POST-MORTEM GLYCOLYTIC AND PHYSICAL CHANGES IN TURKEY BREAST MUSCLE

by

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ABSTRACT

The concentration of glycolytic intermediates and co-factors in and pH values of P. superficialis muscles from each of five 15 week-old and five 25 week-old White Cannon tom turkeys was measured at varying times between 0 and 180 min. post-mortem.

Different rates of post-mortem glycolysis were evident among birds, independent of age. On the basis of ATP catabolism, pH and lactic acid accumulation, two groups categorized as "fast-" and "slow-glycolyzing" were evident. The different rates of glycolysis could not be explained by qualitative or quantitative differences in control of the glycolytic flux.

The patterns of change in concentration of intermediates and co-factors expressed as mass action ratios suggested that regardless of glycolytic rate, post-mortem glycolysis in turkey breast muscle is susceptible to control at the reactions catalyzed by hexokinase, phosphofructo kinase, aldolase and triosephosphate isomerase, glyceraldehyde phosphate dehydrogenase and phosphoglycerokinase and pyruvate kinase.

P. superficialis muscles from six 19 and six 27 week-old White Cannon tom turkeys were analyzed for ATP concentration at "0" and "60" min. post-mortem and muscle pH was determined during a three hour post-mortem period. The time required for excised muscle strips to achieve maximum contraction was determined by periodic measurement of strip length.

Analysis of the data indicated a relationship between

rate of ATP catabolism and time to maximum contraction. "Slow-" and "fast-glycolyzing" groups were evident and were independent of age. The "slow-glycolyzing" group had a higher initial ATP concentration, a larger proportion of initial ATP remaining at 60 min. and required a longer time for the muscle strips to achieve maximum contraction.

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INTRODUCTION

Meat forms an important and substantial part of the human diet in the western world. The 1966 Canadian census (Canada Yearbook, 1967) revealed that the per capita disappearance of all red meats in 1965 was 146.3 pounds (eviscerated weight). The total poultry consumption in the same year was 36.0 pounds per capita. Of this 9.5 pounds was turkey.

The 1970 Who's Who of the Poultry Industry in Canada reported a farm placement in 1969 of 9,774,025 turkey poults for broiler weights and 8,205,635 turkey poults for heavy weights. In the same year 9,188,000 turkeys of 10 pounds and under, 3,217,000 turkeys of over 10 pounds but under 16 pounds and 3,702,000 turkeys of 16 pounds and over were slaughtered.

The important factors which should be considered in the eating quality of meat are tenderness, flavour, juiciness and aroma. However, several consumer studies have shown that tenderness is one of the most important palatability factors in the acceptance of meat.

The quality of meat that is consumed is influenced by ante-mortem and post-mortem factors operative during the conversion of muscle to meat. Briskey (1964), Lister (1970) and Khan et al. (1970) among others, have shown that the metabolism of muscle both ante- and post-mortem, is a major factor influencing the quality attributes of the subsequent meat.

This thesis describes a study of post-mortem glycogen metabolism and rigor development in muscles of turkeys of two ages.

LITERATURE REVIEW

General Muscle Metabolism

Many research papers have been published on the physiology and chemistry of muscle. Bendall (1966), in his monograph (1969), describes in detail the organization of muscle into its component structural and contractile parts. The process of contraction by means of nerve and muscle action potentials, release of Ca^{++} from the sarcoplasmic reticulum, activation of the muscle ATP-ase, the subsequent formation of actomyosin, and the functional aspects of contraction and subsequent relaxation is extensively described.

Bendall (1966, 1969) has summarized the function of ATP in vivo as the provision of energy for three main uses: to drive the $\text{Na}^+ - \text{K}^+$ pump in the plasmalemma; to drive the Ca^{++} pump in the longitudinal elements of the sarcoplasmic reticulum; and as the immediate source of contractile energy. It is clear that to maintain an appropriate energy supply, extremely efficient resynthetic mechanisms are required for the working muscle. It is the extent of interference with the resynthetic mechanism by preslaughter handling and the slaughter processes which are of utmost importance in the conversion of muscle to meat.

Lawrie (1966a) presented an outline of the resynthesis of ATP in muscle. The most immediate source of resynthesis is considered to be from ADP and creatine phosphate by the soluble

sarcoplasmic enzyme creatine kinase. The major source, however, is the resynthesis from ADP by respiration whereby muscle glycogen is oxidized to carbon dioxide and water. When energy in excess of the capacity of the respiratory system to regenerate ATP is needed, the process of anaerobic glycolysis, whereby glycogen is converted to lactic acid, may regenerate ATP, although much less efficiently than the oxidative process. Both Lawrie (1966a) and Kastenschmidt (1970) presented simplified schemes of muscle metabolism whereby ATP is resynthesized.

Post-Mortem Metabolism

Commercial slaughtering of domestic fowl and land mammals results in cessation of the oxygen supply to the muscle and in the establishment of anaerobic glycolysis in the skeletal muscles. The sequence of bio-chemical reactions by which glycogen is converted to lactic acid is essentially the same post-mortem as in vivo when the oxygen supply may become temporarily inadequate for the provision of energy via oxidative metabolism in the muscle. Except when inanition or exercise immediately pre-slaughter has appreciably diminished the reserves of glycogen in muscle, the post-mortem conversion of glycogen to lactic acid will continue until a pH is reached at which the enzymes effecting the catabolism become inactivated. The ultimate pH is generally about 5.5 which is near the iso-electric point of many muscle proteins, including those of the sarcoplasm and myofibril. Lawrie (1966a) has extensively reviewed the importance of post-mortem metabolism, and in particular the

rate and extent of pH decline, to the production of meat of optimal quality. Both the rate and the extent of the post-mortem pH fall are influenced by intrinsic factors such as species, the type of muscle and variability among animals; and by extrinsic factors such as the preslaughter administration of drugs and environmental temperature both ante- and post-mortem.

Post-mortem Metabolism and Meat

A large body of knowledge about post-mortem metabolism and its relation to meat quality has been acquired from study of a number of species exhibiting anomalous post-mortem changes.

Pale Soft Exudative Pork

The muscles of living porcine animals are moderately dark in colour, firm in texture and dry in appearance. After exsanguination the physiological and biophysical changes that take place, regulate the differentiation of muscles on the basis of colour, exudation and gross morphology. When these changes are rapid, a condition results which has been called "pale, soft exudative pork" (P.S.E.), "muscle degeneration" (MD) and "white muscle disease", (Briskey, 1964 and Lawrie, 1966a). In his review, Briskey (1964) indicates some evidence that structural abnormalities in P.S.E. and dark, firm dry musculature may be pathological and dystrophic in nature. However, the major body of evidence suggests this condition is associated with accelerated post-mortem glycolysis and onset of rigor mortis. When these changes are rapid (0.5 to 1.5 hr.) and result in acid conditions at a relatively high temperature, the

muscles usually become pale in appearance, soft in texture and extremely exudative (Briskey, 1964 and Briskey et al. 1966).

Sayre and co-workers (1963 a,b) were able to show that within the strains available, the muscles of Chester White and Hampshire breeds seldom underwent rapid post-mortem glycolytic changes while this abnormality occurred frequently in the muscles of a strain of Poland China animals. Kastenschmidt et al. (1966 and 1968) and Briskey et al. (1966) used these strain differences to study the rate of post-mortem anaerobic glycolysis. Muscles were categorized as "fast-glycolyzing" if the muscle pH had declined to 5.5 or below at 30 min. post-mortem and "slow glycolyzing" if the pH was 6.0 or higher at 60 min. post-mortem (Kastenschmidt et al. 1968). The metabolic intermediate patterns of "fast-" and "slow-glycolyzing" muscles were consistent with the concept that phosphorylase is the primary control site of post-mortem glycolysis and that phosphofructokinase and pyruvate kinase were also involved. Further work substantiated these findings but studies of reconstituted glycolytic systems in vitro, indicated that it is unlikely that the control properties of "fast-" and "slow-glycolyzing" systems are grossly different (Kastenschmidt, 1970).

The same studies of Kastenschmidt et al. (1966, 1968) pointed to the possibility that fast-glycolyzing muscles were in an oxygen-deficient state prior to the time of death. Lister et al. (1970) found that the administration of oxygen or nitrogen immediately before death to anesthetized stress-resistant and stress-susceptible animals resulted in the latter either be-

coming anoxic more easily or responding to anoxia to a greater extent than the former. It was found that there was a very rapid production of lactic acid during the first 2 min. after exsanguination in the stress-susceptible muscles and that this could at least partially explain the high levels of lactic acid in fast-glycolyzing muscles.

Sybesma et al. (1968) attempted to prevent the P.S.E. condition by injection of O_2 into the muscle to delay glycolysis, but found that pre-mortal injection produced gas embolism and no clear cut effect on pH and temperature drop. Post-mortal injection did produce a delay in glycolysis but in neither case was there any improvement in subsequent quality.

Dark Cutting Beef

Dark cutting beef, as the name implies, is a condition in which muscle surfaces remain purplish, long after the cut surface has been exposed to the atmosphere. Investigation into this matter (e.g., Lawrie, 1958; 1966a; 1966b) suggests that in contrast to normal muscle, D.C.B. has an unusually rapid rate of consumption of oxygen, practically no glycogen immediately after slaughter and an ultimate pH considerably higher than the normal 5.4 - 5.6. A survey by Munns and Burrell (1966) indicated that during the years from 1958 to 1961 the average incidence of carcasses yielding D.C.B. in selected Canadian processing plants was eight percent with a range from 3.5 to 12%.

Chalkiness in Halibut

The lowest post-mortem pH which is reached in many species of food fish is about 6.2 to 6.6; but in some species,

such as halibut, tuna, mackerel and shark, it may fall to between 5.5 and 6.0 (Buttkus et al., 1966). Tomlinson et al. (1965, 1966) showed that with increasing storage time and decreasing pH, below about 6.0, the muscle proteins of halibut become more insoluble as the region of the isoelectric zone is reached. As the solubility of the proteins decreased, the appearance of the meat changed from translucent to opaque. All fish with a flesh pH below 6.0 were "chalky", those with a pH above 6.2 nonchalky, while in the pH range between 6.0 and 6.2 intermingling of chalky, borderline chalky, and nonchalky fish occurred. Free drip increased continuously with decreasing pH in the range of pH 6.8 to 5.7.

As the solubility of the proteins decreased and opaqueness increased, free and cooked drip increased, the nitrogen content of drip increased, and the meat became increasingly tough and dry when cooked, (Buttkus et al., 1966).

Poultry

Poultry meat is an important part of the human diet. Continued investigation of this meat product is essential to maintain and increase its acceptance.

The post-mortem biophysical and biochemical changes which take place in poultry are not qualitatively different from those of other species used for meat and include the typical stiffening with the onset of rigor mortis, the disappearance of glycogen, ATP and CP; the appearance of ammonia and inosinic acid

from the deamination of adenylic acid, and; the accumulation of lactic acid as a result of the anaerobic catabolism of glycogen. The accumulation of lactic acid lowers the pH from above 7.0 to ultimate values of 5.7 to 5.9 (de Fremery, 1966a).

In contrast to land mammals, post-mortem tenderization changes are different and poultry requires less time to reach an acceptable level of tenderness (Marion and Goodman, 1967). Numerous factors have been studied to determine their effect on poultry meat tenderness including, breed and strain, sex, ration, grade light vs. dark meat, enzymes, processing methods, and aging time (Marion, 1967).

Shrimpton and Miller (1960) found no differences attributable to sex in the tenderness of breast meat of White Rock broilers, but found differences among male Leghorns, White Rock males and females and Brown Leghorns. Goodwin (1966), however, found that when comparing six strains of 26 week-old Broad Breasted Bronze turkeys strain did not have a significant effect on meat tenderness.

The administration of enzyme preparations ante-mortem, during aging, just prior to cooking or during rehydration of freeze-dehydrated meat, has been shown to increase tenderness (Marion, 1967).

In studying the chemical properties and their relation to tenderness of poultry muscle, de Fremery (1966b) found that when chickens were subjected to severe mechanical feather-plucking immediately post-mortem, the disappearance of ATP was accelerated and toughness increased. Other pre-rigor treatments

such as freezing and thawing, elevated temperatures, excising or cutting the muscle, electron irradiation, and exhaustive electrical stimulation also accelerated ATP and glycogen disappearance as well as the onset of rigor mortis, and also induced toughness that was only partially reversed by prolonged aging.

Studies supporting the findings of de Fremery above, and others of similar nature have been extensively reviewed by Marion (1967).

Heating the muscle as well as beating it as with a mechanical picker, causes an acceleration of post-mortem glycolysis (Marion, 1967; de Fremery, 1966a; Sayre, 1970). Injection of sodium pentobarbital and electrical stunning have been shown to retard glycolysis by virtue of reducing death struggle (Sayre, 1970). Dodge and Stadleman (1960) observed that struggle during slaughter did not affect post-mortem tenderness at 2 or 5 hours post-mortem. However, poultry exhibiting extreme struggling is generally less tender (Marion, 1967).

When post-mortem glycolysis was blocked by the injection of neutralized sodium iodoacetate or epinephrine (de Fremery, 1966a; Sayre, 1970; Khan et al. 1970) it was shown that the meat was tender without aging (de Fremery and Pool, 1963; de Fremery 1966a). Muscles from turkeys anesthetized with sodium pentobarbital were more tender, as measured by shear value, than those from the unanesthetized control birds (Landes et al., 1971).

De Fremery (1966a) concluded that beating, freezing, thawing, excising or cutting the muscle and electrical stimulation causes acceleration of rigor mortis in addition to acceler-

ation of post-mortem glycolysis, but that the latter is responsible for the induced toughness.

Using injections of epinephrine at intervals of time before slaughtering or by allowing the birds to struggle freely just before and during slaughtering, Khan et al. (1970) were able to control the amount of pre- and post-mortem glycolysis in individual birds. By maintaining minimum ante-mortem and maximum post-mortem glycolysis it was possible to achieve a pH of 6.2 just after slaughter and an ultimate pH of 5.7, which were considered desirable for achieving optimum tenderness after cooking.

It may be concluded from the literature cited, that the post-mortem metabolism, in particular glycolysis, is of utmost importance to the ultimate tenderness of poultry meat.

The need to age muscles of avian and mammalian species post-mortem to achieve acceptable degrees of tenderness is well known. The minimum chilling (aging) period for adequate tenderization to occur in young chickens is 2 - 4 hours post-mortem (Hanson et al., 1942; Pool et al., 1959; Dodge and Stadelman, 1959). Older chickens and turkeys require 8 - 12 hours (Klose et al., 1959; Dodge and Stadelman, 1959).

Klose et al., (1961) found that a two-hour chilling period plus a three-hour delay before freezing provided adequate opportunity for tenderization of 20-pound toms and 12-pound hens but was not adequate for six-pound fryer-roasters.

When Large White Bronze and Small White fryer turkeys were subjected to varying chilling procedures prior to freezing,

Marion and Goodman (1967), found a significant effect of treatment on tenderness of 15 week-old birds but no significant effect on 18 or 27 week-old birds. The data indicated that continuous chilling was inadequate for the turkey-fryer tenderization. It was also shown, that smaller turkeys within a group had higher shear values.

Brodine and Carlin (1968) found that for 12 or 20 pound turkeys, the aging that occurs in a spin chiller for one hour following slaughter, was sufficient to ensure adequate tenderness in subsequently frozen and roasted meat.

Scholtyssek and Klose (1967), used two strains and two age groups of turkeys and concluded from their study, that older birds may be limited to a shorter aging period with only a small percentage of resulting tough birds, younger birds aged for the same period of time contain a serious proportion of objectionably tough carcasses.

Attempts have been made to explain this variation in post-mortem aging requirements for different ages or size of turkeys. Marion (1967), hypothesized that the variation might be explained by differences in freezing rate for large and small carcasses. Larger turkey carcasses require a proportionately longer period to reach -4°C thus allowing more time for tenderization to occur.

Physical Changes Post-Mortem

Concurrent with and as a result of post-mortem metabolism in muscle, certain physical changes take place. The con-

dition of rigor-mortis is of primary concern in subsequent meat quality. The changes which take place with the passing of the muscle into rigor-mortis have been reviewed by Cassens (1966), Newbold (1966) and Goll (1968).

Rigor-mortis can literally be translated as "the stiffness of death" but Goll (1968), points out that rigor has two closely associated but quite different aspects. Rigor may be considered as; 1) a shortening or contraction of the muscle fibre, and 2) a loss of muscle extensibility. Loss of extensibility is further divided into a macroscopic phase and a molecular phase. The thesis developed is that, although molecular inextensibility is a necessary condition for the rigidity or stiffness observed in rigor, neither molecular nor macroscopic inextensibility are causes of rigor, i.e., stiffening or hardening. Both are manifestations of rigor and are the result of ATP depletion in post-mortem muscle (Goll, 1968).

Newbold (1966) showed that the normal features of post-mortem changes in muscle are; 1) that pH and the creatine phosphate concentration decline steadily; 2) acid labile P remains virtually constant until the creatine phosphate content has been greatly reduced; and 3) the maximum rate of decrease in extensibility is not attained until the acid labile P has declined appreciably. He reported rapid loss of extension when the ATP level had fallen to about 30% of its initial level in horse muscle and chicken muscle; to about 25% and 50% in rabbit muscle at 17°C and 37°C respectively and to about 67% in beef muscle.

Physical Measurements

In order to assess the relationship of chemical and physical changes post-mortem to ultimate meat quality, measurement of tenderness directly or indirectly is required.

