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Etiology of Dark-Cutting Beef

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SUMMARY AND CONCLUSIONS

One hundred and twenty cattle were used to determine the effect of ante mortem stress on post mortem carcass characteristics and to investigate possible preventive measures against dark-cutting beef. Animals were stressed by excitement and by administration of adrenaline. Preventive measures investigated included administration of hydrocortisone, insulin, Thorazine, Diquel and Reserpine.

Physiological stress induced by excitement over a 24 hour period depleted the muscle glycogen to an extent that the pH of the muscle remained high. High pH and dark color of the muscle were directly related. A short period of excitement just prior to slaughter had little or no effect on subsequent carcass characteristics, except increased residual blood in the subcutaneous fat.

Adrenaline administered subcutaneously or intramuscularly produced hyperglycemia and glycosuria. When administered approximately 24 hours ante mortem at the rate of 3 milligrams per 100 pounds body weight, adrenaline produced dark cutting carcasses. As the level of adrenaline was increased the color of the carcass muscle became darker, the muscle pH higher, and the texture of the muscle more sticky and gummy. Exogenous and/or endogenous adrenaline above normal physiological levels has a profound influence upon metabolic homeostasis, especially those reactions associated with glycolysis.

Dark-cutting beef is caused by cattle being subjected to prolonged ante mortem stress. Any form of stress, such as excitement or trauma, which will arouse the sympathetic nervous system and stimulate the increased release of adrenaline over a prolonged period (approximately 24 hours or longer) can cause the depletion of glycogen and this condition to occur. The intensity and duration of stress as well as the susceptibility of individual cattle to stress will determine the prevalence of dark-cutting carcasses. Also, the efficiency of the stress defense mechanisms involved in gluconeogenesis probably varies among animals. Cattle previously exposed to adverse conditions possibly build up a resistance against future exposure and therefore may be better able to withstand adverse conditions encountered during transportation and marketing.

Muscle glycogen can be depleted much faster than it can be replenished to normal levels. Carcasses from cattle injected with adrenaline at periods from 24 to 96 hours showed improvement as time increased between treatment and slaughter. However, these carcasses were not as light and bright in color as controls.

Administration of hydrocortisone aided in reducing the adverse effects of injected adrenaline when no additional stress was imposed upon the animal. However, hydrocortisone did not prevent dark-cutting beef.

Administration of tranquilizers prior to stress did not prevent dark-cutting beef. There were indications that these compounds caused a reduction in reflexes and alarm responses and delayed the onset of excitement. Animals injected with these compounds appeared listless and drowsy and slight to severe ataxia occurred, depending upon dosage given. Edema was always present in the carcass surrounding the site of injection and constituted an economic loss equivalent to dark-cutting condition.

Insulin administered prior to stress did not prevent dark-cutting beef; however, blood sugar levels were depressed.

The administration of reserpine at levels which produced a noticeable degree of tranquilization produced deleterious side effects which would preclude the use of this compound as a preventive measure against dark-cutting beef.

No significant difference in eating qualities was detected between dark and bright beef from animals of the same approximate age and degree of finish. If the housewife could be assured that the undesirable appearance of dark-cutting beef was not due to deterioration, she could purchase dark beef with the same assurance of eating satisfaction as if she purchased bright beef.

The most economical and feasible method of preventing dark cutting beef is careful and proper handling of cattle from the time they leave the farm until they are slaughtered. Special care should be exercised to prevent excitement, trauma or fatigue while loading cattle at the farm and in transporting and handling them on the way to and at the market or slaughtering plant.

Etiology of Dark-Cutting Beef

H. B. Hedrick, James B. Boillot, D. E. Brady, and H. D. Naumann

INTRODUCTION

The color of beef muscle is an important factor in the grading of beef carcasses and the acceptability of retail cuts by the consumer. Under the conditions encountered in the packing industry the color of beef muscle may vary from a light cherry red to a purplish black. Beef that cuts dark red to purplish black is termed dark-cutting beef. The occurence of dark cutting beef constitutes a sizeable annual loss to the processor and the producer. The consumer associates the dark color of beef with old animals or meat that has deteriorated. Dark cutting beef has to be sold at a reduced price in most instances.

Factors affecting the color of meat have been a matter of interest to research workers for many years. The first recorded observations on dark cutting beef according to Lawrie (1958) were made at least as far back as 1774. However, no detailed information concerning the characteristics of dark cutting beef was published prior to the work of Hall *et al.* (1944a) and the National Live Stock and Meat Board Committee (1949). These workers conducted extensive investigations on the characteristics of dark cutting beef and the causative factors. The characteristics of dark cutting beef reported were abnormally high pH, low glucose, practically no glycogen, high inorganic phosphate, low oxidation potential, and rapid oxygen uptake. Hall *et al.* (1944a) concluded:

> . . . . the cause of dark cutting beef definitely seems to rest upon a deficiency of glycogen in the tissue at the time of slaughter . . . ., it seems probable that glycogen deficiency in muscle tissue may result from a number of unrelated causes, and that extensive investigations will be necessary to determine all the causes upon which may depend the occurrence of dark cutting beef.

Currently there is no means of successfully predicting dark color in the meat before an animal is slaughtered. In some lots of cattle there is a high incidence of dark cutters while in others there are no dark cutters. It has been observed in the packing industry that dark cutters are more prevalent during periods of inclement weather.

From the economical viewpoint of the producer, processor, and the consumer, an investigation into the causative factors and the physiological mechanisms that are involved in the production of dark cutting beef was merited. Once a knowledge of the physiological mechanisms involved has been gained, preventive measures can be more accurately undertaken.

Objectives of this study

The objectives of this study were:

(a) To determine the causes of dark cutting beef; and

(b) to investigate preventive measures which can be taken to alleviate dark cutting beef.

REVIEW OF THE LITERATURE

Influence of Feed on Color and Glycogen Content of Muscle

Mackintosh (1932) reported that many packers consider pasture as the cause of dark cutters and therefore discriminate against grass fed cattle. However, Mackintosh and Hall (1935), Longwell (1936), and Bull and Rusk (1941) reported that grass fed cattle produced beef of equal brightness, compared to grain fed cattle of the same degree of finish. The color of the lean appeared to be related directly to the degree of finish of the beef.

Bull *et al.* (1930) reported that with increased finish the color of beef was lighter in color. Branaman *et al.* (1936) reported that color of lean showed little tendency to change as the degree of finish increased for steers and heifers. Shenk *et al.* (1934) reported that beef cattle fed on pasture had a higher level of muscle myoglobin than animals fed in dry lot. It was postulated that the difference was due to more exercise while on pasture and not due to a nutritional difference. It was also reported that equal concentrations of blood hemoglobin and muscle myoglobin did not have equal power of light absorption because these pigments do not have the same hue. The acid hematin derivatives were reported to be identical. The difference in light absorption has been shown to be due to the globin or protein part of the molecule.

Hall *et al.* (1944a) reported on two lots of cattle shipped to Kansas City and slaughtered: one full fed in dry lot, the other full fed on pasture. Three steers in the lot full fed in dry lot produced dark cutting carcasses. No dark carcasses were produced by the cattle full fed on pasture. The occurrence of dark cutters in the dry-lot steers suggested other factors than feeding were involved in the cause of dark beef. The dry-lot steers were provided with sheds. The pasture animals may have been more resistant and better conditioned to withstand rigorous exposure and sudden change of conditions. Animals maintained under

constant enviromental conditions, especially mild temperatures, have lower resistance to shock from chill and exposure than animals subjected to wide temperature fluctuations.

The color of carcasses of cattle fed on a phosphorus-deficient ration were found by Hall *et al.* (1944b) to be equally as bright as those from cattle fed on a ration containing an ample supply of phosphorus. They reported a trend toward darker color in the rib eyes of cattle receiving 0.1 lb. ground limestone per head daily.

Wilcox *et al.* (1953) reported that beef cattle and swine fed varying amounts of sucrose for different time intervals before slaughter (6 hrs. to 14 days), in general, resulted in slight increases in carbohydrate content, improvement in color, and lower pH values of the fresh muscle. The livers of the sucrose-fed animals contained more sugar, were larger, and had a better flavor and texture when cooked than the controls.

The National Live Stock and Meat Board Committee (1949) reported that an examination of completed questionnaires submitted to 4-H Club members exhibiting calves at the 1938, 1939, 1940, and 1941 International Live Stock Expositions indicated that there was a relationship between the kind and amount of feed fed and the ultimate color of beef muscle. It was observed that calves cutting lightest in color had received a higher ratio of grain to protein supplements than was the case of those cutting dark.

Age of Animal as Related to Color of Muscle

Bull *et al.* (1930), Helser *et al.* (1930), Hostetler *et al.* (1937), Longwell (1936), Mackintosh and Hall (1935) and Trowbridge *et al.* (1937a) reported that with increasing age of the animal the color of the muscle darkens. However, with advanced age the dark color was not comparable to that of dark cutting beef.

Breed and Sex as Related to Characteristics of Muscle

Black *et al.* (1934), Fuller *et al.* (1937), and Starkey *et al.* (1937) reported breeding to have little or no influence on the color of muscle.

Bull *et al.* (1930), Brown *et al.* (1937a, 1937b, 1937c), Trowbridge and U.S.D.A. Workers (1937b), and Hunt *et al.* (1937) found no differences in color of muscle of heifer and steer carcasses attributable to sex.

Munns and Burrell (1958) recorded pH values of 1800 beef carcasses and found 12.2 percent to have a pH value above 6.0. The percentages of carcasses with high pH values by sex were: steers 13.9, heifers 11.7, cows 9.5, yearlings 8.6, and baby beeves 9.9. As a continuation of this work, pH values were taken on 6,589 hot-graded cattle. This group was made up of 4914 cows, 882 steers, and 793 heifers. Out of these, 11.2 percent of the cows, 5.8 percent of the steers and 5.7 percent of the heifers had high loin pH values. The incidence was sig-

nificantly higher for the cows than for steers and heifers. The incidence of carcasses with high pH values by week ranged from 0.9 percent to 26.3 percent. This variation, though not explained, could possibly be attributed to variation in weather conditions or differences in handling conditions.

Relationship of pH and Color of Muscle

A positive relationship between dark cutting beef and high muscle pH was reported by Hall *et al.* (1944a). The color of dark beef was greatly improved by exposure to an atmosphere of oxygen. This indicated that a deficiency of oxygen existed in dark colored beef, but it was not clear whether this deficiency was caused by low permeability of the tissue to oxygen, by an excessive demand for oxygen during the post mortem metabolic processes, or by both.

Winkler (1939) reported that a rise in pH of muscle tissue was accompanied by a darkening of color. This was demonstrated by injecting ammonia into the tissue.

The National Live Stock and Meat Board Committee (1949) conducted extensive studies on the chemical, physiological, and nutritional aspects of dark cutting beef. These studies were initiated on carcasses from 4-H Club cattle exhibited at the 1938 International Live Stock Exposition. The first significant finding in these studies was the abnormally low acidity of dark cutting beef. In dark cutting carcasses, the pH of the longissimus dorsi muscle averaged 6.4 in comparison with a pH of 5.5 found in light cutting carcasses.

Analyses for products of glycogen breakdown were made since the acidity of beef depends, in large measure, on the carbohydrate metabolism of the muscle. In dark cutting beef the amount of water-extractable reducing sugars averaged 0.03 percent compared to 0.18 percent in the light colored beef. Analysis of the longissimus dorsi muscle was made for total reducing sugars after acid hydrolysis. Average values were 0.63 percent and 1.26 percent for dark and light colored beef, respectively.

Oxygen uptake studies were made on samples of light and dark colored beef which were adjusted to pH 7.4. Under these conditions the oxygen uptake for dark colored samples was 35 percent less than that for light colored samples. At pH 6.4 (characteristic of dark cutting beef) the oxygen uptake for dark colored samples was 75 percent greater than for light colored samples. At pH 5.4 (characteristic of light colored beef) the rate of oxygen uptake for dark colored samples was over five times greater than that of light colored beef. Thus the oxygen uptake was found to decrease with decrease in pH.

Lawrie (1958) found that pH influences color of muscle by variation in the manner of oxygen utilization by the tissue. The depth of the layer of oxymyoglobin on the surface of meat is determined by the rate of oxygen utilization of certain tissue enzyme systems and by the rate of diffusion of oxygen inwards from the atmosphere. If the rate of oxygen utilization increases while the rate

of diffusion inwards remains constant, the depth of the oxymyoglobin layer on the surface will diminish and may become so small that the purplish color of reduced myoglobin predominates. Cytochrome oxidase is one of the important enzymes responsible for oxygen uptake in biological tissues. This enzyme catalyzes the conversion of oxygen to water in the presence of certain reducing conditions. Its activity is dependent on pH. If the ultimate pH of meat is abnormally high, as it is in dark cutting beef, the rate of oxygen uptake by cytochrome oxidase would rise considerably and could contribute to the dark color.

Effect of Exercise on Carcass Characteristics

Cattle run for distances up to one and one-half miles prior to slaughter, to study the effect of violent exercise on the ultimate color of beef, failed to produce dark cutting beef according to Hinman (1935) and Bull (1935).

Callow (1936) found that hogs exercised immediately before slaughter resulted in increased pH and increased electrical resistance of the muscle tissue 24 hours post mortem. Resting 24, 48, and 72 hours after exercise and then slaughtering resulted in lower pH. Briskey *et al.* (1957) and Rongey (1958) reported that forced exercise of hogs within 24 hours ante mortem resulted in dark color, high pH, and sticky, gummy surface of the muscles.

Howard and Lawrie (1957) reported that steers which were exercised immediately after being transported by train and then slaughtered had a significant decrease in liver and muscle glycogen, compared with steers from the same shipment which were fed, watered, and rested for 14 days before being slaughtered.

Bate-Smith (1936) suggested that the severity and duration of exercise determined the extent of glycogen depletion. He found that fatigued muscle contained more lactic acid and more residual blood at the time of death than resting muscle. Bate-Smith (1937) suggested that animals be rested at least 24 hours before slaughtering, after a fatiguing or exciting journey. He also recommended that struggling on the killing floor be reduced to a minimum to insure ample reserves of glycogen in the muscles.

Exercise during the growth and fattening period did not affect the color of muscle according to Bull and Rusk (1942).

Prolonged muscular training of cattle was found by Mitchell and Hamilton (1932) to increase the glycogen content of muscle, decrease the nitrogenous extractives, and lower the collagen content of muscle. Proctor and Best (1932) found that exercise for two to three weeks increased the glycogen content of the exercised muscles of dogs but when continued for five to six weeks, there was no further increase in glycogen. Instead the glycogen content was similar to the content prior to exercise, which indicates there is an optimum period of training for the accumulation of glycogen.

These investigations indicate that the pig is much more susceptible to glycogen depletion by activity immediately pre-slaughter than cattle. It is difficult to deplete the muscle glycogen of cattle by enforced exercise alone.

Effect of Partial Starvation on Carcass Characteristics

The National Live Stock and Meat Board Committee (1949) reported that witholding feed and providing water three days prior to slaughtered failed to produce dark cutting beef. However, when cattle were exposed to severe conditions of chill and at the same time deprived of feed, the incidence of dark cutters was 5 percent. Moulton (1920) stated that inanition or partial starvation of cattle does not deplete the glycogen content of the liver. Howard and Lawrie (1956) found the subsequent pH values of carcasses from three steers fasted for seven, 14, and 28 days did not differ from the pH values of controls. However, a steer that was fasted, then forcibly exercised for one and one-half hours immediately prior to slaughter produced a carcass of pH 6.19. These investigations indicate that the stress of fasting alone will not affect the ultimate pH of the carcass but if combined with stresses of enforced exercise or inclement weather the ultimate pH of the carcass might be significantly affected.

The Effect of Stress Upon Physiological Functions

When animals are subjected to conditions more extreme than those to which they are accustomed, various physiological functions are affected. Any change in physiological function caused by abnormal conditions may be referred to as stress. Definitions of stress will vary from one source to another as do the factors which constitute stress. Selye (1950) stated that many factors produce stress in animals, such as trauma, nervous stimuli, muscular exercise, hormones, diet, temperature, hemorrhage, electric injury, anoxia, asphyxia, and drugs.

