

THE INFLUENCE OF PRE- AND POST-MORTEM TREATMENTS  
ON GLYCOGEN CONTENT AND TENDERNESS OF POULTRY MUSCLES

by

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## INTRODUCTION

Consumers want and expect the meat and poultry they purchase to be tender. Consequently, much research has been done on meat tenderness by university, governmental, and industrial laboratories. The subject of meat tenderness is of much economic importance and is of scientific interest to food scientists, biochemists, and biophysicists because of the many physical and biochemical changes that occur during the transition of muscle to meat.

Poultry meat is extremely tough when cooked within 0 to 4 hours after slaughter. This lack of tenderness in broilers is due to rigor, a condition of muscles during the early post-mortem state in which the muscles become quite rigid and stiff. Resolution of rigor aids in tenderizing the muscles, but it ordinarily takes poultry meat 12 to 24 hours post-mortem to reach maximum tenderness (de Fremery, 1963). Rigor presents a problem to the poultry processors because, under commercial processing practices, it is incompatible with the assembly line method of processing and the volume of broilers being processed each day to hold dressed birds for this length of time before freezing or further processing. However, if the chilling or holding time is not long enough to permit resolution of rigor, consumer complaints about toughness increase markedly (White et al., 1964; Palmer et al., 1965).

Considerable variations are noted in poultry in the degree of tenderness before, during and post-rigor (Scholtyssek and Klose, 1967). The reasons for these variations are not understood. Also, the causes of the onset and resolution of rigor have not been determined and should be further investigated so that broilers could be treated in such a manner that rigor could be minimized or resolved in a shorter period of time.

This experiment was designed to study the effects of pre- and post-mortem glycolysis on the onset and resolution of rigor in the Pectoralis major muscle of broilers. In addition, the effect of stretch-tension during rigor on muscle tenderness was also studied.

## REVIEW OF LITERATURE

### Factors Affecting Tenderness of Poultry Meat

Many different factors have been suggested in the literature as possible causes of tenderness, contributors to tenderness, or lack of tenderness in poultry meat. Production factors such as age, breed, sex, feeding practices, and interactions among these factors can significantly affect poultry meat tenderness. Poultry processing factors such as slaughter methods, degree of struggling, scalding, picking, aging, and temperature of carcasses during aging and storage have also been found to be significant in determining the degree of tenderness of poultry meat.

Age, breed, sex and feeding practices. Dodge and Stadelman (1959) reported on factors which influence tenderness of poultry meat comparing chickens and turkeys of similar ages. They found that the age of birds and the class of poultry influenced tenderness. The differences between chickens and turkeys seemed to be more closely related to the actual pattern of tenderness development (time required for rigor onset and resolution) rather than ultimate tenderness reached. Turkey meat required a longer time for aging than chicken meat.

According to Stadelman (1963), age definitely influences tenderness. However, this effect cannot be demonstrated by comparing birds varying in age by only a few weeks but by comparing immature birds with mature birds. Breed and sex have only very

slight influence on tenderness scores. In studies reporting significant breed or sex differences, these differences can usually be associated with degree of finish. Stadelman et al. (1966) noted a strain X sex interaction when studying the tenderness of turkey meat.

Shrimpton and Miller (1960), as reported by Stadelman (1963), observed that when birds were kept on full feed the meat was more tender than when the birds were on a restricted diet. They found that the preferred groups, which were those essentially on full feed and of meat strain chickens, exhibited no sex-related tenderness differences. However, in birds on restricted diet or from egg-type chickens, females were preferred to males with respect to tenderness of breast meat.

Studies on determining the influence of feeding practices on tenderness have led to the speculation that feed influences the deposition of minute quantities of interstitial fat in breast muscles. This fat is believed to be a major factor contributing to the degree of tenderness in white meat. Another possible explanation for tenderness is the effect of the environment on uniformity of growth rate which affects the amount of connective tissue deposited (Stadelman, 1963). In summary, the young fast-growing bird which is raised in confinement and a very limited space is more likely to produce a tender carcass.

Slaughter method and degree of struggling. Dodge and Stadelman (1960b) concluded from their experiments on struggling and tenderness that under normal processing conditions struggling did not exert any effect on post-mortem tenderization. The addition of the tranquilizer, Tyzine, in the feed did not appear to affect either the level of tenderness and struggling or the variation in these factors.

Stadelman and Wise (1961) injected chickens with sufficient Nembutal (pentobarbital sodium) to completely anesthetize the birds before slaughter. Meat from anesthetized birds was not as tough as that from controls when determined by shear values. However, the use of Nembutal as an anesthetizer significantly extended the period of maximum toughness of cooked breast muscles.

Goodwin et al. (1961b) subjected turkeys to six slaughter treatments (Nembutal, debraining, electric knife, CO<sub>2</sub>, tranquilizer, control) to determine their effect upon meat tenderness as determined on a Kramer Shear Press. The control birds received no special ante-mortem treatment prior to the severing of the throat. Method of slaughter had no significant effect on shear values of the breast muscles. Humane slaughter treatments (all treatments except the control) resulted in an increased shear value for the thigh muscles.

Scalding and picking procedures. Studies by a number of researchers have shown that the scalding procedure can have a significant effect on the tenderness of poultry meat. Increasing the severity of the scalding procedure, either by increasing the time

of immersion or by increasing the scald temperature, will increase the toughness of cooked poultry meat (Klose et al., 1956; Shannon et al., 1957; Pool et al., 1959; Klose et al., 1959; Wise and Stadelman, 1959; Wise and Stadelman, 1961). Scald times and temperatures used in the above studies ranged from 5 seconds to 12 minutes and 48.9 to 90.6°C (120 to 195°F). Scald time and temperature ranges used in commercial operations, as surveyed by Mountney (1966), were 30 to 120 seconds and 50.6 to 82.2°C (123 to 180°F), depending on the scald method desired.

Wise and Stadelman (1961) found significant variations in tenderness at different depths of muscle tissues due to the combined effects of scald water, temperature and time. The exterior 6 mm of the muscle tissue scalded at 54.4°C (130°F) for 12.0 minutes was significantly tougher than the interior 6-15 mm of tissue, probably due to the fact that the partially cooked exterior 6 mm of muscle (cooked meat has a lower thermal conductivity than raw meat) tended to decrease the rate of heat penetration to the deeper tissue. Bendall (1951) showed a positive relationship between pre-rigor temperature and the degree of irreversible muscle shortening during rigor. Therefore, permanent toughness produced by excessive scalding may be related to the degree of muscle shortening during rigor onset. Wise and Stadelman (1961) also observed that the presence of skin significantly reduced the scald-toughening effect on the muscles.

Klose et al. (1956) reported that toughness induced by excessive picking action cannot be resolved completely by prolonged aging. The effects of beating were cumulative and were reduced by limiting the beating action to that amount barely essential for complete feather removal.

In a similar study, Pool et al. (1959) found that the ultimate toughness after aging increased with the extent of beating action incurred by the carcass during feather removal. Beating exerted its greatest toughening effect when applied immediately after slaughter. Beating delayed for 1 to 3 hours after slaughter had less effect on muscle toughness.

Klose et al. (1959) compared the tenderness of machine-picked versus hand-picked carcasses. Machine-picked birds resulted in cooked meat about twice as tough as hand-picked controls. The toughening effects of individual picking machines on a commercial line were accumulative. Differences in shear values between machine-picked and hand-picked birds were still detected even after extended chilling periods.

Cutting pre-rigor muscle. Koonz et al. (1954), by cutting or excising poultry muscles before the onset of rigor, were able to induce a toughness in the muscles which could be only partially resolved by aging. Pool et al. (1959) also reported that cutting up the carcass in the early post-mortem period had a small toughening effect. de Fremery and Pool (1960) determined the 24-hour post-mortem

shear values of Pectoralis major muscles which had been excised immediately after the birds were eviscerated and compared them to shear values from control muscles which had not been excised during aging. The mean shear value of the tougher cut muscles was 12.6 compared to 5.8 lbs. for the controls.

Influence of contraction. Klose et al. (1970) studied effects on tenderness caused by contraction of excised chicken muscles induced by immediate post-mortem treatments consisting of electrical stimulation, beating, freeze-thawing and heating (immersion of the muscle in water at 82°C (179.6°F) until 10 min. after the muscle reached an internal temperature of 80°C (176°F)). These treatments were followed by cooking in the pre-rigor state. One member of each pair of muscles was held in restraint while exposed to the same conditions. In terms of percentage of original rest length, electrical stimulation reduced muscle length to 59%, and when followed by cooking to 44%; freeze-thawing reduced the length to 42%, and when followed by cooking to 40%; beating to 96%, and when followed by cooking to 52%; cooking alone to 48-53%. With the exception of the beating-heating combination, all contraction-inducing treatments resulted in a reduction of the shear values of cooked muscle to about one-half those of uncontracted controls. These researchers speculated that in the extreme state of contraction developed in the stimulated non-restrained muscles, the actin filaments slid into the H zone of the sarcomere and changed the

post-cooking adhesiveness between the adjacent filaments and the other structural components of the sarcomeres to produce myofibrils more susceptible to shearing stress.

Aging and temperature during aging and storage. Stewart et al. (1941) reported that chickens which were placed in the oven within 4-1/2 hours after killing almost always went into rigor in the oven and many were still in rigor when served to a taste panel. None of the birds (New York dressed poultry) held for 8 hours or more at 1.7°C (35°F) showed the stiffening characteristics of rigor in the oven or when served. Rigor passed more quickly in the breast than in the thigh muscles. They reported that tenderness in poultry meat increased rapidly with the passing of rigor and that after 24 hours at 1.7°C (35°F) the meat changed little in tenderness.

Klose et al. (1956) reported that chicken fryers and turkey fryer-roasters fried from the frozen state required about 12 hours holding (chilling) above freezing for optimum tenderization. Prolonged periods in frozen storage had no appreciable tenderizing effect upon inadequately chilled birds. Klose et al. (1959) found that holding inadequately aged, frozen turkey fryers for as long as 9 months at -17.8°C (0°F) had no tenderizing effect, but holding at -3.9 to -2.8°C (25 to 27°F) for 1 to 2 weeks produced appreciable tenderization. Holding fryers in a thawed state at 1.7°C (35°F) after frozen storage had essentially as much tenderizing effect as an equal period of chilling before freezing. An elevation of the

holding temperature of freshly processed turkeys prior to freezing to as much as  $37.8^{\circ}\text{C}$  ( $100^{\circ}\text{F}$ ) for 4 hours did not produce adequate tenderization.

