	10	44.6	2	52.7	2	60.3	8	1.14	5	1.15	8	1.30	5	1.11	2	1.72	2	1.24	16	10.00	10	8.74	2	8.12	2	5.67
80	26	45.5	12	47.0	4	49.2	0	----	13	1.10	8	1.06	13	1.22	7	1.18	4	1.70	0	----	14	10.15	6	10.33	4	8.33	0	----
85	22	46.8	7	44.7	2	51.7	2	56.4	11	1.24	3	1.06	11	1.32	4	1.08	2	1.65	2	1.38	10	11.23	4	8.24	2	8.34	2	6.58
90	28	43.1	9	41.8	4	49.0	0	----	15	1.28	5	1.09	13	1.46	4	1.17	4	1.72	0	----	14	9.24	0	----	4	7.87	0	----
95	22	40.8	16	46.3	2	43.0	2	55.1	11	1.34	8	1.05	11	1.75	8	1.07	2	1.81	2	1.50	10	10.06	4	9.45	2	7.53	2	7.85
100	15	39.3	16	45.0	2	50.6	2	55.4	8	1.47	8	1.20	7	2.02	8	1.25	2	2.22	2	1.47	3	10.19	4	9.90	2	9.61	2	7.10
105	3	37.9	--	----	2	43.1	-	----	2	2.12	--	----	1	2.55	--	----	2	2.55	-	----	3	10.07	--	----	2	8.09	-	----

Approx. Temp. Level* °F	UREA NITROGEN, Mg. %				URIC ACID NITROGEN, Mg. %				AMINO ACID NITROGEN, Mg. %			
	Jersey and Holstein				Jersey and Holstein				Jersey and Holstein			
	Experimental		Control		Experimental		Control		Experimental		Control	
	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean
5	18	32.8	12	22.8	12	2.19	12	1.51	18	7.69	12	7.55
15	6	24.9	6	29.2	6	1.53	6	1.43	6	7.22	6	7.64
20	18	27.9	12	23.8	12	1.16	12	1.87	18	7.19	12	7.59
30	18	25.2	6	28.0	18	1.33	6	1.22	18	7.38	6	7.14
40	12	20.4	12	15.6	12	1.12	11	0.91	12	6.79	12	6.81
50	30	29.0	12	28.8	#(a)12	1.40	6	1.43	30	7.42	12	7.63
					(b)24	3.01	6	3.37				
65	6	28.0	6	27.8	6	3.28	6	3.17	6	7.59	6	8.28
70	6	31.4	6	21.7	6	3.76	6	3.21	6	7.68	6	7.65
75	12	33.4	6	27.3	12	3.60	6	3.23	12	7.61	6	8.08
80	6	27.4	6	26.4	6	3.81	6	3.04	6	7.25	6	7.98
85	6	26.1	--	----	6	3.73	--	----	6	7.54	--	----
90	6	23.3	--	----	6	3.46	--	----	6	7.01	--	----
95	6	30.8	--	----	6	3.13	--	----	6	7.54	--	----

N = Total number of samples averaged and includes data from Periods 1 through 4 for NPN and Creatinine, Periods 2 through 4 for Creatine, and Period 2 for the remaining constituents.

* Approximate temperature of Experimental Chamber; Control Chamber maintained at 50°F.

\# Since the Control cows indicate a definite increase in uric acid with regard to advancing time, the 50°F data on the Experimental cows have been divided into: (a) the 50°F period at the beginning of the experiment, and (b) the 50°F period following the decrease in temperature and preceding the increase in temperature. Data on the Control cows were averaged for corresponding time intervals.