The nervous system plays an important role in the pathogenesis of abnormal physiological conditions caused by stress. Himwich (1955) stated when an animal is threatened or under stress, it responds with a number of physiological changes which are triggered by mechanisms in the hypothalamus, particularly in its posterior part. In this portion of the brain are centers which correlate breathing and heart rate with the individual's emotional state, raise the blood pressure, control basal metabolism and body temperature, rouse the body, and put it to sleep. Selye (1950) stated that in the event of exposure to various stresses such as fear, nervous stimuli can be superimposed upon the effect of the main alarming stimulus and thus aggravate its systemic stress effect. Many of the individual reactions which compose the shock syndrome can be mediated by nerves. The production of shock, however, is not dependent upon the integrity of the nervous system.

Systemic defense according to Selye (1950) is made possible through the actions of the two integrating systems of the body, the circulatory and the nervous systems. Systemic nervous defense reactions are mainly regulated through the nerve centers of the hypothalamus. From here, autonomic nerves carry impulses throughout the body to the vascular system and to the organs involved in metabolism. Such stimuli play an important role in gearing the organism for

defense. The adrenal medulla receives secretory impulses through the splanchnics which cause the discharge of adrenaline during exposure to stress.

Every form of stress that has been studied increases the need for hormones which in a normal animal is met by greater output from the adrenal. According to Hartman and Brownell (1940) any type of stress, if of sufficient magnitude and duration, affects the adrenal cortex. Cannon (1911) reported that emotional excitement increased the secretion of the adrenal glands and later (1922) stated evidence had been adduced that adrenal, hepatic, and thyroid secretions were all subject to sympathetic impulses.

Funkenstein (1955) stated that domestic animals and wild animals that live very social lives have a high ratio of adrenaline to noradrenaline. Noradrenaline differs markedly from adrenaline in its physiological effects. Adrenaline elicits profound physiological changes in almost every system of the body. The primary effect of noradrenaline is to stimulate the contraction of small blood vessels and increase the resistance to the flow of blood.

Stress has a pronounced effect upon the circulatory system. Best and Taylor (1955) stated there is a marked reduction in the circulating blood volume of an animal in shock, even though no hemorrhage has occurred. The reduction in the cardiac output and the slowing of the peripheral blood flow occur early in shock and precede the fall in blood pressure. Adrenaline causes vasoconstriction of the splanchnic and cutaneous areas, and dilation of the vessels of the skeletal muscles. It also inhibits the movement of the intestines and increases the rate of respiration, the vasodilation in the muscles increases capillary filtration pressure and transudation of fluid. Any great enlargement of the capillary bed alone would cause a fall in blood pressure without any loss of blood from the vascular system. Continuous infusion of adrenaline in physiological dosage causes a fall in plasma volume.

When an animal is subjected to shock there is a rise in blood sugar resulting from liver glycogenolysis. The blood sugar may rise to extremely high levels. Engel (1952) stated the level which it reaches in any species depends to a considerable degree on how much liver glycogen there is. If the animal has recently eaten and has high liver glycogen, the blood sugar will rise to much higher levels. The rise in blood sugar is most striking during the early phases and lasts well into shock. A fall in blood sugar eventually occurs in the normal animal.

Wiggers (1950) stated that hyperglycemia, once induced, was maintained almost to the terminal stage. The metabolic changes which occur following trauma are dependent upon the nature of the catastrophe, on the physiological stimulation induced, and on the compensatory reactions which follow. The increase in blood glucose appears to start through increased hepatic glycogenolysis; this is followed by the production of more glucose than the tissues can use, though the rate of utilization is also increased. Lactate and pyruvate levels are also slightly raised, but tissue metabolism remains aerobic. During progressive

stages of shock these initial changes are intensified and new ones develop. Blood sugar continues to rise, but after variable periods it falls until hypoglycemia occurs in the terminal stages.

The decline of blood sugar from a maximum appears to be due to intensified utilization by peripheral tissues, to depletion of liver glycogen, and default of hepatic gluconeogenesis, probably associated with inability to metabolize amino acids. Blood amino acids rise progressively, the increase being due in part to greater liberation of amino acids from muscles, but mainly to failure of the liver to metabolize them.

Many factors other than stress influence the blood sugar level in animals. Allcroft (1933) attributed considerable diurnal variation in the blood sugar of lactating cows to the requirements for milk production. Kennedy *et al.* (1939) reported marked diurnal variation in the blood sugar of calves associated with feeding. Reid (1951) reported that blood samples drawn from sheep in the afternoon contained more sugar than those taken in the morning. Fish (1928) and Hodgson *et al.* (1932) reported seasonal variations in blood sugar level in dairy cows. Hewitt (1930) reported blood sugar levels of heifers during estrus as high as 362 milligrams percent. Hodgson *et al.* (1932) reported increases amounting to as much as 40 percent for heifers during estrus. Bate-Smith (1938) suggested that variations in blood sugar level of swine can probably be related to varying degrees of excitability in individual animals.

Fasting and type of ration apparently affects the blood sugar level of ruminants. Hodgson (1932) reported a decrease in blood sugar of heifers fasted for a period of nine days. The decrease was continuous and uniform until the seventh day, at which time a substantial increase was noticed. The blood sugar level decreased to less than 50 percent of its original value during the nine-day study. Forbes (1943) reported that after feeding goats in late pregnancy and at the peak of lactation on hay rations for ten days, the blood sugar level declined steadily from about 50 to 30 milligrams percent.

Administration of adrenaline has been shown to raise the blood sugar level. Courtice *et al.* (1939a) observed, after a subcutaneous injection of adrenaline in the human, a rise in blood sugar, a rise in lactic acid content of the blood, a lowering of the carbon dioxide conbining power of the blood, and a quick rise in the respiratory quotient. A simultaneous subcutantous injection of adrenaline and intravenous injection of insulin produced no change in blood sugar concentration. However, the changes in lactic acid concentration in the blood and the respiratory quotient were practically identical with those of adrenaline alone. An intravenous injection of insulin alone raised the respiratory quotient, signifying an increase of carbohydrate metabolism. There was no accumulation of excess lactic acid in the blood and the effects on the oxygen consumption were slight and inconsistent. Courtice *et al.* (1936) also observed that hyperglycemia in the human, caused by a given subcutaneous dose of adrenaline, was less during exercise than during rest.

The method of adrenaline administration is an important consideration in experimental work. Enikewa (1945) observed that 10 to 50 times as much adrenaline was required to produce toxic effects when administered subcutaneously as when administered intravenously, undoubtedly due to its slower rate of absorption. However, local necrosis may result from subcutaneous absorption. Pekkarinen (1948) reported that large amounts of adrenaline quickly disappear from the circulation. The capillary net is extensive and has a large absorbing and dialyzing surface. Adrenaline is rapidly dialyzed into the tissues in the entire capillary area, where a further splitting takes place. Institute of American Meat Packers *et al.* (1939) failed to produce dark cutting beef with an intravenous injection of 45 milliliters of 1:100 solution of adrenaline. The cattle were slaughtered within a few minutes after injection. The carcasses from treated animals were lighter in color than those of controls but numerous pinpoint hemorrhagic spots occurred throughout the meat. Sokal and Sarcione (1959) found that subcutaneous adrenaline doses of 0.1 mg. per 1 kg. or more consistently produced declines in muscle glycogen of rats. They also concluded that the concentration of adrenaline required to produce glycogenolysis in the liver is at least five to 10 times as high as that effective in the muscle.

Convulsive doses of insulin produce stress and have a very marked effect upon the carbohydrate stores of the body. The normal physiological function of insulin is to enhance glycogen deposition. The Institute of American Meat Packers *et al.* (1939), National Live Stock and Meat Board Committee (1949), and Howard and Lawrie (1956) were able to produce dark cutting beef by injecting massive doses of insulin, ante mortem.

The anterior pituitary, when stimulated, produces an excess of corticotrophin which in turn augments adrenal glucocorticoid secretion. The mechanism by which the anterior pituitary is stimulated to release corticotrophin may involve oxytocin and vasopressin according to Mirsky *et al.* (1955). Observations by Porter and Jones (1956) suggest that blood from the hypophyseal portal vessels contains a substance or substances which accelerate the release of ACTH from the anterior lobe. Schapiro *et tl.* (1956) reported that the release of ACTH involved the hypothalamus.

The discharge of ACTH in response to moderate stress may be completely or partially blocked by the administration of cortical steroids according to Sayers (1951). With increasing intensity of stress the amount of cortical steroid required to suppress pituitary adrenocorticothrophic activity becomes correspondingly greater. Pretreatment with cortical hormone fails to block accelerated discharge of ACTH when the animal is exposed to severe stress. Large toxic doses of adrenaline may have a direct action on the adenohyphophysis to release ACTH.

The response to stress is an involved process. Rhoades *et al.* (1956) stated that nearly every measurable function of the body is affected by trauma. Davis (1956) reported that post combat examinations of attacking infantrymen who went through 18 hours of intense combat in Korea revealed an increased in

steroids and in protein metabolism, smaller ratio of sodium to potassium, and lower eosinophil counts. It was estimated that on the average it took the men about six days to recover physiologically from their intense 18 hour stress in combat. Similar examinations made on men who went through a stress somewhat less intense for five days revealed their physiological profile was completely different. Their adrenal activity was below normal instead of above normal and on the average required 13 days to regain physiological normality. Saroff (1957) found an increase in circulating levels of 17-hydroxycorticosteroids in cattle following the administration of adrenaline or "hot-shot" stimulation.

Rose (1935) and Thomas (1938), in their review of literature regarding the metabolism of creatine and creatinine, reported that creatinuria is a symptom of muscle glycogen depletion. The degree of creatinuria is dependent upon the extent and rapidity with which glycogen is removed rather than upon the absolute amount of glycogen present. Hardy (1952) stated an increased secretion of creatinine following trauma was paralleled by an increase in nitrogen excretion and in adrenocortical activity. Excess liberation of histamine may be related to traumatic shock. West and Todd (1955) stated that histamine depressed blood pressure. Kellaway and Cowell (1922) and Best and Taylor (1955) reported that histamine increased the output of adrenaline from the medulla.

The Endocrine Control of Carbohydrate Metabolism

All body constituents are in a state of flux according to Schoenheimer (1942), being continuously synthesized, interconverted, and degraded. The rates of all these processes are so adjusted that the net concentration of various metabolites within or without the cell remains extraordinarily constant.

Engel (1953) points out that meabolic homeostasis is dependent on the proper supply and distribution of those factors which are concerned with regulation of the rates of metabolic reactions and with the controlled release of energy necessary for the coordination of these reactions. Many factors are involved in this process, among which are vitamins, which serve as coenzymes in enzymatic reactions, and the hormones. The precise mechanism by which hormones influence metabolic processes is as yet unknown. It is generally agreed that hormones do not initiate or halt metabolic reactions, but simply modify the rates of existing reactions. It's not known whether this is brought about by hormones influencing enzyme activity directly or by modifying cell-permeability to specific substrates. Hormones concerned with metabolism are probably being secreted at all times with the amounts of each hormone secreted at any one time being determined in part by the needs of the organism. When either an insufficient or an excessive amount of any one hormone exists, physiological processes are disturbed.

Carbohydrate is being used continuously in the body. It is only periodically renewed. The delivery to the blood, according to Cannon (1932), must not only

be continuous, but must be so adjusted to the demand that there is not an oversupply which results in loss of valuable energy-yielding material from the body or an undersupply which may cause a more or less profound disturbance of the whole organism. Homeostasis of sugar in the blood is maintained by a storage mechanism in the liver and muscle as an intermediary between periodic exogenous supply and constant endogenous need.

The rate of entry of glucose into the circulation is regulated by the activity of the intestine, liver, and cells of the proximal renal tubules. Long (1952a) stated the liver is the most important since it not only releases its preformed stores of glycogen, but manufactures glucose for the needs of the body from smaller molecules derived either from the intermediary metabolism of protein or carbohydrates. Glucose whether derived from dietary sources or furnished to the body by the activity of the liver, is utilized in the tissues by three pathways of metabolism.

1. By transformation to glycogen either in the liver or muscles.
2. Oxidation to carbon dioxide and water.
3. Formation of fatty acids.

Carbohydrate metabolism represents a dynamic balance between blood sugar formation in the liver and its utilization by the body tissues. Soskin (1940) stated any disturbance in carbohydrate metabolism is likely to affect both sides of the equilibrium, whether directly or by secondary compensatory changes. Metabolism of carbohydrates serves as the immediate source of energy for work and synthetic processes. Energy is generated during glycolysis and by way of the Krebs citric acid cycle in which the oxidation of the carbon chain is carried to completion. Carbohydrates entering the metabolic pool either by ingestion or conversion from protein or fat are stored either as glycogen, which is immediately available for energy, or as fat which is an economical form in which to store energy.

Figures 1 and 2 are summaries of the various endocrine factors involved in carbohydrate metabolism which were presented by Engel (1953).

Cannon (1914) showed that adrenaline when liberated in the blood aids in bringing out sugar from the liver's store of glycogen and restores fatigued muscles with their original irritability. Cori and Cori (1928a) reported that adrenaline influences the carbohydrate metabolism of the peripheral tissues, since it leads to the disappearance of muscle glycogen. The increase in liver glycogen after adrenaline injections is explained by the conversion of muscle glycogen into liver glycogen with lactic acid as an intermediary state. Loss of muscle glycogen incurred under different stress conditions is replenished, in part at least, by the liver in terms of glucose.

Cori and Cori (1928b) stated that part of the lactic acid arising from the breakdown of muscle glycogen was carried to the liver where it was reconverted to glycogen. In this way a cycle of carbohydrates is established. Blood sugar derived from liver glycogen is utilized in the muscles and lactic acid derived from

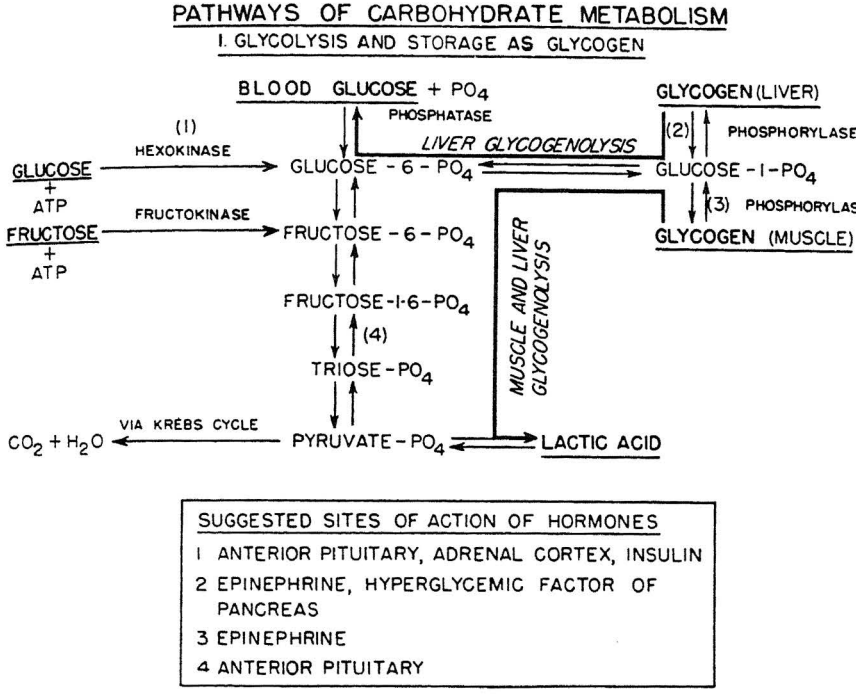

Fig. 1—From Frank L. Engel, Bulletin of the New York Academy of Medicine, Vol. 29, p. 179, 1953.

muscle glycogen is returned to the liver. Adrenaline hyperglycemia develops because the utilization of blood sugar by the liver is diminished in relation to the supply of blood sugar by the liver, even if the supply is not much higher than normal. Cori and Cori (1928c) reported that the absorption of glucose from the intestine of the rat diminished following injection of adrenaline. Cori (1940) states it has been clearly established in experiments in intact animals and on isolated organs that "adrenaline accelerates the reaction glycogen→lactic acid in the muscle and the reaction glycogen→glucose in the liver." Sutherland and Cori (1951) reported that adrenaline was a glycogenolytic agent which increased phosphorylase activity, thus increasing the conversion of glycogen to glucose.