According to Dodge and Stadelman (1959), the time of aging, the temperature of aging, and the media in which poultry carcasses are aged were all important factors in post-mortem tenderization. Fourteen-week-old chicken carcasses aged in water at  $12.8^{\circ}\text{C}$  ( $55.0^{\circ}\text{F}$ ) (2,5,8 hours) were significantly ( $P < .01$ ) more tender than those aged in air at  $12.8^{\circ}\text{C}$  ( $55.0^{\circ}\text{F}$ ) and 90% humidity for 2, 5 and 8 hours.

Pool et al. (1959) made determinations of toughness of chickens as a function of temperature and time of aging. Their results showed that most tenderization occurred within 4 hours at chill temperatures, and very little took place after 12 hours. No appreciable tenderization occurred at  $-17.8^{\circ}\text{C}$  ( $0^{\circ}\text{F}$ ) over a 4-month period following the passing of rigor, but significant tenderization took place in frozen carcasses held at  $-3.9$  to  $-2.8^{\circ}\text{C}$  ( $25$  to  $27^{\circ}\text{F}$ ) for several days. Tenderization arrested by freezing proceeded at about a normal rate on thawing.

Goodwin et al. (1961a) conducted studies to determine the effect of chilling methods and aging on tenderness of 14-week-old turkeys. The methods of chilling were: 1) tap water at  $13.3^{\circ}\text{C}$  ( $56^{\circ}\text{F}$ ), 2) slush ice water with no agitation, 3) slush ice water with agitation supplied by a submersible pump, and 4) a revolving wire basket suspended and revolved in a slush ice bath. Their

results indicated that rapid cooling was not a solution for a more rapid method of tenderization of turkey meat. The flexing of the wings and legs by tumbling during chilling resulted in a retarding of the development of maximum tenderness, but had no significant effect when the turkeys were aged the maximum time of 32 hours. The turkeys subjected to the rotary chill method of chilling had a greater percentage of water uptake and retained more of the water after 32 hours than did the turkeys of the other treatments. Yield of cooked meat was not appreciably affected by chilling procedure. When calculated on the original eviscerated weight, the carcasses which had gained considerable weight during chilling had considerably higher shrinkage during cooking.

Klose et al. (1961) found that holding one-hour ice chilled turkey carcasses for 3 days at  $-2.8^{\circ}\text{C}$  ( $27^{\circ}\text{F}$ ) provided adequate tenderization, and no adverse flavor changes were noted after 14 days at  $-2.8^{\circ}\text{C}$  ( $27^{\circ}\text{F}$ ). They suggested that holding frozen turkeys for a short period in the intermediate thawing range offered a promise of providing tenderness in birds, which, for reasons of economics or convenience, could neither be chilled long enough or held long enough in the thawed condition to provide desired degrees of tenderness.

May et al. (1962) determined the effect of aging in water at 0, 19 and  $37^{\circ}\text{C}$  (32, 66.2 and  $98.6^{\circ}\text{F}$ ) on the tenderness pattern of 10-week-old and 72-week-old chickens. The older birds were less tender, both

initially and throughout aging. Temperature affected the tenderness of 10-week-old birds; 0 and 19°C (32 and 66.2°F) aging water tended to give a more tender product. The older birds showed a similar trend during the first 4 hours of post-mortem aging, but the tenderness of birds from all three temperatures approached the same level after 8 hours of aging. Both ages of birds began to tenderize within 30 minutes at all three temperatures.

Goodwin and Stadelman (1962) reported on the effects of pre-cooling before processing (evisceration) and hand massaging of turkeys during chilling on tenderness of turkey meat. Pre-cooling of turkeys for 3, 5, 6 or 9 minutes prior to processing did not eliminate the surface toughening phenomenon, nor did it reduce the time required to achieve maximum tenderness. Pre-cooling for 25 minutes appeared to reduce the severity of toughening but did not reduce the time to achieve tender meat. Two hours of muscle flexing and massaging increased the shear values (increased toughness) of the turkey meat.

van den Berg et al. (1964) compared the extent of post-rigor tenderization in breast and leg meat of chickens and turkeys stored at 0°C (32°F). Results showed that the leg meat tenderized in two phases rather than in one phase, as did breast meat. Tenderness of breast meat increased markedly during the first 1 to 2 days of storage, but changed little during the next 5 to 6 days. For leg meat, the initial period of tenderization was comparable to that for

breast meat, but a second period of tenderization occurred 2 to 5 days later. The rate of tenderization during this second period was slower for older birds.

Welbourn et al. (1968) studied the relationships among shear values, sarcomere lengths and cooling procedures in turkeys. Their results suggest that turkey processors could possibly increase tenderness of their products by gradually chilling the birds to 0°C (32°F) rather than placing them directly in an ice bath of about 2°C (35.6°F). They concluded that the increase in shear values with decrease in temperature was probably not due to the cold shortening phenomenon because sarcomere measurements showed only a slight amount of shortening with decreasing temperature. This shortening was less for the thigh muscle which had the significant increase in shear values. These researchers suggested that the increase in shear values might be due to the temperature effect on some muscle component such as connective tissue, which was higher in the thigh muscle than in the breast, rather than on muscle shortening.

Smith et al. (1969) conducted experiments which demonstrated that "cold shortening" occurred in chicken breast muscle similar to the "cold shortening" in beef and lamb muscles as reported by Marsh and Leet (1966). Smith et al. (1969) determined the effect of post-mortem temperatures between 0 and 20°C (32 and 68°F) on the degree of shortening in isolated Pectoralis major muscles of chickens and turkeys. The degree of muscle shortening at each temperature after

various post-mortem periods indicated that shortening was essentially complete after 3 hours in chickens and 5 hours in turkeys. Shortening in muscles stored at 0°C (32°F) was significantly greater ( $P < .01$ ) than in the 12 to 18°C (53.6 to 64.4°F) temperature range. Shortening was greatest in muscles stored at 20°C (68°F). The degree of gross shortening observed was directly related to the average sarcomere length of isolated myofibrils. Post-mortem decline in pH was not significantly correlated with shortening. Extractability of myofibrillar and sarcoplasmic proteins after 5 hours at either 0 or 16°C (32 or 60.8°F) was found to be unrelated to the degree of post-mortem shortening. The results of Jungk and Marion (1970) are in conflict with those of Smith et al. (1969). The data of Jungk and Marion (1970) did not show a cold shortening effect in turkey breast muscle, but rather a significant linear relation between extent of shortening and temperature.

#### Biochemistry of Pre- and Post-mortem Muscle

Essential to the understanding of meat is the nature of the phenomenon of rigor and the recognition that tender meat is the result of post-mortem action of the complex biological tissue of muscle. The physiology and biochemistry of pre- and post-mortem muscles are complex areas in which a tremendous amount of research is being conducted. Much additional knowledge has been developed concerning muscles and their behavior and the qualities of meat during the past two decades. Scientists who study the basic and

applied aspects of meat have found that the common ground between life and death, the post-mortem but pre-rigor phase, is as vital to the one area of investigation as to the other (Marsh, 1970).

Muscle function in vivo. A knowledge of muscle function in vivo is necessary before one can understand the changes which occur in post-mortem muscle. Lawrie (1966) presented a brief review of the composition of muscle tissue in the living state. The muscle fiber is composed of smaller units called myofibrils which are cross-striated and composed of numerous parallel filaments. The dark or A (anisotropic) band has a central clear area (the H zone) and the light or I (isotropic) band has a central dark division (the Z line). The distance between two adjacent Z lines is the functional unit of the myofibril, known as the sarcomere. Thick filaments composed of the contractile protein myosin traverse the A band. Thin filaments of the contractile protein actin are continuous through the Z line, but do not traverse the H zone. There are six straight rows of projections or "feet" running longitudinally along the side of each myosin filament, the sets of feet being distributed symmetrically around the periphery of the filament, so that one set of feet is opposite one of the six filaments of actin which surround each myosin filament. Bendall (1966) reported that the actin filaments consist of two helically wound strands composed of globular G-actin monomers.

A brief explanation of the probable sequence of events in contraction was outlined by Lawrie (1966). In muscles at rest in the living animal or in the pre-rigor state following death, the actin filaments are prevented from combining with the myosin "feet" by the magnesium complex of adenosine triphosphate (MgATP) which acts as a plasticizer. When a stimulus arrives from the nervous system at the muscle fiber it causes a depolarization of the sarcolemma. This causes changes in the sarcoplasmic reticulum at the Z lines, releasing  $Ca^{++}$  ions. The  $Ca^{++}$  ions then release ATP from its inert complex with magnesium and also stimulate myosin ATP-ase. This enzyme splits ATP to ADP, yielding the energy necessary for the actin filaments to pull or be pulled into the H zone where they pass between the myosin filaments with which they then unite at the projections to form actomyosin. Following contraction, the Marsh-Bendall factor operates to cause relaxation. Organized as an aspect of the sarcoplasmic reticulum, the relaxation factor pumps  $Ca^{++}$  ions out of the system thus inhibiting ATP-ase. New ATP rushes into the system (reforming the MgATP complex) and breaks up the actomyosin, thus re-establishing the relaxed state. The most immediate source of ATP in the muscle is resynthesis from ADP and creatine phosphate, by the enzyme creatine kinase present in the sarcoplasm. The sarcoplasm also contains a soluble ATP-ase which operates very slowly and is responsible for the small degree of contractility involved in muscle tone. But in vivo the major

source of ATP is its resynthesis from ADP by respiration, in which muscle glycogen is oxidized to carbon dioxide and water. When energy is needed in excess of the power of the respiratory system to restore ATP, anaerobic glycolysis can regenerate ATP, although much less efficiently. Much more ATP can be resynthesized by respiration than by anaerobic glycolysis.

Muscles of mammals can be classified broadly as "red" or "white". In most mammals, the majority of muscles appear to be of mixed fiber types. These two muscle types differ both histologically and biochemically (Beatty and Bocek, 1970). Investigation of the two muscle types in vitro and in vivo have demonstrated differences in carbohydrate, protein and lipid biochemistry. Red or predominantly red muscles are adapted for sustained activity and prolonged energy production and contain greater quantities of mitochondria, respiratory enzymes and myoglobin. White or predominantly white muscles are adapted for sudden bursts of activity with frequent periods of rest; respiratory enzymes and myoglobin are present in relatively small amounts, but lactic dehydrogenase activity is high.