TABLE 6.--HEMATOLOGY OF JERSEY, HOLSTEIN, AND BRAHMAN COWS

Approx. Temp. Level*	HEMOGLOBIN, %								HEMATOCRIT, Vol. %								ERYTHROCYTES, Millions/cmm Blood							
	Jersey & Holstein				Brahman				Jersey & Holstein				Brahman				Jersey & Holstein				Brahman			
	Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control	
°F	N.	Mean	N.	Mean	N.	Mean	N.	Mean	N.	Mean	N.	Mean	N.	Mean	N.	Mean	N.	Mean	N.	Mean	N.	Mean	N.	Mean
5	26	12.6	20	13.2	4	13.8	4	15.4	20	37.2	20	39.1	4	41.2	4	44.9	25	7.40	19	7.12	4	8.43	4	8.74
15	18	12.8	18	12.8	6	12.8	6	15.3	18	37.0	18	36.3	6	38.4	6	45.9	18	7.39	17	7.04	6	7.44	6	9.20
20	26	12.1	16	12.7	4	13.1	2	15.6	26	36.0	16	37.0	4	40.4	2	47.7	26	7.01	16	6.92	4	7.81	2	9.30
30	26	12.1	8	12.5	4	12.7	4	15.4	26	35.7	8	35.2	4	38.1	4	47.1	26	7.26	14	6.90	4	7.30	4	10.24
40	12	12.2	4	12.5	6	13.0	2	15.8	18	34.8	16	36.6	6	38.8	2	47.5	24	6.76	16	6.97	6	7.77	2	9.51
50	40	12.3	22	13.1	8	12.7	8	15.2	46	35.8	28	37.1	8	37.9	8	46.0	58	6.70	28	6.75	8	7.34	8	8.86
60	4	12.1	10	11.7	2	12.5	2	14.0	4	35.4	10	35.1	2	40.4	2	44.0	4	7.00	10	6.50	2	7.87	2	9.64
65	10	12.0	10	12.4	2	13.2	2	14.7	10	35.3	10	38.0	2	40.0	2	47.7	10	6.74	10	7.16	2	8.76	2	9.82
70	22	11.7	19	12.4	2	12.1	2	14.5	25	34.6	19	37.7	2	39.7	2	46.9	28	6.97	19	7.11	2	8.74	2	9.07
75	16	11.6	10	12.2	2	12.3	2	14.5	10	34.7	10	37.2	2	39.6	2	46.1	16	6.40	10	6.74	2	7.50	2	8.70
80	26	11.8	15	12.7	4	12.7	0	----	26	34.1	15	37.5	4	40.1	0	----	24	6.58	15	6.72	4	8.22	0	----
85	22	12.1	7	12.3	2	12.5	2	14.6	22	34.8	7	36.3	2	39.6	2	47.1	21	6.78	7	6.88	2	8.04	2	8.83
90	28	12.3	9	13.4	4	11.7	0	----	28	34.8	9	38.0	4	38.9	0	----	28	6.76	9	7.07	4	7.78	0	----
95	22	12.1	16	13.3	2	10.6	2	15.4	22	33.6	16	37.7	2	33.0	2	45.4	22	6.71	16	7.29	2	6.61	2	9.51
100	15	12.8	16	12.6	2	11.3	2	14.3	15	35.1	16	36.6	2	34.4	2	45.3	13	6.75	16	6.85	2	7.57	2	9.04
105	3	13.0	--	----	2	10.8	-	----	3	37.4	--	----	2	34.2	-	----	3	7.88	--	----	2	7.95	-	----
	LEUCOCYTES, Thousands/cmm Blood								EOSINOPHILES, %								LYMPHOCYTE, %							
5	26	9.5	20	8.3	4	10.9	4	10.5	17	16.2	12	9.7	-	----	-	----	17	52.8	12	69.5	-	----	-	----
15	18	9.1	18	8.1	6	12.3	6	10.2	5	13.6	5	6.8	-	----	-	----	5	53.0	5	69.0	-	----	-	----
20	26	9.2	16	9.0	4	12.3	2	10.8	18	14.2	12	9.3	-	----	-	----	18	58.5	12	56.6	-	----	-	----
30	26	8.9	14	8.5	4	11.6	4	8.9	18	13.8	6	6.3	-	----	-	----	18	57.4	6	56.8	-	----	-	----
40	24	9.5	16	9.4	6	11.0	2	11.1	12	7.2	12	6.5	-	----	-	----	12	64.7	12	71.8	-	----	-	----
50	58	9.2	28	8.8	8	12.2	8	11.0	42	13.8	16	6.7	-	----	2	9.5	42	58.0	16	61.9	-	----	2	41.0
60	4	8.0	10	9.0	2	9.6	2	12.8	4	16.8	10	9.6	2	19.0	2	13.5	4	49.5	10	56.1	2	55.0	2	46.5
65	10	8.4	10	8.8	2	11.3	2	11.2	10	18.4	10	13.3	2	6.0	2	5.5	10	49.8	10	51.5	2	49.0	2	50.0
70	28	9.1	19	8.7	2	10.0	2	11.0	27	10.0	18	9.9	2	9.0	2	11.5	27	53.8	19	57.0	2	51.0	2	59.0
75	16	8.0	10	8.9	2	9.8	2	8.8	16	12.1	10	11.2	2	16.0	2	8.5	16	59.5	10	58.2	2	50.0	2	54.5
80	24	8.8	15	8.4	4	10.1	0	----	26	11.7	15	6.6	4	14.0	0	----	26	51.8	15	56.2	4	54.2	0	----
85	22	9.0	7	9.6	2	11.1	2	9.3	21	9.9	7	9.4	2	18.5	2	9.5	21	58.7	7	56.1	2	51.0	2	55.5
90	28	8.7	9	9.0	4	8.2	0	----	27	7.1	8	6.4	4	13.5	0	----	28	60.5	9	62.0	4	51.0	0	----
95	22	9.0	16	8.1	2	8.7	2	11.0	20	7.3	10	4.5	2	14.0	2	6.0	20	64.6	10	58.9	2	58.5	2	57.0
100	14	8.6	15	8.6	2	11.2	2	11.6	14	3.5	14	5.9	2	12.0	2	9.0	14	62.6	15	63.6	2	57.5	2	47.5
105	3	7.4	--	----	2	10.7	-	----	3	2.3	--	----	2	10.5	-	----	3	67.7	--	----	2	58.0	-	----