Numerous devices have been developed to objectively measure meat tenderness (Pearson, 1966). By far the most commonly employed indicator of tenderness is the force required to shear a sample of meat. The devices commonly used to determine shear force are either the well known Warner-Bratzler or Allo-Kramer shear presses or their modifications. These devices pass a rigid plate of specified thickness through a meat sample of certain dimensions. As the blade passes through the meat, shearing and some compression of the sample take place. This type of measurement has been used widely as an indirect objective approximation of the response which a human would experience when eating the meat in question. Correlations between sensory methods and the Warner-Bratzler are generally in a range of 0.60 to 0.85 (Pearson, 1963). This measure of tenderness has been employed to assess the effect of a variety of factors on meat tenderness. For example it has been used by Buck et al. (1970), to determine the physical and chemical characteristics of free and stretched rabbit muscle; by Scholtyssek and Klose (1967), to measure sources of variability in turkey tenderness; by Wells et al. (1966), to measure the tenderness of freeze-dried chicken meat as related to maturity of birds; by Welbourne et al. (1968), to measure the effect of cooling procedures in turkeys; by Goodwin

et al. (1969), to measure the influence of age, sex and dietary energy level on the tenderness of broilers; by Khan et al. (1970), to determine the effect of pre- and post-mortem glycolysis on poultry tenderness; and by Klose et al. (1970), to measure the effect of contraction on tenderness of poultry muscle cooked in the pre-rigor state.

A fairly recent trend has been to elucidate the sequence of physical changes of post-mortem muscle by the use of tension measuring devices (Jungk et al., 1967; Busch et al., 1967; Schmidt et al., 1968; Jungk et al., 1970; Schmidt et al., 1970). The isometric tension pattern has been shown to closely parallel the tenderness cycle and illustrates the tendency of a muscle to shorten (Jungk et al., 1970).

Forrest (1968) summarized and briefly described the various instruments by which a number of parameters associated with rigor-mortis, can be measured. Elasticity and extensibility can be measured by a rigorometer, a multi-unit isotonic-isometric rigorometer or a Myotron. The usual procedure involves the loading and unloading of a specified weight on an excised strip of parallel muscle fibres at regular intervals. The changes in length of the muscle between the loaded and unloaded condition are measured.

Tension and muscle length changes can be followed by an Isometer or a Myotron. These devices measure tension changes in muscles held isometrically. The muscle length is held constant throughout the development of rigor by a balance beam with a servo mechanism controlling a counterweight position to keep the arm balanced.

An ergometer is available to measure the force and change in length in muscle during either stretching or shortening. The author further discusses the measurement of other physical parameters.

Extensive investigations into the post-mortem glycolytic pattern of turkey muscle have not been reported, nor has the variation in aging requirements for turkeys been satisfactorily or completely accounted for. Since tenderness of turkey remains a critical quality attribute, this study was undertaken to characterize the post-mortem glycolytic and physical changes in two age groups of turkeys.

MATERIALS AND METHODS

Experiment I

Muscle Samples

The birds used in this study were obtained from a commercial turkey grower and transported to the farm facilities of the U.B.C. Poultry Science Department. The birds were White Cannon tom turkeys 14 weeks of age when received. They were fed a commercial 21% protein turkey grower mash during a 13 day period of adjustment before being slaughtered.

The birds were caught and suspended by their legs, exsanguinated by an outside neck cut, and allowed to bleed with restricted movement of wings and legs. Bleeding was allowed to

proceed until completed (5 - 10 min.). Attempts were made to keep struggling to a minimum throughout the catching and slaughtering operations. The breast (muscles and the keel bone) was excised from the carcass. The left Pectoralis superficialis muscle (nomenclature according to Harvey et al. 1969) was dissected from the keel bone and used for pH measurement, and the right P. superficialis was dissected from the bone and used for determination of metabolic intermediate concentrations. Timing of sampling was started 10 minutes after exsanguination. Samples were taken at "0", 15, 30, 60, 120, 180 minutes.

pH

pH was measured continuously with a Corning combination probe electrode (Kastenschmidt et al., 1968 and Greaser et al., 1969) and a Corning model 10 expanded scale pH meter with a photovolt Varicord model 43 recorder. Throughout the pH measurement, the muscle was kept in an ice bath and the temperature change in the muscle over time was compensated for.

Preparation of Muscle Extracts

The muscle used for the determination of intermediate levels was placed in crushed ice immediately after dissection from the keel bone. Samples were obtained by discarding a $\frac{1}{2}$ cm. thick portion of the exposed muscle surface and immediately excising sufficient muscle to make a 25 gm. sample. The sample was cut into $\frac{1}{2}$ cm. cubes and immediately dropped into liquid nitrogen. Sampling proceeded from the anterior to the posterior end of the muscle.

The muscle samples were powdered according to the

following modification of the method of Borchert and Briskey (1965). The muscle was removed from the liquid nitrogen (LN_2) and pulverized in a macro-model Virtis homogenizer for 1.5 minutes at a speed of 11,000 rpm. The powdered samples were then stored in aluminum foil envelopes in LN_2 until extracted.

The powdered samples were extracted in the following manner: ten grams of powder (at LN_2 temperature) was rapidly stirred into a 50 ml plastic tube containing enough 0.6 N perchloric acid to give a 1 g : 3 ml dilution (ratio of wet tissue weight to extract volume, including muscle water assumed to be 73%, Swanson et al. 1962).

The extracts were centrifuged ($15,000 \times g$) for 15 minutes at 0°C . and the supernatant solutions decanted. The extracts were then neutralized to the methyl orange end point with 5 M potassium carbonate and the precipitated potassium perchlorate allowed to settle out at 0°C . for at least 30 minutes, after which duplicate analyses for metabolic intermediates were completed within 72 hours. The analysis for the more labile intermediates, particularly adenosine triphosphate (ATP) and creatine phosphate (CP) were conducted first.

Separate extracts for inorganic phosphate (P_i) were prepared according to a modification of the procedure of Karpatkin et al. (1964). The regularly prepared muscle powder was deproteinized with 0.6 N perchloric acid and immediately neutralized with a mixture containing 0.77 M Trisacetate and 1.25 M KOH. The samples were centrifuged at $15,000 \times g$ to obtain the ortho-phosphate in the supernatant.

Alkaline extracts for determination of reduced nicotinamide-adenine dinucleotide (NADH) were also prepared by methods outlined by Klingenberg (1963b).

Analytical Methods

Analyses for glucose - 6 - phosphate (G-6-P), fructose-6-phosphate (F-6-P), glucose-1-phosphate (G-1-P) and glucose were carried out in the same cuvette according to the method of Hohorst (1963a). Assays for fructose-1,6-diphosphate (FDP) glyceraldehyde-3-phosphate (GA3P) and dihydroxy acetone phosphate (DHAP) were conducted by the procedures outlined by Bucher and Hohorst (1963). The methods outlined by Czok and Eckert (1963) were used to determine 3-phosphoglyceric acid (3PGA), 2-phosphoglyceric acid (2PGA), phosphoenol-pyruvate (PEP), and pyruvate in the same assay. Lactate was determined by the method of Hohorst (1963b) and α -glycerophosphate (α -GP) by the methods of Hohorst (1963c).

Creatine phosphate (CP) and adenosine triphosphate (ATP) were determined in the same reaction vessel after the methods of Lamprecht and Stein (1963) and Lamprecht and Trautschold (1963). Adenosine diphosphate (ADP) and adenosine monophosphate (AMP) were determined using the methods of Adam (1963).

Nicotinamide-adenine dinucleotide (NAD) was determined by the method of Klingenberg (1963a) while reduced nicotinamide-adenine dinucleotide (NADH) was estimated by the method of Klingenberg (1963b).

Glycogen was determined by the methods of Pfeleiderer

(1963) in which the glucose liberated by acid hydrolysis of isolated glycogen is estimated.

In all the above determinations, the procedures were used as indicated with only slight modifications in the concentrations of intermediates, cofactors and enzymes. Some of these modifications were necessary because only cuvettes of 10 mm were used. All analyses were carried out with a Unicam-SP 800 model recording spectrophotometer.

Total creatine was determined on the diluted neutralized perchloric acid extracts by the method of Ennor (1953). The resulting colour solutions were read in a spectrophotometer at 590 nm and the concentration determined from a standard curve.

Inorganic phosphate was determined by the method of Berenblum and Chain (1938) on the neutralized extracts prepared as previously outlined by Karpatkin et al. (1964). The extraction and reduction were carried out in separate flasks as outlined by Ennor and Stocken (1950). The resulting colour complex was read in a spectrophotometer at 710 nm and the concentration determined from a standard curve.

Chemicals

All chemicals used were of reagent grade. Distilled water was used throughout the experiment. Enzymes, purified glycolytic intermediates and co-factors were purchased from Sigma Chemical Company, St Louis, Missouri.

Experiment II

The animals used in this study were of the same population as those in Experiment I, and were maintained in the same facilities and fed the same ration. The birds were slaughtered when they were 25 weeks old and all procedures of pH determination and sample preparation and analyses were identical to those of Experiment I.

In making up the extracts, the muscle was taken to contain 73% water as determined by actual moisture determinations on the muscle samples (Appendix A - Table I).

Experiment III

Two additional 25-week old birds were used to determine live muscle pH. The birds were anesthetized with ether to a "deep plane" of anesthesia (Fedde et al. 1963). With the bird restrained on a modified surgical board, the skin was cut and the P. superficialis muscle exposed. A slit was made into the muscle and the combination pH probe electrode inserted. pH was recorded for 5 - 10 minutes as previously described (Exp. I). The bird was then given an overdose of ether and bled by an outside neck cut. The muscle was excised and pH recorded for 3 hours as previously described. Sodium iodoacetate (5mm) slurries were also made at the time intervals used in Experiment I and pH measured as described by Marsh (1952) (Appendix A - Table II).

Experiment IV

Five White Cannon tom turkeys of 14 weeks and 5 of 22 weeks were obtained from the same source and kept under conditions similar to those described in Experiment I until they were 19 and 27 weeks old respectively. Slaughter conditions and sampling of muscle were conducted as described previously. The left P. superficialis was used to obtain three muscle strips of 10 cm x 1.5 cm x 1.5 cm and also to obtain samples for the determination of ATP. The muscle strips were used to determine the amount of shortening prior to rigor mortis and the extent of lengthening after rigor mortis. Sampling for ATP determinations was started 15 minutes after exsanguination, and samples were taken at "0" and "60" minutes. Preparation of the muscle extracts and the determination of ATP levels was as described for Experiment I.

The device used to measure the rigor development was designed from the idea put forth by Smith et al. (1969) and Schmidt et al. (1968) and was constructed by the Agricultural Engineering Department at U.B.C. It consists of a plexiglass tank 16.5 cm x 30 cm x 6 cm (outside measurements) and having 3 fixed plexiglass clamps at one end and moveable stainless steel and plexiglass clamps at the other. Each moveable clamp was attached by 6 lb. test monofibre fishing line running under and over two 6.5 cm diameter plexiglass pulleys to a set of weights. The pulleys were mounted on a brass shaft by means of sealed bearings (Plate 1). The weights attached to the moveable

clamps exerted a load of 20 grams at the clamp as calibrated by a dynamometer. The whole tank was maintained in a water bath.

The muscle strips were allowed to contract freely in 15°C distilled water in the tank.

Muscle strip length was measured as soon as the strips were placed in the apparatus and at intervals and when contraction had ceased (no change in length for 10 - 20 min.) the weights were attached to the strips via the clamp-pulley system. The muscle was then allowed to lengthen until maximum length was attained as measured by muscle strip length.

Plate 1. Rigor measuring device showing the fixed (A) and moveable (B) clamps and weights (C).

RESULTS AND DISCUSSION

Experiments I to III

In the subsequent discussion of changes in concentration of metabolites during the post-mortem period of measurement, the bird-to-bird variability in absolute concentration was large and therefore statistical significance of changes over time could not be generally demonstrated. However, the qualitative changes in concentration were relatively consistent from bird to bird and are reflected by the pattern of changes in average concentration

pH

Changes in post-mortem muscle pH of 15 week-old birds were quite varied for individual birds in Experiment I (Appendix A - Table VI). Some birds exhibited a rapid decline during the first 15 min. with a subsequent slow decline to the ultimate pH value; others showed a rapid initial decline with a slight increase and then a steady decline or a continued increase to the ultimate pH value (Appendix B - Figure I). The lowest pH value reached was not always identical to the ultimate pH value attained. The lowest pH occurred anywhere from 0 to 180 minutes post-mortem.

The average pH values for the five birds indicate that pH dropped to a value of 5.99 at 15 min., rose to 6.03 at 30 min. and then declined to an ultimate value of 5.93 at 180 min. post-mortem.

The 25 week-old birds of Experiment II showed considerable variation in post-mortem pH values. However, the patterns of

pH decline were similar (Appendix A - Table VII). All birds exhibited an initial rapid decline and then a considerable increase over the next 45 minutes followed by a gradual increase to the ultimate value. The lowest pH value attained did not correspond to the final ultimate value and for all but one bird occurred at 15 min. post-mortem, (Appendix B - Figure 2).

TABLE I - PH OF P. SUPERFICIALIS AT VARYING TIMES POST-MORTEM FOR 15 AND 25 WEEK-OLD TURKEYS (EXP. I AND II).

Time (min.) Post-mortem	Age (wks.)	
	15	25
0	6.11 $\pm$ 0.17 ¹	6.17 $\pm$ 0.14
15	5.99 $\pm$ 0.16	5.83 $\pm$ 0.10
30	6.03 $\pm$ 0.18	5.88 $\pm$ 0.09
60	6.01 $\pm$ 0.15	5.93 $\pm$ 0.07
120	5.95 $\pm$ 0.08	5.96 $\pm$ 0.07
180	5.93 $\pm$ 0.10	5.95 $\pm$ 0.06

¹Values expressed as mean $\pm$ standard deviation.

The average pH values for the five 25 week-old birds dropped to a low of 5.83 at 15 min., then rose gradually to the ultimate value of 5.96 (Table I and Fig. 10B). At each measurement time the standard deviation of pH was lower for the 25 week-old birds than for the 15 week-old birds.

To verify that the low pH values attained at "0" minutes post-mortem were realistic, Experiment III was conducted to determine the pH of muscle of an anesthetized bird. The pH, determined as previously described, was in the range of 6.90 to 7.10. Following the pH pattern of these muscles over the 180 min. post-

mortem period showed similar rapid drops in pH, with an average ultimate pH of 6.19 and 6.06 as determined by the probe and slurry method respectively (Appendix A - Table II). The very high initial pH values of bird 15 can be accounted for by the fact that the probe was not properly inserted into the muscle and was repositioned at about 30 min. post-mortem.

The fact that the post-mortem pH values for these muscles were somewhat higher than those shown for the birds of Experiments I and II can probably be accounted for by the fact that the birds had been anesthetized with ether. This finding corresponds with that of Landes et al. (1971), who found that muscle pH of anesthetized birds declined considerably slower than that of unanesthetized control birds, particularly during the first 6 hr. post-mortem.

It can be seen from the limited results of Experiment III and also from the report of Kastenschmidt et al. (1968), that the difference in pH between the two methods of measurement is relatively small, especially for the last two time periods. Briskey (1964) had reported earlier that pH values taken with probe electrodes at the time of death are usually 0.2 pH units lower than the pH of macerated tissue at room temperature. The results of Experiment III indicate that the pH of the slurries was consistently lower than pH measured by the probe.

Glycolytic Intermediates and Co-factors

All the glycolytic intermediates and co-factors, with the exception of creatine and inorganic phosphate were determined by enzymatic methods. These methods depended on the coupling of a series of reactions, the end result of which was the reduction of NAD and NADP or the oxidation of NADH and NADPH. The concentration of the intermediate or co-factor was calculated by dividing the increase or decrease in absorbance, from the start to the completion of the reaction, by the appropriate molar extinction value of the NAD or NADP.

Creatine and inorganic phosphate were measured by colorimetric means with comparison to a standard curve determined in the same manner.

Intermediates of 15 week-old birds

The concentration of each intermediate and co-factor determined for individual 15 week-old birds is shown in Appendix A - Table VIII. These values represent the average of duplicate determinations. Table II contains the average levels of intermediates and co-factors at varying times post-mortem for the 15 week-old birds.

The concentration of G-1-P shows a slight increase over time from 0.2579 μ Moles/g to 0.4288 μ Moles/g of tissue. Glucose-6-phosphate, was present at about ten times the concentration of G-1-P for the initial 30 minutes. Its concentration increased markedly, particularly during the period, from 60 to 180 minutes post-mortem. Fructose-6-phosphate shows a

TABLE II

CONCENTRATION¹ OF THE GLYCOLYTIC INTERMEDIATES AND CO-FACTORS IN P. SUPERFICIALIS MUSCLE AT VARYING TIMES POST-MORTEM FOR 15 WEEK OLD TURKEYS (EXP. I).

Intermediate or Co-factor	Time (min.) Post-mortem		
	0	15	30
ATP ²	2.8253 ± 2.6459 ⁵	2.5619 ± 2.4879	2.1446 ± 2.6029
ADP	1.0690 ± 0.1877	0.9560 ± 0.3596	0.9595 ± 0.2272
AMP	0.2815 ± 0.0766	0.3164 ± 0.1596	0.2460 ± 0.1342
NAD	1.0363 ± 0.0949	0.9446 ± 0.1524	0.8914 ± 0.1845
NADH	0.5247 ± 0.2757	0.5399 ± 0.1959	0.3370 ± 0.1520
CP	0.9933 ± 1.5579	1.7303 ± 2.8600	0.9063 ± 1.4289
Total Creatine ³	2.7389 ± 1.0619	3.0119 ± 1.2959	3.0379 ± 1.5410
P _i	14.3639 ± 5.7949	16.6087 ± 3.2139	16.1761 ± 2.8310
G-1-P	0.2579 ± 0.2039	0.2835 ± 0.1473	0.2343 ± 0.1661
G-6-P	2.4715 ± 1.1200	2.6020 ± 1.1105	2.6787 ± 1.1250
F-6-P	0.4156 ± 0.1873	0.4622 ± 0.2015	0.3892 ± 0.2172
FDP	0.0395 ± 0.0366	0.0378 ± 0.0417	0.0400 ± 0.0503
DHAP	0.0288 ± 0.0206	0.0248 ± 0.0201	0.0307 ± 0.0260
GA3P	0.0160 ± 0.0077	0.0159 ± 0.0051	0.0114 ± 0.0066
3PGA	0.0097 ± 0.0066	0.0080 ± 0.0051	0.0112 ± 0.0097
2PGA	0.0061 ± 0.0021	0.0089 ± 0.0073	0.0065 ± 0.0065
PEP	0.0114 ± 0.0059	0.0075 ± 0.0054	0.0174 ± 0.0169
Pyruvate	0.3983 ± 0.2193	0.3350 ± 0.2462	0.2982 ± 0.2979
Lactate	320.8059 ± 126.1000	344.4151 ± 148.6000	344.6626 ± 180.7000
α-GP	1.0926 ± 0.4740	0.9591 ± 0.3479	0.8742 ± 0.2269
Glucose	3.4799 ± 0.9104	3.5761 ± 1.0329	3.4190 ± 1.3329
Glycogen ⁴	5.8759 ± 0.5195	6.2489 ± 0.2173	6.0705 ± 0.3174

Continued....