The effect of the administration of adrenaline upon the quantity of liver glycogen varies with the past history of the animal according to Stetten (1940). In a well nourished animal there is a decrease in the quantity of liver glycogen and a rise in the level of blood glucose. In a previously fasted animal there is an insufficient amount of liver glycogen for this effect to be of any importance.

Recent evidence by West (1955) indicates a differential secretion of adrenaline and noradrenaline exists dependent upon the functional requirements of the

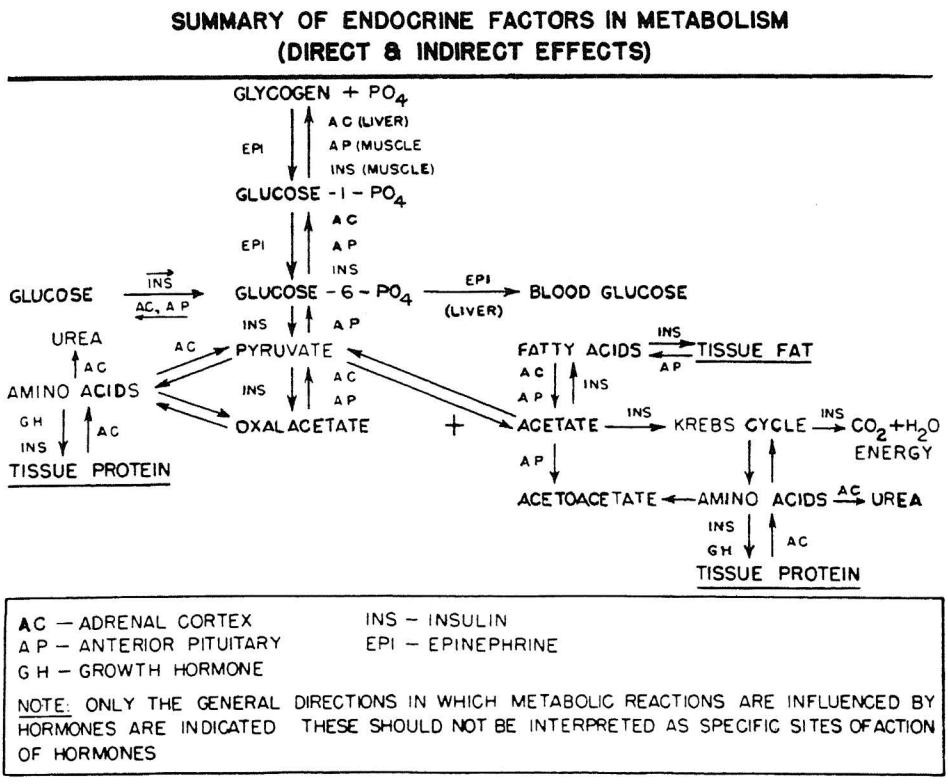

Fig. 2.—From Frank L. Engel, Bulletin of the New York Academy of Medicine, Vol. 29, p. 199, 1953.

animal. Noradrenaline is released during conditions involving circulatory needs while adrenaline becomes available in local metabolic processes where increased regional blood supply is demanded.

Long (1952b) reported that hypoglycemia provokes adrenaline discharge by its excitatory action on the sympathetic centers. The discharge of ACTH that follows adrenaline release enables the organism to call upon the virtually unlimited stores of carbohydrate precursers in the form of the tissue proteins to meet the threat imposed by severe hypoglycemia. Increased secretion of adrenal cortical hormones accelerates the rate of gluconeogenesis from protein. The release of adrenaline, not the level of blood glucose, determines the activity of the pituitary, according to Gershberg and Long (1948).

Engel (1953) cited evidence that insulin, growth hormone and 11-oxycorticosteroids influenced the hexokinase catalyzed phosphorylation of glucose. The 11-oxycorticosteroids inhibit the hexokinase catalyzed reaction while insulin and

the growth hormone overcome this inhibition. When insulin action predominates, glucose utilization will be accelerated. When adrenal and pituitary activity predominate and insulin is decreased, glucose utilization is decreased.

From the reports by Stadie (1954) and Best and Taylor (1955) it appears that insulin increases the permeability of cell membranes to glucose and thus accelerates the transfer of glucose into the cell.

There are many factors which interfere with the action of insulin. Best and Taylor (1955) state that growth hormone, cortisone, and adrenaline are antagonistic to the action of insulin. Toxic products from micro-organism infections may interfere with the action of insulin. All anesthetics interfere with the action of insulin because adrenaline is liberated in most instances and acid products tend to accumulate.

Olsen and Klein (1947) reported that when insulin is injected intravenously there is a transient hyperglycemia. Dudley and Marrian (1923) found that convulsive doses of insulin given to a normal animal decreased liver glycogen and essentially depleted muscle glycogen. Later West and Todd (1955) stated that the injection of insulin into normal animals generally caused a fall in both muscle and liver glycogen and hypoglycemia, because of an increase in the rate of carbohydrate oxidation.

Glucagon raises blood sugar level by releasing glucose from hepatic glycogen according to Pincus and Rutman (1953). Ferner (1953) stated that the relation of the two antagonistic systems, A-and B-cells, as a source of glucagon and insulin respectively, is more important than the total volume of the islet organ in maintaining a physiological blood sugar level.

Long *et al.* (1940) found when C-11 oxygenated cortical steroids were adminstered to rats, liver glycogen, blood sugar and nitrogen excretion increased without significant alterations in muscle glycogen. They attributed the increases in liver glycogen and blood sugar to gluconeogenesis from protein. Engel (1951a) stated the action of the adrenal cortex in nitrogen metabolism was predominately at the level of whole protein, serving to facilitate the mobilization of amino acids, presumably by promoting protein catabolism and inhibiting protein anabolism. Russell (1955) reported that insulin, cortical hormones, and growth hormone all appeared influential in some aspect of the synthesis or breakdown of proteins within most or many tissues.

Engel (1951b) stated that it was not known whether hormones were used up at the site of their action or elsewhere. Since it is a general rule in endocrinology that the administration of a hormone suppresses the endogenous secretion of that same hormone, it may be accepted that any metabolic effect of a hormone achieved in an intact animal is quantitatively or qualitatively an overdosage effect. The measurable effects of a hormone under given conditions may be greatly influenced by the dosage used. These effects may be modified by changing the internal or external environment. Since the functions of the several endocrine glands are interrelated, Sprague (1951) anticipated that the introduc-

tion of excess amounts of a secretory product of one gland might influence the structure or function of another gland.

Use of Tranquilizers to Alleviate Stress.

Tranquilizers comprise a group of drugs which relieve anxiety or tension without inducing sleep or deadening pain. Luther (1958) classified the available tranquilizers into four major chemical categories: (1) rauwolfia alkaloids, (2) phenothiazine derivatives, (3) diphenylmethane derivatives, and (4) substituted propane diols. These various compounds are used extensively in human medicine and the veterinarian and livestock producer are making increased use of these compounds.

Tranquilizers also are being used as growth stimulants. The mechanism by which tranquilizers stimulate growth is not yet apparent. Luther (1958) postulated that small doses of tranquilizing drugs exert a protective effect against environmental stress which does not present itself in the animals' gross behavior. The action of these drugs may conceivably be mediated through the nervous system to the organs of digestion and assimilation.

If the sympathetic portions of the autonomic nervous system are suppressed, an animal does not respond to a stress. Huber (1958) pointed out that tranquilizers act on the lower brain center to diminish anxiety and agitation and thus reduce the intensity of the "alarm reaction," enabling the animal to adjust adequately without going into a stage of hyperexcitement. There is wide variability among animals as to the dosage which can be tolerated without various side effects becoming evident. The side effects of over-dosage are muscle tremors, bradycardia, and increased urination.

Reserpine is a white crystalline compound which is produced from the root of the Rauwolfia plant. Leake (1955), Saniz (1956), and Himwich (1957) reported that reserpine reduced the quantity of the cerebral response, lowered blood pressure, combatted aggressiveness and induced tranquilization. In addition, Meyers (1956) stated that injections of reserpine caused the release of antidiuretic hormone and depressed respiratory rate and adrenal medulla activity.

Leake (1955), Grenell (1957), and Himwich (1957) stated that chloropromazine depressed the hypothalamus. Dobkin *et al.* (1954) found that small doses of chloropromazine block all sympathetic vasopressor reflexes, while large doses block vagal reflexes. The release of adrenaline, acetycholine, and histamine was suppressed. Ohler (1954) stated chloropromazine induced adrenal ascorbic acid depletion, which represented an inhibition of stress-induced ACTH secretion. Lehmann and Harrahan (1954) observed that the effects of chloropromazine persisted for about 48 hours after treatment. In animals, it produced a type of depression which increased progressively with the dosage.

Other effects are strong antiemetic properties, inhibition of the secretion of gastric juice and production of hyperthermia. Lancaster and Jones (1954) found

that chlorpromazine had no significant effect on fasting blood sugar level following insulin injection.

Smith *et al.* (1955) cited evidence that thorazine depressed activity of the central nervous system.

Ritchie (1958) reported that swine injected with chloropromazine gave no indication of pain or irritation at the site of injection. When it was given intravenously, the animal appeared dazed and its respiratory rate was increased for about 15 minutes. If undisturbed, the animal would pass into a deep sleep from which it could be aroused only by vigorous stimuli. When chloropromazine was given intramuscularly, the effect was marked and even vicious boars became docile.

EXPERIMENTAL PROCEDURE

A total of 120 cattle were utilized in this study. The cattle were yearlings and two-year olds. Seventy were Shorthorns, four Herefords and 40 of dairy breeding. The Shorthorn and Hereford cattle ranged in grade from high Standard to average Choice. Those of mixed dairy breeding ranged from low Utility to low Standard.

Methods Used to Stress Animals

Cattle were stressed by excitement and by administration of adrenaline. The cattle which were excited were prodded periodically with an electric "hot shot," while closely confined in a holding pen to avoid injury.

Two adrenaline preparations were used; a suspension of basic adrenaline in peanut oil (2 mg. adrenaline/1 ml.) and an aqueous solution of adrenaline, chlorobutanol, sodium chloride and sulfer dioxide saturated with carbon dioxide (1 mg. adrenaline/1 ml.). Both preparations were satisfactory for use in this study.

The first method of administering adrenaline was intramuscular injection in the shoulder in the area of the scapula. Edema occurred in most of the animals in the area where adrenaline was injected. It was later found that subcutaneous injections of adrenaline in the shoulder would stress the animals comparable to intramuscular injections and would not cause intramuscular edema. Adrenaline injected intramuscularly or subcutaneously is absorbed slowly and produces stress comparable to a prolonged period of excitement. It is not practicable to administer adrenaline intravenously to cattle to simulate a prolonged period of stress because adrenaline in the blood is quickly absorbed and dialyzed in the tissues (Pekkarinen, 1948). Therefore, to simulate a prolonged period of stress, adrenaline would have to be administered slowly and continuously over a prolonged period.

Methods Used to Counteract Stress

Hydrocortisone was administered intramuscularly in the area of the scapula to 23 cattle prior to and/or after the administration of adrenaline to determine if an exogenous source of this hormone would aid in the restoration of muscle glycogen. Dosage ranged from 30 to 160 mg. per 100 pounds body weight.

Protamine zinc insulin was administered intramuscularly to three cattle prior to being stressed by excitement. One unit of insulin was given per pound of body weight.

Tranquilizers were given to 34 cattle of mixed dairy breeding to determine if these compounds would alleviate stress. Three cattle were used as controls. Not all animals were slaughtered following treatment. Some were used later for other treatments before being slaughtered. Thorazine HCL (chlorpromazine HCL) was given to 14 cattle, Diquel (phenothiazine HCL) to 12 cattle, and Serpasil (crystalline alkaloid of Rauwolfia root) to five cattle. The dosages, time intervals and method of administration are presented in the results section.

Collection of Blood and Urine Samples and Methods of Analysis

Blood samples were taken from the jugular vein. The blood was collected in a flask to which had been added a small amount of heparin to prevent coagulation.

Duplicate 1 ml. aliquots of the blood were then placed immediately in 50 ml. test tubes, to which 9.5 ml. of 0.3 N. barium hydroxide was added while rotating the test tube. After adding the barium hydroxide, 9.5 ml. of a 5 percent zinc sulfate solution was added while mixing. The contents of each test tube were then filtered through Whatman No. 1 paper or equivalent. A fraction of a drop of toluene was added to the protein-free filtrate to preserve the sample. The samples were then stored at 45°F. until all samples from each animal had been collected. All blood samples from individual animals were analyzed at the same time. The Nelson-Somogyi method for blood glucose as published by the American Association of Clinical Chemists (1953) was followed in making the blood sugar determinations. The absorbance was measured at 540 mu. by means of a Bausch & Lomb Spectronic 20 Colorimeter, using a blank for setting the zero. A standard glucose curve was used to calculate the amount of glucose present in the sample.

Urine samples were collected and analyzed for glucose according to the previously described Nelson-Somogyi method for glucose. In instances where the sugar content of the urine was high, dilutions were made of the barium-zinc filtrate before the determinations were made.

Urine samples for nitrogen determinations were stored at 45° F. in rubber stoppered Erlenmeyer flasks until all samples from each animal were collected. Duplicate 10 ml. aliquots were used to determine total nitrogen according to the official A.O.A.C method (1950) using salicylic acid.

Determination of Muscle pH

Muscle pH was determined 24 hours post mortem. A Beckman pH meter, Model G, was used to make the determinations. The instrument was standardized to pH 7.0 prior to each reading, using a phosphate buffer. The glass electrodes were inserted into the longissimus dorsi muscle at the 12th rib and readings taken to the nearest 0.05 pH unit.

Determination of Muscle and Liver Sugar

The Hall method as described by Henrickson (1954) was used to determine glucose in the muscle and liver. Muscle samples from the longissimus dorsi and liver samples were taken two to three hours post mortem, wrapped in aluminum foil and frozen. Later the samples were finely ground while frozen.

Ten grams were placed in a 50 ml. centrifuge tube with 30 ml. of distilled water and dispersed well with a stirring rod. The tubes were placed in a boiling water bath for five minutes and the contents stirred frequently. The tube and contents were centrifuged for five minutes and the supernatant liquid decanted through a Whatman No. 12 filter paper into a 100 ml. volumetric flask. The residue was washed twice with boiling distilled water and the washings poured through the filter and collected in the volumetric flask. The filtrate was made up to 100 ml. volume with distilled water.

One ml. of cadmium sulfate reagent was mixed well with 8 ml. of the extract. One ml. of 0.75 N. sodium hydroxide was added slowly while mixing, followed by vigorous shaking. After standing a few minutes the contents were filtered through a dry quantitative filter.

Two ml. of the cadmium-treated filtrate and 2 ml. of alkaline copper tartrate were pipetted into a Folin sugar tube. The tubes were placed in a vigorously boiling water bath for 15 minutes, then chilled immediately in cold water without stirring. Two ml. of phosphomolybdic acid reagent were added and mixed well to completely dissolve the precipitate. The solution was diluted to 25 ml. with distilled water and read in a Bausch & Lomb Spectronic 20 Colorimeter at 420 mu. A standard glucose curve was used to culculate the amount of glucose present in the sample.

Sensory Panel Procedure

The left sides of carcasses from 33 animals used in this study were aged three, six or 14 days at 36°F. Rib steaks (cut ¾ inch thick) were removed at the end of the aging period, packaged, frozen, and stored at 0°F. for three to four days prior to the taste panel evaluation.

The rib steaks were removed from 0°F. storage, placed in a 36° F. cooler and allowed to thaw 24 hours prior to cooking. The weight of each steak was recorded to the nearest gram before and after thawing to obtain drip loss. The gas broiler was preheated one hour to 350° F., using a Micromax Temperature

Recorder to regulate the temperature. Thermocouples were placed in the center of each steak and the rise in temperature was recorded. No seasoning was added to the steaks. Steaks were placed on the broiler grill approximately five inches from the flame. On reaching an internal temperature of 104° F., the steaks were turned over and allowed to continue broiling to an internal temperature of 160° F. Cooking loss was determined by weights taken prior to and after broiling.