N = Total number of samples averaged and includes data from Periods 1 through 4 except for Eosinophiles and Lymphocytes which includes Periods 1 through 3.
* Approximate temperature of Experimental Chamber; Control Chamber was maintained at 50°F.

TABLE 7.--INORGANIC CONSTITUENTS FOR JERSEY, HOLSTEIN, AND BRAHMAN COWS

Approx. Temp. Level* °F	BLOOD SODIUM, meq./liter												PLASMA SODIUM, meq./liter												CALCIUM, mg. %			
	Jersey				Holstein				Brahman				Jersey				Holstein				Brahman				Jersey & Holstein			
	Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control	
	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean
5	4	112	4	107	4	112	4	113	4	125	4	112	4	139	4	142	4	147	4	143	4	145	4	145	18	12.9	12	12.7
15	6	105	6	113	6	118	6	111	6	120	6	114	6	150	6	151	6	147	6	146	6	146	6	147	6	12.4	6	13.0
20	4	94	2	131	4	115	2	137	4	117	2	136	4	148	2	156	4	147	2	148	4	148	2	152	18	13.3	12	14.0
30	4	123	4	100	4	125	4	98	4	134	4	100	4	151	4	130	4	148	4	140	4	147	4	140	18	13.2	6	13.2
40	6	93	2	88	6	96	2	97	6	99	2	104	6	128	2	142	6	130	2	134	6	130	2	145	12	13.7	12	13.4
50	6	107	6	101	6	116	6	116	6	130	6	117	6	159	5	144	6	142	6	135	6	147	6	134	36	13.7	12	13.1
60	2	92	2	100	2	101	2	113	2	100	2	108	2	148	2	155	2	142	2	144	2	151	2	140	0	----	0	----
65	2	97	2	106	2	114	2	113	2	112	2	106	2	158	2	146	2	166	2	138	2	152	2	143	6	13.2	6	13.1
70	2	91	2	99	2	96	2	106	2	101	2	100	2	135	2	134	2	129	2	137	2	131	2	135	20	14.9	14	14.5
75	0	——	2	97	0	---	2	108	0	---	2	103	0	---	2	141	0	---	2	138	0	---	2	139	12	13.5	6	13.7
80	4	89	0	---	4	102	0	---	4	101	0	---	4	134	0	---	4	128	0	---	4	136	0	---	18	15.4	15	15.5
85	2	89	2	90	2	101	2	99	2	101	2	96	2	130	2	124	2	123	2	126	2	125	2	128	18	14.2	3	12.5
90	4	91	0	---	4	98	0	---	4	99	0	---	4	127	0	---	3	120	0	---	4	126	0	---	19	12.9	9	13.7
95	2	90	2	88	2	102	2	100	2	104	2	96	2	133	2	131	2	124	2	135	2	129	2	141	12	12.0	12	11.8
100	2	89	2	91	1	100	2	105	2	101	2	105	2	127	2	129	1	133	2	127	2	124	2	132	12	12.3	12	13.2
105	2	90	-	---	1	89	-	---	2	104	-	---	2	127	-	---	1	135	-	---	2	126	-	---	--	----	--	----