ATP concentration. The chemical and physical reactions which occur in post-mortem muscle have been reviewed by Bate-Smith (1948). The chemical event most closely associated with the onset of rigor is the disappearance of adenosine triphosphate (ATP). The living muscle cell maintains a high level of ATP by oxidation of organic compounds via the glycolytic and Embden-Meyerhof pathways. When an

animal dies and oxygen is no longer available to the cells, ATP and creatine phosphate can be maintained only by anaerobic glycolysis. After the muscle glycogen has been depleted, the ATP concentration decreases rapidly and the muscle passes into rigor, a state characterized by a sharp increase in the modulus of elasticity of the muscle and the development of a rigid, nonplastic texture.

Bendall (1951), in a study of rigor in rabbit muscles, showed that during the course of rigor in rested muscles at 37 and 17°C (98.6 and 62.6°F), creatine phosphate was the first chemical compound to be broken down. The ATP started to break down when 70% or more of the creatine phosphate had disappeared. The ATP concentration then decreased relatively quickly, regardless of how vigorously glycolysis proceeded. This researcher suggested that muscle shortening in rigor can best be explained as a very slow irreversible contraction, and that disappearance of ATP from the muscle is a fundamental prerequisite for both shortening in rigor as well as physiological contraction. He demonstrated the disappearance of creatine phosphate before there were any changes in muscle texture and concluded that this compound played no part in rigor other than as a reserve of phosphate-bond energy.

de Fremery and Pool (1960) reported on changes in ATP concentration and the modulus of elasticity in chicken muscle. The modulus of elasticity was generally about  $1 \text{ to } 2 \times 10^3 \text{ g/cm}^2$  in the pre-rigor state. This value increased 5 to 10 times when rigor was fully

established. In general, the muscle began to lose its extensibility when about two-thirds of the ATP had disappeared. The decrease in ATP and increase in the modulus of elasticity were much more rapid when the muscles were held at 43°C (109.4°F) than at 14°C (57.2°F). The loss of extensibility continued for as much as 8 hours in some cases. The loss of extensibility that occurred during the onset of rigor was not resolved during the resolution of rigor.

de Fremery and Pool (1960) also investigated the chemical changes accompanying the toughening effect of mechanical feather pickers. Two groups of eleven-week-old cockerels were slaughtered and subjected to identical conditions with the exception that one group was hand-picked and the other group had the feathers removed by a drum-type mechanical picker. At 2.5 hours post-mortem, the average ATP-phosphorus content of muscle from the beaten birds was 5.9% of the total TCA (trichloroacetic acid)-soluble phosphorus while that of the hand-picked group was 23.8%.

Study of the effect of mechanical beating on post-mortem muscles was extended to an experiment involving paired Pectoralis major muscles from the same bird (de Fremery and Pool, 1960). Both muscles were removed immediately after the chicken was killed, and one muscle was struck gently with a wooden mallet for two minutes. Periodic determinations of ATP and pH showed that both the pH and ATP dropped more rapidly in the muscle that had been beaten. The time required for the ATP concentration to fall to 50% of its initial

value (about 4.8 mg/g) was reduced from 3.2 to 2.3 hours. Twenty-four-hour post-mortem shear values of the beaten muscles averaged 12.6 lbs. compared to 4.7 lbs. for the control muscles.

de Fremery and Pool (1960) determined the rates of ATP disappearance in chicken Pectoralis major muscles held at different temperatures during the early post-mortem period. Generally, the higher the temperature, the faster was the rate of ATP disappearance. However, it was observed that ATP disappeared more rapidly at 0°C (32°F) than at 10°C (50°F). Shear force values when compared with relative rate of ATP disappearance were strongly indicative of a correlation between rate of ATP disappearance and muscle toughness.

Other factors, including electrical stimulation, electron irradiation, and freezing and thawing of pre-rigor muscles also resulted in a more rapid loss of ATP, more rapid development of rigor, and increased muscle toughness (de Fremery and Pool, 1960).

It is becoming increasingly obvious that it is those factors which affect the rate and extent of the changes brought about by the disappearance of ATP that confer on meat its tenderness or unacceptable toughness. Commercial handling practices after slaughter can influence the subsequent quality of meat, but they can only do this within limits set by the physiological and biochemical characteristics of an animal before and at the time of slaughter (Lister, 1970).

Glycolysis and pH. Marsh (1954), reporting on rigor in beef Longissimus dorsi muscle, stated that the onset of rigor coincided with the rapid phase of ATP decomposition, that the ATP decomposition was directly related to pH when the ultimate pH was low, and that the onset of rigor could be followed approximately by pH determinations. It was found that a period of 36 hours post-mortem would allow all glycolytic changes to reach completion. This researcher suggested that a 36-hour interval between killing and the commencement of freezing of beef would prevent problems associated with thaw-rigor, due to freezing before the completion of the onset of rigor.

Mellor et al. (1958) studied the influence of muscle glycogen concentration on tenderness of poultry. Nine-week-old broilers were assigned to four groups and were either fasted, or for a 16-hr. period immediately prior to slaughter were allowed access to only water, to broiler mash and water, or to a sugar-broiler mash mixture and water. The fasted group of broilers had a higher muscle glycogen level than did the group which had been fed the sugar-broiler mash mixture (55% sugar). The other two groups were intermediate in glycogen level and did not differ significantly from either the fasted birds or the sugar-fed birds. Shear values obtained from the Pectoralis minor muscle of the 18 carcasses of highest glycogen concentrations were lower (muscles more tender) than the shear values of corresponding muscles from the birds of

lowest glycogen concentrations. The pH of the Pectoralis major muscle decreased from an initial value of 6.4 to a final pH of 5.9 for the group of carcasses with highest glycogen concentration. The pH of the group of carcasses with the lowest glycogen level did not change.

de Fremery and Pool (1960) compared the ultimate pH of chicken muscle with data in the literature for other species. Values were similar for the several species. Ultimate pH of chicken muscle was approximately 5.8 to 5.9 as compared to 5.9 for the rabbit and 5.4 for pork and beef. However, larger animals tended to require more time for the onset of rigor. Under normal processing procedures the time of rigor onset in the chicken was 2 to 4.5 hours post-mortem as compared to 10 hours for beef.

de Fremery and Pool (1960) reported on a number of factors affecting glycogen breakdown and pH change in post-mortem chicken muscle. They found that severe mechanical handling of fresh muscle tissue induced muscle toughness and caused a rapid decline in muscle pH when compared to controls. A very rapid breakdown of glycogen and a rapid decline in pH occurred following the freezing and thawing of fresh muscle (muscle frozen immediately after death) as compared to fresh unfrozen controls. Exhaustive electrical stimulation and electron irradiation of chicken muscle excised immediately after death resulted in rapid pH decline and increased muscle toughness.

McLoughlin (1970) studied muscle contraction and post-mortem pH changes in Landrace pig skeletal muscle. The gastrocnemius muscle was stimulated to contract via the sciatic nerve in vivo and the pattern of pH change was compared to that of the paired unstimulated muscle. Stimulation caused a reduction in initial pH and an acceleration in the subsequent rate of pH fall in the excised muscle under a stream of moist nitrogen at 37°C (98.6°F). It was concluded that neural stimuli entering the muscle at the time of death were the main factors involved in the rapid post-mortem glycolysis observed in the pigs studied.

de Fremery and Pool (1963) prevented or minimized post-mortem glycolysis in young birds by three different techniques. Antemortem injections of epinephrine were given to deplete muscle glycogen stores prior to slaughter, as described by Cori and Cori (1928). Injections of sodium iodoacetate were given the birds to inhibit post-mortem glycolysis by inhibiting phosphoglycericaldehyde dehydrogenase. The third technique involved very rapid cooking before glycolysis had time to proceed very far. Rapid post-mortem disappearance of ATP and consequent rapid onset of rigor were not accompanied by toughness in all three conditions. This led to the conclusions that post-mortem glycolysis caused toughness and that the faster the glycolysis the greater the toughness. However, it is not yet known whether a rapid rate of glycogen breakdown, a rapid rate of pH decrease (lactic acid formation), or some other post-

post-mortem change related to glycolysis induces toughness when normal muscle goes into rigor. An accelerated formation of lactic acid may be involved since acids do affect protein stability. It may be that rapid post-mortem glycolysis influenced the normal post-mortem changes in the muscle fibers by increasing the degree of inter- and intramolecular bonds that changed the muscle from an elastic to an inelastic fiber. These researchers also noted that the elevation of the ultimate pH by adrenaline appeared to have little or no effect on meat flavor.

Pool (1963) reported on the elasticity of muscle of epinephrine-treated chickens. Maximum loss of extensibility occurred sooner in the epinephrine-treated muscles (7 to 8 hrs.) than in the control group (10 to 12 hrs.). Length (under very light load) of epinephrine-treated muscle strips decreased to about 70% of initial length, then increased slowly over the remainder of a 24-hour period to about 80% of initial length. The controls shortened slightly, to about 90% of initial value, then recovered almost their original length. Epinephrine-treated muscles showed changes in extensibility and length similar to muscles stimulated by various physical agents (de Fremery and Pool, 1960). However, epinephrine-treated muscles were generally tender immediately post-mortem in contrast to stimulated muscles which were usually permanently tough. From these observations, Pool (1963) concluded that the extensibility changes in muscle during the course of rigor and the development of tenderness

in muscle were separate phenomena. The first was closely related to the concentration of ATP present and might involve some irreversible protein-protein interaction (de Fremery and Pool, 1960). The second, the tenderization-toughening phenomenon, appeared to be quite sensitive to the rate of anaerobic degradation of glycogen to lactic acid. The mechanism of this glycolytic effect is yet unknown.

Khan and Nakamura (1970) studied the effects of pre- and post-mortem glycolysis upon poultry tenderness. Pre-mortem glycolysis, occurring as a result of death struggle or epinephrine administration 1 to 2 hours before slaughtering, lowered the pH of the meat at the time of death and caused toughness. Minimization of post-mortem glycolysis by epinephrine administration more than 5 hours before slaughtering increased the ultimate pH of meat and the tenderness. Their results indicated that a pH value above 6.2 just after slaughtering and an ultimate pH near 5.7 were desirable for maintaining quality of poultry breast meat, and that these pH values were maintained by minimum pre-mortem and maximum post-mortem glycolysis. Accumulation of lactic acid in muscle tissue immediately before and after slaughter apparently caused some changes in certain muscle tissue components and rendered the meat tough.