A 1-inch segment was carved from the center of each steak and cut into six individual servings for the taste panel. Each member of the committee was served a warm sample from the same relative position of each steak throughout the experiment. Evaluations, using the hedonic scale method, were made for aroma, flavor, juiciness and tenderness. The values ranged from a score of 1 (very undesirable) to 7 (very desirable).

RESULTS

Effect of Excitement on Carcass Characteristics

Four cattle were used to determine the effect of excitement upon carcass quality. The cattle were held 24 hours ante mortem in a holding pen at the University abattoir. No feed or water was given during this period. During the 24-hour ante mortem period animals No. 12 and 13 were handled and slaughtered with minimum excitement, while animals No. 14 and 15 were prodded periodically with an electric "hot shot." The prodding was carried out for approximately eight hours.

It can be seen from data in Table 1 that the excitement induced by the electrical stimulation had a pronounced effect upon the resulting pH and color of the muscle. Carcasses from the two stressed animals were sticky and gummy and were considered extreme examples of dark cutting beef. The control animals

TABLE 1--THE EFFECT OF PERIODIC EXCITEMENT FOR 24 HOURS ANTE MORTEM ON BEEF CARCASS CHARACTERISTICS

Animal No.	Live Weight	Carcass Grade	Ante mortem Treatment	pH of Carcass[1]	Color of Muscle[2]
12	900	Good	Control	5.4	A- 4
13	850	Choice	Control	5.4	A- 4
14	765	Choice	Electric "hot shot"	6.7	A-10
15	860	Good /	Electric "hot shot"	6.4	A-10

[1] Determined in longissumus dorsi at 12th rib.

[2] Munsell Color Paddles, Meat Scale A.

produced bright cutting carcasses. The color of the muscle from the two stressed animals was brownish black, while that of the controls was a light cherry red.

This experiment demonstrated that strong emotional excitement inflicted for 24 hours ante mortem will produce dark cutting beef. Excitement arouses the sympathetic nervous system. Secretory impluses are then transmitted via the splanchnic nerves to the adrenal medulla, causing an increased amount of adrenaline to be released (Selye 1950). Adrenaline, when released into the blood stream, is carried to all parts of the body and affects many physiological processes (Best and Taylor 1955). Phosphorylase in the muscle and liver is activated by adrenaline and produces hyperglycemia and glycosuria (Sutherland and Cori, 1948). These are the series of reactions Cannon (1914) referred to which enable an animal to run or fight for its life. If these physiological reactions are stimulated over an extended period, exhaustion will eventually ensue. As demonstrated in this experiment, the glycogen stores of the stressed animals were greatly diminished due to increased adrenaline release.

It is commonly believed that all animals do not respond equally to a given stimulus. In this experiment the carcass from animal No. 14 had a higher pH than that of animal No. 15, indicating less glycogen present in this carcass at the time of slaughter for conversion to lactic acid. The difference between these two animals may be attributed to one or more of the following factors: a difference in the original glycogen stores; the sympathetic-adrenal mechanism of animal No. 14 was more easily aroused; the pituitary-adrenal cortex defense mechanism of animal No. 14 was less efficient in replenishing glycogen stores; or animal No. 15 was more successful in avoiding contact with the source of electrical shock.

Effect of a Short Period of Excitement on Blood Sugar and Carcass Characteristics.

There are volumes of literature dealing with stress and the effect of various kinds of stress on blood sugar levels. To determine the effect of a short period of excitement, such as might be encountered in loading cattle, two cattle were periodically excited with an electric "hot shot" for approximately two hours. Animal No. 33 was placed on the experiment 41 hours ante mortem and animal No. 44 65 hours ante mortem. Samples for blood sugar analysis were taken prior to excitement, after two hours of excitement, and at the time of slaughter. In addition to the original two-hour period of excitement, both animals were subjected to 30-minute periods of excitement 10 and six hours ante mortem. During the entire period of ante mortem treatment the cattle were held in a holding pen at the University abattoir. Feed and water were available to the cattle during the entire period. Both animals seemed well contented except during the time when they were prodded with the electric "hot shot."

Table 2 indicates that the short periods of intermittent excitement had no recognizable effect upon carcass characteristics. Both of the carcasses cut bright

TABLE 2--THE EFFECT OF TWO HOURS OF EXCITEMENT[1] ON BLOOD SUGAR AND BEEF CARCASS CHARACTERISTICS

Animal Number	Live Weight	Carcass Grade	pH of Muscle[2]	Color of Muscle[3]	Muscle Sugar Percent	Liver Sugar Percent	Blood Sugar (mg./100 ml.)
33	780	Choice-	5.5	A-4	0.156	2.73	27.3[4] 58.2[5] 37.0[6]
44	780	Choice	5.5	A-3	0.125	3.25	31.9[7] 61.5[8] 34.8[6]

[1] Excitement induced by electric "hot shot".
[2] Determined in longissimus dorsi at 12th rib.
[3] Munsell Color Paddles, Meat Scale A.
[4] Forty-one hours ante mortem prior to excitement.
[5] Thirty-nine hours ante mortem after two hours excitement.
[6] Time of slaughter.
[7] Sixty-five hours ante mortem prior to excitement.
[8] Sixty-three hours ante mortem after two hours excitement.

with pH values near the isoelectric point. The water-extractable reducing sugars of the longissimus dorsi were in agreement with values for light colored beef reported by the National Live Stock and Meat Board Committee (1949). Excitement caused an approximate two-fold increase in blood sugar in both animals. However, this increase in blood sugar did not have any apparent effect on subsequent carcass characteristics. These results indicate that short periods of intermittent excitement will not produce dark cutting beef. Stress must be prolonged to produce dark cutting carcasses.

Effect of Adrenaline on Carcass Characteristics

It has been established by many investigators that when an animal is subjected to stress there is an increase in the secretion of adrenaline by the adrenal medulla. Hyperglycemia and glycosuria occur due to the increased secretion of adrenaline. This process rapidly depletes the glycogen stores of body.

The effect on beef quality of injecting adrenaline five hours ante mortem: Two cattle which had been fasted for 24 hours were injected with adrenaline 5 hours prior to slaughter. Table 3 gives dosages and methods of administering the adrenaline. Marked symptoms of stress were noted within 1 hour in the animal given 5 milligrams intramuscular injection per 100 pounds body weight. The animal given 2.5 milligrams subcutaneously per 100 pounds body weight did not show marked symptoms of stress until approximately two hours after the injection. The characteristic symptoms of stress produced were frequent urination, dyspnea, restlessness, and trembling. Effects of the adrenaline stress were still very apparent at the time the animals were slaughtered. As indicated by the pH values, both the 5.0 and 2.5 milligram injections of adrenaline reduced the

TABLE 3--EFFECT ON CARCASS CHARACTERISTICS DUE TO ADRENALINE ADMINISTERED TO BEEF CATTLE FIVE HOURS ANTE MORTEM

Animal No.	Live Weight	Carcass Grade	Ante Mortem Treatment	pH of Carcass[1]	Color of Muscle[2]
17	860	Choice-	5 mg. adrenaline/100 lbs. body wt.[3]	5.8	A-7
18	785	Choice-	2.5 mg. adrenaline/100 lbs. body wt.[4]	5.6	A-5

[1] Determined in longissimus dorsi at 12th rib.

[2] Munsell Color Paddles, Meat Scale A.

[3] Adrenaline in oil (1:500 dilution) injected intramuscularly.

[4] Adrenaline aqueous solution (1:1000 dilution) injected subcutaneously.

muscle glycogen stores. The color of the muscle 24 hours post mortem was shady. Even though these animals were rather severely stressed, neither produced a typical dark cutting carcass. These results indicated that a longer period of stress was required to deplete the muscle glycogen sufficiently to cause the carcass to cut dark.

The effect of injecting high levels of adrenaline over a 24-hour ante mortem period: Since injection of adrenaline five hours ante mortem did not produce dark cutting carcasses, three cattle were next given high levels of adrenaline over a 24-hour period. The cattle were deprived of feed and water during the ante mortem stress period. The levels of adrenaline administered and the resulting carcass color and pH values are in Table 4. Stress symptoms produced were the same as those described previously. Animals No. 19 and 21 were stressed on the same

TABLE 4--EFFECT ON CARCASS CHARACTERISTICS DUE TO ADRENALINE ADMINISTERED INTRAMUSCULARLY TO BEEF CATTLE DURING A 24 HOUR ANTE MORTEM PERIOD

Animal Number	Live Weight	Carcass Grade	Adrenaline (mg./100 lbs. body wt.[1])	Hours Ante Mortem	pH of Muscle[2]	Color of Muscle[3]
19	755	Good	2.6	24	6.75	A-10
			2.6	12		
			4.0	2		
21	715	Good	2.8	24	6.4	A-10
			2.8	12		
			4.2	2		
24	560	Good	3	24	6.35	A-10
			3	13		

[1] Adrenaline in oil (1:500 dilution).

[2] Determined in longissimus dorsi at 12th rib.

[3] Munsell Color Paddles, Meat Scale A.

date and animal No. 24 at a later date. Figure 3 shows rib steaks from the carcasses of animals 19 and 21.

Carcasses from the three animals cut dark; all were considered extreme examples of dark cutting beef. The muscles of all three carcasses were very sticky and gummy. These results demonstrated that severe stress for 24-hours will diminish the glycogen stores until an insufficient amount of glycogen remains to produce the desired biochemical changes necessary for bright cutting beef.

The effect of administering varied levels of adrenaline over a 24-hour period and immobilizing with carbon dioxide. To determine the minimum level of adrenaline that would produce a dark cutting carcass, three cattle were given different dosage levels of 1, 2, and 3 milligrams of adrenaline per 100 pounds body weight during a 23-hour ante mortem period. The cattle were held in their accustomed feed lot until the day of slaughter. Feed and water were provided during the stress period. Adrenaline was injected subcutaneously. Injections were given 23, 15, and seven hours ante mortem. One-third of the total dosage was injected at each of the time intervals.

The more noticeable signs of stress were observed in the animal that received the 3-milligram level. However, only slight dyspnea, restlessness, and trembling were observed in this animal.

Another animal was immobilized with carbon dioxide and served as a control. The objective of immobilizing with carbon dioxide was to determine if anoxia could be produced in the muscle to the extent that it would affect the subsequent color of the muscle. Dark cutting beef has been reported deficient in oxygen (Hall *et al.* 1944b). A mask connected to a spirometer was placed over the animal's muzzle to administer the carbon dioxide. The spirometer was then filled with carbon dioxide from a connecting cylinder. After the spirometer was filled with carbon dioxide the animal was forced to breathe the gas for four minutes before the mask was removed. The animal was then hoisted and bled. After breathing the carbon dioxide for three minutes the animal's corneal reflex no longer functioned. At the time of hoisting, the heart rate of the animal had decreased to approximately one stroke per three seconds.

From Table 5 it is concluded that immobilizing with carbon dioxide did not affect the subsequent color of the muscle. The 1-milligram level of adrenaline did not appreciably affect the color of the muscle. However the amount of reducing sugar present in the muscle and pH of the muscle indicated that muscle glycogen had been diminished. The 2-milligram level of adrenaline produced a shady cutting carcass while the 3-milligram level produced a dark cutting carcass. As the level of adrenaline was increased the color of the muscle became progressively darker and the pH values of the muscle were higher. There was a further reduction in muscle sugar as the level of adrenaline was increased as compared to the control and to the animal that received the 1-milligram level. The

TABLE 5--EFFECT ON CARCASS CHARACTERISTICS DUE TO VARIED LEVELS OF ADRENALINE ADMINISTERED SUBCUTANEOUSLY TO BEEF CATTLE DURING A 23 HOUR ANTE MORTEM PERIOD

Animal Number	Live Weight	Carcass Grade	Ante Mortem Treatment	pH of Muscle[2]	Color of Muscle[3]	Muscle Sugar (percent)	Liver Sugar (percent)
25	775	Good	Control	5.45	A-4	0.375	3.44
26	700	Good	1 mg. adrenaline/ 100 lbs. body wt.	5.55	A-4	0.150	2.18
27	700	Good	2 mg. adrenaline/ 100 lbs. body wt.	5.75	A-8	0.125	3.58
28	700	Good	3 mg. adrenaline/ 100 lbs. body wt.	6.15	A-10	0.088	2.75

[1] Control was immobilized with CO_2. The adrenaline was administered as follows: One third of the total dosage at 23, one-third at 15, and one third at 7 hours ante mortem respectively.

[2] Determined in longissimus dorsi at 12th rib.

[3] Munsell Color Paddles, Meat Scale A.

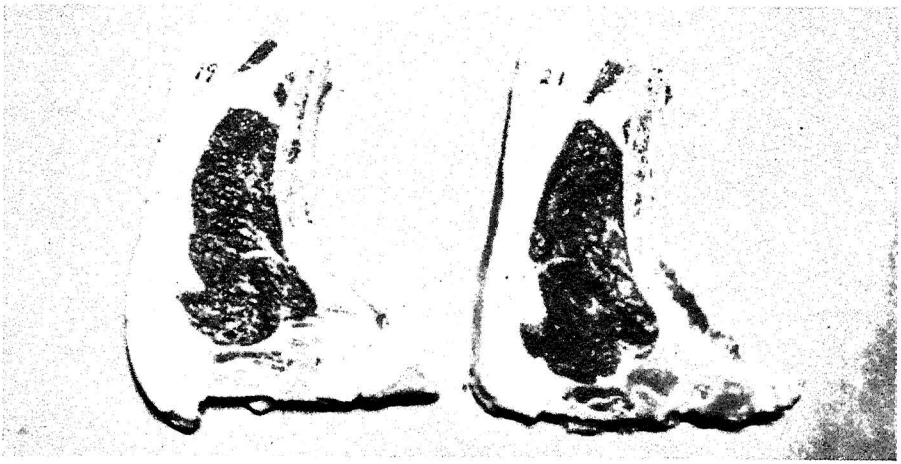

Fig. 3—Dark Color of the longissimus dorsi muscle of beef as a result of the administration of high levels of adrenaline to the live animal. Steaks 19 and 21 were taken from cattle which received 9.2 and 9.8 mg. adrenaline per 100 lbs. body weight during the 24-hour ante mortem period.

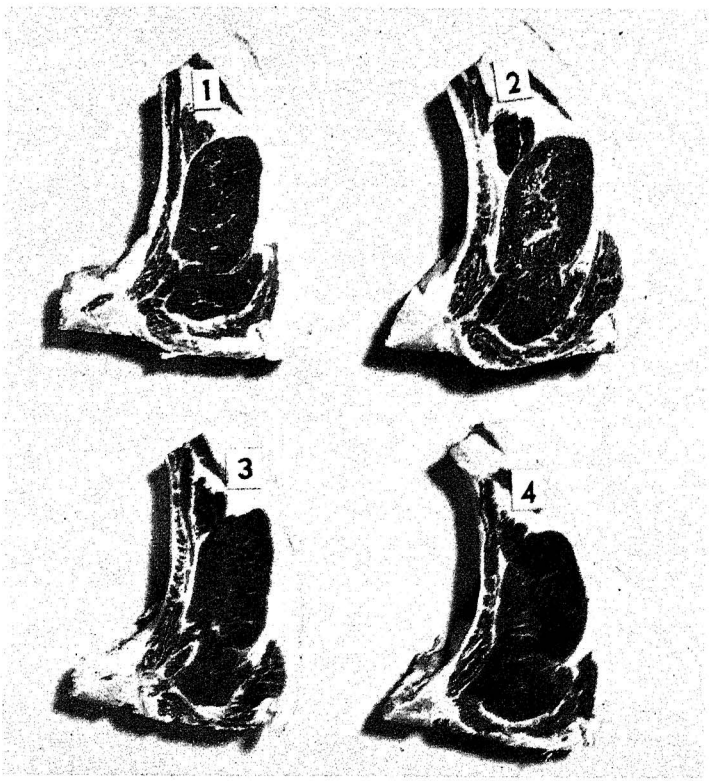

Fig. 4—The effect of varied levels of adrenaline on the color of the longissimus dorsi muscle of beef. Animal 1 was a control and animals 2, 3, and 4 received 2, 3, and 4 mg. adrenaline per 100 pounds body weight during 24-hour ante mortem period.

amount of reducing sugar in the liver samples varied among treatments and did not necessarily decrease as the level of adrenaline was increased.