	PHOSPHORUS, mg. %												PLASMA POTASSIUM, meq./liter											
5	13	6.43	10	5.93	13	5.88	10	5.80	4	8.28	4	7.29	4	4.07	2	4.63	4	4.78	2	4.50	4	4.48	2	4.68
15	9	5.76	9	5.33	8	5.67	9	5.67	6	7.09	6	6.87	6	4.68	6	4.40	6	4.88	6	4.76	6	4.94	6	4.71
20	13	5.95	8	5.22	13	6.01	7	5.35	4	7.94	2	7.40	4	4.53	2	4.73	4	4.99	2	4.76	4	4.73	2	5.06
30	11	5.92	5	5.93	11	6.03	5	6.39	2	6.68	1	7.16	4	4.99	4	4.45	4	4.81	4	4.96	4	4.86	4	4.96
40	12	5.76	8	6.56	12	6.27	8	6.22	6	7.26	2	7.39	6	3.50	2	4.30	6	3.50	2	4.81	6	3.35	2	5.04
50	24	7.13	11	6.47	24	6.79	12	6.52	6	6.77	6	7.54	2	4.58	-	----	2	4.40	-	----	2	4.71	-	----
60	0	----	3	5.35	0	----	3	7.07	0	----	0	----												
65	3	7.13	3	6.73	3	5.99	3	6.27	0	----	0	----												
70	13	5.90	6	5.76	14	6.02	8	6.53	2	8.22	0	----												
75	6	6.78	5	6.02	6	6.27	5	6.67	0	----	2	8.11												
80	11	5.96	8	5.73	11	5.19	7	5.97	1	4.93	0	----												
85	9	5.56	3	4.96	9	5.76	4	5.50	0	----	2	7.75												
90	12	6.24	5	4.99	11	6.87	4	6.61	2	8.62	0	----												
95	9	5.99	8	4.97	9	6.21	8	6.43	0	----	2	8.30												
100	8	7.25	8	5.49	7	7.54	8	5.42	2	9.57	2	8.09												

BLOOD POTASSIUM,# meq./liter									
Temp. Level,* °F									
5	4	8.46	2	10.43	4	9.92	2	9.26	
15	6	9.10	6	10.51	6	9.72	6	9.51	
20	4	9.61	2	10.61	4	9.79	2	10.00	
30	4	9.72	4	11.23	4	10.28	4	9.74	
40	6	8.13	2	8.49	6	8.18	2	8.77	
50	2	9.85	-	----	2	10.71	-	----	

N = Total number of samples averaged and include data from Periods 1 through 4 for Phosphorus; Periods 3 and 4 for Sodium; Periods 1 and 2 for Calcium; and Period 4 for Potassium.

* Approximate temperature of Experimental Chamber; Control Chamber was maintained at 50 F.

See Fig. 00 for Blood Potassium level of Jersey cows; level not given because of extreme variability between the two cows.