Tests made on Pectoralis major muscles having post-slaughter pH values ranging from 6.1 to 7.0, indicated that holding poultry meat at 30 to 37°C (86 to 98.6°F) during the onset of rigor caused toughness (Khan, 1971). This toughening effect of high temperature

occurred when the pH level of the meat dropped from a value of about 6.3 to its ultimate low value and the ATP content dropped below 40% of its initial concentration. Holding temperatures at 10, 15 and 25°C (50, 59 and 77°F) during onset of rigor, or cooling to 15°C (59°F) before the pH dropped to 6.3 produced meat more tender than that held at 30 to 37°C (86 to 98.6°F). After the completion of post-mortem glycolysis and dephosphorylation of high energy phosphates, high temperature had no deleterious effect on tenderness. The effects of post-mortem dephosphorylation of ATP, and the mode or extent of stiffening on tenderness are not yet fully understood, but results of this study indicated that the harmful effects of these changes can be minimized by ensuring that the final phases of dephosphorylation and glycolysis occurred at temperatures below 25°C (77°F).

Muscle proteins. Many of the attributes which determine the acceptability of muscle as a food are established during post-mortem onset and resolution of rigor. Therefore, it is important to understand the nature of the changes that occur in muscle proteins during this period. Principal emphasis has been placed on the myofibrillar proteins since they constitute over 50% of the total protein in muscle and are also directly implicated in the water-holding capacity, emulsification properties, and tenderness of muscle.

Weinberg and Rose (1960) studied changes in protein extractability during post-rigor tenderization of Pectoralis major muscles of young chickens. They suggested that post-rigor tenderization results from the dissociation of the actomyosin complex (the contractile protein formed during rigor) into actin and myosin.

Khan (1962) compared different methods of extraction and fractionation of chicken breast and leg muscle from fresh-killed (stored in crushed ice 24 to 48 hours) 10-week-, 4-month-, and one-year-old birds. Protein fractionation in KCl-borate buffer showed that in one-year-old chicken meat, stroma-, myofibrillar-, and sarcoplasmic-protein nitrogen, respectively, contributed 13, 42 and 30% of total nitrogen in breast muscle and 27, 30 and 22% in leg muscle. Results with different chickens indicated that, with increase of age, stroma increased and myofibril decreased in both breast and leg muscles. These two protein fractions also differed in breast and leg muscle, and varied with source of supply of the chickens. The difference was small between birds of the same flock and between left and right halves of the same bird.

Chajuss and Spencer (1962b) aged chicken Pectoralis major muscles in water, potassium iodate, sodium hydrosulfite, and sodium sulfite. Iodate caused the muscles to be considerably tougher, probably due to the oxidation effect of iodate on the labile sulfhydryl groups (-SH) of cysteine, glutathione, and thioglycollate to form nonlabile disulfide bonds (-S-S-) with a small amount of

sulfinates ( $-S_2H$ ) and sulfonates ( $-SO_3H$ ). Non-significant differences were observed between hydrosulfite and water treatments. Sodium sulfite treated muscles were more tender, possibly due to the reduction of disulfide bonds. The results suggested that changes in tenderness of poultry meat during the resolution of rigor and post-mortem aging may be due to cleavage or reorientation of intermolecular and/or intramolecular disulfide bonds. Further research by Chajuss and Spencer (1962a) showed a rapid decrease in sulfhydryl group content of chicken muscle aged in air during the development of rigor.

Partmann (1963) reported that contraction normally occurred when ATP was added to fiber fragments of aged meat. This implies that the actomyosin complex formed during rigor development became dissociated in aged meat, and that tenderness changes in the aging period were correlated with this process.

Fischer (1963) reported on changes in the chemical and physical properties of protein during aging of meat. Samples removed from the Pectoralis major muscle of chickens at various times after slaughter were extracted with water, dilute salt, and dilute acid or base in order to separate the proteins into separate fractions. When the water-soluble fractions were subjected to diethylaminoethyl (DEAE) cellulose column chromatography, an unidentified substance was isolated which in a limited number of experiments was found to increase during the aging process and with tenderness. The salt-soluble fractions when subjected to disc electrophoresis showed an

increase in one band which appeared to be actomyosin and a decrease in another band thought to be myosin.

Miller et al. (1964) studied the relationship between free amino acid content and tenderness of chicken muscle tissue. Free amino acid analyses were conducted on young (tender) and old (tough) and fresh and aged chicken samples. In general, ammonia nitrogen remained fairly constant throughout the study. Storage resulted in increases in free amino acids with proline being a major exception. Light meat contained less free amino acids than dark meat with major exceptions being lysine and histidine. Broilers had more free amino acids than hens in most cases. No relationship was found between tenderness and the general pattern of free amino acid concentration nor between tenderness and the concentration of any single free amino acid.

Khan and van den Berg (1964) extracted and fractionated the muscle proteins from breast and leg muscles from 6-, 9- and 12-month-old chicken carcasses held for aging at 0°C (32°F). Buffer-extractable nitrogen (% extractable nitrogen of total nitrogen) rapidly decreased after death during the onset of rigor and gradually increased to a maximum value during post-rigor aging. The changes in extractable nitrogen occurred mainly as a result of changes in the solubility of myofibrillar proteins. Changes in sarcoplasmic and stroma (connective tissue) protein fractions were small. The nonprotein - nitrogen content decreased slightly during the onset of

rigor as a result of the interaction of amino-acid-containing polymers with proteins. During the post-rigor tenderization period, peptides and amino acids increased in the meat as a result of proteolysis. The reason that tenderization occurred more slowly in leg muscle than in breast muscle appeared to be related to the fact that leg muscle contains more than twice as much stroma protein as does breast muscle. These researchers concluded that proteolysis appeared to weaken or break the bonds that bound myofibrils to the matrix of the muscle and caused protein changes which were responsible for post-rigor tenderization.

Neelin and Rose (1964) studied progressive changes in starch gel electrophoretic patterns of chicken muscle proteins during post-mortem aging. Myofibrillar proteins revealed no detectable consistent change during a two-day aging period. Myogen proteins also remained unchanged in white muscles, but an additional electrophoretic component, possibly derived from myoglobin, slowly appeared in red muscle extracts. The delay in the development of this component suggested a secondary relation with tenderizing processes. Important constituents of myogen were lacking in sarcoplasmic proteins extracted from breast muscle, pre-rigor or in-rigor. Some of the myogen components absent from sarcoplasm gradually reappeared as tenderization proceeded. It was suggested that the additional components obtained in sarcoplasm of tenderized muscle reflected soluble proteins escaping into the extract because

of the breakdown of intracellular barriers or subcellular particles. These components may include enzymes instrumental in initiating changes in the myofibril, ultimately leading to tenderization.

de Fremery (1967) noted that raw muscles do not have transverse breaks in the region of the I-band of the sarcomeres in poultry muscle. Such breaks were present in post-rigor cooked muscle where they occurred to a much greater extent than in pre-rigor cooked muscle. It was suggested that although it is possible that this appearance could result from a proteolysis of the I-band components (chiefly actin) into fragments of low molecular weight, it would seem more reasonable to hypothesize that this appearance results from a labilization of F-actin leading to dissociation upon heating.

Sayre (1968) measured protein extractability from chicken pectoralis after the muscle was aged in ice for various periods from 30 min. to 24 hrs. Sarcoplasmic protein, non-protein nitrogen and stromal protein remained constant for all aging periods at 33, 16 and 7% of the total nitrogen, respectively. Myosin extractability decreased rapidly during the first 3 to 4 hours of aging while the alkali soluble protein increased and actomyosin was extracted at a low constant level. The alkali soluble protein became constant after 4 to 6 hours of aging, and actomyosin appeared in the extract in increasing quantities as myosin continued to decline. The sum of myosin, actomyosin and alkali soluble protein was constant for all aging times at 44% of the muscle nitrogen. The initial accumulation

of alkali soluble protein and the subsequent release of actomyosin corresponded to the time course of toughening and tenderization in chicken muscle. It was suggested that these observations reflected initial binding of myosin to the nonextractable thin filaments, followed by disintegration or detachment of these filaments from the Z line.

de Fremery and Streeter (1969) found that post-mortem tenderization of chicken meat was not related to changes in connective tissue but must be ascribed to some other fraction of muscle tissue. Maximum shear-resistance values occurred in breast muscles 3 to 4 hrs. post-mortem; minimum values were reached 12 hrs. post-mortem and did not change significantly during aging for 8 days. Maximum shear-resistance values occurred in thigh muscles 3 hrs. post-mortem. In these muscles, tenderization continued during 8 days of aging. In contrast, alkali-insoluble connective tissue determined in either raw or cooked muscle (as measured by alkali-insoluble hydroxyproline) did not change significantly as a function of post-mortem aging time (1 hr. vs. 24 hrs. for breast meat, 1 day vs. 8 days for thigh meat). Cooking solubilized considerable amounts of the connective tissue.

Caldwell and Lineweaver (1969) found no significant changes in total or non-protein sulfhydryl concentrations in chicken breast muscle during the first 6 hrs. post-mortem. They rejected the postulate of Chajuss and Spencer (1962a, 1962b) that a correlation existed between sulfhydryl concentration and rigor or tenderization.

Fukazawa et al. (1970) extracted myofibrillar proteins from pre- and post-rigor chicken pectoral muscles. They proposed that an increasing amount of post-rigor protein which indicated the release of  $\alpha$ -actinin (a protein component of the Z-line) along with the destruction and final dissolution of the Z-line structure occurred during post-mortem storage of chicken pectoral muscle.

Landes et al. (1971) determined changes in pH and protein extractability in turkey breast muscles from anesthetized and nonanesthetized birds from 0 to 72 hours post-mortem. Total extractable-, total soluble fibrillar protein-, soluble actomyosin-, sarcoplasmic protein-, nonprotein-, and unextracted alkali soluble protein-nitrogen values remained fairly constant during the first hour post-mortem in muscles from anesthetized birds, but changed immediately in muscles from control birds. Total extractable nitrogen, total soluble fibrillar protein nitrogen and soluble actomyosin nitrogen extracted from muscles of control birds increased during post-mortem aging (beginning at 1 hour post-mortem). These fractions in muscles from other species (chicken, beef, pig, rabbit) either remained the same during rigor development or decreased. An explanation for this species difference was not evident from the results of this study. The muscles of anesthetized birds were more tender (as measured by shear value) than those from the control birds.