There was an excessive amount of residual blood in the flank, diaphragm and round muscles of the carcass from the animal that was immobilized with carbon dioxide.

The effect on carcass characteristics of adrenaline administered to beef cattle at varied ante mortem periods: Twenty-six cattle were used to determine the effect of administering 2.0, 2.6, 2.8, 3.0, 4.0, and 5.0 milligrams of adrenaline per 100 pounds body weight on the subsequent carcass characteristics. All animals were given feed and water up to the date of slaughter. Table 6 lists the levels of adrenaline and the hours prior to slaughter the adrenaline was given.

Administration of 3 milligrams of adrenaline per 100 pounds body weight 22 to 30 hours ante mortem produced carcasses with pH values ranging from 5.7 to 6.25. Color of the muscle was related directly to the pH of the muscle; the higher the pH, the darker the color. Texture of the muscle became more sticky at the higher pH values. Figure 4 shows rib steaks from a control animal and three animals that received 2.0, 3.0, or 4.0 milligrams of adrenaline per 100 pounds body weight.

Less reducing sugar was present in the muscle of the animals given adrenaline than in the control animal. Liver reducing sugar varied among animals. In some instances liver samples from animals given adrenaline contained more sugar than the control. The presence of adrenaline may have caused increased glycogen deposition in the liver. Cori and Cori (1928b) reported that rats injected with adrenaline deposited more liver glycogen than control rats due to a decreased utilization of blood sugar in the peripheral tissues.

In some instances it was observed that more highly finished animals were more adversely affected by a given level of adrenaline than animals with less finish. Adrenaline affects the glycogen stores of the muscle and liver and has no affect on the fat deposits. When a given level of adrenaline was given on a body weight basis, animals with more finish received more adrenaline per unit of muscle. This explains in part the variation among animals given the same amount of adrenaline per unit body weight. Probably differences in the amount of glycogen present in the muscles of the various animals would also influence the final results attained.

Variation among animals in the activity of the adrenal cortex in response to stress would influence the amount of glycogen present in the muscle at the time of slaughter. When an animal is stressed an increased secretion of adrenaline occurs, which affects the hypothalamus to release a substance (Porter and Jones 1956). This substance released by the hypothalamus is carried in the blood to the anterior pituitary and stimulates the release of ACTH. ACTH is then carried in the blood to the adrenal cortex and there stimulates the release of glucocorticoids, which promote gluconeogenesis. This physiological mechanism enables

TABLE 6--EFFECT ON CARCASS CHARACTERISTICS DUE TO ADRENALINE ADMINISTERED SUBCUTANEOUSLY TO BEEF CATTLE AT VARIED ANTE MORTEM PERIODS

Animal Number	Live Weight	Adrenaline (mg./100 lbs. body wt.)[1]	Hours Ante Mortem	Carcass Grade	pH of Muscle[2]	Color of Muscle[3]	Muscle Sugar (percent)	Liver Sugar (percent)
29	720	-	--	Good	5.45	A-4	0.139	2.73
30	850	-	--	Choice	5.45	A-5	---	---
31	600	-	--	Good	5.50	A-4	---	---
1	800	-	--		5.45	A-4		
2	750	1	24		5.55	A-5		
		1	12					
32	550	2	22	Good/	5.75	A-7	0.069	3.00
33-1	680	3	22	Good-	6.0	A-9	0.063	1.54
34	680	3	24	Std. /	5.70	A-7	0.075	2.40
35	720	3	24	Good	5.7	A-7	0.088	2.18
36	765	3	24	Good/	6.0	A-9	---	---
37	640	3	24	Good	5.9	A-9	---	---
38	630	3	24	Choice-	6.15	A-10	---	---
39	600	3	24	Good/	6.20	A-10	---	---
3	750	1.5	24		5.85	A-8		
		1.5	12					
4	750	2	24		5.75	A-7		
		2	12					
40	680	2	27	Good	6.0	A-9	---	---
		1	15					
41	800	2.5	27	Choice	6.1	A-9	0.087	3.0
		2.5	16					
42	680	2	30	Good	6.25	A-10	---	3.12
		1	18					
43	780	3	45	Good	5.95	A-8	0.044	1.93
44	565	3	48	Good	5.6	A-7	---	---
45	725	3	48	Choice-	5.65	A-7	---	---
46	760	2.6	68	Good/	5.45	A-4	0.048	2.0
47	855	2.8	68	Choice-	5.55	A-4	---	1.85
48	720	3	72	Choice	5.6	A-7	---	---
49	750	3	72	Choice	5.7	A-7	---	---
50	860	2.8	96	Good/	5.45	A-4	0.088	3.75

[1] Adrenaline Aqueous solution (1:1000 dilution).

[2] Determined in longissimus dorsi at 12th rib.

[3] Munsell Color paddles, Meat Scale A.

an animal to recover when subjected to stress. The efficiency of this mechanism governs an animal's rate of recovery from stress. The administration of adrenaline would activate this physiological process of recovery. Animals held under optimal conditions following stress will regain physiological normality. However the severity and duration of the stress will influence the rate of recovery (Davis 1956).

Blood samples were taken from animals No. 34 and 35 prior to the injection of adrenaline and at four, seven, 13 and 24 hours after injection of adrenaline. These samples were analyzed for sugar; results are presented in Figure 5. The initial blood sugar level of animal No. 35 was higher than that of animal No. 34. This difference may be due to excitement of the animal. Within four hours after adrenaline was injected a five-fold increase in blood sugar occurred in animal 34. This rise in blood sugar was due to glycolytic action induced by the adrenaline. Cori (1940) reported that "adrenaline accelerates the reactions:

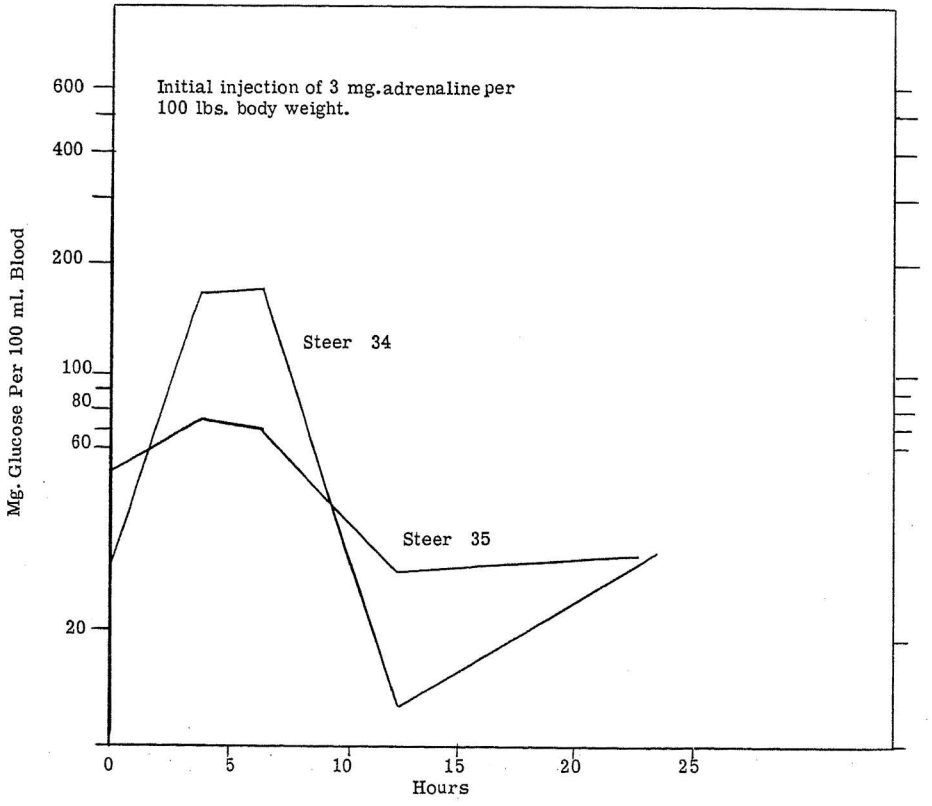

Fig. 5—The Effect of Subcutaneous Injections of Adrenaline on Blood Sugar Levels of Beef Cattle.

glycogen → lactic acid in muscle; and glycogen → glucose in the liver." The maximum blood sugar level measured in animal No. 35 was less than one-half that found at the same time in animal No. 34. A higher peak of blood sugar in animal No. 35 may have occurred between the intervals at which the blood samples were taken or there may have been less glycogen present initially. Stetten (1949) reported that the level to which blood sugar rose due to a given stress was dependent upon the amount of glycogen present initially in the liver and muscle. The level to which blood sugar rises will also vary among animals due to differences in the renal threshold. The blood sugar level was the same in both animals at the time of slaughter and the resulting pH and color of the muscle was the same. These results indicate that animals react physiologically different to the same stress.

Blood and urine samples were also taken from animals No. 40 and 42 before adrenaline was injected and at various intervals after adrenaline was injected. The blood samples were analyzed for sugar and the urine samples for nitrogen. Figure 6 gives results. There was a considerable variation between the two animals in both blood sugar and urinary nitrogen level. The blood sugar level of animal No. 42 remained at a high level for a longer period after the adrenaline injection than did that of animal No. 40. The darker color and higher pH of the muscle from animal No. 42 may be attributed partly to additional stress the animal was under due to a leg injury. The animal injured its leg in the holding chute while we were taking a blood sample. The greater increase in nitrogen level of animal No. 42 during the later phase of the stress period can be attributed to greater gluconeogenesis activity promoted by glucocorticoids.

Blood and urine samples were also taken from animal No. 36 before adrenaline was injected. The blood samples were analyzed for sugar and the urine samples for sugar and nitrogen. Figure 7 depicts the results. A precipitous rise in blood sugar and urine sugar occurred following the injection of adrenaline. The high level of sugar in the urine occurred when the blood sugar level exceeded the renal threshold. Spilling of sugar over into the urine and subsequent elimination accounts for the disposition of a major portion of the glycogen lost from the muscle stores during stress. The initial decline in urinary nitrogen was probably due to an increased urine volume and the later increase due to nitrogen coming from the deaminization of amino acids and protein by the glucocorticoids.

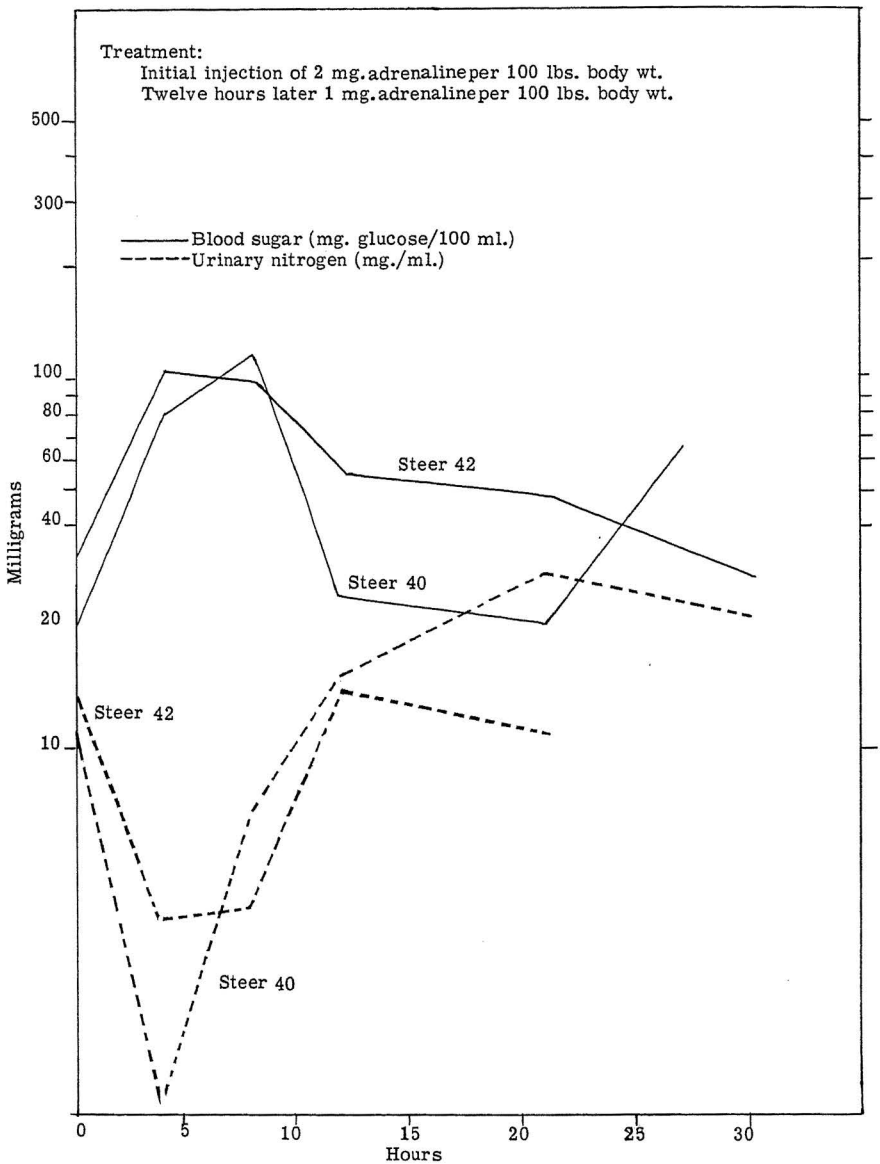

Fig. 6—The effect of subcutaneous injections of adrenaline on blood sugar and urinary nitrogen levels of beef cattle.

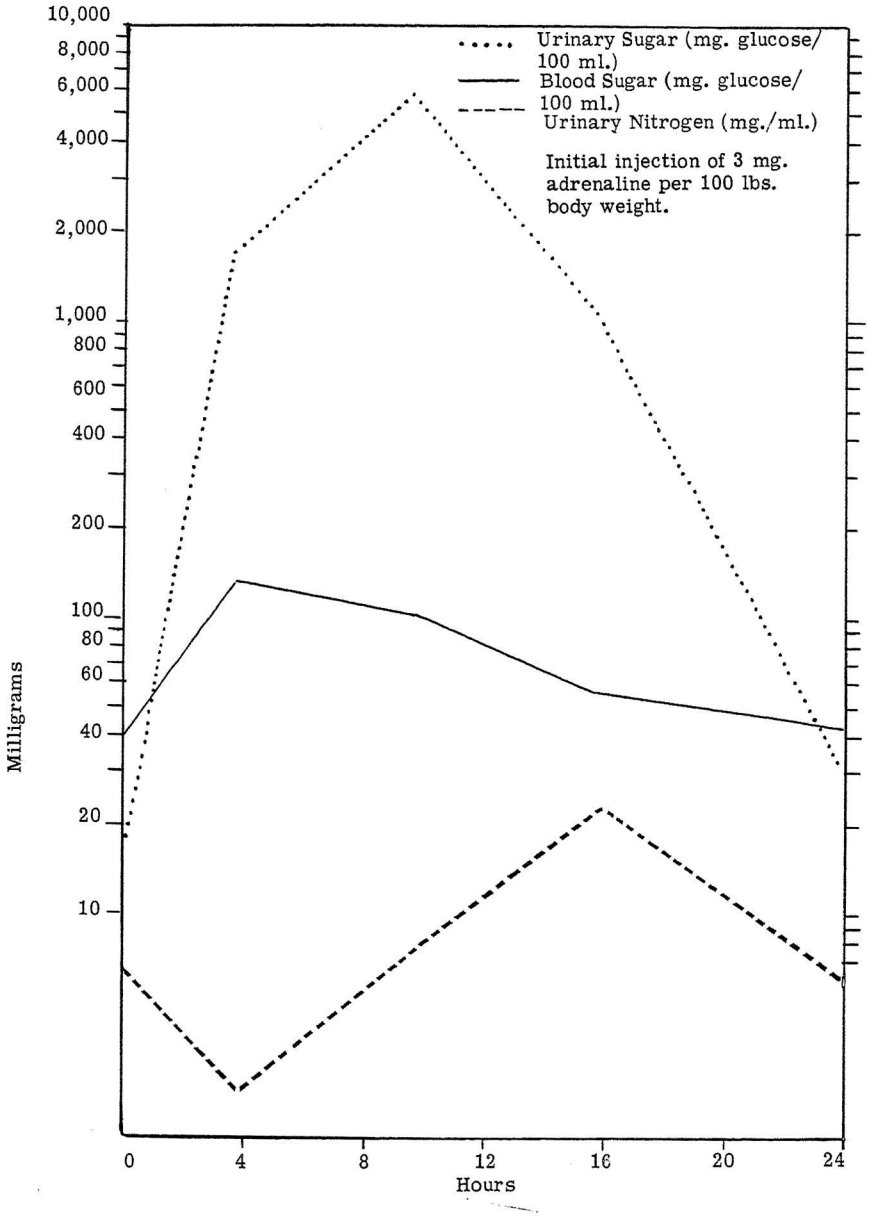

Fig. 7—The effect of a subcutaneous injection of adrenaline on urinary sugar, blood sugar, and urinary nitrogen levels of beef cattle.