TABLE 8.--ADDITIONAL BLOOD CONSTITUENTS FOR JERSEY, HOLSTEIN, AND BRAHMAN COWS

Approx. Temp. Level* °F	PLASMA PROTEIN, %								ASCORBIC ACID,** mg. %												PROTEIN BOUND IODINE, mcg. %							
	Jersey & Holstein				Brahman				Jersey				Holstein				Brahman				Jersey & Holstein				Brahman			
	Exper.		Control		Exper..		Control		Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control	
	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean
5	26	8.82	20	8.99	4	9.07	4	9.51	4	0.50	2	0.51	4	0.55	2	0.46	4	0.51	2	0.58	8	5.05	0	----	4	5.36	0	----
15	18	9.05	18	8.95	6	8.69	6	9.24	6	0.59	6	0.56	6	0.59	6	0.55	6	0.62	6	0.62	4	4.33	4	4.10	2	5.60	2	5.55
20	26	8.91	16	8.93	4	8.37	1	8.47	4	0.63	2	0.52	4	0.59	2	0.57	4	0.58	2	0.65	4	3.96	0	----	2	4.66	0	----
30	26	9.40	14	9.02	4	9.16	3	9.37	4	0.58	4	0.50	4	0.58	4	0.54	4	0.62	4	0.66	4	4.15	8	3.75	2	5.20	4	5.84
40	24	8.75	16	8.31	6	8.15	2	8.80	6	0.58	2	0.44	6	0.60	2	0.47	6	0.62	2	0.64	4	3.58	4	3.62	2	5.88	2	5.99
50	52	9.05	27	9.11	8	8.09	8	8.95	8	0.62	6	0.52	8	0.59	6	0.50	8	0.67	6	0.63	12	3.76	8	3.87	6	5.10	4	5.36
60	0	----	6	8.54	0	----	0	----	2	0.67	2	0.98	2	0.48	2	1.23	2	0.74	2	1.14	4	4.02	4	3.40	2	4.70	2	5.24
65	10	8.55	10	9.40	2	7.96	2	8.70	2	0.77	2	0.91	2	0.72	2	1.01	2	1.05	2	1.18	0	----	0	----	0	----	0	----
70	21	8.45	19	8.51	2	7.89	2	9.20	2	0.71	2	0.86	2	0.68	2	1.04	2	0.94	2	1.15	4	5.88	0	----	2	6.61	0	----
75	16	8.82	6	8.77	2	8.53	0	----	2	0.80	2	0.81	2	0.64	2	0.88	2	1.25	2	0.98	0	----	0	----	0	----	0	----
80	25	8.62	12	8.60	4	7.97	0	----	4	0.73	0	----	4	0.55	0	----	4	1.04	0	----	4	5.73	0	----	2	6.33	0	----
85	22	9.05	7	9.52	2	8.66	2	9.62	2	0.76	2	1.04	2	0.40	2	0.98	2	0.61	2	1.18	4	5.78	4	3.78	2	6.92	2	5.21
90	28	8.94	9	8.88	4	8.21	0	----	4	0.80	0	----	4	0.48	0	----	4	1.10	0	----	0	----	0	----	0	----	0	----
95	21	9.03	16	8.82	2	8.10	2	8.42	2	0.59	2	1.07	2	0.37	2	0.96	2	1.13	2	1.24	4	5.08	4	3.60	2	5.37	2	4.31
100	14	8.64	16	9.02	2	7.98	2	9.44	2	0.35	2	1.03	1	0.26	2	1.04	2	0.92	2	0.99	0	----	4	3.91	0	----	2	6.18
105	3	8.47	--	----	2	8.13	-	----	2	0.35	--	----	1	0.18	--	----	2	0.72	-	----	2	5.30	--	----	2	3.86	-	----