Wu and Sayre (1971) extracted myosin from chicken muscle which had been aged for 30 min. and 24 hrs. post-mortem. When a protein fraction (component T) was separated by chromatography from the myosin extraction, the ATP-ase activity and sedimentation pattern of the resulting myosin were similar to those of chromatographed myosin from unaged muscle. The number of sulfhydryl groups in the myosin was not affected by aging. Lowering the pH of myosin extracted from unaged muscle to the pH values found in muscle aged for 24 hrs. caused aggregation and loss of ATP-ase activity.

Goll et al. (1970) summarized the present knowledge of changes that occur in muscle during post-mortem aging. Post-mortem storage causes at least two kinds of biochemical changes in the myofibrillar apparatus: 1) disruption and degradation of the Z line, and 2) weakening of the actin-myosin interaction. Post-mortem degradation of the Z line is indicated biochemically by a post-mortem increase in the rate of actin extractability. Post-mortem weakening of the actin-myosin interaction is indicated by an increased susceptibility of the actin-myosin complex to dissociation by ATP, and possibly also by the increased nucleosidetriphosphatase activity of actomyosin preparations made from post-mortem muscle. Recent results indicate that post-mortem Z-line degradation is caused by  $Ca^{++}$ , which is released when the sarcoplasmic reticular membranes in post-mortem muscle lose the ability to accumulate  $Ca^{++}$  against a concentration gradient. The cause of post-mortem weakening of

the actin-myosin interaction is not yet clear, but the cause probably originates from post-mortem changes in actin and myosin themselves. Post-mortem disruption and removal of Z lines would cause loss of the ability of post-mortem muscle to maintain isometric tension and would also have important effects on the use of muscle as a food. Future studies on the effects of  $Mg^{++}$ ,  $Ca^{++}$ , ATP and pH changes on the myofibrillar proteins may be expected to clarify our understanding of post-mortem changes in the molecular structure of muscle.

Moisture content. Dodge and Stadelman (1960a) studied relationships between tenderness and moisture levels during early post-mortem aging of turkey meat. Water uptake and rates of cooling were not shown to affect tenderness, whereas dehydration of hot carcasses was found to produce a toughening effect which could not be eliminated by aging. Total moisture content of the tissue did not appear to be associated with water uptake, nor was it shown to be related with tenderness. Percent free moisture in the meat did not appear to be related to tenderness.

It is well known that meat frozen without aging loses considerable water on thawing and tends to become drier and less palatable. Khan and Lentz (1965) tested chickens frozen before, during, and after rigor and found that the amount of drip exuded upon thawing was greatest from poultry frozen during rigor. The loss of nitrogenous constituents and ribose increased proportionally with the amount of drip. Protein solubility was minimum and cooking losses

maximum in poultry frozen during rigor. It is probable that more water was released from the frozen muscle during rigor which caused a higher solute concentration. This may have affected the solubility of proteins and their ability to reabsorb water upon thawing and thus affected tenderness and loss of drip.

#### Histological Changes in Post-mortem Muscle

Hanson et al. (1942) presented photomicrographs showing the disintegration of the striated to a granular-like structure in muscle fibers of broilers in which rigor had been resolved. Stewart et al. (1945) and Carlin (1949) reported that both the rate of freezing and time of aging before freezing affected the histological appearance of poultry muscle fibers. Stewart et al. (1945) observed vacuoles within the fibers of breast and thigh muscles of broiler carcasses frozen at  $-67.8^{\circ}\text{C}$  ( $-90^{\circ}\text{F}$ ) within 2 hours after slaughter. These vacuoles were very numerous in both raw and cooked sections from some broilers, less so in other broilers. These vacuoles were considered an indication of intra-fibrillar freezing; the ice crystals formerly occupying the site of the vacuoles. Intra-fibrillar freezing also occurred in all breast muscles and half of the thigh muscles of broilers frozen within 2 hours post-mortem at  $-45.5^{\circ}\text{C}$  ( $-49.9^{\circ}\text{F}$ ). No intra-fibrillar freezing occurred in broilers frozen at  $-20.5^{\circ}\text{C}$  ( $-4.9^{\circ}\text{F}$ ) within 2 hours post-mortem. Generally, intra-fibrillar freezing did not occur in any broilers held 18 hours before freezing, regardless of the freezing temperature used. Both

passive and active rigor nodes were observed in the histological sections of the broilers. The waves and kinks of passive rigor were nearly always found near connective tissue, either collagenous or elastic or both, as if the contractures might have been caused by shrinking of the connective tissues. The waves and kinks were found in tissues of all broilers regardless of time held before freezing or the temperature of freezing. The active rigor nodes were found more frequently in broilers frozen within two hours post-mortem. Thin spots and breaks in muscle fibers, which are characteristic of the passing of rigor, were far more numerous in birds held 18 hours than in those held 2 hours before freezing. The breaks were more numerous in the breast muscle than in the thigh muscle fibers. The cross striations disappeared in the thin spots and in the breaks of the fibers in the uncooked tissue, leaving an apparently empty space. Cooking further altered the histological appearance of the fiber, in that a granular-like tissue replaced the apparently empty spaces of breaks of the uncooked tissues. Cooking also accentuated the disruption of the elastic fibers into short, needle-like rods.

Koonz and Robinson (1946) studied the histological composition of twelve of the principal muscles composing the poultry carcass. The various muscles showed some variation in the amount and distribution of connective tissue and of fat and in the size and arrangement of muscle bundles. Elastic connective tissue was almost completely

absent. Breast muscles contained less fat and connective tissue than did thigh muscles.

Lowe (1948) studied histological changes in poultry muscle. She photomicrographed the turbulence phenomenon in fowl muscle 30 minutes post-mortem. Turbulence is a disorganization of the cross striae caused by exposing the muscle fibers to heat and possibly by other factors. It is much more likely to develop if the muscles have been aged for only a short time. Turbulence has been described as caused by thermal agitation of the molecules and colloidal particles sufficient to destroy the cross-striated internal structure. Turbulence was found more often in cooked than in uncooked tissues. In the uncooked tissues it appeared as if causes other than the scalding might have brought about some of the turbulence. It is possible that the spasmodic quivers of some birds (instead of the usual type of death struggle) might have caused turbulence. When turbulence was found in uncooked fibers it usually persisted through cooking.

Photomicrographs by Lowe (1948) also revealed that the shape of rigor nodes in chicken muscles varied from very long to a rounded shape with some arranged in bead-like rows. Some nodes were perpendicular to the long axis fibers, some were obliquely placed across the fiber, and some did not cover the entire fiber diameter. Rigor nodes are the result of strong contraction waves in muscle and are of two types, those occurring in the normal onset of rigor and those induced by heat.

Disintegration is the term applied to the disappearance of the cross striations of the muscle fibers and their replacement with a granular-like substance. Muscle tenderness increased with the beginning of disintegration (Lowe, 1948).

Takahashi et al. (1967), using a phase-contrast microscope, observed that myofibrils of chicken pectoral muscles were fragmented into progressively smaller sections (consisting of 1 to 4 sarcomeres) when homogenized in buffer after increased periods of aging. Post-mortem aging caused initial fiber contraction and a dense band to develop in the mid-region of the sarcomere (2 hours post-mortem). The fibrils then became shorter and subsequently the number of fragments increased. Sayre (1970), however, reported that the myofibril fragmentation pattern was not an accurate index of tenderness.

Fukazawa et al. (1969) examined changes in the morphology of myofibrils prepared from chicken pectoral muscle during post-mortem storage at 5°C (41°F) using light and electron microscopy. Electron photomicrographs of homogenized 24-hour stored samples showed two types of destruction in the Z-lines of sarcomeres and myofibrillar fragments: 1) the degradation and/or disappearance of Z-lines and 2) the breakdown of the junction of the Z-line and I-filaments. A change in the state of the Z-line and the junction of the Z-line and I-filaments appeared to be indispensable for the fragmentation of the myofibrils. It was also shown through phase contrast microscopic

examinations that sarcoplasmic proteins, participating in the glycolytic cycle, may play a role in the fragmentation of the myofibrils.

#### Effects of Stretch Tension on Post-mortem Muscle

Buck and Black (1967) subjected bovine Longissimus dorsi muscles to stretch-tension during rigor. A 2000-gram weight was suspended from muscle strips measuring 2.5 x 2.5 x 23 cm for 72 hours post-mortem. Control strips of the same size were weighted with 500 grams to maintain the muscle strips at a constant length. Stretching the muscles significantly decreased individual muscle fiber diameter and extensibility and significantly increased tenderness. There were essentially no differences in pH values between the control and stretched samples. The mean pH at 10 minutes post-mortem was 6.7. Seventy-two hours post-mortem the pH had dropped to a mean of 5.5.

Histological examination of cooked tissue samples from these same Longissimus dorsi muscles revealed greater amounts of perimysial tissue denaturation (as indicated by the degree of tissue granulation) in the stretched muscle sections (Buck and Black, 1968). These results suggest that the increased tenderness caused by stretching may be accounted for by a mechanical thinning of connective tissues.

Gillis and Henrickson (1969) studied the effect of stretch-tension during rigor on bovine semitendinosus and semimembranosus muscle strips (6 x 6 x 21 cm). The samples were subjected to four levels of tension (0, 1000, 2500, 5000 grams) for 48 hours. Results showed that the stretched muscles had reduced fiber diameters, increased sarcomere lengths and increased tenderness as compared to controls.

Buck et al. (1970) studied shear strength, sarcomere length and protein solubility in rabbit Longissimus dorsi muscles allowed to pass through rigor in free and stretched physical states. Stretched muscles were significantly more tender and exhibited significantly longer sarcomeres as compared to their paired non-stretched controls. Greater amounts of total protein and significantly greater amounts of actomyosin were extracted from the stretched muscles. The actomyosin results were unexpected since it had been suggested by several researchers that actomyosin formation is directly related to toughness in muscle. Two possible explanations were offered for the unexpected results. First, stretching may stimulate muscle so that it uses ATP more rapidly and more completely, forming actomyosin which does not dissociate upon extraction. Free muscle, on the other hand, might contain greater amounts of residual ATP which would tend to dissociate actomyosin during extraction. Another possible explanation for the greater extraction of total protein and actomyosin from stretched muscle may be that the stretching caused a

disruption and dissolution of the Z-band structures similar to that which occurred during aging. Earlier workers found that extraction of myofibrillar protein increased as aging progressed.