Effect of adrenaline administered to cattle of dairy breeding on carcass characteristics: Nine cattle were used to determine the effect varied levels of adrenaline would have on subsequent carcass characteristics of low grade cattle. The cattle used were Utility grade, with the exception of No. 57 which was low Standard. The animals were divided into three equal groups; two animals in each group received adrenaline and one served as a control. Levels of adrenaline given and the color and pH values of the subsequent muscle of the carcasses are given in Table 7. The carcasses of control animals were brighter in color and had lower

TABLE 7--EFFECTS OF VARIOUS LEVELS OF SUBCUTANEOUS ADRENALINE INJECTIONS ON CARCASS CHARACTERISTICS OF LOW GRADE BEEF

Animal Number	Live Weight	Time of Ante Mortem Injection (hrs.)	Treatment (mg./100 lb.)	Color of Muscle	pH of Loin Eye
Group I:					
51	730	No injection	Control	A- 7	5.5
52	715	24 hours	3.5	A- 9	5.9
53	685	24 hours	3.5	A- 9	5.8
Group II:					
54	755	24 and 12 hrs.	6.0*	A-10	5.9
55	765	24 and 12 hrs.	6.0*	A-10	5.9
56	880	No injection	Control	A- 5	5.5
Group III:					
57	790	24 hours	3.0	A- 5	5.45
58	785	24 and 12 hrs.	6.0*	A-10	5.8
59	690	No injection	Control	A- 7	5.4

*One-half of the dosage given at each time interval.

pH values, with the exception of No. 57, than the carcasses of treated animals. There was a considerable variation in color of muscles throughout the carcasses of the treated animals. In several instances throughout the study the various muscles in the chuck, flank and round were darker than the longissimus dorsi. The color of the longissimus dorsi is not always a true representative of the color of all muscles throughout the carcass. This variation in color, often referred to as two-toned color, can be attributed to differences in pH and possibly to some extent to differences in myoglobin. It has been pointed out by many investigators that all muscles do not contain the same amount of glycogen and that the rate of glycogen loss during stress is not the same for all muscles.

Blood samples were taken from six of the cattle and analyzed for blood sugar. Table 8 lists the blood sugar values. In all instances there was a precipitious rise in blood sugar following the injection of adrenaline. This rise in blood

TABLE 8--EFFECTS OF SUBCUTANEOUS INJECTIONS OF ADRENALINE ON THE BLOOD SUGAR CONTENT OF UTILITY GRADE CATTLE

Animal Number	Treatment*	Blood Sugar (mg./100 ml.)	Hours After Injection
Group I:			
51	Control	57.0	0
		57.0	4
		57.0	24
52	3.5	48.5	0
		181.0	4
		52.0	24
53		47.0	0
		157.0	4
		57.0	24
Group II:			
54	6.0	55.0	0
		205.0	5
		95.0	12
		52.4	24
55	6.0	57.0	0
		130.0	5
		80.0	12
		65.0	24
56	Control	75.0	0
		75.0	5
		75.0	12
		75.0	24

*Treatment presented as milligrams of adrenaline per 100 pounds live weight.

sugar varied considerably among the treated animals, while in the control animals the blood sugar level remained constant throughout the 24-hour period.

Period Required for Recovery Following Stress

In the previous section it was shown that animals injected with adrenaline 48 to 96 hours ante mortem, in most instances produced carcasses that were darker than the controls. Ten yearling Shorthorn cattle were used to determine the time required to replenish the muscle glycogen level required for bright cutting beef after the animals had been subjected to stress. Eight animals were injected subcutaneously with 3 milligrams of adrenaline per 100 pounds live weight and two served as controls. All animals were taken off feed 24 hours before slaughter but were given free access to water. The time interval of slaughter and carcass data are given in Table 9.

TABLE 9--EFFECT OF ADRENALINE ADMINISTERED TO BEEF CATTLE ON CARCASS CHARACTERISTICS

Animal Number	Live Weight	Carcass Grade	Interval Between Injection and Slaughter[1]	Color of Muscle[2]	pH of Muscle[3]
801	790	Choice -	Control	A-4	5.4
807	700	Choice -	Control	A-4	5.4
828	735	Choice -	24 hours	A-9	6.0
829	760	Choice	24 hours	A-9	6.2
802	890	Choice	48 hours	A-6	5.75
803	780	Choice	48 hours	A-7	5.95
819	825	Choice -	72 hours	A-8	5.95
806	750	Choice -	72 hours	A-9	6.0
809	755	Choice	96 hours	A-6	5.55
810	735	Choice -	96 hours	A-8	5.9

[1]All animals except 801 and 807 were injected subcutaneously with 3 milligrams of adrenaline per 100 pounds body weight.
[2]Munsell Color Paddles, Meat Scale A.
[3]Determined in the longissimus dorsi at twelfth rib.

Figure 8 pictures rib steaks taken from a representative carcass of each time interval. The cattle which were slaughtered 24 hours after injection exhibited the darkest color, highest pH value, and stickiest, gummiest cut surfaces of the muscle. These carcasses were typical of dark cutting beef. There was a gradual lowering of the color values and decrease in pH value as the period of time between injection and slaughter was lengthened. The cattle which were injected 96 hours ante mortem were not as light and bright in color or as low in pH value as the controls. These results indicate that muscle glycogen replenishment is a much slower process than that of depletion. It would seem impractical to consider holding cattle after they had been shipped to market as a preventive measure for dark cutting beef.

Effect of Hydrocortisone in Counteracting Stress.

Twenty-three cattle were used to determine the effect on carcass characteristics of administering hydrocortisone prior to and/or after the administration of adrenaline. Adrenaline was administered subcutaneously to produce stress and to deplete glycogen stores. The hydrocortisone was administered intramuscularly prior to and/or after adrenaline to determine if an exogenous source of this hormone would aid in the restoration of muscle glycogen. All animals were given feed and water up to the date of slaughter. Hydrocortisone promotes gluconeogenesis and should, therefore, enable an animal to maintain physiological normality when subjected to stress.

The various levels of hydrocortisone and adrenaline that were administered, as well as the resulting carcass characteristics, are given in Table 10. Varying

TABLE 10--EFFECT OF HYDROCORTISONE ADMINISTERED PRIOR TO AND/OR AFTER THE ADMINISTRATION OF ADRENALINE TO BEEF CATTLE ON CARCASS CHARACTERISTICS

Animal Number	Live Weight	Carcass Grade	pH of Muscle[1]	Color of Muscle[2]	Muscle Sugar (percent)	Liver Sugar (percent)	Hydrocortisone (mg./100 lbs. body wt.)	Hours Ante Mortem Hydrocortisone Administered	Adrenaline (mg./100 lbs. body wt.)	Hours Ante Mortem Adrenaline Administered
60	700	Good	5.5	A-5	0.175	3.12	75	48	3	30
61	700	Choice-	5.55	A-4	....	...	75	47	3	22
62	700	Choice	5.6	A-6	....	...	75	48	3	24
63	800	Choice-	6.0	A-9	....	...	75	48	3	24
64	880	Good+	5.7	A-6	0.144	1.93	115	50	2.5	26
65	775	Choice	5.6	A-5	....	...	75	50	3	26
66	680	Choice-	5.45	A-4	....	...	75	50	3	26
67	730	Choice-	5.8	A-8	....	...	75	52	3	24
68	685	Good	6.25	A-9	....	...	75	52	3	24
69	700	Choice-	6.2	A-9	....	...	75	52	3	24
70	700	Choice-	5.95	A-8	....	...	75	52	3	24
71	700	Choice-	6.0	A-7	0.063	1.78	140	69	2.5	45
72	700	Good	5.6	A-7	....	...	75	71	3	46
73	650	Choice-	5.55	A-7	....	...	75	72	3	48
74	650	Choice-	5.7	A-8	....	...	75	72	3	48
75	785	Choice	6.15	A-9	0.188	3.12	30	72	3	30
							75	48		
76	830	Good+	5.7	A-8	0.088	3.00	75	48	3	20
							25	24		
77	650	Good	5.6	A-7	0.112	...	15	36	2.3	24
							15	27		
							20	12		
78	980	Choice-	5.6	A-7	0.20	3.35	20	48	3	24
							75	30		
							25	24		
79	725	Std+	5.8	A-9	0.162	2.80	75	24	3	30
80	580	Std+	5.5	A-6	0.106	1.93	160	26	3.5	50
81	920	Choice	6.1	A-9	0.087	3.5	15	26	2	25
							15	14	2	14
82	535	Good-	5.4	A-4	0.063	1.69	150	69	2.5	45
							100	20		

[1] Determined in longissimus dorsi at 12th rib.

[2] Munsell Color Paddles, Meat Scale A.

results were obtained from different animals given the same levels of adrenaline and hydrocortisone. The results obtained in some animals indicated that the administration of hydrocortisone had a beneficial effect, while in other animals hydrocortisone appeared to have no recognizable effect.

Animals No. 67, 68, 69 and 70 were subjected to an additional stress of taking frequent blood samples. Animal No. 63 was given an intraperitoneal injection of chlortetracycline one hour prior to slaughter according to the manufacturer's recommendation. It was believed that the administration of chlortetracycline would not stress the animal. However, at the time of slaughter it was discovered that the injection needle had not punctured the peritoneum and the antibiotic was deposited in the flank. It was also discovered that the antibiotic had caused necrosis of the tissue. The antibiotic probably produced an adverse effect in regard to the subsequent carcass characteristics. Animal No. 75 was very fat and "wasty" and therefore received a higher dosage of adrenaline per unit of muscle than the other animals. It was discovered at the time of slaughter that animal No. 81 had necrosis of the liver, which probably interfered with carbohydrate metabolism.

The administration of hydrocortisone appeared to have a beneficial effect on animals No. 60, 61, 62, 64, 65, 66, 72, 73, 76, 77, 78, 80 and 82. These animals were not subjected to additional stress nor were any physiological abnormalities noted. In the case of animals 71, 74, and 79, the administration of hydrocortisone appeared to have no beneficial effect.

The amount of water-extractable reducing sugar present in liver samples varied among animals with the same treatment. In general, the values for water-extractable muscle sugar are in agreement with values reported by the National Live Stock and Meat Board Committee (1949).

Blood samples were taken from six cattle prior to and subsequent to the administration of hydrocortisone and analyzed for sugar. Table 11 gives results

TABLE 11--THE EFFECT ON BLOOD SUGAR LEVEL CAUSED BY HYDROCORTISONE[1] ADMINISTERED INTRAMUSCULARLY TO BEEF CATTLE

Animal Number	Blood Sugar Prior to Hydrocortisone (mg./100 ml.)	Blood Sugar After Hydrocortisone (mg./100 ml.)	Hours[2]
83	40.0	44.8	2
84	51.2	53.6	2
85	41.6	34.4	6
86	58.4	101.6[3]	2
87	34.0	28.4	6
88	46.8	43.2	4
89	44.8	53.6	6

[1] Seventy-five milligrams per 100 pounds body weight.
[2] Hours after hydrocortisone was given when a second blood sample was taken for the determination of blood sugar.
[3] Animal was excited when blood sample was taken.

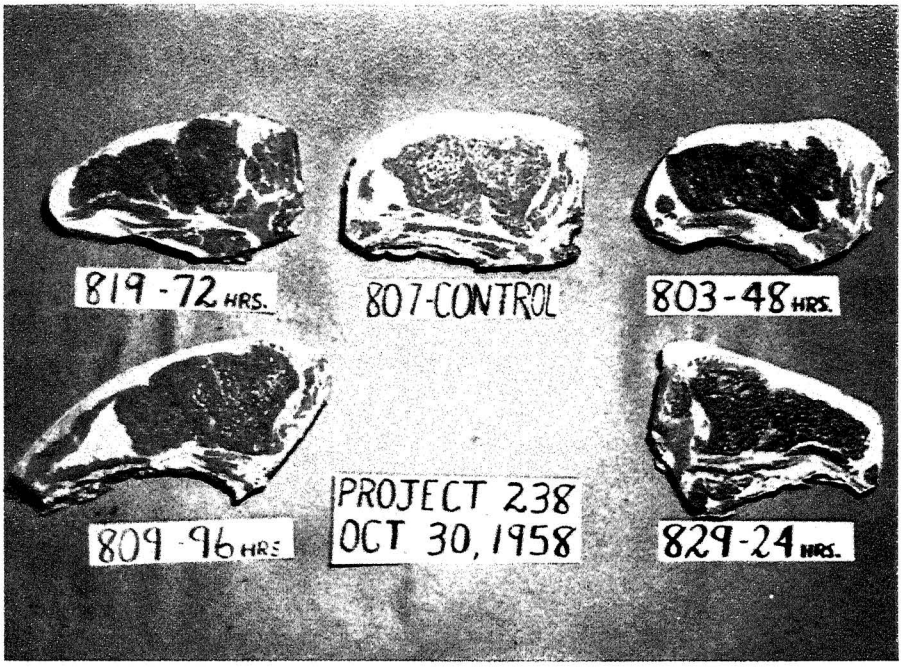

Fig. 8—The effect of adrenaline, injected at varying time intervals ante mortem, on the color of the longissimus dorsi muscle of beef.

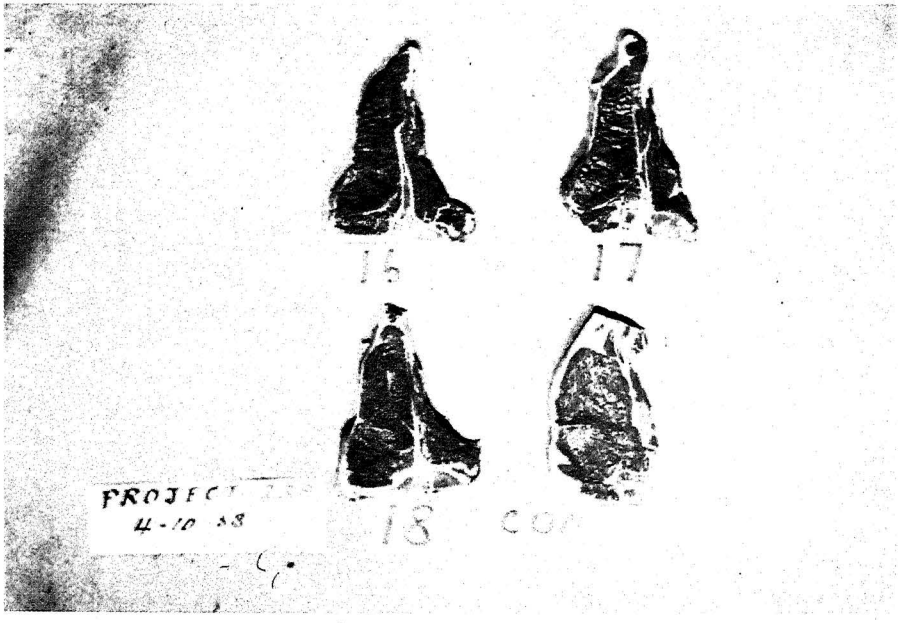

Fig. 9—The effect of excitement on the color of muscle of cattle.

of these analyses. Animal No. 86 was the only animal that had a marked change in blood sugar level after the administration of hydrocortisone. This animal was excited when the second blood sample was taken, which probably accounts for the rise in blood sugar level.