	FATTY ACIDS, mg. %							
	Lactating Jersey & Holstein				Non-lactating Holstein			
	N	Mean	N	Mean	N	Mean	N	Mean
5	14	170	9	147	3	187	2	188
15	5	185	4	233	1	246	1	288
20	14	196	7	185	3	233	1	295
30	8	241	5	241	2	135	1	223
40	10	239	9	193	2	167	1	280
50	#(a)10	220	5	197	2	197	0	---
	(b)15	163	4	168	3	210	1	165
60	0	---	0	---	0	---	0	---
65	5	131	5	126	1	174	1	159
70	5	128	5	131	1	165	1	178
75	10	158	5	200	2	184	1	180
80	5	162	5	310	1	169	-	---
85	5	157	--	---	1	186	-	---
90	5	121	--	---	1	135	-	---
95	5	106	--	---	1	111	-	---
100	--	---	--	---	-	---	-	---
105	--	---	--	---	-	---	-	---

	GLUCOSE, mg. %												CARBON DIOXIDE CAPACITY, vol. %							
	Jersey				Holstein				Brahman				Jersey & Holstein				Brahman			
	Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control		Exper.		Control	
	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean	N	Mean
5	13	65.7	10	59.6	13	65.7	10	65.4	4	69.5	4	64.7	26	60.7	20	57.8	4	67.6	4	59.2
15	9	62.7	9	62.7	9	63.6	9	65.9	6	68.9	6	66.6	11	59.6	18	56.8	6	59.9	6	57.9
20	13	62.7	8	56.5	13	63.2	8	62.5	4	61.7	2	77.4	26	59.7	16	58.0	4	63.2	2	59.2
30	13	56.4	7	56.0	13	56.5	7	60.3	4	59.2	4	70.4	26	59.9	14	56.3	4	60.0	3	58.1
40	12	54.8	8	54.1	12	53.0	8	58.4	6	61.2	2	74.4	24	59.7	16	57.0	6	59.0	2	52.8
50	24	59.0	14	57.4	24	60.0	13	62.0	6	60.2	7	56.7	48	56.7	26	57.6	6	57.9	8	58.9
60	2	55.8	5	54.9	2	59.0	5	58.4	2	62.9	2	62.0	4	55.5	10	49.7	2	53.7	2	50.8
65	5	54.3	2	54.5	5	55.1	2	51.4	2	54.3	2	61.0	10	51.3	10	53.9	2	51.2	2	53.0
70	12	52.0	9	56.6	13	55.7	10	58.9	2	56.0	2	63.7	28	55.4	19	53.2	2	54.6	2	47.2
75	8	52.5	5	53.7	8	53.1	5	57.2	2	55.2	2	60.0	16	52.2	10	58.3	2	52.0	2	52.0
80	13	52.3	8	60.1	13	55.9	7	65.2	4	52.3	0	----	26	53.2	15	57.9	4	49.4	0	----
85	11	50.0	3	51.1	11	52.6	4	48.6	2	50.7	2	57.2	22	53.3	7	51.3	2	50.5	2	52.6
90	15	55.0	5	63.2	13	57.0	4	69.2	4	53.6	0	----	28	49.4	9	55.7	4	52.2	0	----
95	11	53.0	8	57.8	11	56.4	8	59.9	2	55.7	2	55.1	22	45.1	10	56.4	2	53.1	2	49.8
100	8	46.0	8	55.5	7	51.5	8	55.7	2	50.7	2	56.5	3	36.1	4	56.4	2	47.4	2	56.6
105	2	52.0	--	----	1	50.7	--	----	2	49.0	-	----	3	34.5	--	----	2	39.7	-	----

N = Total number of samples averaged and include data from Periods 1 through 4 for Plasma Protein, Glucose, CO_2 Capacity; Periods 3 and 4 for Ascorbic Acid and Protein Bound Iodine; and Period 2 (see Research Bulletin 433 for Period 1) for Fatty Acids.

* Approximate temperature of Experimental Chamber; Control Chamber was maintained at 50°F.

** Note that the Ascorbic Acid values obtained during Period 3 (60° up to 105°F) were considerably higher than those obtained during Period 4 (50° to 5°F) especially on the Control Group as is shown graphically in Fig. 8.

Since the Control cows indicate a definite increase in Fatty Acids with regard to advancing time, the 50°F data on the Experimental cows have been divided into: (a) the 50°F period at the beginning of the experiment, and (b) the 50°F period following the decrease in temperature and preceding the increase in temperature. Data on the Control cows were averaged for corresponding time intervals.