#### Enzymatic Tenderization of Poultry Meat

Some attempts have been made to improve the tenderness of poultry meat by ante-mortem injection and post-mortem application of the enzyme papain. Huffman et al. (1961) found that ante-mortem injection of papain into the peritoneal cavity significantly increased the tenderness of poultry breast muscle, but that certain levels of the enzyme caused overtenderization which rendered the breast muscle lacking in sufficient body or texture to be fully acceptable.

Fry et al. (1966) reported that ante-mortem injection of papain solution into the circulatory system of 15-month-old broiler breeder males resulted in thighs and breasts significantly more tender (shear press and taste panel values) than those of controls. Post-mortem application of the enzyme resulted in a serious lack of uniformity of action, and was not considered an acceptable means of tenderizing poultry. The enzyme solution injected into the chicken rolls tended to migrate to the bottom of the roll during cooking. As a result, the muscle portions at the top of the roll were not tenderized, and those at the bottom were mushy.

### Effects of Cooking Methods on Poultry Meat Tenderness

Mickelberry and Stadelman (1962) reported that cooking chicken meat before freezing resulted in significantly less tender products, a smaller freezer drip, and greater total weight losses than freezing in the uncooked condition, slow thawing, and subsequent cooking.

Goodwin et al. (1962) cooked turkey fryer-roasters by six different methods to an internal temperature of 85°C (185°F) and determined shear values on the Pectoralis major and Biceps femoris muscles using a Kramer shear press. Methods of cooking (microwave oven, deep-fat frying, steam pressure, rotary reel oven, a combination of steam and deep-fat frying, and the combination of deep-fat frying and microwave oven) had no significant effects upon shear values of the turkey meat.

Goodwin et al. (1962) studied effects of end-point temperature and cooking rate on turkey meat tenderness. The turkeys (weighing 18 to 20 lbs.) were wrapped in aluminum foil and cooked in a conventional electric range. No significant differences were found in shear values of turkeys cooked to internal temperatures of 77, 82, 88 and 94°C (170.6, 179.6, 190.4 and 201.2°F), but meat cooked to 55°C (131°F) had significantly higher shear values than meat cooked to 77°C (170.6°F) or above. Breast cooked to 88 and 94°C (190.4 and 201.2°F) appeared drier and tended to crumble and fall apart more than that cooked to 77 or 82°C (170.6 or 179.6°F). Rate of cooking (oven temperature) had no significant effect upon shear values.

Essary et al. (1963) found that shear values of the Pectoralis major and Biceps femoris muscles of broilers were significantly increased (tenderness decreased) by cooking boned meat as compared to cooking meat attached to the bone and by cooking poultry meat in water at different initial water temperatures. Their results indicate that a more tender product was obtained when meat was placed in cold tap water before cooking as compared to placing it in warm or boiling water.

## OBJECTIVES

The objectives of this study were:

1. To further study the relationship between pre- and post-mortem glycolysis and tenderness of poultry Pectoralis major muscles.
2. To determine the effects of speeding up pre-mortem glycolysis on the rigor pattern and ultimate tenderness of poultry meat.
3. To determine how reduced struggling during slaughter affects the rigor pattern, glycogen content, and tenderness of poultry meat.
4. To compare the effects of fasting and full-feeding prior to slaughter on post-mortem glycolysis and tenderization.
5. To study the effects of stretch-tension during rigor on glycogen content and tenderization of poultry meat.

## EXPERIMENTAL PROCEDURE

### Experiment 1

The birds used in this experiment were nine-week-old Arbor Acre X Vantress 60 Cross male broilers purchased from Rockingham Poultry Marketing Co-op., Inc., Broadway, Virginia. The live broilers were brought from the poultry processing plant in coops in an air-conditioned station wagon, placed in wire cages and fed a broiler mash and water ad libitum for several days at the Virginia Polytechnic Institute and State University poultry processing laboratory until slaughter. For Experiment 1, thirty-six broilers were divided into three groups of twelve birds each.

Group I (off-feed control group) birds were fasted for 16 hours and then were killed, scalded at  $58.9^{\circ}\text{C}$  ( $138^{\circ}\text{F}$ ) for 40 seconds, and picked using a rotating drum picker and warm eviscerated. Average dressed weight (without giblets) of birds in Experiment 1 was 2.6 pounds. The Pectoralis major muscle from the right side of each bird was removed immediately after evisceration, and a two-inch C-clamp was attached to each end of the muscle. The muscle was then stretched by attaching approximately 200 grams (weight determined while suspended in water) to the C-clamp on the smaller end of the muscle. The C-clamp attached to the larger end of the muscle was anchored to a rod suspended in such a manner to permit the muscle to be in slush ice during the chilling operation. The left Pectoralis major muscle of each bird was left intact in the carcass during the chilling process.

At each of four post-mortem time intervals (1 hour, 4 hours, 8 hours, and 24 hours), the paired Pectoralis major muscles (one stretched and one non-stretched) from three carcasses were evaluated for shear values and glycogen content. A strip of each muscle 15 mm wide and the thickness of the muscle was removed and cut into four sections (1/2 to 3/4 inch apart) on the Warner-Bratzler shear press (Bratzler, 1932). Results from many experiments indicated that the correlation between sensory methods and the Warner-Bratzler shear were generally in a range of 0.60 to 0.85, with an average value of about 0.75, which means that the Warner-Bratzler shear gives a fairly good estimate of tenderness (Pearson, 1963). Approximately one-gram samples of the muscle tissue strips were then digested and the glycogen precipitated by the method of Hassid and Abraham (1957), and the tissue glycogen content was determined by the anthrone method of Seifter et al. (1950).

Group II (adrenaline group) birds were fasted for 24 hours, injected subcutaneously in the neck with 1 mg/kg body weight of adrenaline suspended in 0.85% saline solution, and then fasted an additional 16 hours prior to slaughter. These birds were processed and the Pectoralis major muscles evaluated as described above for Group I.

Group III (alcohol group) birds were fasted for 16 hours and were then given 10 ml of 30% ethyl alcohol per kilogram of broiler weight by pipette into the crop in order to reduce struggling

during processing. The alcohol was permitted to take effect upon the birds (approximately 30 minutes), and they were processed and evaluated as described above for Group I.

The data were treated to analysis of variance by the methods of Snedecor and Cochran (1967) and to the multiple range and multiple F test by Duncan (1955).

### Experiment 2

The procedure for Experiment 2 was the same as that of Experiment 1, except that an additional experimental group was added. For Experiment 2, forty-eight nine-week-old male broilers from the same flock as those of Experiment 1 were divided into four groups of twelve birds each. Groups I-III were treated the same as those in Experiment 1.

Group IV (full-feed control group) birds were not fasted prior to slaughter but were kept on full-feed until a few minutes before slaughter. The birds were then processed and the carcasses evaluated as described above. The purpose of this additional control group was to determine the effect of fasting versus full-feeding prior to slaughter on the glycogen content and shear values of the Pectoralis major muscles.

## RESULTS AND DISCUSSION

### Expt. 1. Effects of Treatment Group X Post-mortem Aging Interaction on Broiler Muscle Shear Values

According to data in Table I, the ante-mortem treatment of the broilers affected the post-mortem aging pattern of the Pectoralis major muscles. Group I (off-feed controls) carcasses were significantly tougher at 1 hour post-mortem than at any of the remaining post-mortem time intervals. Breast muscles from Group II (adrenaline group) carcasses were not significantly different in tenderness at any of the four post-mortem time intervals (1, 4, 8 and 24 hours). However, the birds which had been given alcohol prior to slaughter did not reach maximum toughness until 4 hours post-mortem, after which time there was a gradual increase in tenderness at 8 and 24 hours post-mortem. Statistical analyses of data in Table I are presented in Tables IX and X in the Appendix. Group III (alcohol group) had the highest muscle glycogen content (Table VII) upon death which means that it probably required a longer time for glycolysis and decreased ultimate pH to lead to rigor in these muscles.

### Expt. 1. Effects of Stretch-Tension on Broiler Muscle Tenderness

Statistical analyses (Table IX in Appendix) of data from Table II indicated no significant differences in tenderness between stretched and non-stretched Pectoralis major muscles for any of the

Table I. Expt. 1.\* Shear values of broiler Pectoralis major muscles

	Group I	Group II	Group III
post-mortem hrs	off-feed control lbs.	adrenaline lbs.	alcohol lbs.
1	4.76 <sup>a**</sup>	4.44	3.79 <sup>a</sup>
4	2.82 <sup>b</sup>	4.45	4.91 <sup>c</sup>
8	2.63 <sup>b</sup>	4.38	3.12 <sup>ab</sup>
24	3.12 <sup>b</sup>	3.98	2.54 <sup>b</sup>

\* Each value is the average of 6 muscles (one pair of muscles from 3 different broilers).

\*\* Values in the same column having different superscripts are significantly different ( $P \leq .01$ ).

Table II. Expt. 1. Shear values of non-stretched and stretched broiler Pectoralis major muscles\*

post-mortem hrs	<u>Group I (off-feed control)</u>		<u>Group II (adrenaline)</u>		<u>Group III (alcohol)</u>	
	non-stretched** lbs.	stretched lbs.	non-stretched lbs.	stretched lbs.	non-stretched lbs.	stretched lbs.
1	4.86	4.65	4.54	4.34	3.90	3.67
4	2.50	3.13	4.83	4.06	5.12	4.69
8	2.46	2.79	4.55	4.21	3.04	3.19
24	3.13	3.11	4.09	3.86	2.63	2.44

\* Each value is the average of 3 muscles.

\*\* Non-stretched and stretched muscles under each group are paired muscles from the same broiler.

three treatment groups in Expt. 1. The 200-gram weight used in this experiment apparently did not cause sufficient stretch-tension in the muscles to result in the increased muscle tenderness as reported by Buck and Black (1967) with bovine muscles and by Buck et al. (1970) with rabbit muscles. Either the use of a smaller strip of muscle rather than the entire Pectoralis major muscle and/or the use of a heavier weight would probably have resulted in the expected increased tenderness of the poultry meat.

Expt. 1. Effects of Stretch-Tension on  
Broiler Muscle Glycogen Content

Data presented in Table III were subjected to statistical analyses (Table XI in Appendix) and no significant differences in glycogen content between stretched and non-stretched Pectoralis major muscles at each of the post-mortem time intervals were found for any of the three treatment groups in Expt. 1. Since the 200-gram weight used as stretched-tension in this experiment was probably not sufficient to increase tenderness of the breast muscles, it is difficult to predict what effect stretch-tension would have on glycogen content of these muscles. Buck and Black (1967) found essentially no differences in pH values between control and stretched samples of bovine Longissimus dorsi muscle as post-mortem aging progressed from 10 minutes to 72 hours.