Blood and urine samples were taken periodically from animals 67, 68, 69 and 70. The blood was analyzed for sugar and the urine for sugar and total nitrogen. The intervals at which the samples were taken and the results of these analyses are presented in Figure 10 and in Table 12. A slight rise in blood and

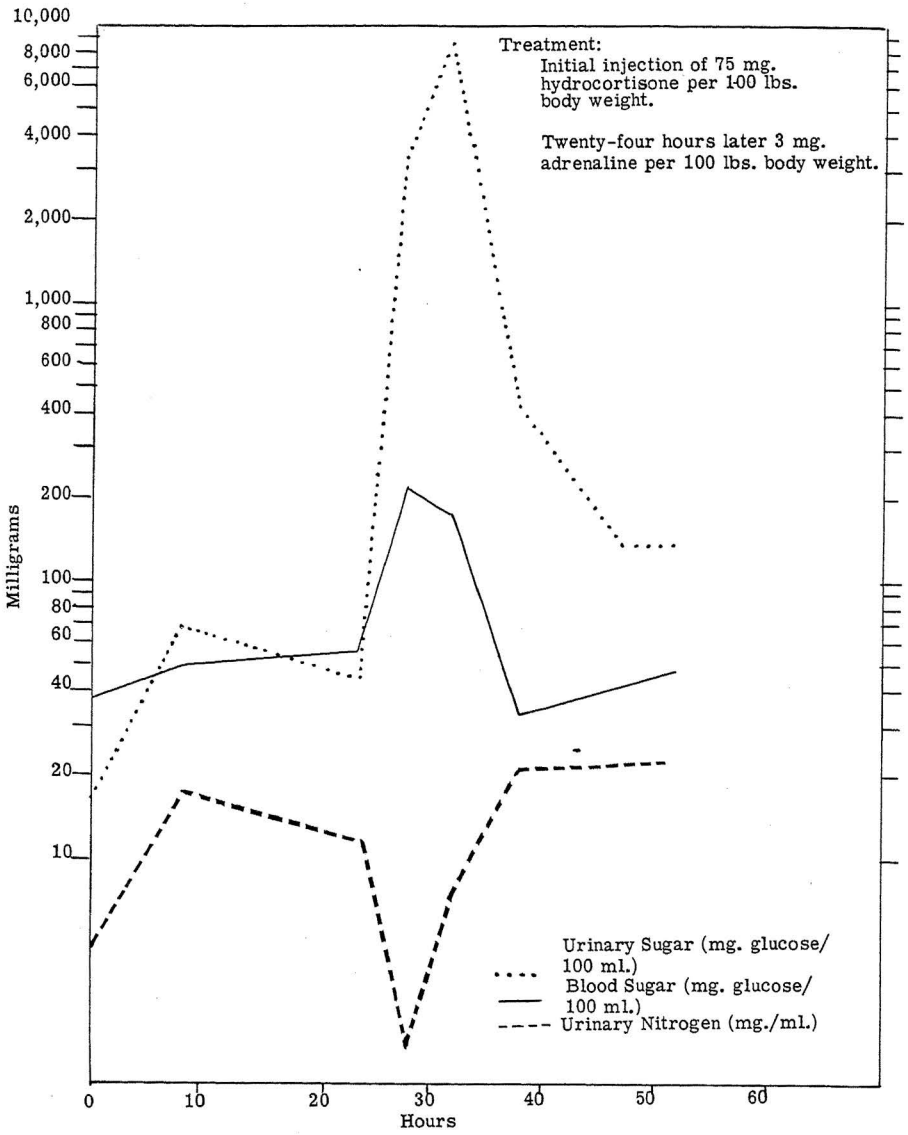

Fig. 10—The average effect of hydrocortisone and adrenaline injections on urinary sugar, blood sugar and urinary nitrogen levels of four beef cattle.

TABLE 12--EFFECT OF A SUBCUTANEOUS INJECTION OF ADRENALINE[1] PRECEDED BY HYDROCORTISONE[2] ON BLOOD SUGAR, URINE SUGAR AND URINARY NITROGEN LEVELS OF BEEF CATTLE

Animal Number	Blood Sugar (mg./100 ml.)	Urine Sugar (mg./100 ml.)	Urinary Nitrogen (mg./ml.)	Hours After Hydro-cortisone	Hours After Adrena-line
67	39.0		4.35	0	
68	27.0	31.2	11.03	0	
69	37.6	4.8	0.80	0	
70	44.8	14.4	1.98	0	
	37.1	16.8	4.54		
67	44.8	40.0	15.47	8	
68	44.8	48.0	13.62	8	
69	51.2	88.0	20.59	8	
70	53.6	95.2	16.56	8	
	48.6	67.8	16.56		
67	68.0	35.2	10.08	24	
68	50.4	8.0	3.51	24	
69	48.0	62.4	17.38	24	
70	59.2	72.0	15.82	24	
	56.4	44.4	11.70		
67	220.0	2016.0	1.14		4
68	196.0	2720.0	1.39		4
69	232.0	4000.0	2.32		4
70	226.4	4772.0	3.52		4
	218.6	3377.0	2.09		
67	141.6	10880.0	6.80		8
68	256.0	5312.0	2.53		8
69	153.6	9024.0	12.84		8
70	146.4	6784.0	7.94		8
	174.4	8000.0	7.53		
67	33.6	102.4	16.08		14
68		1004.0	15.50		14
69	26.4	192.0	19.49		14
70	40.0	400.0	20.91		14
	33.3	424.6	20.25		
67		214.4	27.21		23
68		120.0	18.22		23
69	36.0	90.4	30.84		23
70	48.8	124.0	29.04		23
	42.4	137.2	26.33		
67	60.0	112.2	21.45		28
68	41.6	58.4	12.68		28
69	47.2	164.8			28
70	42.4	210.4	34.24		28
	47.8	136.4	17.09		

[1] Three milligrams adrenaline per 100 pounds body weight.

[2] Seventy-five milligrams hydrocortisone per 100 pounds body weight.

urine sugar was noted following the injection of hydrocortisone. After the injection of adrenaline there was a significant rise in blood sugar (.001 level) and in urine sugar (.001 level). Hyperglycemia and glycosuria still existed eight hours after the administration of the adrenaline. Hyperglycemia was caused by the actuation of phosphorylase in the muscle and liver by adrenaline and glycosuria, by the blood sugar liver exceeding the renal threshold.

A significant rise in urinary nitrogen (.01 level) was noted eight hours after the administration of hydrocortisone. The rise in urinary nitrogen is attributed to the exogenous hydrocortisone stimulating gluconeogenesis and the subsequent release of nitrogen in the urine. After the administration of adrenaline there was a decrease in the urinary nitrogen level, probably due to an increased urine volume. Following the decrease in urinary nitrogen, a rise in the nitrogen level occurred. The nitrogen levels at 14 and 23 hours after the administration of adrenaline were significantly higher (.001 level) and significantly higher at 28 hours (.05 level) as compared to the level prior to the administration of hydrocortisone. This significant increase in urinary nitrogen subsequent to the administration of adrenaline is attributed to the stimulation of gluconeogenesis by endogenous glucocorticoids and possibly to some extent by exogenous hydrocortisone. Endogenous glucocorticoid release would occur subsequent to the administration of the adrenaline (Sayer 1951).

Insulin and Tranquilizers as Measures to Alleviate Stress

Preliminary work was carried out to determine dosage level of tranquilizer and method of administration to use. Nine cattle were given varying levels of Thorazine to determine the effect dosage had on the appearance and activity of the animal. These animals were not slaughtered following treatment. Four animals were injected intravenously at the rate of 25, 30, 50, and 50 milligrams per 100 pounds body weight. Within a few minutes the animals were sedated, palpebral fissure of the eye became inflammed, and increased ataxia was apparent as the dosage level was increased. However, the effects were apparent for only one to two hours following treatment. Five animals were given intramuscular injections at rates of 40, 50, 60, 100, and 100 milligrams per 100 pounds body weight. The same effects were observed in these animals as in the ones receiving intravenous injections, except the symptoms were not evident until approximately one hour after injection and were evident for several hours.

Nine Utility grade cattle of mixed dairy breeding were used in this study. The cattle were divided into three groups of three animals each. One animal in each group was injected subcutaneously with one unit of insulin per pound body weight, one was injected intramuscularly with 1 milligram Thorazine per pound body weight and the remaining animal served as a control and received no injection. After injection the animals were subjected to periodic excitement with a hot-shot while confined in a small holding pen. The stimulation was not con-

tinuous but was of such a nature as to keep the animals moving in the holding pen. The animals received no feed or water during the ante mortem stress period.

The cattle were slaughtered 24 hours following the beginning of the stress period. Blood samples were taken prior to injection, prior to stress, and at various periods during stress.

Group I consisted of three Jersey cattle weighing 745 to 780 pounds. Before injection all animals were of similar temperament. Stress consisted of periodic excitement beginning 24 hours ante mortem for 3½ hours; 18 hours ante mortem for 20 minutes; 16 hours ante mortem for 20 minutes; and 13 hours ante mortem for 20 minutes. The animals were allowed to rest between periods of excitement.

Definite differences were noted in the response of the individual animals when they were stimulated with the hot-shot. During the initial period of excitement, the control animal (No. 16) became completely exhausted before the steer receiving Thorazine became excited. The animal receiving insulin appeared to become excited somewhat slower than the control, but more rapidly than the steer receiving Thorazine. The control animal became extremely excited. Respiratory rate of the control animal increased much more rapidly than the tranquilized steer. This could be explained in part by increased activity. After a period of 1½ hours of excitement the control animal would lie down whenever possible and when stimulated with the hot-shot would make little or no attempt to arise. It was only with much effort that this animal could be made to stand. The animal receiving insulin exhibited the same indications of excitement; however, these indications were less pronounced than those of the control. After a period of two hours of stress the insulin-injected animal would lie down, but differed from the control in that he would get up when stimulated. The tranquilized animal had a drowsy, depressed appearance and when stimulated with the hot-shot would move away but did not become as excited and nervous as the other animals. After 1¼ hours the tranquilized animal began to exhibit the same reactions as the other two animals. The tranquilized animal showed indications of tiring at the end of the initial period but was not completely exhausted as were the other animals. During each of the subsequent periods of excitement the animal receiving the tranquilizer appeared to have more energy than the other two animals. At the time of slaughter the tranquilized animal appeared dopey, but responded quickly to stimulation from the hot-shot. The other two animals in his group were much slower in their response to stimulation.

As can be noted from the data presented in Table 13, there was little difference in the pH values of the longissimus dorsi muscles of the carcasses in Group I. All carcasses were dark cutters. A photograph of short loin steaks taken from each of the three carcasses and an unstressed control is presented in Figure

TABLE 13--EFFECT OF EXCITEMENT ON CARCASS CHARACTERISTICS OF UTILITY GRADE CATTLE PREVIOUSLY INJECTED WITH INSULIN OR TRANQUILIZER

Animal Number	Preventive Treatment	Period of Stress[1]	pH of Muscle[2]
Group I:			
16	Control, no injection	4.5 hrs. during 24 hr. ante mortem period	6.5
17	1 unit insulin/lb. injected subcutaneously 28 hrs. ante mortem	4.5 hrs. during 24 hr. ante mortem period	6.35
18	1 mg. Thorazine/lb. injected intramuscularly 28 hrs. ante mortem	4.5 hrs. during 24 hr. ante mortem period	6.3
Group II:			
90	Control, no injection	1.5 hrs. at beginning of 24 hr. ante mortem period	6.7
91	1 unit insulin/lb. injected subcutaneously 30 hrs. ante mortem	1.5 hrs. at beginning of 24 hr. ante mortem period	5.7
92	1 mg. Thorazine/lb. injected intramuscularly 30 hrs. ante mortem	1.5 hrs. at beginning of 24 hr. ante mortem period	6.35
Group III:			
93	Control, no injection	40 min. at beginning of 24 hr. ante mortem period	5.9
94	1 unit insulin/lb. injected subcutaneously 24 hrs. ante mortem	40 min. at beginning of 24 hr. ante mortem period	6.8
95	1 mg. Thorazine/lb. injected intramuscularly 24 hrs. ante mortem	40 min. at beginning of 24 hr. ante mortem period	6.0

[1] Stress was periodic stimulation with an electric hot-shot.

[2] Determined in the longissimus dorsi at the twelfth rib.

9. The period of excitement was much longer than necessary to cause the subsequent carcass of the control to cut dark. The ante mortem stress was severe enough to overcome any stress inhibitory effects of the Thorazine or insulin.

The cattle in Group II ranged in weight from 710 to 725 pounds. The injections given were the same as those for Group I. Stress consisted of periodic excitement beginning 24 hours ante mortem for 1½ hours.

There were definite variations in temperament of the animals in this group prior to treatment. The animal receiving Thorazine was nervous and easily excited. The animal receiving insulin was the opposite, being very sluggish and docile. It should be noted that the animal receiving insulin had an unusually thick hide. During the excitement period the control and tranquilized animals responded similarily as the control and insulin injected animals of Group I. The control and tranquilized animal was slower to respond to stimulation. After 20 minutes of stimulation the tranquilized animal became very excited. Although this animal showed indications of fatigue, it would still respond to stimulation with the hot-shot. During the excitement period the control animal showed signs of exhaustion and would lie down if given an opportunity. The animal which received the insulin did not become excited when stimulated with the hot-shot but showed signs of fatigue at the end of the excitement period.

All carcasses in Group II cut dark; however, the carcass from the animal receiving insulin was not as dark as the control or the one receiving Thorazine. Data in Table 13 show the range in pH values of the longissimus dorsi muscle.

As in Group I, the period of excitement for the control appeared to exceed the minimum required to cause dark cutting beef. Due to the nervous disposition of the animal receiving Thorazine, it is possible that the dosage level was not sufficient to cause tranquilization. The animal receiving insulin was not as susceptible to stress as the control or the animal receiving the tranquilizer, probably because of docile disposition and extremely thick hide.

The animals in Group III ranged in weight from 565 to 645 pounds. The animals in Group III were handled different from those in Groups I and II in that they were allowed free access to water during the evening and night following the stress period. The excitement period was 40 minutes in duration at the beginning of the 24-hour ante mortem period. The animals in this group were of the same temperament. Animals No. 93 and 94 were Holstein and 95 was a Guernsey. At the beginning of the stress period, the tranquilized animal was definitely tranquilized but became excited when stimulated with the hot-shot. All three cattle in this group reacted similarly when stimulated with the hot-shot. These cattle were beginning to show signs of fatigue when the stress period was ended.

At the time of slaughter there were wide variations in appearance and reactions of the animals in Group III. Animal No. 94 which received insulin was very aggressive and alert. There was marked reduction in the reflexes and alarm

responses of animal No. 95, which appeared to be almost completely sedated. Animal No. 93 appeared normal.

Table 13 shows the variation in pH values of the carcasses in this group. Although there were variations in the color of the longissimus dorsi muscle, all carcasses were dark cutters.

Under the conditions of this study Thorazine did not protect the animals from the stress imposed upon them. The pH values of the animals receiving tranquilizers were near those of the controls regardless of the period of stress. However, there were indications that Thorazine would cause a reduction in reflexes and alarm responses and prolong the time required for the animal to reach maximum excitement. The use of insulin appeared to have little or no effect on the behavior of the animal or consistently improve the carcass characteristics as compared to the control.

Two additional animals were injected intramuscularly with 50 and 100 milligrams of Thorazine per 100 pounds of body weight and slaughtered 20 hours later. There was extensive edema surrounding the site of injection in both carcasses. In addition there was extensive swelling of the muscles surrounding the site of injection of the animal that received the 100 milligram dosage.

Many deleterious side effects were encountered from Thorazine at the dosage levels used. Outstanding among these effects were large areas of edema at the site of injection. In addition, the animals appeared listless and dopey. Their ears drooped and partial closure of the palpebral fissure and ataxia were noted.

Table 14 shows the effects of various periods of excitement with a hot-shot on the blood sugar levels of Utility grade cattle injected ante mortem with insulin and Thorazine. In all instances, the animals receiving Thorazine had the highest blood sugar level at the end of the excitement period. The animal in each group which received insulin had the lowest blood sugar level.

It is possible that the blood sugar of the controls had reached a peak and began to decline prior to the time the blood samples were taken. While in the case of the animals receiving Thorazine, the tranquilizer prolonged the time required to reach maximum state of excitement and therefore the blood samples possibly were taken when the blood sugar level was at the highest peak. In the case of the animals receiving insulin, the dosage level given should promote glycogen deposition and retard glycolysis.