¹Concentrations expressed as μMoles/g tissue.

²Abbreviations as given in text.

³Concentration expressed as mg/g tissue.

⁴Concentration expressed as μMoles glucose/g tissue.

⁵Values are means ± standard deviation.

TABLE II (CONTINUED)

Intermediate or Co-factor	Time (min.) Post-mortem					
	60		120		180	
ATP	1.5783 ±	2.2749	0.2742 ±	0.3343	0.0914 ±	0.1075
ADP	1.0023 ±	0.1932	0.7879 ±	0.2528	0.8149 ±	0.2429
AMP	0.3162 ±	0.1707	0.6481 ±	0.5531	0.7619 ±	0.4727
NAD	0.8904 ±	0.1371	0.8478 ±	0.1708	0.9478 ±	0.1585
NADH	0.3386 ±	0.0837	0.3132 ±	0.1004	0.3247 ±	0.0520
CP	0.2018 ±	0.3679	0.1444 ±	0.2413	0.0869 ±	0.1288
Total Creatine	2.5679 ±	1.4049	2.8460 ±	1.5280	2.9890 ±	1.6509
P _i	16.0325 ±	2.9089	15.1777 ±	2.1369	17.8505 ±	6.7479
G-1-P	0.2855 ±	0.2456	0.3268 ±	0.1894	0.4288 ±	0.3485
G-6-P	3.6916 ±	1.9300	5.9251 ±	3.0609	8.0341 ±	3.5370
F-6-P	0.6025 ±	0.3772	0.9960 ±	0.4214	1.3431 ±	0.5537
FDP	0.0364 ±	0.0394	0.0239 ±	0.0306	0.0072 ±	0.0020
DHAP	0.0210 ±	0.0094	0.0133 ±	0.0058	0.0100 ±	0.0064
GA3P	0.0123 ±	0.0059	0.0153 ±	0.0130	0.0105 ±	0.0101
3PGA	0.0067 ±	0.0056	0.0114 ±	0.0068	0.0110 ±	0.0051
2PGA	0.0074 ±	0.0061	0.0046 ±	0.0057	0.0072 ±	0.0045
PEP	0.0094 ±	0.0062	0.0119 ±	0.0122	0.0167 ±	0.0334
Pyruvate	0.3348 ±	0.3396	0.3425 ±	0.2761	0.4152 ±	0.3031
Lactate	273.3271 ±	84.3210	355.9400 ±	180.1000	358.4657 ±	155.3900
α-GP	0.7744 ±	0.2907	0.8690 ±	0.1750	0.9586 ±	0.2722
Glucose	3.8463 ±	0.9783	4.1060 ±	0.9918	4.0545 ±	1.1919
Glycogen	6.0923 ±	0.0228	6.0938 ±	0.7776	5.7133 ±	1.5309

similar rapid increase from 30 to 180 minutes. In comparison with the intermediates prior to FDP in the glycolytic cycle (Appendix B - Figure 3) those from FDP to pyruvate were present at much lower concentrations at all sampling times. Generally, the concentration of each of these intermediates, i.e., FDP, DHAP, GA3P, 3PGA, 2PGA, PEP and pyruvate, declined or remained relatively static over time post-mortem.

Lactate concentration declined rapidly between 30 and 60 minutes but then increased to a level slightly above that of the initial 30 minute period.

Glucose increased gradually in concentration whereas glycogen decreased slightly in concentration (particularly during the period from 120 to 180 minutes) over the post-mortem time period measured.

Co-factors of 15 week-old birds.

Adenosine triphosphate concentration declined almost linearly from 2.8253 to 0.0914 μ Moles/g over the three hour period. Adenosine diphosphate concentration declined in a manner similar to that of ATP, whereas AMP concentration remained relatively constant over the first 60 minute period, after which it increased from 0.3162 to 0.7619 μ Moles/g between 60 and 180 minutes (Table II).

Creatine phosphate concentration exhibited a very rapid decline during the period from 0 to 60 minutes and then a more gradual decrease to almost zero at 180 minutes. Total creatine, although showing some fluctuation over the three hour period of measurement, remained relatively constant.

TABLE III

CONCENTRATION¹ OF THE GLYCOLYTIC INTERMEDIATES AND CO-FACTORS IN P. SUPERFICIALIS MUSCLE AT VARYING TIMES POST-MORTEM FOR 25 WEEK OLD TURKEYS² (EXP. II).

Intermediate or Co-factor	Time (min.) Post-mortem					
	0		15		30	
ATP ²	3.9219	± 1.9100 ⁵	2.6369	± 1.5849	2.2245	± 1.4319
ADP	0.9609	± 0.1905	0.9882	± 0.1614	0.9991	± 0.2250
AMP	0.2643	± 0.0625	0.2367	± 0.0545	0.2449	± 0.0982
NAD	0.7184	± 0.0479	0.6939	± 0.0419	0.6314	± 0.1207
NADH	0.2626	± 0.0973	0.3226	± 0.1776	0.2750	± 0.1016
CP	0.8452	± 0.6853	0.5580	± 0.4042	0.2644	± 0.1901
Total Creatine ³	5.4219	± 0.7507	5.3277	± 1.1239	5.5129	± 0.8949
P _i	16.8082	± 3.4769	18.2879	± 3.9400	19.8231	± 3.3429
G-1-P	0.3369	± 0.2795	0.2265	± 0.1269	0.2440	± 0.1322
G-6-P	3.2238	± 1.4289	2.7225	± 1.4879	2.7007	± 1.0000
F-6-P	0.3646	± 0.1754	0.3064	± 0.1988	0.3222	± 0.1959
FDP	0.0547	± 0.0450	0.0158	± 0.0112	0.0132	± 0.0093
DHAP	0.0250	± 0.0178	0.0168	± 0.0080	0.0122	± 0.0028
GA3P	0.0165	± 0.0057	0.0129	± 0.0026	0.0149	± 0.0038
3PGA	0.0126	± 0.0049	0.0103	± 0.0048	0.0126	± 0.0049
2PGA	0.0119	± 0.0042	0.0113	± 0.0001	0.0114	± 0.0069
PEP	0.0225	± 0.0089	0.0208	± 0.0054	0.0157	± 0.0061
Pyruvate	0.6570	± 0.1401	0.4691	± 0.1216	0.3041	± 0.0947
Lactate	368.4849	± 121.6000	358.8349	± 96.3590	338.4682	± 102.8900
α-GP	0.7786	± 0.2532	0.6899	± 0.1921	0.5702	± 0.2034
Glucose	4.1604	± 0.6736	4.0102	± 0.6586	4.3341	± 0.4369
Glycogen ⁴	6.1342	± 2.8290	5.9008	± 2.5500	6.0559	± 2.7090

Continued.....

¹Concentrations expressed as μ Moles/g tissue.

²Abbreviations as given in text.

³Concentration expressed as mg/g tissue.

⁴Concentration expressed as μ Moles glucose/g tissue.

⁵Values are means $\pm$ standard deviation.

TABLE III (CONTINUED)

Intermediate or Co-factor	Time (min.) Post-mortem					
	60		120		180	
ATP	1.4744	± 1.4259	0.3482	± 0.2060	0.1290	± 0.0797
ADP	0.9826	± 0.3688	0.9171	± 0.3577	0.7642	± 0.2597
AMP	0.2779	± 0.1150	0.8205	± 0.5853	0.9001	± 0.5916
NAD	0.5660	± 0.1430	0.5783	± 0.1499	0.6222	± 0.1355
NADH	0.2710	± 0.1021	0.2946	± 0.2464	0.3473	± 0.1974
CP	0.1616	± 0.1669	0.1029	± 0.0652	0.0737	± 0.0732
Total Creatine	5.3591	± 0.9237	5.0163	± 1.2909	5.4213	± 0.9578
P _i	19.2841	± 2.4679	19.3791	± 3.8819	17.8615	± 4.5229
G-1-P	0.2325	± 0.1836	0.2905	± 0.2250	0.2963	± 0.2195
G-6-P	3.0829	± 0.9379	5.1260	± 3.3019	7.3486	± 4.0029
F-6-P	0.3264	± 0.1202	0.7559	± 0.5037	0.9935	± 0.6617
FDP	0.0078	± 0.0045	0.0072	± 0.0021	0.0047	± 0.0028
DHAP	0.0148	± 0.0028	0.0133	± 0.0028	0.0112	± 0.0023
GA3P	0.0139	± 0.0034	0.0134	± 0.0028	0.0092	± 0.0045
3PGA	0.0126	± 0.0026	0.0132	± 0.0038	0.0131	± 0.0069
2PGA	0.0091	± 0.0050	0.0102	± 0.0047	0.0113	± 0.0057
PEP	0.0140	± 0.0063	0.0157	± 0.0025	0.0163	± 0.0028
Pyruvate	0.2599	± 0.1242	0.2756	± 0.1225	0.2415	± 0.1024
Lactate	307.4510	± 91.0390	328.2378	± 94.7290	369.2962	± 118.6000
α-GP	0.5290	± 0.1720	0.5079	± 0.2092	0.5958	± 0.2116
Glucose	4.1258	± 0.5307	4.7160	± 0.4356	4.8607	± 0.4096
Glycogen	6.2120	± 2.9049	5.9791	± 2.6280	5.5128	± 2.2690

Inorganic phosphate concentration increased over the total time period but declined from 15 to 120 minutes (Table II).

The concentration of NAD decreased slightly from 0 to 120 minutes and then increased. NADH was present at about 50% of the concentration of NAD and dropped rapidly between 15 and 30 minutes after which it remained essentially constant (Table II).

Intermediates of 25 week-old birds.

The concentration of each intermediate and co-factor determined for individual 25 week-old birds is shown in Appendix A - Table IX. These values represent the average of duplicate determinations. Table III contains the average levels of intermediates and co-factors over varying times post-mortem for the 25 week-old birds.

Glucose-1-phosphate concentration fluctuated during the post-mortem period but showed a slight net decrease from 0 to 180 minutes. Glucose-6-phosphate was present in the tissue at a considerably higher concentration than G-1-P and increased from 3.2238 to 7.3486 μ Moles/g tissue between 0 and 180 minutes. Fructose-6-phosphate concentration was quite stable up to 60 minutes post-mortem after which it increased rapidly to a maximum at 180 minutes. Fructose-1, 6-diphosphate and the intermediates following it, were contained in the tissue at levels considerably lower than the intermediates prior to FDP. These intermediates, i.e., FDP, DHAP, GA3P, 3PGA, 2PGA, PEP and pyruvate, generally declined or remained relatively constant over time.

Lactate concentration declined steadily from 0 to 60 minutes after which it increased steadily to a concentration of 369.2962 μ Moles/g at 180 minutes, slightly above the initial concentration.

Glucose concentration, although fluctuating somewhat, slowly increased over the three hour period, whereas glycogen concentration, similarly fluctuating, decreased slightly over time.

Co-factors of 25 week-old birds.

Adenosine triphosphate concentration declined rapidly whereas ADP concentration increased slightly for the first 30 minutes and then decreased rapidly over the next 150 minutes. Adenosine monophosphate concentration remained essentially constant for the initial 60 minutes after which it increased very rapidly from 60 to 120 minutes and continued to increase, but more slowly during the last 60 minutes post-mortem (Table III).

Creatine phosphate concentration declined very rapidly during the initial 60 minutes and continued to decrease to 0.0737 μ Moles/g tissue at 180 minutes post-mortem. Creatine concentration although fluctuating, remained essentially constant throughout the time period of measurement. Inorganic phosphate concentration increased considerably during the initial 30 minutes and then decreased gradually to 180 minutes post-mortem (Table III).

The concentration of NAD decreased slightly whereas NADH concentration increased slightly over the total time period of measurement (Table III).

Figure 1. Comparisons of concentrations of some glycolytic intermediates in *P. superficialis* muscles from 15 week-old (O—O) and 25 week-old turkeys. (Δ—Δ)

Figure 2. Comparisons of concentrations of some glycolytic intermediates in *P. superficialis* muscles from 15 week-old (O—O) and 25 week-old turkeys. (Δ—Δ)

Figure 3. Comparisons of concentrations of some glycolytic intermediates in *P. superficialis* muscles from 15 week-old (O—O) and 25 week-old turkeys. (Δ—Δ)

Figure 4. Comparisons of concentrations of some glycolytic intermediates in and pH values of *P. superficialis* muscles from 15 week-old (O—O) and 25 week-old turkeys (Δ—Δ). Glycogen concentration is expressed as μMoles of glucose/g.

Figure 5. Comparisons of concentrations of some glycolytic co-factors in *P. superficialis* muscles from 15 week-old (O—O) and 25 week-old turkeys. (Δ — Δ)

Figure 6. Comparisons of concentrations of some glycolytic co-factors in *P. superficialis* muscle from 15 week-old (O—O) and 25 week-old turkeys (Δ—Δ). Creatine concentration is expressed as mg/g.

Comparison of Intermediates and Co-factors for 15 and 25 week-old birds.

The standard deviation associated with the concentration of each intermediate and co-factor was relatively large regardless of age group or post-mortem time (Tables II and III). The magnitude of the variability in the data is further demonstrated by the average coefficients of variability (Table IV). It can be ascertained readily from the means and standard deviations presented that with few exceptions no significant differences ($P=0.05$) in concentration of intermediates and co-factors existed between the two age groups.¹ The exceptions were NAD and total creatine which were significantly higher in the 15 and 25 week-old birds respectively at a probability level of 5% in all cases except one.

The large variability encountered, however, is not unique to the results reported here. Bodwell et al. (1965), reported similar variations for beef muscle at slaughter; e.g. lactic acid, 13.1 ± 6.0 $\mu\text{Moles/g}$; ATP, 6.4 ± 1.2 $\mu\text{Moles/g}$; creatine phosphate, 9.1 ± 3.2 $\mu\text{Moles/g}$; ortho P, 22.1 ± 3.1 micro-atoms/g. Bodwell et al. (1966), reported ATP concentrations of 5.6 ± 4.1 and 3.2 ± 3.7 $\mu\text{Moles/g}$ tissue in pork muscle at 13 and 166 minutes post-mortem respectively. Hall et al. (1968)

¹ Because almost every datum contained in Tables II and III is based on $n=5$ (for the few exceptions $n=3$ or 4 ; see Appendix A - Tables VIII and IX) the standard deviations presented are each approximate estimates of $\frac{1}{2}$ the 95% confidence interval for their corresponding mean, i.e. $1.96 \frac{s}{\sqrt{5}} \approx s$. Thus, the signi-

TABLE IV

AVERAGE COEFFICIENTS OF VARIABILITY¹ ASSOCIATED WITH THE
PLOTS OF THE MEAN CONCENTRATIONS OF INTERMEDIATES
AND CO-FACTORS OVER TIME POST-MORTEM

Intermediate or co-factor	Age Group	
	15 Weeks	25 Weeks
ATP	116.01	65.17
ADP	26.66	29.23
AMP	55.59	44.98
NAD	16.38	17.47
NADH	34.45	51.20
CP	162.90	81.89
Total Creatine	48.87	18.64
P _i	24.54	19.54
G-1-P	71.19	70.61
G-6-P	46.33	47.55
F-6-P	48.44	57.31
FDP	98.80	61.67
DHAP	64.94	33.71
GA3P	61.20	29.08
3PGA	68.00	37.75
2PGA	80.88	41.36
PEP	98.23	30.41
Pyruvate	80.34	35.51
Lactate	43.28	30.14
α-GP	31.16	34.21
Glucose	28.84	12.20
Glycogen	10.14	44.32

¹Average coefficient of variability is the arithmetic average of the coefficients of variability for each of the 6 post-mortem sampling times.

reported ATP, ADP and AMP concentration in turkey muscle 4.5 minutes after slaughter, of 3.1 ± 1.0 , 1.34 ± 0.52 and 0.18 ± 0.08 μ Moles/g respectively.

The average concentration of all intermediates and co-factors in turkey breast muscle are similar in magnitude to those reported by Kastenschmidt et al. (1968) for "slow" and "fast-glycolyzing" porcine muscles.

The very rapid decline of creatine phosphate for both 15 and 25 week-old birds parallels the rapid decline of ATP concentration. Creatine phosphate in fact is depleted at an earlier time post-mortem than is ATP, particularly when considering individual birds (Appendix A - Table VIII and IX). This is in agreement with the findings of Newbold (1966) and Kadtenschmidt et al. (1968).

ficance of differences between corresponding means from each of the two age groups can be approximated from an assessment of the degree of overlap of the ranges of concentrations represented by $\bar{x} \pm s$ as presented in the tables.

Possible sites of post-mortem glycolytic control

The fact that only few significant differences in concentration of intermediates and co-factors were found between 15 and 25 week-old birds did not preclude the possibility of differences in the glycolytic control sites for the two age groups. Some pairs of means, both between age groups and between intermediates within an age group, differed by several orders of magnitude and the lack of statistical significance is likely predominately a reflection of the relatively low number of samples (5) for the variability encountered. For this reason and because the author was unaware of any studies of glycolytic control sites in post-mortem turkey muscle it was considered germane to estimate the susceptibility of the various glycolytic reactions to control. To accomplish this, mass action ratios of the majority of the glycolytic enzymes were calculated for each age group according to the methods of Minakami et al. (1966) as summarized in Table V.