Table III. Expt. 1. Glycogen contents of broiler Pectoralis major muscles\*

post-mortem hrs	<u>Group I (off-feed control)</u>		<u>Group II (adrenaline)</u>		<u>Group III (alcohol)</u>	
	non-stretched** mcg/gm	stretched mcg/gm	non-stretched mcg/gm	stretched mcg/gm	non-stretched mcg/gm	stretched mcg/gm
1	61.53	56.47	78.21	63.20	810.59	1518.04
4	98.38	140.20	68.49	71.28	140.66	343.13
8	52.12	58.01	66.32	79.99	50.19	61.08
24	70.11	104.49	54.70	51.96	57.95	52.59

\* Each value is the average of 3 muscles.

\*\* Non-stretched and stretched muscles under each treatment are paired muscles from the same broiler.

Table VII. Effects of ante-mortem treatments on shear values and glycogen contents of broiler Pectoralis major muscles\* for combined post-mortem time intervals

	Expt. 1		Expt. 2	
	Shear Values lbs.	Glycogen mcg/gm	Shear Values lbs.	Glycogen mcg/gm
Group I (Off-feed Control)	3.33 <sup>a**</sup>	80.16	3.25 <sup>a</sup>	150.63 <sup>a</sup>
Group II (Adrenaline)	4.31 <sup>b</sup>	66.77	5.88 <sup>b</sup>	101.41 <sup>a</sup>
Group III (Alcohol)	3.58 <sup>a</sup>	379.28	4.06 <sup>c</sup>	455.52 <sup>b</sup>
Group IV (Full-feed Control)	-- <sup>***</sup>	--	4.66 <sup>c</sup>	230.92 <sup>a</sup>

\* Each value is the average of 24 muscles (one pair of muscles from 12 different broilers).

\*\* Values in the same column having different superscripts are significantly different ( $P \leq .01$ ).

\*\*\* Group IV not included in design of Expt. 1.

Table IV. Expt. 2\* Shear values of broiler Pectoralis major muscles

post-mortem hrs	Group I off-feed control lbs.	Group II adrenaline lbs.	Group III alcohol lbs.	Group IV full-feed control lbs.
1	3.27	7.16 <sup>a**</sup>	4.19	5.30 <sup>a</sup>
4	3.33	5.94 <sup>b</sup>	4.31	4.96 <sup>a</sup>
8	3.18	5.72 <sup>b</sup>	4.02	5.44 <sup>a</sup>
24	3.22	4.72 <sup>b</sup>	3.73	2.94 <sup>b</sup>

\* Each value is the average of 6 muscles (one pair of muscles from 3 different broilers).

\*\* Values in the same column having different superscripts are significantly different ( $P \leq .01$ ).

Expt. 2. Shear Values of Muscles  
Subjected to Stretch-Tension

Analysis of variance (Table XII in Appendix) of shear value data from Expt. 2 showed, as in Expt. 1, no significant differences in tenderness between stretched and non-stretched Pectoralis major muscles (Table V). The stretch-tension applied to the muscle during rigor was not sufficient to alter muscle tenderness.

Expt. 2. Effects of Stretch-Tension and  
Aging on Broiler Muscle Glycogen Content

Analysis of variance (Table XIV in Appendix) of data in Table VI again showed no significant differences in glycogen contents between stretched and non-stretched muscles for any treatment group or any post-mortem aging time. However, in Group III (alcohol group), aging had a significant effect (Tables XIV and XV) on muscle glycogen content. Glycogen content was significantly higher at 1 hour post-mortem than at any of the remaining aging periods. For the non-stretched muscles of alcohol-treated birds, glycogen content dropped from 1377.09 mcg/gm at 1 hour post-mortem to 109.64 mcg/gm at 24 hours post-mortem. For the stretched muscles, glycogen content dropped from 1146.26 mcg/gm at 1 hour post-mortem to 119.95 mcg/gm after 24 hours of aging. These data again indicate that reduced struggling prior to and during slaughter as a result of ante-mortem administration of 30% ethyl alcohol resulted in less glycogen depletion in the muscles immediately post-mortem as compared to the glycogen content of muscles from other treatments.

Table V. Expt. 2. Shear values of non-stretched and stretched broiler Pectoralis major muscles\*

post-mortem hrs	<u>Group I (off-feed control)</u>		<u>Group II (adrenaline)</u>		<u>Group III (alcohol)</u>		<u>Group IV (full-feed control)</u>	
	non-stretched** lbs.	stretched lbs.	non-stretched lbs.	stretched lbs.	non-stretched lbs.	stretched lbs.	non-stretched lbs.	stretched lbs.
1	3.25	3.29	7.25	7.06	4.54	3.83	5.90	4.69
4	3.77	2.89	6.19	5.69	4.27	4.35	4.62	5.29
8	2.88	3.48	6.00	5.44	3.94	4.09	5.85	5.02
24	3.46	2.98	4.81	4.63	3.94	3.52	3.02	2.86

\* Each value is the average of 3 muscles.

\*\* Non-stretched and stretched muscles under each treatment are paired muscles from the same broiler.

Table VI. Expt. 2. Glycogen contents of broiler Pectoralis major muscles\*

post-mortem hrs	<u>Group I (off-feed control)</u>		<u>Group II (adrenaline)</u>		<u>Group III (alcohol)</u>		<u>Group IV (full-feed control)</u>	
	non-stretched** mcg/gm	stretched mcg/gm	non-stretched mcg/gm	stretched mcg/gm	non-stretched mcg/gm	stretched mcg/gm	non-stretched mcg/gm	stretched mcg/gm
1	197.91	180.28	121.96	121.54	1377.09 <sup>a***</sup>	1146.26 <sup>a</sup>	317.47	526.48
4	125.78	153.96	129.81	118.87	251.87 <sup>b</sup>	243.16 <sup>b</sup>	330.91	327.88
8	159.27	161.14	78.81	78.51	185.10 <sup>b</sup>	211.12 <sup>b</sup>	105.63	93.30
24	118.10	108.59	82.51	79.27	109.64 <sup>b</sup>	119.95 <sup>b</sup>	55.32	90.35

\* Each value is the average of 3 muscles.

\*\* Non-stretched and stretched muscles under each treatment are paired muscles from the same broiler.

\*\*\* Values in the same column having different superscripts are significantly different ( $P \leq .01$ ).

Treatment Group Effects on Muscle Shear Values  
and Glycogen Content

Table VII shows the effects of ante-mortem treatments on shear values and glycogen contents of the broiler Pectoralis major muscles for all post-mortem time intervals combined. Statistical analyses (Tables IX and X in Appendix) showed that Group II (adrenaline injected birds) in Expt. 1 were significantly less tender ( $P \leq .01$ ) than the other two groups. The purpose of the adrenaline injection was to reduce muscle glycogen stores at the time of slaughter, thereby resulting in decreased post-mortem glycolysis. de Fremery and Pool (1963) and Khan and Nakamura (1970) reported that ante-mortem injections of epinephrine resulted in minimized post-mortem glycolysis, higher ultimate pH and increased tenderness of poultry meat. The reason for the conflicting results may be due to the fact that the L-form of adrenaline was used in the present experiments rather than the D-form which is more active. Glycogen stores may not have been sufficiently depleted to cause a significant increase in tenderness of the muscles used in this study. There was no significant difference in shear values between Group I (off-feed control) and Group III (alcohol) in Expt. 1.

In Expt. 2 shear values of muscles from birds given ante-mortem adrenaline injections were also significantly higher than those of the other treatment groups (Table VII). Group I (off-feed control) birds were significantly more tender than those in Groups II, III and IV. Shear values of Groups III (alcohol) and IV (full-feed

Table VII. Effects of ante-mortem treatments on shear values and glycogen contents of broiler Pectoralis major muscles\* for combined post-mortem time intervals

	Expt. 1		Expt. 2	
	Shear Values lbs.	Glycogen mcg/gm	Shear Values lbs.	Glycogen mcg/gm
Group I (Off-feed Control)	3.33 <sup>a**</sup>	80.16	3.25 <sup>a</sup>	150.63 <sup>a</sup>
Group II (Adrenaline)	4.31 <sup>b</sup>	66.77	5.88 <sup>b</sup>	101.41 <sup>a</sup>
Group III (Alcohol)	3.58 <sup>a</sup>	379.28	4.06 <sup>c</sup>	455.52 <sup>b</sup>
Group IV (Full-feed Control)	-- <sup>***</sup>	--	4.66 <sup>c</sup>	230.92 <sup>a</sup>

\* Each value is the average of 24 muscles (one pair of muscles from 12 different broilers).

\*\* Values in the same column having different superscripts are significantly different ( $P \leq .01$ ).

\*\*\* Group IV not included in design of Expt. 1.

control) were not significantly different from each other, but both were significantly tougher than Group I. The off-feed control group had an average shear value of 3.25 lbs. as compared to 4.66 lbs. for the full-feed control group. This may be partly explained by the fact that the full-feed control group was significantly heavier than the other three treatment groups in Expt. 2 (average dressed weight of 3.00 lbs. for full-feed controls as compared to 2.64 lbs. for the off-feed controls, 2.66 lbs. for the adrenaline group, and 2.76 lbs. for the alcohol group). Muscles of larger birds are sometimes thicker and thus have higher shear values than those of smaller birds. Analysis of variance and Duncan's multiple range tests for these data are presented in Tables XII and XIII in the Appendix.

Ante-mortem administration of alcohol significantly reduced struggling of the birds in both experiments immediately before and during slaughter. As would be expected from this reduced struggling, glycogen content of the Pectoralis major muscles was higher (significantly higher in Expt. 2) in those birds given alcohol prior to killing than in those birds in the other treatment groups. There were no significant differences in muscle glycogen content among the other three groups (Table VII). This lack of significant differences may be due to the large error term as shown in the statistical analyses of glycogen contents presented in Tables XI and XIV in the Appendix.