Injection of Diquel

Preliminary work was carried out to determine dosage level and method of administration. Two cattle were given intravenous injections of 0.25 milligram Diquel per pound body weight and two were given 0.50 milligram. Only slight tranquilization was observed in the cattle which received the 0.25 milligram level, whereas the cattle which received the 0.50 milligram level were immediately tranquilized and had difficulty standing because of ataxia.

TABLE 14--EFFECTS OF VARYING PERIODS OF EXCITEMENT WITH A HOT-SHOT ON THE BLOOD SUGAR LEVELS OF UTILITY GRADE CATTLE INJECTED ANTE MORTEM WITH VARIOUS COMPOUNDS TO INHIBIT STRESS

Animal Number	Ante Mortem Treatment	Blood Sugar Level (mg./100 ml.)	Hours After Injection
Group I:			
16	Control	81	0
	No injection, 4.5 hrs.	83	4
	excitement with hot-shot	95	9
	during 24 hr. ante mortem	67	16
	period	52	23
		71	28
17	1 unit insulin/lb.	76	0
	subcutaneously, 4.5 hrs.	69	4
	excitement with hot-shot	69	9
	during 24 hr. ante mortem	36	16
	period	36	24
		23	28
18	1 mg. Thorazine/lb.	84	0
	intramuscularly, 4.5 hrs.	52	4
	excitement with hot-shot	138	9
	during 24 hrs. ante mortem	62	16
	period	71	23
		52	24
Group II:			
90	Control	61	0
	No injection, 1.5 hrs.	79	5.5
	excitement with hot-shot	128	7.0
	at beginning of 24 hr.	74	24.5
	ante mortem period	79	29.5
91	1 unit insulin/lb.	56	0
	subcutaneously, 1.5 hrs.	52	5.5
	excitement with hot-shot	74	7
	at beginning of 24 hr.	39	24.5
	ante mortem period	70	29.5
92	1 mg. Thorazine/lb.	70	0
	intramuscularly, 1.5 hrs.	74	5.5
	excitement with hot-shot	177	7
	at beginning of 24 hr.	70	19.5
	ante mortem period	74	24.5
		74	29.5
Group III:			
93	Control, no injection	29	5
	40 min. excitement with	97	6
	hot-shot at beginning of	16	13
	ante mortem period		
94	1 unit insulin/lb.	29	5
	subcutaneously, 40 min.	86	6
	excitement with hot-shot	14	13
	at beginning of 24 hr.		
	ante mortem period		
95	1 mg. Thorazine/lb.	34	5
	intramuscularly, 40 min.	124	6
	excitement with hot-shot	34	13
	at beginning of 24 hr.		
	ante mortem period		

Three cattle were given 0.20, 0.40 and 0.40 milligram Diquel per pound intramuscularly. No indications of sedation were observed.

Four Utility grade cattle were given 1 milligram per pound body weight intramuscularly in the shoulder area. One animal was slaughtered at each of the following time intervals post-treatment: 24, 48, 72 and 168 hours. There was extensive adema and hemorrhage throughout the chuck and shank of the animals slaughtered 24, 48 and 72 hours post-treatment. There was also extensive edema surrounding the trachea and esophagus of the animal slaughtered 24 hours post-treatment. The edematous and hemmorrhagic condition of muscle in the chuck and shank of these carcasses was extensive enough that the entire chuck and shank had to be removed and the carcasses subsequently condemned. In the case of the animal slaughtered 168 hours post-treatment, there was a hemorrhagic and edematous area about 6 inches in diameter surrounding the site of injection and extending approximately 1 inch deep into the muscle.

Three Utility grade cattle were injected intramuscularly with 0.25, 0.75 and 1 milligram of Diquel per pound body weight 28 hours ante mortem. Five and one-half hours after treatment the animals were excited for 30 minutes with an electric hot-shot. The animals which received the 0.75 and 1 milligram dosage were sedated and did not become excited when stimulated with the hot-shot. In contrast, the animal that received the 0.25 milligram dosage became very excited when touched with the hot-shot.

Blood samples were taken prior to injection, prior to excitement, and immediately following the excitement period. The blood sugar values are in Table 15. Animal No. 98 had the greatest increase in blood sugar level. These results

TABLE 15--THE EFFECT OF 30 MINUTES EXCITEMENT ON THE BLOOD SUGAR LEVEL AND MUSCLE pH OF UTILITY GRADE CATTLE WHICH HAD BEEN INJECTED WITH DIQUEL

Animal Number	Ante Mortem Treatment	Blood Sugar Level (mg./100 ml.)	Hours After Injection	Muscle pH
96	1 mg. Diquel/lb. intramuscularly, 30 minutes excitement with hot-shot 24 hrs. ante mortem	43 56 56	0 5.5 6.5	5.4
97	3/4 mg. Diquel/lb. intramuscularly, 30 minutes excitement with hot-shot 24 hrs. ante mortem	43 43 56	0 5.5 6.5	5.7
98	1/2 mg. Diquel/lb. intramuscularly, 30 minutes excitement with hot-shot 24 hrs. ante mortem	52 56 119	0 5.5 6.5	5.65

indicate that the higher dosage levels of Diquel inhibited the physiological mechanisms affected by excitement, which increase blood sugar levels.

Muscle pH values are presented in Table 15. The color of muscle of animal No. 98 was cherry red, characteristic of bright cutting beef, while that of animals 97 and 98 was dark red and dark purplish black, respectively. Diquel at the 1 milligram per pound dosage level appeared to give protection against stress, resulting in the subsequent carcass having a brighter color than the carcasses of animals receiving lower levels. However, there was extensive edema in the area surrounding the site of injection. Prior to slaughter the animals had a drowsy, depressed appearance and in some instances considerable ataxia.

Physiological Effects of Serpasil

Five cattle were injected intramuscularly with varying dosage levels of Serpasil. Dosage levels are given in Table 16. The cattle had access to feed and water during the treatment period. Observations were made over a period of 13 days.

TABLE 16--DOSAGE LEVELS OF SERPASIL ADMINISTERED INTRAMUSCULARLY TO BEEF CATTLE

Animal No.	Live Weight (lbs.)	Treatment (mg./100 lbs.)	Total Dosage (mg.)
99	590	0.5	3.0
100	600	1.0	6.0
101	750	1.0	7.5
102	600	2.0	12.0
103	660	3.0	19.8

Two hours after treatment animal No. 101 appeared normal except for a looseness of the feces. This condition grew worse and eight hours post-treatment a watery discharge from the eyes and nostrils was noted. Twenty-four hours post-treatment, a return to normal was noted in the condition of the feces; however, there was still a discharge from the eyes and nostrils. The animal appeared to be normal 48 hours post-treatment. At no time was the animal off feed.

Within the first 18 hours post-treatment, animals No. 99, 100, and 102 appeared normal in every respect. However, at 24 hours post-treatment animal 102 appeared very sick. The eyes were inflamed and swollen, there was a pronounced nasal discharge and breathing was short and rapid. The bowels were very loose and watery and the animal would not eat or drink. Muscle tremours were in evidence and the animal was highly nervous. The rectal temperature was 103°F. Thirty-two hours post-treatment showed little improvement. The animal was markedly depressed, with the eyes remaining red and swollen. The nasal discharge was still marked and there was a loss of saliva from the mouth. The rectal temperature was 105°F.

Forty-eight hours post-treatment some improvement was noted in the condition of animal No. 102; however, the animal was still deeply depressed and dyspnea was apparent. The animal ate a little hay and drank some water; this appeared to require considerable effort. Seventy-two hours post-treatment the animal appeared to be making good recovery. There was little nasal discharge and the condition of the eyes was approaching normal. The condition of the feces was firm and the animal was eating hay and grain. Breathing was still rapid and the animal appeared to be markedly sedated.

Seven days post-treatment animal 102 had returned to normal. During this period animals 99 and 100 were normal and showed no sign of tranquilization, even when prodded with a hot-shot.

Animal 103 appeared normal two hours after receiving Serpasil. Four hours post-treatment this animal appeared to be in a state of sedation, was very docile, had muscle tremours, short rapid breathing and diarrhea. Five hours post-treatment, in addition to the above conditions, the animal's eyes were inflammed, watery, and swollen and saliva was flowing from its mouth. Seven hours post-treatment the animal was lying down and had to be urged to rise. The animal walked with a staggering gait, appeared very dazed, had a pronounced nasal discharge, and a rectal temperature of 105°F. Eleven hours post-treatment the animal was lying down in a position characteristic of an animal with tetany and no amount of urging would cause the animal to rise. The rate of breathing was abnormal and the rectal temperature was 106.2° F.

On the following day animal No. 103 could stand up only for a short period, because of ataxia. There was no improvement in any of the conditions noted above. The animal would not eat or drink. The next day some improvement was noted and the steer attempted to eat. During the next few days the animal continued to show improvement. On the thirteenth day the animal appeared to be approaching normal physiological reactions.

The administration of Serpasil at a level which produced a noticeable degree of tranquilization resulted in deleterious side effects which would preclude the use of this compound as a preventive measure against stress.

Sensory Panel

The relationships between carcass pH and organoleptic characteristics, cooking losses, and drip loss of rib steaks are presented in Figure 11 and Table 17. The correlations which were obtained between carcass pH and percent cooking loss, percent drip loss, aroma, flavor, tenderness, or juiciness of the steaks were not significant. These results indicate the color of beef muscle due to glycogen depletion has no significant effect upon subsequent eating qualities, providing the animals from which the meat came are approximately of the same age and grade. The pH of muscle and color of the muscle are closely related.

The consumer relates dark beef with meat from old animals or meat that has deteriorated. No significant differences in eating qualities can be detected

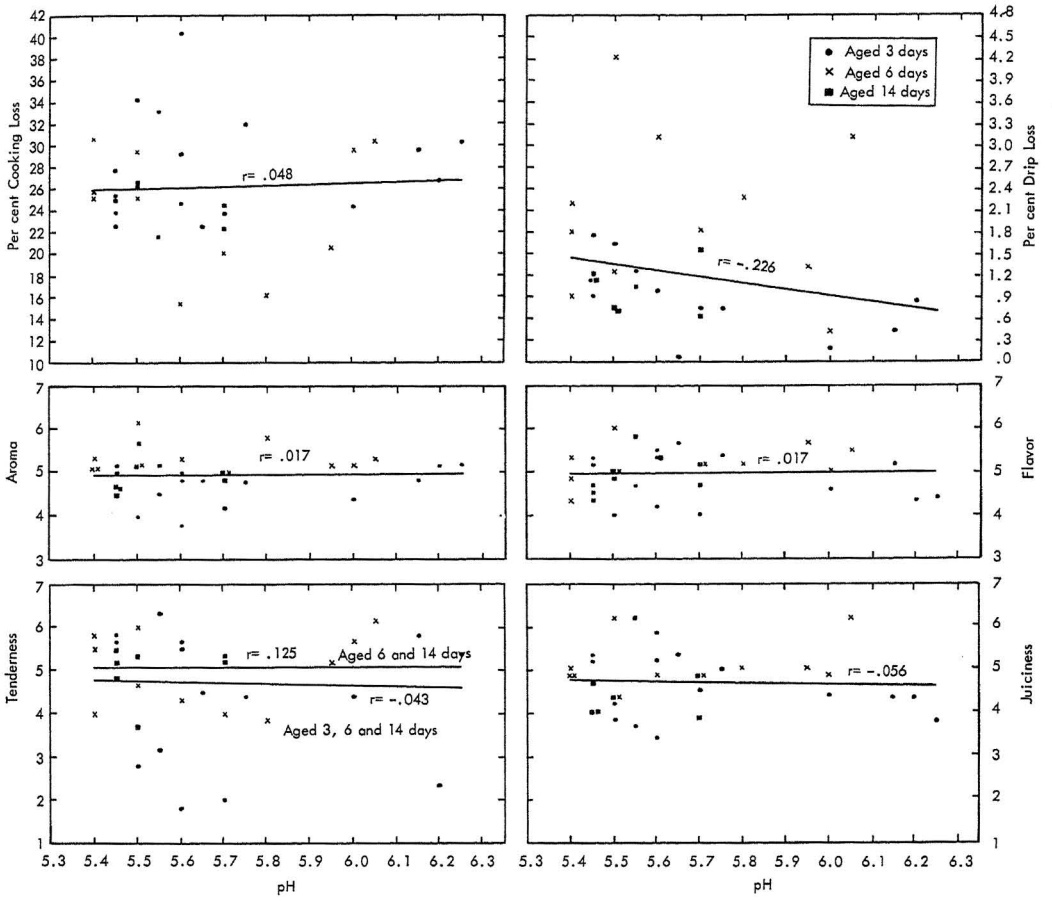

Fig. 11—The relationship of carcass pH and subsequent organoleptic characteristics of beef rib steaks.

between dark and bright beef, if the dark color is due to high pH caused by glycogen depletion. If the housewife could be assured that the undesirable appearance of dark beef was not due to deterioration, she could purchase dark beef with the same assurance of eating satisfaction as if she purchased bright beef.

TABLE 17--EFFECT OF CARCASS pH ON COOKING LOSS, DRIP LOSS AND ORGANOLEPTIC CHARACTERISTICS OF RIB STEAKS

Animal Number	Grade	Days Aged	Carcass pH	Aroma	Flavor	Juiciness	Tenderness	Drip (percent)	Cooking Loss (percent)
25	Good+	6	5.4	5.17	4.83	4.83	4.00	.92	25.30
26	Good+	3	5.55	4.50	4.66	3.66	3.16	1.29	33.22
27	Good	3	5.75	4.80	5.40	5.00	4.40	.77	32.06
28	Good+	3	6.15	4.83	5.16	4.33	5.83	.46	29.79
29	Good-	14	5.45	4.67	4.50	4.67	5.50	1.78	25.05
33	Choice-	14	5.5	5.17	5.00	4.17	3.67	.78	26.72
33-1	Good-	6	6.0	5.17	5.00	4.83	5.67	.45	29.76
34	Std.	14	5.7	4.83	5.17	3.82	5.33	1.56	24.68
35	Good	14	5.7	5.00	4.67	4.83	5.17	.64	22.45
40	Good	3	6.0	4.40	4.60	4.40	4.40	.24	24.50
41	Choice	6	6.05	5.33	5.50	6.17	6.17	3.14	30.63
42	Good	3	6.25	5.20	4.40	3.80	5.40	.00	30.48
43	Good	6	5.95	5.17	5.67	5.00	5.17	1.34	20.74
44	Choice	14	5.5	5.67	4.83	4.33	5.33	.73	26.88
46	Good+	14	5.45	4.67	4.67	4.00	4.83	1.23	25.41
47	Choice-	14	5.55	5.17	5.83	6.17	6.33	1.08	21.85
50	Good+	14	5.45	4.50	4.33	4.00	5.17	1.15	27.86
63	Choice	6	5.4	5.17	4.33	4.83	5.83	2.20	25.85
64	Good+	6	5.7	5.00	5.17	4.83	4.00	1.83	20.17
65	Choice	3	5.6	5.00	5.50	5.83	5.50	.04	29.40
72	Good	3	5.6	4.83	5.33	5.17	5.67	.07	24.81
75	Good+	6	5.4	5.17	4.83	4.83	4.00	.92	25.30
77	Good	3	5.6	3.80	4.20	3.40	1.80	1.01	40.49
80	Std.	6	5.5	5.17	5.00	4.33	4.67	1.28	29.77
82	Good-	6	5.4	5.33	5.33	5.00	5.50	1.81	30.68
85	Choice-	3	5.7	4.17	4.00	4.50	2.00	.78	23.93
86	Good+	3	6.2	5.17	4.33	4.33	2.33	.86	26.95
88	Choice-	3	5.45	5.17	5.33	5.17	5.83	.91	22.70
89	Choice-	3	5.65	4.83	5.67	5.33	4.50	.09	22.86
104	Good+	6	5.5	6.17	6.00	6.17	6.00	4.24	25.21
105	Good+	3	5.5	4.00	4.00	3.80	2.80	1.68	34.26
106	Good-	6	5.8	5.83	5.17	5.00	3.83	2.30	16.20

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