The results appear in Table VI along with the apparent equilibrium constants taken from Williamson (1965) and Minakami et al. (1966). By comparing mass action ratios with the known equilibrium constants of the enzymes concerned some indication of their susceptibility to control can be achieved. Minakami et al. (1966), consider any reaction with a mass action ratio differing from the equilibrium constant by a factor

TABLE V
MASS ACTION RATIOS OF THE GLYCOLYTIC REACTIONS

Enzymes	Mass Action Ratio
Hexokinase (HK)	$\frac{(G-6-P)(ADP)}{(Glucose)(ATP)}$
Phosphoglucomutase (PGM)	$\frac{(G-6-P)}{(G-1-P)}$
Phosphoglycoisomerase (PGI)	$\frac{(F-6-P)}{(G-6-P)}$
Phosphofructo kinase (PFK)	$\frac{(FDP)(ADP)}{(F-6-P)(ATP)}$
Aldolase (Ald)	$\frac{(GA3P)(DHAP)}{(FDP)}$
Phosphotriose isomerase (PTI)	$\frac{(GA3P)}{(DHAP)}$
Ald x PTI	$\frac{(GA3P)^2}{(FDP)}$
Glyceraldehyde phosphate dehydrogenase x phosphoglyceric kinase (GAPDH) x (PGK)	$\frac{(3PGA)(ATP)(NADH)}{(GA3P)(ADP)(P_i)(NAD)}$
Phosphoglycerate mutase (PGM)	$\frac{(2-PGA)}{(3-PGA)}$
Enolase (Enol)	$\frac{(PEP)}{(2-PGA)}$
Pyruvate kinase (PK)	$\frac{(Pyruvate)(ATP)}{(PEP)(ADP)}$
Lactate dehydrogenase (LDH)	$\frac{(Lactate)(NAD)}{(Pyruvate)(NADH)}$

of less than 10^2 to 10^3 , as being in quasi-equilibrium. Because the free energy change for reactions close to equilibrium is small, these reactions are not normally susceptible to control.

TABLE VI
MASS ACTION RATIOS CALCULATED FROM "0" MINUTE DATA
ON GLYCOLYTIC INTERMEDIATES AND CO-FACTORS
IN MUSCLES OF 15 and 25 WEEK-OLD BIRDS

Reaction ¹	Apparent Equilibrium Constant	Mass Action Ratios	
		15 wks	25 wks
HK	4×10^{-4}	2.69×10^{-1}	1.91×10^{-1}
PGM	5.5×10^{-2}	9.58	9.56
PGI	0.28 - 0.45	1.68×10^{-1}	1.13×10^{-1}
PFK	$1-1.2 \times 10^3$	3.59×10^{-2}	3.70×10^{-2}
Aldolase	$0.7-1.3 \times 10^{-4}$	1.16×10^{-2}	7.30×10^{-3}
PTI	$4-5 \times 10^{-2}$	5.55×10^{-1}	6.60×10^{-1}
Ald. x PTI	$4-6.5 \times 10^{-6}$	6.44×10^{-3}	4.81×10^{-4}
GAPDH x PGK	$2-15 \times 10^2$	5.65×10^{-2}	6.72×10^{-2}
PGM	$1-1.8 \times 10^{-1}$	6.29×10^{-1}	9.44×10^{-1}
Enol.	2.8 - 6.3	1.87	1.89
PK	$2-20 \times 10^3$	92.24	118.67
LDH	1.6×10^4	1.59×10^3	1.52×10^3

¹Abbreviations as given in Table V.

The data of Table VI indicate no apparent difference in the post-mortem metabolism of the 15 and 25 week-old age groups. With the exception of the Aldolase x phosphotriose isomerase reaction, the mass action ratios of the two groups differ by less than two-fold. Kastenschmidt et al. (1968)

reported on the basis of their investigations, that the different post-mortem glycolytic rates exhibited by the various strains of swine used, could probably be explained by differences in post-mortem glycolytic control. In a subsequent report, Kastenschmidt (1970) stated that control properties of the two rates of glycolysis are unlikely to be different.

For both age groups, the probable control sites are the reaction steps catalyzed by hexokinase, phosphofructokinase, aldolase and triosphosphate isomerase, glyceraldehyde phosphate dehydrogenase and phosphoglycerokinase and pyruvate kinase. The reactions at these points can be considered to be severely displaced from equilibrium. Manipulation of the 120 minute data in a manner similar to that shown in Table VI gave similar results.

Williamson (1965), showed that in the glycolysis of rat heart tissue, hexokinase, phosphofructkinase, glyceraldehyde phosphate dehydrogenase and pyruvate kinase were far displaced from equilibrium and were therefore operational in the glycolytic flux. Minakami et al. (1966) showed that the reactions catalyzed by hexokinase, phosphofructokinase and pyruvate kinase were rate limiting steps in the glycolytic process in erythrocytes. Phosphorylase, phosphofructokinase and pyruvate kinase have been implicated in the post-mortem glycolytic control of porcine muscle (Kastenschmidt et al., 1968). Southard et al. (1969), describe the preparation of

a chicken breast muscle mitochondrial fraction which is capable of aerobic respiration or lactate production and in which the hexokinase catalyzed phosphorylation of glucose is the rate limiting step under both the aerobic and anaerobic conditions.

If no control were exerted on the glycolytic flux post-mortem, then glycogen would be stoichiometrically converted to lactic acid and all intermediate concentrations would remain constant. From Tables II and III and Figures 1 - 6 it can be seen that while glycogen concentration decreased, glucose, G-1-P, G-6-P and F-6-P concentrations increased with time post-mortem. All four of these intermediates remained relatively constant over the initial 30 minute period, after which the concentration began to rise, indicating an inhibition of phosphofructokinase and thus a buildup of intermediates prior to F-6-P phosphorylation. The intermediates subsequent to this reaction were markedly reduced in concentration and declined in concentration over time. The decline was approximately parallel in DHAP, GA-3-P, 3PGA, 2PGA and PEP. These findings substantiate the evidence from mass action ratios which indicates that the phosphofructokinase catalysed reaction is a glycolytic control site.

ATP Metabolism

Because of the well established importance of ATP concentration and rate of disappearance to post-mortem muscle

extensibility and subsequent physical and chemical changes (e.g., Newbold, 1966; de Fremery et al., 1963; Khan, 1971) and because the data for ATP concentration (Tables II and III) suggested differences among birds, an attempt was made to categorize the 10 birds on the basis of rate of ATP disappearance.

The ATP concentration at 15, 30, 60, 120 and 180 min. post-mortem as a percentage of initial ATP concentration was calculated for each bird (Table VII). These values tend to fall into two distinct groups, each containing several birds of each age. The two groups were evident at the 15, 30 and 60 min. but not at the 120 and 180 min. time periods. The groups are most clearly distinguished on the basis of the 60 min. data for which the groups exhibited ranges of ATP concentration as a percentage of initial ATP concentration of 5.16 to 32.49% and 52.81 to 76.00%. The latter group contained birds numbered 2, 6, 10 and 11 and hereafter will be referred to as Group I. The former group contained birds numbered 3, 4, 5, 12, 13 and 14 and will henceforth be referred to as Group II.

Lactic acid and pH

Inspection of the lactic acid concentration and pH data for both age groups, on either an individual bird or mean basis, shows a lack of agreement. It is generally reported, that since lactic acid is the end product of glycolysis, pH can

be taken as an indicator of the extent of post-mortem glycolysis (e.g., Kastenschmidt et al., 1968 and Khan et al., 1970).

Bodwell et al. (1965), report that in beef during the initial 24 hour post-mortem period, 46.5 μM of lactic acid/g of muscle were produced for every unit decrease in pH. The workers report values showing similar relationships for other species (Bodwell et al., 1966). Gunther et al. (1966), however, after investigating the changes of lactate concentration and pH values at varying times post-mortem on different samples of beef and pork muscles, reported that the results indicated no simple correlation between lactate concentration and pH values. They further reported a considerable difference in the results obtained between the chemical and enzymatic method of determining lactic acid.

The concentration of lactic acid reported in the present study (273.3 - 369.3 $\mu\text{Moles/g}$) appears quite high in comparison to other reported concentrations. Bodwell et al. (1965), report lactic acid concentrations in beef ranging from 13.1 to 84.6 $\mu\text{Moles/g}$ for initial and 180 minutes post-mortem respectively. Kastenschmidt et al. (1968), report concentrations ranging from about 30 to 115 $\mu\text{Moles/g}$ tissue. Shrimpton (1960), reported concentrations of approximately 1 mg/g one minute after slaughter to a maximum of about 8 mg/g fresh weight 10 minutes after slaughter. Depending on the method of slaughtering or the treatment before slaughter, lactic acid concentrations after slaughter has been reported to range from 2.6 to 9.0 mMoles/100g of chicken muscle (Khan et al., 1970).

The method of measurement and the results obtained in the present study were rechecked. It is suggested that the differences in lactic acid concentration between the present study and those reported in the literature might be explained in part by species differences.

TABLE VII
ATP CONCENTRATION AS A PERCENTAGE OF INITIAL
ATP CONCENTRATION

Bird No.	Age (wks)	Time (min.) Post-mortem				
		15	30	60	120	180
2	15	117.05%	111.37	61.37	9.09	2.27
3	15	57.14	28.57	14.28	14.28	2.39
4	15	49.93	29.95	5.16	2.58	5.16
5	15	143.77	31.89	32.49	0	0
6	15	88.29	83.77	74.25	11.42	3.79
10	25	79.77	68.54	52.81	8.99	2.25
11	25	72.00	68.00	76.00	16.00	4.00
12	25	58.18	50.91	14.54	7.27	0
13	25	57.32	44.10	11.76	2.94	2.94
14	25	55.17	27.59	20.69	17.24	20.69

pH Pattern of Group I and Group II

Average muscle pH for each group at varying times post-mortem appear in Table VIII and Figure 10B. Although the zero time pH values were similar, the two groups exhibited a considerable difference in the rate of decline. The pH of Group I dropped rapidly from 6.11 at 0 min. to 6.01 at 15 min., rose to 6.05 at 30 min., remained constant from 30 to

60 min. and subsequently decreased to 5.99 at 120 minutes. The final pH was 5.99. Group II exhibited an extremely rapid pH drop from 6.16 at 0 min. to 5.85 at 15 min., after which it gradually rose to 5.93 during the next 105 min. and subsequently declined slightly to the ultimate pH of 5.92 at 180 minutes.

TABLE VIII

COMPARISON OF pH VALUES AT VARYING TIMES POST-MORTEM
FOR GROUP I AND GROUP II TURKEYS

Time (min.) Post-mortem	Group I	Group II
0	6.11 $\pm$ 0.17 ¹	6.16 $\pm$ 0.14
15	6.01 $\pm$ 0.16	5.85 $\pm$ 0.11
30	6.05 $\pm$ 0.18	5.89 $\pm$ 0.10
60	6.05 $\pm$ 0.14	5.91 $\pm$ 0.07
120	5.99 $\pm$ 0.30	5.93 $\pm$ 0.08
180	5.99 $\pm$ 0.28	5.92 $\pm$ 0.09

¹Values expressed as mean $\pm$ standard deviation.

Glycolytic Intermediates and Co-factors
of Groups I and II

Using the data of Appendix A - Table VIII and XI, averages for every intermediate and co-factor over time post-mortem were calculated for each group. The averages and standard deviations are tabulated in Tables IX and X for Groups I and II respectively.

TABLE IX

CONCENTRATION¹ OF THE GLYCOLYTIC INTERMEDIATES AND CO-FACTORS IN P. SUPERFICIALIS MUSCLE
AT VARYING TIMES POST-MORTEM FOR GROUP I TURKEYS

Intermediate or Co-Factor	Time (min.) Post-mortem		
	0	15	30
ATP ²	5.0731 ± 2.0300 ⁵	4.4282 ± 1.6740	4.0886 ± 1.5540
ADP	0.9702 ± 0.1312	0.8967 ± 0.3167	0.9887 ± 0.2589
AMP	0.2013 ± 0.0645	0.2485 ± 0.1696	0.1808 ± 0.1056
NAD	0.8354 ± 0.1791	0.8483 ± 0.1519	0.8444 ± 0.1616
NADH	0.2985 ± 0.1744	0.4287 ± 0.2523	0.3336 ± 0.1975
CP	1.7099 ± 1.5480	2.3318 ± 3.0090	1.1355 ± 1.5400
Total Creatine ³	4.4551 ± 2.2000	4.5442 ± 2.2260	4.8319 ± 2.3580
P _i	12.5319 ± 5.4870	14.3706 ± 1.0910	15.6725 ± 3.0920
G-1-P	0.2967 ± 0.1568	0.2338 ± 0.1061	0.1734 ± 0.1041
G-6-P	3.6812 ± 1.2630	3.4548 ± 1.2000	3.1420 ± 1.0680
F-6-P	0.4853 ± 0.1015	0.4846 ± 0.1441	0.3539 ± 0.1907
FDP	0.0628 ± 0.0392	0.0509 ± 0.0393	0.0561 ± 0.0463
DHAP	0.0321 ± 0.0214	0.0286 ± 0.0204	0.0342 ± 0.0288
GA3P	0.0127 ± 0.0052	0.0159 ± 0.0031	0.0159 ± 0.0043
3PGA	0.0093 ± 0.0063	0.0116 ± 0.0050	0.0095 ± 0.0059
2PGA	0.0087 ± 0.0051	0.0138 ± 0.0038	0.0085 ± 0.0049
PEP	0.0156 ± 0.0085	0.0137 ± 0.0083	0.0111 ± 0.0045
Pyruvate	0.4738 ± 0.2257	0.2818 ± 0.1556	0.1302 ± 0.1010
Lactate	236.7775 ± 99.8900	265.2485 ± 119.8000	239.0618 ± 110.9000
α-GP	0.7456 ± 0.2943	0.6955 ± 0.2579	0.6210 ± 0.2379
Glucose	3.6599 ± 0.3851	3.4585 ± 0.5621	3.3538 ± 1.1570
Glycogen ⁴	6.8436 ± 1.3470	7.0212 ± 0.8788	6.9922 ± 1.3910

Continued.....

¹Concentrations expressed as μMoles/g tissue.

²Abbreviations as given in text.

³Concentration expressed as mg/g tissue.

⁴Concentration expressed as μMoles glucose/g tissue.

⁵Values are means ± standard deviation.

TABLE IX (CONTINUED)

Intermediate or Co-factor	Time (min.) Post-mortem		
	60	120	180
ATP	3.3431 ± 1.4890	0.5633 ± 0.2224	0.1578 ± 0.0853
ADP	1.1837 ± 0.0966	0.9686 ± 0.3835	0.7919 ± 0.2756
AMP	0.2183 ± 0.0445	1.1779 ± 0.6378	1.1922 ± 0.6258
NAD	0.8075 ± 0.1931	0.7887 ± 0.1717	0.7825 ± 0.1589
NADH	0.3737 ± 0.1013	0.2501 ± 0.2068	0.2924 ± 0.1044
CP	0.3616 ± 0.3794	0.2351 ± 0.2281	0.1083 ± 0.1375
Total Creatine	4.5284 ± 2.4380	4.6228 ± 2.4750	4.8783 ± 2.4570
P _i	15.1368 ± 2.7530	14.4796 ± 1.5900	17.7213 ± 7.9790
G-1-P	0.1877 ± 0.1844	0.3116 ± 0.1769	0.3031 ± 0.2428
G-6-P	3.5855 ± 1.2660	8.6594 ± 1.1670	10.7076 ± 0.5615
F-6-P	0.4430 ± 0.2719	1.2177 ± 0.2876	1.6747 ± 0.2172
FDP	0.0449 ± 0.0402	0.0308 ± 0.0310	0.0065 ± 0.0024
DHAP	0.0227 ± 0.0099	0.0171 ± 0.0036	0.0089 ± 0.0063
GA3P	0.0138 ± 0.0037	0.0105 ± 0.0043	0.0039 ± 0.0049
3PGA	0.0086 ± 0.0050	0.0109 ± 0.0053	0.0132 ± 0.0065
2PGA	0.0061 ± 0.0039	0.0052 ± 0.0047	0.0113 ± 0.0057
PEP	0.0079 ± 0.0039	0.0089 ± 0.0066	0.0112 ± 0.0057
Pyruvate	0.1195 ± 0.0823	0.1903 ± 0.0675	0.1610 ± 0.0428
Lactate	245.3914 ± 98.1300	259.9055 ± 92.4200	247.6486 ± 110.7000
α-GP	0.5162 ± 0.1709	0.5628 ± 0.2617	0.6155 ± 0.2269
Glucose	3.8333 ± 0.5330	4.7309 ± 0.5383	4.7871 ± 0.6946
Glycogen	7.1153 ± 1.2460	6.5375 ± 1.4010	5.6754 ± 1.8540

The concentrations of glycolytic intermediates and co-factors do not differ greatly between Groups I and II (Figures 7 - 12). The differences between the two groups are of a magnitude similar to those between the two age groups. The large variation from bird to bird, evident on the basis of age grouping, is also present in the ATP - grouped data. The data for each of the intermediates and co-factors indicates that the standard deviations are of very similar magnitude, whether grouped on the basis of age or ATP disappearance rate.