7. ABSTRACT

Voluminous data with interpretations are presented on the effects of environmental temperature, 0° to 105°F (about −18° to 41°C), on the blood composition of Holstein, Jersey, Brown Swiss (European-evolved), and Brahman or Zebu (Indian-evolved) cows, and Brown Swiss and Brahman yearling heifers. There were no significant changes between 0° and 65°F. On raising the environmental temperature from 65° to 105°F, the creatinine level was doubled; and the carbon dioxide-combining capacity, ascorbic acid, and cholesterol were halved. There were no significant disturbances in water balance—in electrolytes, protein colloids, and sugar. The protein-bound iodine and radioiodine data were not sufficient for drawing conclusions concerning the effect of temperature change on thyroid activity.

RESEARCH BULLETINS IN THE "ENVIRONMENTAL PHYSIOLOGY" SERIES

Mo. Res. Bul. No.		
423	I.	Physiological Backgrounds (Samuel Brody).
425	II.	Influence of Temperature, 50° to 105°F, on Milk Production and Feed Consumption in Dairy Cattle (A. C. Ragsdale, et al.).
433	III.	Influence of Ambient Temperature, 50° to 100°F, on the Blood Composition of Jersey and Holstein Cows. (S. Brody and Coworkers.)
435	IV.	Influence of Temperature, 50° to 105°F, on Heat Production and Cardiorespiratory Activities in Dairy Cattle (H. H. Kibler, et al.).
436	V.	Influence of Temperature, 50° to 105°F, on Water Consumption in Dairy Cattle (H. J. Thompson, et al.).
449	VI.	Influence of Temperature, 50° to 0°F and 50° to 95°F, on Milk Production, Feed and Water Consumption and Body Weight in Jersey and Holstein Cows. (A. C. Ragsdale, et al.).
450	VII.	Influence of Temperature, 50° to 5°F and 50° to 95°F, on Heat Production and Cardiorespiratory Activities of Dairy Cattle. (H. H. Kibler, et al.).
451	VIII.	Influence of Ambient Temperature, 0° to 105°F, on Insensible Weight Loss and Moisture Vaporization in Holstein and Jersey Cattle (H. J. Thompson, et al.).
460	IX.	Milk Production and Feed and Water Consumption Responses of Brahman, Jersey, and Holstein Cows to Changes in Temperature, 50° to 105°F and 50° to 8°F (A. C. Ragsdale, et al.).

461 X. Influence of Temperature, 5° to 95°F, on Evaporative Cooling From the Respiratory and Exterior Body Surfaces in Jersey and Holstein Cows (H. H. Kibler, et al.).

464 XI. Effects of Temperature, 50° to 105°F and 50° to 9°F, on Heat Production and Cardiorespiratory Activities in Brahman, Jersey, and Holstein Cows (H. H. Kibler, et al.).

471 XII. Influence of Increasing Temperature, 40° to 105°F, on Milk Production in Brown Swiss Cows, and on Feed and Water Consumption and Body Weight in Brown Swiss and Brahman Cows and Heifers (A. C. Ragsdale, et al.).

473 XIII. Influence of Increasing Temperature, 40° to 105°F, on Heat Production and Cardiorespiratory Activities in Brown Swiss and Brahman Cows and Heifers (H. H. Kibler, et al.).

479 XIV. Influence of Temperature on Insensible Weight Loss and Moisture Vaporization in Brahman, Brown Swiss, Holstein, and Jersey Cattle (H. J. Thompson, et al.).

481 XV. Influence of Environmental Temperature, 0° to 105°F, on Hair and Skin Temperatures and on the Partition of Heat Dissipation Between Evaporative and Non-evaporative Cooling in Jersey and Holstein Cattle (H. J. Thompson, et al).

484 XVI. Effect of Increasing Temperature, 65° to 95°F, on the Reflection of Visible Radiation From the Hair of Brown Swiss and Brahman Cows (R. E. Stewart, et al).