Concentration of G-6-P was essentially the same for the two groups during the first 60 minutes but from 60 to 180 min. the Group I concentration rose sharply to 2.1 times the concentration of Group II at 180 min. Fructose-6-phosphate concentration followed a similar pattern. The FDP concentration for Group I birds decreased steadily and almost linearly over the 180 min. but for Group II birds there was a rapid decrease over the initial 30 minute period, after which the concentration remained essentially constant. The initial concentrations of DHAP for the two groups were somewhat different, with Group I showing a higher initial concentration which gradually decreased over the 3 hours, while Group II showed an initial rapid drop over the first 30 min. and a relatively constant concentration over the next 150 min. Group I concentration of GA3P showed a slight initial increase followed by a gradual decrease to its lowest level at 180 min. whereas the concentration of GA3P for the Group II birds showed a sharp de-

TABLE X

CONCENTRATION¹ OF THE GLYCOLYTIC INTERMEDIATES AND CO-FACTORS IN P. SUPERFICIALIS MUSCLE
AT VARYING TIMES POST-MORTEM FOR GROUP II TURKEYS

Intermediate or Co-factor	Time (min.) Post-mortem		
	0	15	30
ATP ²	2.2403 ± 1.6800 ⁵	1.3803 ± 0.9074	0.9152 ± 0.8838
ADP	1.0448 ± 0.2238	1.0224 ± 0.2388	0.9731 ± 0.2055
AMP	0.3206 ± 0.0485	0.2953 ± 0.0871	0.2886 ± 0.0973
NAD	0.9054 ± 0.1949	0.7999 ± 0.1910	0.7061 ± 0.2187
NADH	0.4571 ± 0.2691	0.4330 ± 0.2033	0.2877 ± 0.0586
CP	0.3922 ± 0.2369	0.3523 ± 0.2145	0.2186 ± 0.2029
Total Creatine ³	3.8310 ± 1.3630	3.9154 ± 1.3630	3.9049 ± 1.3620
P _i	17.6219 ± 3.0040	19.5308 ± 2.9220	19.5510 ± 3.0080
G-1-P	0.2979 ± 0.2900	0.2692 ± 0.1566	0.2830 ± 0.1544
G-6-P	2.2920 ± 1.0180	2.1459 ± 1.0380	2.3880 ± 0.9249
F-6-P	0.3267 ± 0.1890	0.3175 ± 0.2245	0.3569 ± 0.2210
FDP	0.0367 ± 0.0395	0.0107 ± 0.0052	0.0069 ± 0.0035
DHAP	0.0235 ± 0.0169	0.0156 ± 0.0088	0.0130 ± 0.0024
GA3P	0.0186 ± 0.0064	0.0134 ± 0.0047	0.0114 ± 0.0055
3PGA	0.0125 ± 0.0052	0.0076 ± 0.0043	0.0135 ± 0.0081
2PGA	0.0092 ± 0.0043	0.0077 ± 0.0044	0.0096 ± 0.0082
PEP	0.0178 ± 0.0105	0.0144 ± 0.0096	0.0202 ± 0.0145
Pyruvate	0.5635 ± 0.2269	0.4822 ± 0.1885	0.4152 ± 0.1813
Lactate	416.5578 ± 64.7400	409.2092 ± 81.4900	409.9011 ± 115.0000
α-GP	1.0623 ± 0.4245	0.9105 ± 0.3170	0.7897 ± 0.2700
Glucose	3.9271 ± 1.0650	4.0163 ± 0.9775	4.2215 ± 0.9130
Glycogen ⁴	5.4460 ± 2.1520	5.4439 ± 1.9090	5.4439 ± 1.9090

¹Concentrations expressed as μMoles/g tissue.

Continued.....

²Abbreviations as given in text.

³Concentration expressed as mg/g tissue.

⁴Concentration expressed as μMoles glucose/g tissue.

⁵Values are means ± standard deviation.

TABLE X (CONTINUED)

Intermediate or Co-factor	Time (min.) Post-mortem					
	60		120		180	
ATP	0.3152	± 0.2039	0.1431	± 0.1080	0.0782	± 0.0874
ADP	0.8650	± 0.2908	0.7751	± 0.2359	0.7881	± 0.2385
AMP	0.3496	± 0.1586	0.4386	± 0.1648	0.5903	± 0.2437
NAD	0.6754	± 0.2308	0.6626	± 0.2287	0.7868	± 0.2695
NADH	0.2589	± 0.0612	0.3398	± 0.1652	0.3650	± 0.1567
CP	0.0618	± 0.0500	0.0493	± 0.0552	0.0617	± 0.0724
Total Creatine	3.5867	± 1.4720	3.4668	± 1.1090	3.8593	± 1.4340
P _i	19.3393	± 1.9750	19.1442	± 3.5470	17.9459	± 3.8170
G-1-P	0.3065	± 0.2224	0.3067	± 0.2260	0.4022	± 0.3231
G-6-P	3.2551	± 1.6890	4.0551	± 2.2860	5.6805	± 3.2770
F-6-P	0.4783	± 0.3435	0.6484	± 0.4106	0.8306	± 0.5352
FDP	0.0069	± 0.0023	0.0054	± 0.0029	0.0055	± 0.0030
DHAP	0.0147	± 0.0024	0.0108	± 0.0026	0.0118	± 0.0031
GA3P	0.0127	± 0.0055	0.0169	± 0.0107	0.0139	± 0.0058
3PGA	0.0107	± 0.0052	0.0132	± 0.0056	0.0117	± 0.0063
2PGA	0.0097	± 0.0060	0.0089	± 0.0063	0.0086	± 0.0055
PEP	0.0142	± 0.0067	0.0171	± 0.0086	0.0189	± 0.0115
Pyruvate	0.4160	± 0.2473	0.3882	± 0.2318	0.3975	± 0.2320
Lactate	320.3875	± 66.5100	396.8778	± 139.2000	441.3691	± 72.5400
α-GP	0.7412	± 0.2816	0.7723	± 0.2501	0.8851	± 0.3084
Glucose	4.0881	± 0.9103	4.1978	± 0.9025	4.2380	± 1.0780
Glycogen	5.5098	± 2.1420	5.7024	± 2.1220	5.5715	± 1.9880

Figure 7. Comparisons of concentrations of some glycolytic intermediates in *P. superficialis* muscles in Group I turkeys (Δ—Δ) and Group II turkeys (○—○).

Figure 8. Comparisons of concentrations of some glycolytic intermediates in *P. superficialis* muscles in Group I turkeys (Δ — Δ) and Group II turkeys ($\circ$ — $\circ$).

Figure 9. Comparisons of concentrations of some glycolytic intermediates in *P. superficialis* muscles in Group I turkeys (Δ — Δ) and Group II turkeys ($\circ$ — $\circ$).

Figure 10. Comparisons of concentrations of some glycolytic intermediates in and pH values of *P. superficialis* muscles in Group I turkeys (Δ — Δ) and Group II turkeys ($\circ$ — $\circ$). Glycogen concentration is expressed as μMoles of glucose/g.

Figure 11. Comparisons of concentrations of some glycolytic co-factors in *P. superficialis* muscles in Group I turkeys (Δ—Δ) and Group II turkeys (O—O).

Figure 12. Comparisons of concentrations of some glycolytic co-factors in *P. superficialis* muscle in Group I turkeys (Δ—Δ) and Group II turkeys (O—O). Creatine concentration is expressed as mg/g.

crease over the initial 30 min. then an increase almost to the initial level at 120 min. and then a slight decline to 180 min. The concentration of α -GP for both Groups showed a fairly rapid decrease over the first hour after which it rose gradually over the last two hours of measurement. The concentration of 3PGA and 2PGA for both Groups exhibited considerable fluctuation over the three hours period. PEP concentration for Group I decreased rapidly while for Group II it fluctuated considerably for the first 60 min. The concentration of this intermediate increased slightly over the next two hours for both Groups. Pyruvate concentration dropped for both Groups over the initial 30 min. but remained essentially constant for the remainder of the period. Glucose concentration of Group I muscles increased quite sharply between 30 and 120 min. Glycogen concentration, was essentially constant for the first hour, then showed a considerable drop over the last two hours of measurement. Both glucose and glycogen concentration of Group II muscles, showed very little increase over the three hours.

Initial ATP concentration was 5.0731 μ Moles/g and 2.2403 μ Moles/g for Groups I and II respectively, but the concentration for both Groups was essentially zero at the end of the three hour period. The formation of the two groups on the basis of %-ATP at 60 min. accounts for some of the differences in ATP concentration for the groups at varying times. However, it is evident from the data (Tables IX and X and Figure 11A) that a two-fold difference in initial ATP concentration also existed

between the groups and was maintained or increased during the first 60 min. after which the rate of disappearance of ATP from Group I muscles increased and that of Group II muscles decreased.

Both Groups showed a general decrease in ADP concentration, however, Group I concentration was somewhat higher than that of Group II during the period from 60 to 180 min. post-mortem.

The concentration of AMP for both Groups was essentially the same and constant during the first hour. Group II concentration rose only slightly but Group I concentration increased 5.5 fold over the next 2 hour period.

Initial CP concentration was higher for Group I but the concentration at 180 min. was essentially zero for both Groups. Total creatine concentration showed very little net change for either group.

Group I NAD concentration decreased sharply over the initial 30 min. period, continued to decline during the next 1.5 hours and sharply increased during the last hour. The Group II concentration showed only a slight decrease over the total three hour period. The NADH concentration for both Groups showed a slight net decrease over the three hour period.

Inorganic phosphate concentration showed an overall increase over the three hours for Group I but a slight initial increase and then a gradual decrease to the initial level for Group II.

Kastenschmidt et al. (1968), categorized pork muscles as "slow-" or "fast-glycolyzing" on the basis of post-mortem pH decline and reported that slow-glycolyzing muscles had a 40% higher initial ATP concentration. The results of the present study, clearly indicate that Group I muscles can be classified as "slow-" and Group II as "fast-glycolyzing". This classification is compatible with the data on pH and lactic acid concentrations over the three hour post-mortem period. "Fast-glycolyzing" muscles (Group II) had a lower initial ATP concentration, larger percentage of ATP breakdown between 0 and 60 minutes, more rapid initial pH drop and a higher lactic acid concentration than did the "slow-glycolyzing" muscles (Group I). These differences between the two groups are statistically significant at probability levels ranging from 0.0001 to 0.20 (Appendix A - Table XI).

From the studies described here, it is impossible to determine whether the initial ATP level of the "fast-glycolyzing" birds is a reflection of the general in vivo ATP concentration or of some physiological state which makes these birds more susceptible to the stress of slaughter. However, the data clearly establish the existence of considerable variation in post-mortem glycolytic rate among turkeys.

Lactic acid and pH for Group I and Group II

The data showing lactic acid concentrations and pH values over time (Table IX and X and Figures 10 A and B) do not

demonstrate a consistent relationship. This confirms the findings reported by Gunther et al. (1966).

Possible sites of post-mortem glycolytic control for
Group I and Group II

Mass action ratios using the "0" min. data of Groups I and II were calculated as outlined previously. The mass action ratios indicated that for each group, the possible post-mortem glycolytic controlling points are at the reactions catalyzed by hexokinase, phosphofructokinase, aldolase and triosephosphate isomerase, glyceraldehyde phosphate dehydrogenase and phosphoglycerokinase, and pyruvate kinase (Table XI).

Further evidence that the post-mortem glycolytic flux may be controlled at these points might be presented. As was the case for the data of the 15 and 25 week-old birds, the intermediates prior to the point of action of phosphofructokinase in the cycle, showed an increase in concentration over time, whereas the metabolites after this point were markedly reduced in concentration and generally exhibited a decrease or maintained a relatively stable level over time.

It is known (e.g. Kastenschmidt et al., 1968; Williamson, 1965; Minakami et al., 1966) that both ATP and G-6-P counteract the activating influences of AMP and P_i on phosphorylase; that phosphofructokinase activity is influenced by F-6-P, ATP, ADP, AMP, P_i ; and pyruvate kinase by ATP. It must be accepted that the intermediates and co-factors of the glycolytic cycle may control its flux by either activating

TABLE XI

MASS ACTION RATIOS CALCULATED FROM "0" MINUTE DATA
ON GLYCOLYTIC INTERMEDIATES AND CO-FACTORS IN
MUSCLES OF GROUP I AND II TURKEYS

Reaction	Apparent Equilibrium Constant	Mass Action Ratios	
		Group I	Group II
HK	4×10^{-4}	1.92×10^{-1}	2.72×10^{-1}
PGM	5.5×10^{-2}	12.41	7.69
PGI	0.28 - 0.45	1.32×10^{-1}	1.43×10^{-1}
PFK	$1-1.1 \times 10^3$	2.47×10^{-2}	5.23×10^{-2}
Aldolase	$0.7-1.3 \times 10^{-4}$	6.40×10^{-3}	1.09×10^{-2}
PTI	$4-5 \times 10^{-2}$	3.96×10^{-1}	7.92×10^{-1}
Ald. x PTI	$4-6.5 \times 10^{-6}$	3.20×10^{-3}	8.20×10^{-3}
GAPDH x PGK	$2-15 \times 10^2$	1.09×10^{-1}	4.13×10^{-2}
PGM	$1-1.8 \times 10^{-1}$	9.36×10^{-1}	7.36×10^{-1}
Enolase	2.8 - 6.3	1.79	1.94
PK	$2-20 \times 10^3$	1.59×10^2	1.02×10^2
LDH	1.6×10^4	1.40×10^3	9.76×10^2

¹Abbreviations as given in Table V.

or inhibiting the various enzymes of the cycle. If activating and inhibiting factors were unbalanced between groups for any of several reactions as outlined, the mass action ratios would be expected to be different for the two groups. This is clearly not the case (Table XI) and therefore the difference in post-mortem glycolysis between the groups cannot be said to be mediated through variable rates of reaction at the inhibited

control sites.

EXPERIMENT IV

pH

The post-mortem changes in pH showed considerable variation from bird to bird in both the 19 and 27 week-old age groups (Appendix A - Table X), but were generally similar to those described in Experiment I, i.e., a rapid initial decline with a continued gradual decline to the ultimate pH or a rapid initial decline followed by a slight and gradual increase to the ultimate pH.

The average pH values for each age group followed patterns which were quite similar (Table XII). The 19 week-old birds had an initial pH of 6.27 which declined very rapidly to pH 5.89 at 15 min. and then gradually rose to an ultimate of 5.98 at 180 min. The 27 week-old birds on the other hand, had an initial pH of 6.44 which decreased very rapidly to 6.04 at 15 min. and then continued to decline gradually to an ultimate of pH 5.95 at 180 min.

TABLE XII

pH OF P. SUPERFICIALIS MUSCLE AT VARYING TIMES POST-MORTEM
FOR 19 AND 27 WEEK-OLD TURKEYS (EXP. IV)

Time (min.) Post-mortem	Age (wks.)	
	19	27
0	6.27 $\pm$ 0.12 ¹	6.44 $\pm$ 0.32
15	5.89 $\pm$ 0.22	6.04 $\pm$ 0.22
30	5.90 $\pm$ 0.24	5.99 $\pm$ 0.20
60	5.93 $\pm$ 0.22	5.97 $\pm$ 0.17
120	5.98 $\pm$ 0.21	5.96 $\pm$ 0.13
180	5.98 $\pm$ 0.20	5.95 $\pm$ 0.10

¹Values expressed as mean $\pm$ standard deviation.

ATP

Adenosine triphosphate concentration was determined at "0" and "60" min. post-mortem to establish the rate of disappearance. As in Experiments I and II initial ATP concentration showed great variability among birds in each age group. The average initial ATP concentration was 5.4129 ± 2.2900 and 4.4858 ± 2.6400 μ Moles/g for 19 and 27 week-old birds respectively (Table XIII). ATP concentration at 60 minutes as a percent of initial ATP concentration (%-ATP) was $53.59 \pm 31.47\%$ and $55.20 \pm 19.05\%$ for the 19 and 27 week-old birds respectively.

TABLE XIII

COMPARISON ATP CONCENTRATION IN P. SUPERFICIALIS MUSCLES
AT "0" AND "60" MINUTES POST-MORTEM
FOR 19 AND 27 WEEK-OLD TURKEYS (EXP. IV).

Bird No.	Age (Wks.)	Time (min.) Post-mortem.		
		0	60	(60/0)x100
20	19	9.4403 ¹	8.8767 ¹	94.03 ²
21	19	4.2975	1.5499	36.07
22	19	4.2270	1.2681	30.00
23	19	6.7632	5.9178	87.50
24	19	4.5088	2.5362	56.25
25	19	3.2407	0.5636	17.39
	$\bar{x}^3$	5.4129±2.2900	3.4521±3.2580	53.59±31.47
30	27	3.0294	2.2544	74.42
31	27	8.7358	6.6928	76.61
32	27	6.6928	2.3953	35.79
33	27	1.8317	.5636	30.77
34	27	3.3488	1.8317	54.70
35	27	3.2760	1.9292	58.89
	$\bar{x}^3$	4.4858±2.6400	2.6110±2.1020	55.20±19.05
overall	$\bar{x}^3$	4.9490±2.4060	3.0320±2.6510	54.40±24.82

¹Values are averages of duplicate analysis expressed as μ Moles/g tissue.

²Values expressed as 60 minute concentration as a percent of initial concentration (%-ATP).

³Values are expressed as means $\pm$ standard deviations.

Rigor mortis measurement.

To relate the physical changes that take place to the chemical changes, an attempt was made to assess the contraction of the muscle post-mortem. To achieve a measure of this physical parameter the instrument previously described was used. Although not designed to allow the loading and unloading cycle

on the muscle strips, nor to allow the quantitative measurement of tension developed over time in the strips held at constant length, the attachment of a constant load on the strip during the post-mortem period was possible. By measuring the muscle strip length under constant load, it was felt that an approximation of the tension development pattern could be achieved.

In preliminary experiments it was found that the amount of applied weight that would allow the muscle strip to contract and shorten was insufficient to cause lengthening of the strip after it had passed into rigor, even up to 36 hours post-mortem. Conversely, the amount of applied weight sufficient to cause relengthening of the muscle strip post-rigor, was too great to allow contraction and shortening of the muscle strip pre-rigor.

In order to achieve an estimate of the contraction pattern, therefore, three muscle strips were excised from the P. superficialis immediately post-exsanguination. The strips were excised in order from the anterior to the posterior location and were 10 x 1.5 x 1.5 cm before excision. The strips were then allowed to contract freely and the weights attached after the completion of contraction as previously described.

An example of the contraction pattern is shown in Figure 13. The length measurements were made by Vernier calipers and the data expressed as $S=1-L/L_0$ (Davey et al. 1970). S defines the degree of shortening (+ ve) or stretching (- ve), L_0 is the initial length of the strip and L is the length at the

Figure 13. Typical contraction patterns of P. superficialis muscle strips.

time of measurement.

The typical pattern (Fig. 13) obtained in the present study is very similar to the isometric tension pattern described by Jungk et al. (1970). They described an initial phase to maximum tension development and a subsequent decrease in the relative tension. This tension development prior to the onset of rigor is equated with the tendency for muscle to shorten while being restrained by attachment to the carcass.

In the present study the parameter of importance was taken to be the time at which contraction was maximum i.e., no further shortening took place. Table XIV shows the time to maximum contraction for each of the three muscle strips from each individual bird. The overall average time to maximum contraction was 130, 128.2 and 125 minutes for strips 1, 2 and 3 respectively.

It has been established (e.g. Jungk et al. 1970) that excision of strips from the muscle induces some shortening. The amount of shortening induced by excision can be determined easily by comparison of the length of the strip on the carcass with that immediately after excision. Smith et al. (1969), reported shortening due to excision to be 10.4% and Jungk et al. (1970) reported a shortening of 10%. The data of Experiment IV indicated that the average amount of shortening due to excision was 8.7%.

Jungk et al. (1970), reported the average time for turkey breast muscle strips to achieve maximum tension to be 3.85 ± 0.19 hours. T-I Ma et al. (1971), reported considerable

variability in the time course of rigor mortis in turkey muscle strips. Their data showed a range of from 25 to 391 min. required to complete the decrease in extensibility. The data from the present study is also highly variable with a range of 60 to 210 minutes required for the muscle strips to attain maximum contraction.

TABLE XIV

TIMES TO MAXIMUM CONTRACTION FOR INDIVIDUAL
19 AND 27 WEEK-OLD BIRDS (EXP. IV).

Bird No.		Time (min.) to Maximum Contraction		
		Strip 1	Strip 2	Strip 3
20	19	130	150	150
21	19	105	105	105
22	19	110	84	96
23	19	165	165	165
24	19	90	90	90
25	19	90	75	60
		$\bar{x}^1$ 115 $\pm$ 28.64	111.5 $\pm$ 37.25	111 $\pm$ 39.34
30	27	105	105	105
31	27	210	210	195
32	27	180	180	180
33	27	105	105	90
34	27	135	135	135
35	27	135	135	135
		$\bar{x}^1$ 145 $\pm$ 42.07	145 $\pm$ 42.07	140 $\pm$ 40.99
overall		$\bar{x}^1$ 130 $\pm$ 37.72	128 $\pm$ 41.73	125 $\pm$ 41.19

¹Values are expressed as means $\pm$ standard deviations.

Rigor mortis vs. ATP

The post-mortem catabolism of ATP is known to be associated with the post-mortem physical changes of muscle (e.g. Newbold, 1966; Jungk et al., 1970; Khan, 1971). Sybesma (1969), indicated that in three classes of rigor, the muscles that were in complete rigor showed the lowest ATP concentration, those at the onset of rigor had intermediate and those without any stiffness had the highest ATP concentration.

Table XVII contains the correlation coefficients (r) between ATP concentration and time to maximum contraction. When the data for the 19 and 27 week-old birds was combined, the ATP concentration at 0 min. post-mortem showed the highest correlation with the time to maximum contraction. When the data for each age group was considered separately however, time to maximum contraction was most strongly correlated with %-ATP for 19 week-old birds and with initial ATP for the 27 week-old birds. The data clearly support the relationship between post-mortem ATP catabolism and physical changes in muscle.

Regrouping of Data on Basis of ATP Disappearance Rates

The data for the 12 birds were regrouped on the basis of ATP disappearance rates as described for Experiments I and II. The ATP concentration at 60 min. as a percent of initial

TABLE XVII

RELATIONSHIP BETWEEN ATP CONCENTRATION AND TIME
TO MAXIMUM MUSCLE CONTRACTION (EXP. IV).

	Simple Correlation Coefficients (r)		
	Strip 1	Strip 2	Strip 3
	(n= 12)	(n= 12)	(n= 12)
ATP-0 ¹	0.68 *	0.74 **	0.75 **
ATP-60 ²	0.57 n.s.	0.68 *	0.68 *
ATP-% ³	0.43 n.s.	0.56 n.s.	0.60 *
	(n= 10)	(n= 10)	(n= 10)
ATP-0 ¹	0.47 n.s.	0.58 n.s.	0.62 n.s.
ATP-60 ²	0.57 n.s.	0.73 *	0.74 *
ATP-% ³	0.65 *	0.80 **	0.83 **

¹ATP concentration at "0" minutes post-mortem.

²ATP concentration at "60" minutes post-mortem.

³ATP concentration at "60" minutes as a percent of ATP concentration at "0" minutes post-mortem.

**Significant at P 0.01

* Significant at P 0.05

n.s. Not significant at P 0.05

ATP concentration indicated two general groups with a %-ATP range of 55 to 94 for Group I and 17 to 37 Group II. Group I, included birds numbered 20, 23, 24, 30, 31, 34 and 35 had a mean %-ATP of 71.77 ± 15.66 and a mean initial ATP concentration of 5.5860 ± 2.7140 μ Moles/g. Group II included birds numbered 21, 22, 25, 32 and 33 had a mean %-ATP of 30.07 ± 7.58 and a mean initial ATP concentration of 4.0579 ± 1.7790 μ Moles/g.

Group I muscles had mean contraction times of 138.6 ± 39.55 , 141.4 ± 39.55 and 139.3 ± 35.41 min. for muscle strips 1,

2 and 3 respectively and an overall mean of 140.01 min. Group II had mean contraction time of 118.0 ± 35.46 , 109.8 ± 41.38 and 106.2 ± 44.58 min. for muscle strips 1, 2 and 3 respectively and an overall mean of 111.73 min. These data are consistent with the previously quoted literature, in that muscles with faster glycolytic rates (i.e. Group II) achieved rigor at an earlier time.

The regrouping served to reduce the variability of the ATP concentration data only slightly but showed no reduction in the variability in the data for time required to achieve maximum contraction. Regrouping of the data on the basis of ATP disappearance does not improve the correlation between ATP disappearance and time to maximum contraction. The regrouped data show higher correlations between initial ATP and contraction time than between %-ATP at 60 min. and time to maximum contraction.

The average time to maximum contraction appears to be linearly related to percent ATP (Figure 14). The relationship is considerably improved (Table XV) if the birds numbered 31 and 32 (Figure 14) are not considered. These two birds deviate severely from the relationship presented by the other 10 birds and although no explanation is readily apparent for this deviation the remainder of the data suggest a relationship worthy of consideration. The average time to maximum contraction for Group I shows a variability reduced from 38.1 to 35.0 min. and for Group II from 40.5 to 19.0 min., when these birds

Figure 14. Plot of maximum contraction time vs. %-ATP (ATP conc. at 60 min. as a percent of ATP conc. at 0 min.). (45-100% Group I; 0-45% Group II).

are omitted from the data. The correlation between %-ATP and time to maximum contraction is improved slightly for Group I and quite markedly for Group II.

It appears from the present study that the time required for muscle strips to reach maximum contraction is related to some extent to the post-mortem metabolic pathway, but that this relationship is not particularly strong.

SUMMARY AND CONCLUSIONS

The concentration of glycolytic intermediates and co-factors in breast muscle of five 15 week-old and five 25 week-old White Cannon tom turkeys was measured at six times post-mortem. pH was also measured over this time period.

The data showed large bird-to-bird variability in both age groups. It was concluded that different rates of post-mortem glycolysis occur in turkey breast muscle. However, the difference in rates is not correlated with age.

The patterns of change in concentration of the intermediates and co-factors suggest that post-mortem glycolysis in general, in turkey muscle is subject to some control at various reaction points. Mass action ratios as an indication of reaction equilibrium constants, suggest that controlling points might occur at the reaction steps catalyzed by hexokinase, phosphofructo kinase, aldolase and triosphosphate isomerase, glyceraldehyde phosphate dehydrogenase and phosphoglycerokinase and pyruvate kinase. Phosphofructo kinase appears to be the site of primary control.

When the data were regrouped on the basis of the disappearance of ATP over time post-mortem two distinct groups became evident. The large bird-to-bird variability contributed to the large variation evident for the two groupings. The two groups could be categorized as "fast-" and "slow-glycolyzing" on the basis of ATP catabolism, pH and lactic acid accumulation.

The data of the two groups indicated that post-mortem metabolism can be controlled at a number of sites but that the different rates of glycolysis can not be attributed to different extents of glycolytic control.

An additional six 19 week-old and six 27 week-old White Cannon tom turkey breast muscles were analyzed for ATP at "0" and "60" minutes post-mortem. pH during the three hour post-mortem period was also measured. The time course of rigor for three muscle strips from each bird was measured.

The pattern of muscle strip length over time post-mortem very closely resembled a pattern of tension development in muscle strips over time post-mortem.

On the combined data of the 19 and 27 week-old birds, time to maximum contraction showed the highest correlation ($r = .68 - .75$) with the initial ATP concentration, whereas for the 19 week-old birds the highest correlation was with ATP concentration at "60" minutes as a percent of "0" minute concentration. For the 27 week-old bird muscles the highest correlation was between initial ATP concentration and time to maximum contraction.

When the data for the 19 and 27 week-old birds were regrouped on the basis of ATP disappearance, a "slow-" and "fast-glycolyzing" group became evident. The group with the higher initial ATP concentration, had a larger percentage of the initial ATP remaining at "60" minutes and longer times for muscle strips to achieve maximum contraction.

LITERATURE CITED

- Adam, H. 1963. Adenosine-5¹-diphosphate and adenosine-5¹-monophosphate. in Bergmeyer, H.U. (ed.) *Methods of Enzymatic Analysis*, p.573. Academic Press, New York. 1965.
- Bendall, J.R. 1966. Muscle as a contractile machine. in Briskey, E.J., Cassens, R.G. and Trautman, J.C. (eds.). *The Physiology and Biochemistry of Muscle as a Food*. p.7. Univ. of Wisconsin.
- Bendall, J.R. 1969. *Muscles, Molecules and Movement*. Heinemann Educational Books Ltd. London.
- Berenblum, I. and Chain, E. 1938. XI. An improved method for the colorimetric determination of phosphate. *Bioch. J.* 32:295.
- Bodwell, C.E., Pearson, A.M. and Spooner, M.E. 1965. Post-mortem changes in muscle. 1. Chemical changes in beef. *J. Food Sci.* 30:766.
- Bodwell, C.E., Pearson, A.M., Wismer-Pedersen, J. and Bratzler, L.J. 1966. Post-mortem changes in muscle II. Chemical and physical changes in pork. *J. Food Sci.* 31:1.
- Borchert, L.L. and Briskey, E.J. 1965. Protein solubility and associated properties of porcine muscle as influenced by partial freezing with liquid nitrogen. *J. Food Sci.* 30:138.
- Briskey, E.J. 1964. Etiological status and associated studies of pale, soft exudative porcine musculature. *Adv. Food Res.* 13. Academic Press, New York.
- Briskey, E.J., Kastenschmidt, L.L., Forrest, J.C., Beecher, G.R., Judge, M.D., Cassens, R.G. and Hoekstra, W.G. 1966. Biochemical aspects of post-mortem changes in porcine muscle. *J. Agr. Food Chem.* 14(3):201.
- Brodine, M.V. and Carlin, A.F. 1968. Chilling and thawing methods and their effect on cooked whole turkeys. *J. Food Tech.* 22(5):607.
- Buck, E.M., Stankey, D.W. and Commissiong, E.A. 1970. Physical and chemical characteristics of free and stretched rabbit muscle. *J. Food Sci.* 35(1):100.
- Bucher, T. and Hohorst, H.J. 1963. Dihydroxyacetone phosphate, fructose-1, 6-diphosphate and D-glyceraldehyde-3-phosphate. Determination with glycerol-1-phosphate dehydrogenase,

- aldolase and triosephosphate isomerase. in Bergmeyer, H.U. (ed.) *Methods of Enzymatic Analysis*, p. 246. Academic Press, New York. 1965.
- Bush, W.A., Parrish, F.C. and Goll, D.E. 1967. Molecular properties of post-mortem muscle. 4. Effect of temperature on adenosine triphosphate degradation, isometric tension parameters and shear resistance of bovine muscle. *J. Food Sci.* 32:390.
- Buttkus, H. and Tomlinson, N. 1966. Some aspects of post-mortem changes in fish muscle. in Briskey, E.J., Cassens, R.G. and Trautman, J.C. (eds.) *The physiology and Biochemistry of Muscle as a Food*. p. 197. Univ. of Wisconsin.
- Canada's Who's Who of the Poultry Industry, 1970. 15th edition. Farm Papers Ltd., New Westminster, B.C.
- Canada Year Book, 1967. Dominion Bureau of Statistics, Canada Yearbook Division, Ottawa.
- Cassens, R.G. 1966. General aspects of post-mortem changes. in Briskey, E.J., Cassens, R.G. and Trautman, J.C. (eds.) *The Physiology and Biochemistry of Muscle as a Food*. p. 181. Univ. of Wisconsin.
- Czok, R. and Eckert, L. 1963. D-3-phosphoglycerate, D-2 phosphoglycerate, phosphoenolpyruvate. Determination with phosphoglycerate mutase, enolase, pyruvic kinase and lactic dehydrogenase. in Bergmeyer, H.U. (ed) *Methods of Enzymatic Analysis*. p. 229. Academic Press, New York. 1965.
- Davey, C.L. and Dickson, M.R. 1970. Studies in meat tenderness 8. Ultra-structural changes in meat during aging. *J. Food Sci.* 35(1):56.
- de Fremery, D. 1966a. Some aspects of post-mortem change in poultry muscle. in Briskey, E.J., Cassens, R.G. and Trautman, J.C. (eds.) *The Physiology and Biochemistry of Muscle as a Food*. p. 205. Univ. of Wisconsin.
- de Fremery, D. 1966b. Relationship between chemical properties and tenderness of poultry muscle. *J. Agr. Food Chem.* 14(3):214.
- de Fremery, D. and Pool, M.F. 1963. The influence of post-mortem glycolysis on poultry tenderness. *J. Food Sci.* 28(2):173.
- Dodge, J.W. and Stadelman, W.J. 1959. Post-mortem aging of poultry meat and its effects on the tenderness of breast muscle. *Food Tech.* 13(2):81.

- Dodge, J.W. and Stadelman, W.J. 1966. Variability in tenderness due to struggling. *Poultry Sci.* 39(3):672.
- Ennor, A.H. 1953. Determination and preparation of N-phosphates of biological origin. I. Phosphocreatine. in Colwick, S.P. and Kaplan, N.O. (eds.) *Methods in Enzymology*. p. 850. Academic Press, New York.
- Ennor, A.H. and Stocken, L.A. 1950. The determination of inorganic phosphate in the presence of labile organophosphorus compounds. *Australian J. Exp. Biol. Med. Sci.* 28(6):647.
- Fedde, M.R., Burger, R.E. and Kitchell, R.L. 1963. The effect of anesthesia and age on respiration following bilateral cervical vagotomy in the fowl. *Poultry Sci.* 42(5):1212.
- Forrest, J.C. 1968. Instruments used in the measurement of rigor mortis. *Proc. 21st Annual Rec. Meat Conf. of the Am. Meat Sci. Assn., National Live Stock and Meat Board, Chicago.*
- Goll, D.E. 1968. The resolution of rigor mortis. *Pro. 21st Annual Rec. Meat Conf. of the Am. Meat Sci. Assn., National Live Stock and Meat Board, Chicago.*
- Goodwin, T.L. 1966. The influence of strain on the tenderness of twenty-six week-old turkeys. *Poultry Sci.* 45(3):594.
- Goodwin, T.L., Andrews, L.D. and Webb, J.E. 1969. The influence of age, sex and energy level on the tenderness of broilers. *Poultry Sci.* 48(2):548.
- Greaser, M.L., Cassens, R.G., Briskey, E.J. and Hoekstra, W.G. 1969. Post-mortem changes in subcellular fractions from normal and pale soft exudative porcine muscle. *J. Food Sci.* 34(2):120.
- Gunther, H. and Schweiger, A. 1966. Changes in the concentration of lactic acid and free sugars in post-mortem samples of beef and pork muscle. *J. Food Sci.* 31:300.
- Hall, K.N. and Spencer, J.V. 1968. Quantitative comparison of certain nucleotides and related compounds in chicken and turkey meat. *Poultry Sci.* 47(1):300.
- Hanson, H.L., Stewart, G.F. and Lowe, B. 1942. Palatability and histological changes occurring in New York dressed broilers held at 1.7°C (35°F). *Food Research* 7:148.

- Harvey, E.B., Kaiser, H.E. and Rosenberg, R.E. 1969. Atlas of the Domestic Turkey (*Meleagris Gallopavo*). Myology and Osteology. United States Atomic Energy Commission, Div. of Biology and Medicine.
- Hohorst, H.J. 1963a. D-glucose-6-phosphate and D-fructose-6-phosphate. Determination with glucose-6-phosphate dehydrogenase and phosphoglucose isomerase. in Bergmeyer, H.U. (ed.) Methods of Enzymatic Analysis. p. 134. Academic Press, New York. 1965.
- Hohorst, H.J. 1963b. L-(+)-Lactate. Determination with lactic dehydrogenase and DPN. in Bergmeyer, H.U. (ed.) Methods of Enzymatic Analysis. p. 266. Academic Press, New York. 1965.
- Hohorst, H.J. 1963c. L-(-)-Glycerol-1-phosphate. Determination with glycerol-1-phosphate dehydrogenase. in Bergmeyer, H.U. (ed.) Methods of Enzymatic Analysis. p. 215. Academic Press, New York. 1965.
- Jungk, R.A., Snyder, H.E., Goll, D.E. and McConnell, K.G. 1967. Isometric tension changes and shortening in muscle strips during post-mortem aging. *J. Food Sci.* 32:158.
- Jungk, R.A. and Marion, W.W. 1970. Post-mortem isometric tension changes shortening in turkey muscle strips held at various temperatures. *J. Food Sci.* 35(2):143.
- Karpatkin, S., Helmreich, E. and Cori, C.F. 1964. Regulation of glycolysis in muscle. II. Effect of stimulation and epinephrine in isolated frog Sartorius muscle. *J. Biol. Chem.* 239(10):3139.
- Kastenschmidt, L.L. 1970. The metabolism of muscle as a food. in Briskey, E.J., Cassens, R.G. and Marsh, B.B. (eds.) *The Physiology and Biochemistry of Muscle as a Food*, 2. Univ. of Wisconsin.
- Kastenschmidt, L.L., Briskey, E.J. and Hoekstra, W.G. 1966. Metabolic intermediates in skeletal muscles with fast and slow rates of post-mortem glycolysis. *Nature* 212:288.
- Kastenschmidt, L.L., Hoekstra, W.G. and Briskey, E.J. 1968. Glycolytic intermediates and co-factors in "fast" and "slow glycolysing" muscles of the pig. *J. Food Sci.* 33:151.
- Khan, A.W. 1971. Effect of temperature during post-mortem glycolysis and dephosphorylation of high energy phosphates on poultry meat tenderness. *J. Food Sci.* 36(1):120.

- Khan, A.W. and Nakamura, R. 1970. Effects of pre- and post-mortem glycolysis on poultry tenderness. *J. Food Sci.* 35(3):266.
- Klingenberg, M. 1963a. Diphosphopyridine nucleotide (DPN). in Bergmeyer, H.U. (ed.) *Methods of Enzymatic Analysis*. p. 528. Academic Press, New York. 1965.
- Klingenberg, M. 1963b. Reduced diphosphopyridine nucleotide (DPNH). in Bergmeyer, H.U. (ed.) *Methods of Enzymatic Analysis*. p. 531. Academic Press, New York.
- Klose, A.A., Pool, M.F., Wiele, M.B. Hanson, H.L. and Lineweaver, H. 1959. Poultry tenderness. I. Influence of processing on tenderness of turkeys. *Food Tech.* 13(1):20.
- Klose, A.A., Campbell, A.A., Hanson, H.L. and Lineweaver, H. 1961. Effect of duration and type of chilling and thawing on tenderness of frozen turkeys. *Poultry Sci.* 40(3):683.
- Klose, A.A., Luyet, B.J. and Menz, L.J. 1970. Effect of contraction on tenderness of poultry muscle cooked in the pre-rigor state. *J. Food Sci.* 35(5):577.
- Landes, D.R., Dawson, L.E. and Price, J.F. 1971. Protein extractability of turkey breast muscle exhibiting different rates of post-mortem glycolysis. *J. Food Sci.* 36(1):122.
- Lamprecht, W. and Stein, P. 1963. Creatine phosphate. in Bergmeyer, H.U. (ed.) *Methods of Enzymatic Analysis*. p. 610. Academic Press, New York. 1965.
- Lamprecht, W. and Trautschold, I. 1963. Adenosine-5¹-triphosphate. Determination with hexokinase and glucose-6-phosphate dehydrogenase. in Bergmeyer, H.U. (ed.) *Methods of Enzymatic Analysis*. p. 543. Academic Press, New York. 1965.
- Lawrie, R.A. 1958. Physiological stress in relation to dark-cutting beef. *J. Sci. Food Agric.* 9:721.
- Lawrie, R.A. 1966a. *Meat Science*. Pergamon Press Ltd. Oxford.
- Lawrie, R.A. 1966b. Metabolic stresses which affect muscle. in - Briskey, E.J., Cassens, R.G. and Trautman, J.C. (eds.) *The physiology and Biochemistry of Muscle as a Food*. p. 137. Univ. of Wisconsin.
- Lister, D. 1970. The physiology of animals and the use of their muscle for food. in Briskey, E.J., Cassens, R.G. and Marsh, B.B. (eds.) *The Physiology and Biochemistry of Muscle as a Food*, 2. p.705. Univ. of Wisconsin.

- Lister, D., Sair, R.A., Will, J.A., Schmidt, G.R., Cassens, R.G., Hoekstra, W.G. and Briskey, E.J. 1970. Metabolism of striated muscle of "stress-susceptible" pigs breathing oxygen or nitrogen. *Amer. J. Physiol.* 218:102.
- Marion, W.W. 1967. Meat tenderness in the avian species. *World's Poultry Sci. J.* 23(1):6.
- Marion, W.W. and Goodman, H.M. 1967. Influence of continuous chilling on tenderness of turkey. *Food Tech.* 21(3):89.
- Marsh, B.B. 1952. Observations on rigor mortis in whale muscle. *Biochem. Biophys. Acta* 9:127.
- Minakami, S. and Yoshikawa, H. 1966. Studies on erythrocyte glycolysis. II. Free energy changes and rate limiting steps in erythrocyte glycolysis. *J. Bioch.* 59(2):139.
- Munns, W.O. and Burrell, D.E. 1966. The incidence of dark-cutting beef. *Food Tech.* 20(12):95.
- Newbold, R.P. 1966. Changes associated with rigor mortis. in Briskey, E.J., Cassens, R.G. and Trautman, J.C. (eds.) The Physiology and Biochemistry of Muscle as a Food. p. 213. Univ. of Wisconsin.
- Pearson, A.M. 1963. Objective and subjective measurements for meat tenderness. *Proc. Meat Tenderness Symposium*, Campbell Soup Company, Camden, New Jersey.
- Pfleiderer, G. 1963. Glycogen. Determination as D-glucose with hexokinase, pyruvic kinase and lactic dehydrogenase. in Bergmeyer, H.U. (ed.) Methods of Enzymatic Analysis. p. 59. Academic Press, New York. 1965.
- Pool, M.F., de Fremery, D., Campbell, A.A. and Klose, A.A. 1959. Poultry tenderness. II. Influence of processing on tenderness of chicken. *Food Tech.* 13(1):25.
- Sayre, R.N. 1970. Chicken myofibril fragmentation in relation to factors influencing tenderness. *J. Food Sci.* 35:7.
- Sayre, R.N., Briskey, E.J. and Hoekstra, W.G. 1963a. Comparison of muscle characteristics and post-mortem glycolysis in three breeds of swine. *J. Animal Sci.* 22:9012.
- Sayre, R.N., Briskey, E.J. and Hoekstra, W.G. 1963b. Porcine muscle glycogen structure and its association with other muscle properties. *Proc. Soc. Exptl. Biol. Med.* 112:164.
- Schmidt, G.R., Cassens, R.G. and Briskey, E.J. 1968. Development of an isotonic and isometric rigorometer. *J. Food Sci.* 33:239.

- Schmidt, G.R. and Briskey, E.J. 1970. Phosphocreatine and nucleotide changes in pig longissimus muscle during the development of rigor mortis under controlled environmental conditions. *J. Food Sci.* 35(5):568.
- Scholtyssek, S. and Klose, A.A. 1967. Sources of variability in turkey tenderness. *Poultry Sci.* 46:936.
- Shrimpton, D.H. 1960. Some causes of toughness in broilers (young roasting chickens). I. Packing station procedure, its influence on the chemical changes associated with rigor mortis and on the tenderness of the flesh. *Brit. Poultry Sci.* 12:101.
- Shrimpton, D.H. and Miller, W.S. 1960. Some causes of toughness in broilers (young roasting chickens). II. Effects of breed, management and sex. *Brit. Poultry Sci.* 12:111.
- Smith, Jr., M.C., Judge, M.D. and Stadelman, W.J. 1969. A "cold shortening" effect in avian muscle. *J. Food Sci.* 34(1):42.
- Snedecor, G.W. 1956. *Statistical Methods*. 5th ed. Iowa State College Press, Iowa.
- Southard, J.H. and Hultin, H.O. 1969. Glycolytic activity of chicken breast muscle mitochondria. *J. Food Sci.* 34:622.
- Swanson, M.H., Froning, G.W. and Richards, J.F. 1962. Moisture levels in frozen poultry as related to thawing losses, cooking losses and palatability. 2. Turkey fryer-roasters. *Poultry Sci.* 51(1):272.
- Sybesma, W. 1969. The significance of rigor measurement in relation to the quality of meat and to the environmental influences. *Abst. of Research Reports of the 15th European Meeting of Meat Research Workers. American Meat Science Assn.*
- Sybesma, W. and Logetestijn, J.G. Van. 1968. Experiments in improving meat quality in pigs by ante- and post-mortem injections of oxygen. *Fleischwirtschaft* 48(7):926.
- T-I Ma, R., Addis, P.B. and Allen, E. 1971. Response to electrical stimulation and post-mortem changes in turkey Pectoralis major muscle. *J. Food Sci.* 36(1):125.
- Tomlinson, N., Geiger, S.E. and Dollinger, E. 1965. Chalkiness in Halibut in relation to muscle pH and protein denaturation. *J. Fish. Res. Bd. Canada.* 22:653.

- Tomlinson, N., Geiger, S.E. and Dollinger, E. 1966. Free Drip, flesh pH and chalkiness in halibut. J. Fish. Res. Bd. Canada. 23(5):673.
- Welbourne, J.L., Harrington, R.B. and Stadelman, W.J. 1968. Relationships among shear values, sarcomere lengths and cooling procedures in turkeys. J. Food Sci. 33(5):450.
- Wells, G.H. and Dawson, L.E. 1966. Tenderness and juiciness of freeze-dried chicken meat as related to maturity of birds. Poultry Sci. 45(5):1004.
- Williamson, J.R. 1965. Glycolytic control mechanisms. I. Inhibition of glycolysis by acetate and pyruvate in the isolated perfused rat heart. J. Biol. Chem. 240(6):2308.

APPENDIX A

TABLE I
MOISTURE CONTENT AS PERCENT OF WET WEIGHT FOR
25 WEEK-OLD BIRDS (EXP. II).

Bird No.	% Moisture
10	73.7 ¹
11	75.0
12	74.3
13	73.7
14	73.7
<hr/>	
	$\bar{x}$ 74.3

¹Average of duplicate determinations.

TABLE II
COMPARISON OF METHOD OF MEASURING pH (EXP. III).

Time p.m. ¹ (min.)	pH			
	Bird 15		Bird 16	
	Probe	Slurry	Probe	Slurry
0 ¹	-	6.35	7.10	6.49
15	7.64	6.31	6.81	6.45
30	7.50	6.21	6.75	6.48
60	6.73	6.22	6.65	6.41
120	6.25	6.20	6.32	6.13
180	6.17	5.99	6.21	6.13

¹Timing was started three minutes after exsanguination.

TABLE III
THE AGE AND WEIGHT OF TURKEYS AT TIME OF SLAUGHTER FOR
EXPERIMENT I.

Bird No.	Age (Wks-Days)		Weight (lbs.)
2	15	5	14.25
3	15	6	14.00
4	15	6	14.00
5	16	0	12.50
6	16	0	14.00
ave.	15	6	13.75

TABLE IV
THE AGE AND WEIGHT OF TURKEYS AT TIME OF SLAUGHTER FOR
EXPERIMENT II.

Bird No.	Age (Wks - Days)		Weight (lbs.)
10	24	6	22.75
11	24	6	27.25
12	25	0	19.25
13	25	0	23.25
14	25	1	22.25
ave.	25	0	22.95

TABLE V
THE AGE AND WEIGHT OF TURKEYS AT TIME OF SLAUGHTER FOR
EXPERIMENT IV.

Bird No.	Age (Wks - Days)		Weight (lbs.)
20	18	3	18.75
21	18	4	19.25
22	18	6	19.75
23	19	3	18.25
24	19	4	18.25
25	19	5	18.50
	ave.	<u>19 0</u>	<u>18.79</u>
30	27	2	20.00
31	27	3	26.50
32	27	3	27.25
33	27	4	31.00
34	27	4	23.50
35	27	5	22.25
	ave.	<u>27 3</u>	<u>25.08</u>

TABLE VI

THE TIME COURSE OF pH POST-MORTEM (EXP. I)

Bird No.	Time (min.) Post-mortem ¹					
	0	15	30	60	120	180
2	6.01	6.19	6.28	6.24	6.03	6.02
3	5.92	5.78	5.85	5.90	5.94	5.94
4	6.26	5.87	5.87	5.86	5.82	5.77
5	6.05	6.05	6.05	5.97	5.97	5.97
6	6.32	6.06	6.08	6.06	5.98	5.97

¹Timing was started 10 minutes after exsanguination.

TABLE VII

THE TIME COURSE OF pH POST-MORTEM (EXP. II).

Bird No.	Time (min.) Post-mortem ¹					
	0	15	30	60	120	180
10	6.17	5.98	5.99	5.96	6.00	6.00
11	5.93	5.80	5.86	5.94	5.96	5.96
12	6.25	5.80	5.85	5.90	5.93	5.94
13	6.26	5.72	5.76	5.83	5.86	5.87
14	6.23	5.86	5.96	6.02	6.03	6.03

¹Timing was started 10 minutes after exsanguination.

TABLE VIII

CONCENTRATION¹ OF THE GLYCOLYTIC INTERMEDIATES AND CO-FACTORS IN *P. SUPERFICIALIS* MUSCLE
AT VARYING TIMES POST-MORTEM FOR 15 WEEK-OLD TURKEYS (EXP. I).

Intermediate or Co-factor	Bird No.	Time (min.) Post-mortem					
		0	15	30	60	120	180
ATP ²	2	3.1640 ⁵	3.7034	3.5236	1.9416	0.2876	0.0719
	3	1.5052	0.8601	0.4301	0.2150	0.2150	0.0359
	4	1.3938	0.6959	0.4175	0.0719	0.0360	0.0719
	5	0.7774	1.1177	0.2479	0.2526	0.0000	0.0000
	6	7.2862	6.4331	6.1038	5.4102	0.8323	0.2759
ADP	2	0.9123	0.4293	0.6171	1.0732	0.4293	0.4025
	3	1.1767	1.2836	1.2302	1.0162	0.8558	0.8291
	4	1.2877	1.2877	1.0463	1.0731	0.9390	1.0195
	5	1.1298	0.8070	0.8877	0.6725	0.6456	0.8608
	6	0.8383	0.9725	1.0162	1.1766	1.0697	0.9627
AMP	2	0.2976	0.5005	0.3111	0.2706	0.2841	0.2570
	3	0.3505	0.4045	0.3505	0.5932	0.5797	0.7415
	4	0.3517	0.3787	0.3517	0.3516	0.4057	0.4869
	5	0.2308	0.1629	0.1629	0.2037	0.3530	0.8140
	6	0.1769	0.1354	0.0539	0.1618	1.6179	1.5102
NAD	2	0.9643	1.0045	1.0045	1.0196	0.8438	0.9041
	3	1.2029	1.1468	1.0969	1.0161	1.0346	1.1468
	4	1.0045	0.8941	0.7082	0.7635	0.6580	0.7232
	5	0.9975	0.7305	0.6851	0.7305	0.7002	1.0378
	6	1.0125	0.9474	0.9624	0.9223	1.0025	0.9274
NADH	2	0.5182	0.6357	0.5989	0.4223	0.3647	0.3309
	3	0.4827	0.4838	0.3160	0.2586	0.2488	0.2496
	4	0.4475	0.2832	0.2217	0.2530	0.2249	0.3065
	5	0.9672	0.8092	0.3075	0.3350	0.2625	0.3857
	6	0.2079	0.4875	0.2410	0.4240	0.4653	0.3507

Continued.....

TABLE VIII (CONTINUED)

Intermediate or Co-factor	Bird No.	Time (min.) Post-mortem					
		0	15	30	60	120	180
CP	2	0.2227	0.5938	0.2227	0.0000	0.0742	0.0000
	3	0.0740	0.0740	0.0740	0.0370	0.0370	0.1480
	4	0.5034	0.5753	0.5753	0.0371	0.0370	0.0000
	5	0.4026	0.5772	0.2188	0.0745	0.0000	0.0000
	6	3.7638	6.8309	3.4408	0.8602	0.5735	0.2867
Total Creatine ³	2	3.9676	4.4254	5.3410	4.5780	5.0358	5.6462
	3	2.5942	3.5098	3.2046	2.5942	3.0520	3.2046
	4	3.6744	3.8275	3.3682	3.1539	3.2611	2.6793
	5	1.9200	1.8586	1.9047	1.4439	1.8739	2.1658
	6	1.5410	1.4391	1.3734	1.0682	1.0072	1.2513
P ₁	2	13.9385	13.7949	13.5298	13.2537	13.8059	13.2537
	3	16.4235	16.1474	18.0802	17.2629	16.0149	16.0149
	4	16.6996	20.4328	17.8041	17.8041	15.1976	13.8832
	5	20.0131	19.4719	18.7650	19.1958	18.2238	16.4235
	6	4.7448	13.3862	12.7014	12.6462	12.6462	29.6772
G-1-P	2	0.4854	0.3426	0.2855	0.3426	0.4568	0.6282
	3	0.2562	0.2562	0.3985	0.5408	0.5123	0.5977
	4	0.4283	0.4597	0.3712	0.4854	0.3997	0.7995
	5	0.0000	0.0573	0.0573	0.0000	0.0573	0.0000
	6	0.1199	0.3019	0.0589	0.0589	0.2078	0.1187
G-6-P	2	3.4983	3.7041	3.8584	5.3503	8.6428	10.0833
	3	2.4612	2.4869	3.0765	4.5122	5.4352	7.6400
	4	3.1896	3.5754	3.4468	5.1960	6.1734	8.8485
	5	0.6192	0.9804	1.2126	0.9030	1.0320	2.2188
	6	2.5893	2.2632	1.7990	2.4966	8.3421	11.3801

Continued.....

TABLE VIII (CONTINUED)

Intermediate or Co-factor	Bird No.	Time (min.) Post-mortem					
		0	15	30	60	120	180
F-6-P	2	0.4155	0.5713	0.3116	0.8310	1.4023	1.4542
	3	0.3624	0.3106	0.4141	0.7247	1.0094	1.4494
	4	0.6752	0.7531	0.7531	1.0387	1.1426	1.4022
	5	0.1563	0.2605	0.2084	0.2084	0.2866	0.4429
	6	0.4688	0.4154	0.2588	0.2071	1.1391	1.9667
FDP	2	0.0710	0.0710	0.0761	0.0837	0.0343	0.0101
	3	0.0189	0.0126	0.0076	0.0064	0.0064	0.0051
	4	0.0095	0.0037	0.0037	0.0113	0.0056	0.0075
	5	0.0113	0.0076	0.0019	0.0057	0.0000	0.0076
	6	0.0870	0.0939	0.1108	0.0751	0.0732	0.0057
DHAP	2	0.0602	0.0552	0.0678	0.0201	0.0151	0.0000
	3	0.0187	0.0200	0.0150	0.0175	0.0100	0.0125
	4	0.0200	0.0075	0.0113	0.0150	0.0075	0.0075
	5	0.0075	0.0075	0.0111	0.0150	0.0113	0.0150
	6	0.0375	0.0336	0.0485	0.0373	0.0224	0.0150
GA3P	2	0.0202	0.0177	0.0177	0.0104	0.0050	0.0000
	3	0.0187	0.0176	0.0151	0.0101	0.0151	0.0151
	4	0.0150	0.0075	0.0075	0.0075	0.0075	0.0150
	5	0.0264	0.0191	0.0019	0.0226	0.0376	0.0226
	6	0.0151	0.0150	0.0150	0.0113	0.0113	0.0000
3PGA	2	0.0000	0.0140	0.0169	0.0028	0.0168	0.0168
	3	0.0084	0.0111	0.0084	0.0084	0.0111	0.0056
	4	0.0169	0.0037	0.0252	0.0140	0.0196	0.0140
	5	0.0094	0.0019	0.0019	0.0019	0.0057	0.0075
	6	0.0141	0.0094	0.0038	-	0.0038	-

Continued.....

TABLE VIII (CONTINUED)

Intermediate or Co-factor	Bird No.	Time (min.) Post-mortem					
		0	15	30	60	120	180
2 PGA	2	0.0028	0.0194	0.0028	0.0055	0.0000	0.0111
	3	0.0083	0.0028	0.0028	0.0139	0.0000	0.0028
	4	0.0056	0.0074	0.0167	0.0139	0.0139	0.0111
	5	0.0075	0.0019	0.0037	0.0018	0.0056	0.0037
	6	0.0065	0.0130	-	0.0018	0.0038	-
PEP	2	0.0138	0.0055	0.0165	0.0055	0.0055	0.0055
	3	0.0137	0.0137	0.0109	0.0109	0.0082	0.0109
	4	0.0183	0.0019	0.0467	0.0193	0.0330	0.0412
	5	0.0019	0.0037	0.0074	0.0074	0.0110	0.0092
	6	0.0093	0.0128	0.0055	0.0037	0.0019	-
Pyruvate	2	0.3334	0.2077	0.0820	0.1312	0.1312	0.1530
	3	0.6752	0.6425	0.6643	0.6507	0.6530	0.6425
	4	0.5681	0.5571	0.5684	0.7487	0.6367	0.7105
	5	0.1840	0.1620	0.1656	0.1436	0.1454	0.1546
	6	0.2308	0.1060	0.0110	0.0000	0.1462	-
Lactate	2	339.1830	365.2737	281.7828	333.9648	354.8376	313.0920
	3	416.0928	299.0667	603.3346	369.2824	301.6673	421.2940
	4	294.8283	534.8655	333.9648	260.9100	657.4930	524.4291
	5	434.5288	395.2614	397.8790	251.2920	285.3212	418.8200
	6	119.3996	127.6080	106.3519	151.1865	180.3812	114.6932
α -GP	2	0.6321	0.5531	0.5794	0.4741	0.7901	0.7375
	3	0.8400	0.7875	0.8400	0.6300	0.7613	0.8400
	4	1.0009	0.8955	0.8428	0.8428	0.8955	0.9482
	5	1.8761	1.4800	1.2155	1.2369	1.1626	1.4269
	6	1.1140	1.0792	0.8933	0.6831	0.7357	0.8407

Continued.....

TABLE VIII (CONTINUED)

Intermediate or Co-factor	Bird No.	Time (min.) Post-mortem					
		0	15	30	60	120	180
Glucose	2	3.9518	3.8665	3.8490	4.4351	4.8902	5.2885
	3	3.8539	4.1373	4.3306	4.4207	4.4490	4.2223
	4	4.4067	4.8047	4.8331	4.6057	4.7763	4.7194
	5	2.0824	2.2250	2.4247	2.2820	2.4532	2.1394
	6	3.1051	2.8470	1.6559	3.4882	3.9616	3.9032
Glycogen ⁴	2	5.3063	6.0643	6.0643	6.0643	6.0643	6.0643
	3	6.0844	6.0844	6.0844	6.0844	5.3238	6.0844
	4	6.4859	6.4859	6.4859	6.1043	6.8674	6.4859
	5	5.3587	6.1242	6.1242	6.1242	6.8898	6.8898
	6	6.1441	6.4855	5.5938	6.0842	5.3237	3.0421

¹Concentrations expressed as μ Moles/g tissue.

²Abbreviations as given in text.

³Concentration expressed as mg/g tissue.

⁴Concentration expressed as μ Moles glucose/g tissue.

⁵Values are means of duplicates.

TABLE IX

CONCENTRATION¹ OF THE GLYCOLYTIC INTERMEDIATES AND CO-FACTORS IN P. SUPERFICIALIS MUSCLE
AT VARYING TIMES POST-MORTEM FOR 25 WEEK OLD TURKEYS² (EXP. II).

Intermediate or Co-factor	Bird No.	Time (min.) Post-mortem					
		0	15	30	60	120	180
ATP ²	10	6.2949 ⁵	5.0217	4.3144	3.3242	0.5658	0.1415
	11	3.5478	2.5544	2.4125	2.6963	0.5676	0.1419
	12	3.8900	2.2633	1.9804	0.5658	0.2829	0.0000
	13	4.8119	2.7584	2.1219	0.5658	0.1415	0.1415
	14	1.0632	0.5866	0.2933	0.2200	0.1833	0.2200
ADP	10	1.1453	1.0907	1.1998	1.3088	1.3361	1.0089
	11	0.9848	1.0942	1.1216	1.1763	1.0395	0.7933
	12	1.0907	1.0635	1.0907	1.0089	0.8726	0.8999
	13	0.9271	0.9816	0.9544	1.0635	0.9817	0.7908
	14	0.6565	0.7112	0.6292	0.3557	0.3557	0.3283
AMP	10	0.1650	0.1925	0.1925	0.2200	1.1548	1.5672
	11	0.1655	0.1655	0.1655	0.2206	1.6548	1.4342
	12	0.3299	0.2613	0.3712	0.4537	0.5912	0.5637
	13	0.3024	0.2887	0.3299	0.3299	0.5362	0.7699
	14	0.3585	0.2758	0.1655	0.1655	0.1655	0.1655
NAD	10	0.7093	0.7552	0.7144	0.6327	0.6123	0.6021
	11	0.6554	0.6861	0.6963	0.6554	0.6963	0.6963
	12	0.7807	0.6531	0.6735	0.6531	0.6531	0.6939
	13	0.6990	0.7144	0.6531	0.5715	0.6123	0.7246
	14	0.7476	0.6605	0.4195	0.3175	0.3175	0.3943
NADH	10	0.3491	0.5296	0.3547	0.4267	0.1705	0.3515
	11	0.1189	0.0620	0.1396	0.2217	0.0000	0.1364
	12	0.3204	0.3915	0.3282	0.3026	0.5494	0.3192
	13	0.3189	0.3875	0.3461	0.2495	0.2000	0.2608
	14	0.2056	0.2426	0.2065	0.1546	0.5530	0.6684

Continued.....

TABLE IX (CONTINUED)

Intermediate or Co-factor	Bird No.	Time (min.) Post-mortem					
		0	15	30	60	120	180
CP	10	1.9733	1.1694	0.4385	0.1462	0.1462	0.0000
	11	0.8798	0.7332	0.4399	0.4399	0.1466	0.1466
	12	0.2923	0.1462	0.0000	0.0000	0.0000	0.0000
	13	0.7780	0.4385	0.2923	0.1462	0.1462	0.1462
	14	0.3027	0.3027	0.1514	0.0757	0.0757	0.0757
Total Creatine ³	10	6.2240	6.0684	6.5352	6.3796	6.2240	6.6840
	11	6.0879	6.2440	6.0779	6.0879	6.2240	5.9318
	12	4.5124	5.6016	4.8236	4.9792	4.8236	5.4460
	13	5.4460	5.2604	5.7572	5.2904	4.6680	5.6016
	14	4.8391	3.4342	4.2708	4.0586	3.1220	4.0586
P ₁	10	13.8059	14.4244	17.1856	18.6325	16.2910	13.8059
	11	17.6384	15.8771	19.2731	16.0149	15.1755	14.1483
	12	18.0802	18.4889	19.4608	19.4608	19.1847	19.3946
	13	12.9776	17.9367	17.6605	19.3946	21.5372	17.1193
	14	21.5372	24.7071	25.5354	22.9178	24.7071	24.8396
G-1-P	10	0.2322	0.1742	0.1161	0.0000	0.1161	0.1161
	11	0.3494	0.1165	0.2329	0.3494	0.4658	0.3494
	12	0.8125	0.3482	0.2322	0.2322	0.2322	0.3482
	13	0.1741	0.3772	0.4643	0.4643	0.5804	0.6094
	14	0.1165	0.1165	0.1747	0.1165	0.0582	0.0582
G-6-P	10	5.4899	5.0194	4.1305	2.8757	7.4245	10.4570
	11	3.1472	2.8324	2.7800	3.6192	10.2283	10.9101
	12	2.9280	2.0914	2.8300	3.3463	5.0194	5.4376
	13	3.0325	2.7973	3.0848	3.9998	5.4115	8.6270
	14	1.5213	0.9442	1.6784	1.5735	1.2588	1.3113

Continued.....

TABLE IX (CONTINUED)

Intermediate or Co-factor	Bird No.	Time (min.) Post-mortem					
		0	15	30	60	120	180
F-6-P	10	0.6334	0.6334	0.6334	0.4223	0.8466	1.6891
	11	0.4236	0.3177	0.2118	0.3117	1.4827	1.5886
	12	0.2111	0.2111	0.2111	0.2111	0.5279	0.5279
	13	0.3431	0.2639	0.3959	0.4751	0.8182	1.0026
	14	0.2118	0.1059	0.1589	0.2118	0.1059	0.1589
FDP	10	0.0052	0.0052	0.0077	0.0052	0.0052	0.0052
	11	0.0879	0.0336	0.0297	0.0155	0.0103	0.0052
	12	0.1095	0.0180	0.0103	0.0052	0.0077	0.0052
	13	0.0554	0.0142	0.0103	0.0077	0.0052	0.0000
	14	0.0155	0.0078	0.0078	0.0052	0.0078	0.0078
DHAP	10	0.0153	0.0153	0.0102	0.0153	0.0153	0.0102
	11	0.0154	0.0102	0.0102	0.0180	0.0154	0.0102
	12	0.0306	0.0153	0.0153	0.0153	0.0102	0.0102
	13	0.0537	0.0306	0.0153	0.0102	0.0102	0.0102
	14	0.0102	0.0128	0.0102	0.0154	0.0154	0.0154
GA3P	10	0.0154	0.0154	0.0205	0.0180	0.0154	0.0103
	11	0.0154	0.0129	0.0103	0.0154	0.0103	0.0051
	12	0.0103	0.0103	0.0154	0.0103	0.0154	0.0103
	13	0.0154	0.0103	0.0154	0.0103	0.0103	0.0051
	14	0.0258	0.0155	0.0129	0.0155	0.0154	0.0154
3PGA	10	0.0114	0.0171	0.0057	0.0114	0.0114	0.0171
	11	0.0115	0.0057	0.0115	0.0115	0.0115	0.0057
	12	0.0057	0.0057	0.0114	0.0114	0.0171	0.0171
	13	0.0171	0.0114	0.0171	0.0114	0.0086	0.0057
	14	0.0172	0.0115	0.0172	0.0172	0.0172	0.0201

Continued.....

TABLE IX (CONTINUED)

Intermediate or Co-factor	Bird No.	Time (min.) Post-mortem					
		0	15	30	60	120	180
2PGA	10	0.0142	0.0113	0.0113	0.0113	0.0113	0.0170
	11	0.0114	0.0114	0.0114	0.0057	0.0057	0.0057
	12	0.0057	0.0113	0.0057	0.0057	0.0113	0.0113
	13	0.0113	0.0113	0.0057	0.0057	0.0057	0.0057
	14	0.0170	0.0114	0.0227	0.0170	0.0170	0.0170
PEP	10	0.0281	0.0253	0.0112	0.0112	0.0168	0.0168
	11	0.0113	0.0113	0.0113	0.0113	0.0113	0.0113
	12	0.0168	0.0224	0.0224	0.0112	0.0168	0.0168
	13	0.0337	0.0224	0.0112	0.0112	0.0168	0.0168
	14	0.0225	0.0225	0.0225	0.0253	0.0169	0.0187
Pyruvate	10	0.6814	0.3574	0.2178	0.1676	0.2039	0.1229
	11	0.6496	0.4564	0.2100	0.1792	0.2800	0.2072
	12	0.7819	0.6702	0.4329	0.4636	0.4468	0.3742
	13	0.4245	0.3910	0.3016	0.2932	0.3240	0.3184
	14	0.7476	0.4704	0.3584	0.1960	0.1232	0.1848
Lactate	10	296.9910	365.9353	365.9353	326.1597	323.5080	360.6319
	11	191.5362	202.1771	202.1771	170.2544	180.8953	202.1771
	12	429.5762	445.3362	486.7628	424.6229	403.9096	471.2278
	13	434.8797	429.5762	307.5978	296.9910	418.9694	493.2172
	14	489.4414	351.1497	329.8679	319.2270	313.9066	319.2270
α -GP	10	0.8166	0.6124	0.6890	0.6124	0.5104	0.5614
	11	0.4197	0.5371	0.3223	0.2954	0.2148	0.3223
	12	0.8166	0.6124	0.4083	0.4083	0.4083	0.4849
	13	0.7145	0.6635	0.6124	0.6124	0.6635	0.7656
	14	1.1259	1.0239	0.8191	0.7167	0.7423	0.8447

Continued.....

TABLE IX (CONTINUED)

Intermediate or Co-factor	Bird No.	Time (min.) Post-mortem					
		0	15	30	60	120	180
Glucose	10	3.6984	3.1205	3.6984	3.2939	4.8542	4.5652
	11	3.8842	4.0001	4.2320	4.1161	5.2176	5.3915
	12	5.0853	4.6230	4.5074	4.6230	4.9121	5.1432
	13	3.4960	3.6405	4.3339	4.0450	4.0739	4.3918
	14	4.6381	4.6670	4.8990	4.5511	4.5221	4.8120
Glycogen ⁴	10	7.7550	7.7550	8.5305	7.7550	6.2040	6.2040
	11	8.1690	7.7800	7.7800	8.5579	8.5579	7.3910
	12	8.5305	7.7550	7.7550	8.5305	8.5305	7.7550
	13	2.3265	3.1020	3.1020	2.3265	3.1020	3.1020
	14	3.8900	3.1120	3.1120	3.8900	3.5010	3.1120

¹Concentrations expressed as μ Moles/g tissue.

²Abbreviations as given in text.

³Concentration expressed as mg/g tissue.

⁴Concentration expressed as μ Moles glucose/g tissue.

⁵Values are means of duplicates.

TABLE X

pH OF P. SUPERFICIALIS MUSCLE AT VARYING TIMES POST-MORTEM
FOR 19 AND 27 WEEK-OLD TURKEYS

(EXP. IV).

Bird No.	Age (wks)	Time (min.) Post-mortem ¹					
		0	15	30	60	120	180
20	19	6.18	6.11	6.11	6.10	6.07	6.05
21	19	6.31	5.76	5.73	5.77	5.80	5.80
22	19	6.08	5.75	5.75	5.79	5.84	5.85
23	19	6.43	6.23	6.30	6.31	6.34	6.33
24	19	6.31	5.75	5.76	5.86	5.98	6.02
25	19	6.26	5.75	5.75	5.78	5.83	5.85
30	27	6.35	5.88	5.81	5.81	5.83	5.85
31	27	6.79	6.30	6.23	6.21	6.13	6.07
32	27	6.49	5.85	5.85	5.85	5.84	5.84
33	27	5.94	5.85	5.85	5.86	5.89	5.91
34	27	6.76	6.33	6.25	6.16	6.09	6.06
35	27	6.29	6.02	5.95	5.92	5.95	5.96

¹Timing was started 15 minutes after exsanguination.

TABLE XI

CALCULATED AND SELECTED TABLED t VALUES FOR CERTAIN
PARAMETERS FOR EXPERIMENT I AND II.

Parameter	t calc.	t (0.05)	t (0.01)
<u>15 wk. vs. 25 wk. NAD</u>			
0 min.	1.276	2.306	3.355
15 min.	3.586	2.306	3.355
30 min.	2.642	2.306	3.355
60 min.	3.674	2.306	3.355
120 min.	2.671	2.306	3.355
180 min.	3.607	2.306	3.355
<u>15 wk. vs. 25 wk. Total Creatine</u>			
0 min.	4.625	2.306	3.355
15 min.	3.027	2.306	3.355
30 min.	3.189	2.306	3.355
60 min.	3.717	2.306	3.355
120 min.	2.418	2.306	3.355
180 min.	2.999	2.306	3.355
<u>Group I vs. Group II</u>			
Initial ATP	3.060	2.306	3.355
%-ATP	7.499	2.306	3.355
pH at 15 min.	1.839	2.306	3.355
Lactic acid at 30 min.	2.332	2.306	3.355

APPENDIX B

Figure 1. The pH of *P. superficialis* muscle at varying times post-mortem for 15 week-old turkeys. (O—O Bird 2; Δ — Δ Bird 3; $\square$ — $\square$ Bird 4; O---O Bird 5; Δ --- Δ Bird 6).

Figure 2. The pH of *P. superficialis* muscle at varying times post-mortem for 25 week-old turkeys. (O—O Bird 10; Δ — Δ Bird 11; $\square$ — $\square$ Bird 12; O---O Bird 13; Δ --- Δ Bird 14).

Figure 3. Glycolytic cycle showing intermediates, co-factors and enzymes.