Effects of Post-mortem Aging on Muscle  
Shear Values and Glycogen Content

Table VIII presents the effects of post-mortem aging on broiler muscle shear values and glycogen content. For all treatments combined, shear values were highest (muscle toughest) at 1 hour post-mortem during rigor onset and gradually decreased during aging until 24 hours post-mortem at which time the muscles had become significantly more tender (shear values lowest) as a result of the resolution of rigor. Muscle glycogen content followed a pattern similar to the shear values during post-mortem aging. Glycogen content was highest at 1 hour post-mortem and gradually decreased as post-mortem glycolysis occurred in the muscles. In Expt. 1, for all treatment groups combined, muscle glycogen decreased from 431.34 mcg/gm at 1 hour post-mortem to 65.30 mcg/gm at 24 hours post-mortem. These same values for Expt. 2 were 498.62 mcg/gm and 95.47 mcg/gm. Again, the lack of significant differences in muscle glycogen content between post-mortem time intervals is the result of the large error terms as shown in the statistical analyses (Tables XI and XIV in Appendix).

This work demonstrates that glycolysis and tenderness of poultry meat are closely related. However, the mechanism of glycolysis and other biochemical reactions as they affect the onset of rigor in muscle is yet unknown. The question of what causes the resolution of rigor associated with post-mortem tenderization as a result of

Table VIII. Effects of post-mortem aging on shear values and glycogen contents of broiler Pectoralis major muscles\* of combined groups

post-mortem hrs	Expt. 1		Expt. 2	
	Shear Values lbs.	Glycogen mcg/gm	Shear Values lbs.	Glycogen mcg/gm
1	4.32 <sup>a**</sup>	431.34	4.98 <sup>a</sup>	498.62 <sup>a</sup>
4	4.06 <sup>a</sup>	143.69	4.64 <sup>a</sup>	210.28 <sup>b</sup>
8	3.37 <sup>b</sup>	61.29	4.59 <sup>a</sup>	134.11 <sup>b</sup>
24	3.21 <sup>b</sup>	65.30	3.65 <sup>b</sup>	95.47 <sup>b</sup>

\* Each value for Expt. 1 is average of 18 muscles (one pair of muscles from 9 different broilers); each value for Expt. 2 is average of 24 muscles (one pair of muscles from 12 different broilers).

\*\* Values in the same column having different superscripts are significantly different ( $P \leq .01$ ).

aging is yet unanswered. After the biochemistry of post-mortem muscle is completely understood, we can further deal with the practical aspects of presenting more tender and better quality meat and poultry products to the consumer.

## SUMMARY

This study was undertaken to determine the effects of pre- and post-mortem treatments on the glycogen content and tenderness of broiler Pectoralis major muscles. Pre-mortem treatments of the broilers included fasting, full-feeding, subcutaneous adrenaline injection, and oral administration of ethyl alcohol. The effect of stretch-tension during rigor on muscle tenderness was also studied.

Eighty-four nine-week-old broilers were divided into two experimental groups of 36 and 48 birds, respectively. Three treatment groups of twelve birds each were used in Experiment 1. Group I birds were fasted for 16 hours prior to slaughter. Group II birds were fasted for 24 hours, injected subcutaneously in the neck with 1 mg/kg body weight of adrenaline and then fasted an additional 16 hours prior to slaughter. Group III birds were fasted for 16 hours and then, approximately 30 min. before slaughter, were given 10 ml of 30% ethyl alcohol by pipette into the crop per kilogram of body weight. The Pectoralis major muscle from the right side of each carcass was removed immediately after evisceration and subjected to stretch-tension during the chilling operation. The left Pectoralis major muscle of each carcass was left intact in the carcass during the chilling process. At each of four post-mortem time intervals (1 hour, 4 hours, 8 hours, and 24 hours), the paired Pectoralis major muscles (one stretched and one non-stretched) from three carcasses were evaluated for shear values and glycogen content.

The procedure for Experiment 2 was the same as that of Experiment 1, except that an additional treatment group (Group IV) was added. Group IV birds were not fasted prior to slaughter but were kept on full-feed until a few minutes before slaughter.

The ante-mortem treatments of the broilers affected the post-mortem aging patterns of the Pectoralis major muscles. Muscles from all carcasses reached maximum toughness at either 1 hour or 4 hours post-mortem and gradually became more tender as aging proceeded. Muscle glycogen contents decreased with aging time. The degree of stretch-tension used in this experiment caused no significant differences in tenderness or glycogen content between stretched and non-stretched muscles.

In both experiments adrenaline injection caused reduced muscle glycogen stores but significantly tougher muscles. It was expected that adrenaline would result in more tender muscles as a result of reduced glycogen stores and minimized post-mortem glycolysis. Ante-mortem administration of alcohol significantly reduced struggling of the birds before and during slaughter. Glycogen contents of muscles were higher for birds given alcohol than for those birds in the other treatment groups. Shear values of muscles from alcohol-treated birds, were higher (less tender) than those from controls and lower (more tender) than those from adrenaline-treated birds. Muscles from full-feed control birds had higher glycogen content and significantly higher shear values than those from off-feed control birds.

This work demonstrates that glycolysis and tenderness of poultry meat are related. However, the mechanisms of glycolysis and other biochemical reactions as they affect the onset and resolution of rigor are still unanswered.

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APPENDIX

Table IX. Experiment 1. Analysis of variance of shear values of bivalve Pectoralis major muscles

Source of variation	df	ms	F
Treatment groups	2	6.2264	7.3442**
Hours post-mortem	3	5.1648	6.0920**
Sides (stretched, non-stretched)	1	.2913	-
T x H	6	3.5167	4.1480**
T x S	2	.4973	-
H x S	3	.0634	-
T x H x S	6	.1664	-
Error	48	.8478	
Total	71		
-----			
Hours within T <sub>1</sub>	3	5.6565	6.6720**
Hours within T <sub>2</sub>	3	.3099	-
Hours within T <sub>3</sub>	3	6.2318	7.3506**

\*\* P ≤ .01

Table X. Expt. 1. Duncan's multiple range tests\* on shear values of broiler Pectoralis major muscles

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<u>Treatment groups</u>			
Off-feed control	Alcohol	Adrenaline	
<u>3.3271</u>	<u>3.5838</u>	<u>4.3092</u>	
<u>Hours post-mortem</u>			
24 hrs	8 hrs	4 hrs	1 hr
<u>3.2067</u>	<u>3.3728</u>	<u>4.0561</u>	<u>4.3244</u>
<u>Hours within T<sub>1</sub> (off-feed control)</u>			
8 hrs	4 hrs	24 hrs	1 hr
<u>2.63</u>	<u>2.81</u>	<u>3.12</u>	<u>4.75</u>
<u>Hours within T<sub>3</sub> (alcohol)</u>			
24 hrs	8 hrs	1 hr	4 hrs
<u>2.53</u>	<u>3.12</u>	<u>3.78</u>	<u>4.91</u>

---

\* Means not underlined by the same line are significantly different at  $P \leq .05$ .

Table XI. Expt. 1. Analysis of variance of glycogen contents of broiler Pectoralis major muscles

Source of variation	df	ms	F
Treatment groups	2	749,254.9924	2.7988
Hours post-mortem	3	549,926.7394	2.0542
Sides (stretched, non-stretched)	1	12,280.5500	-
T x H	6	572,825.0506	2.1398
T x S	2	152,104.4340	-
H x S	3	85,098.6183	-
T x H x S	6	40,964.4391	-
Error	48	267,696.2703	
Total	71		

Table XII. Expt. 2. Analysis of variance of shear values of broiler Pectoralis major muscles

Source of variation	df	ms	F
Treatment groups	3	29.4970	26.98**
Hours post-mortem	3	7.7308	7.07**
Sides (stretched, non-stretched)	1	1.9694	1.80
T x H	9	2.2626	2.07*
T x S	3	.0614	-
H x S	3	.1713	-
T x H x S	9	.5947	-
Error	64	1.0932	
Total	95		
-----			
Hours within T <sub>1</sub>	3	.0269	-
Hours within T <sub>2</sub>	3	6.0089	5.4966**
Hours within T <sub>3</sub>	3	.3838	-
Hours within T <sub>4</sub>	3	8.0991	7.4086**

\*  $P \leq .05$ \*\*  $P \leq .01$

Table XIII. Expt. 2. Duncan's multiple range tests\* on shear values of broiler Pectoralis major muscles

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<u>Treatment groups</u>			
Off-feed control	Alcohol	Full-feed control	Adrenaline
<u>3.25</u>	<u>4.06</u>	<u>4.66</u>	<u>5.88</u>
<u>Hours post-mortem</u>			
24 hrs	8 hrs	4 hrs	1 hr
<u>3.65</u>	<u>4.59</u>	<u>4.64</u>	<u>4.98</u>
<u>Hours within T<sub>2</sub> (adrenaline)</u>			
24 hrs	8 hrs	4 hrs	1 hr
<u>4.72</u>	<u>5.72</u>	<u>5.94</u>	<u>7.16</u>
<u>Hours within T<sub>4</sub> (full-feed control)</u>			
24 hrs	4 hrs	1 hr	8 hrs
<u>2.94</u>	<u>4.96</u>	<u>5.29</u>	<u>5.44</u>

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\* Means not underlined by the same line are significantly different at  $P \leq .05$ .

Table XIV. Expt. 2. Analysis of variance of glycogen contents of broiler Pectoralis major muscles

Source of variation	df	ms	F
Treatment groups	3	588,893.1297	4.69**
Hours post-mortem	3	798,055.4079	6.36**
Sides (stretched, non-stretched)	1	17.0606	-
T x H	9	379,935.5462	3.03**
T x S	3	11,721.9405	-
H x S	3	358.7183	-
T x H x S	9	12,728.9144	-
Error	64	125,558.9369	
Total	95		
-----			
Hours within T <sub>1</sub>	3	6,155.1888	-
Hours within T <sub>2</sub>	3	3,757.1323	-
Hours within T <sub>3</sub>	3	1,751,012.7376	13.9457**
Hours within T <sub>4</sub>	3	176,936.9879	1.4092

\*\* P ≤ .01

Table XV. Expt. 2. Duncan's multiple range tests\* on glycogen contents of broiler Pectoralis major muscles

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<u>Treatment groups</u>			
Adrenaline	Off-feed control	Full-feed control	Alcohol
<u>101.41</u>	<u>150.63</u>	<u>230.92</u>	<u>455.52</u>

<u>Hours post-mortem</u>			
24 hrs	8 hrs	4 hrs	1 hr
<u>95.47</u>	<u>134.11</u>	<u>210.28</u>	<u>498.62</u>

<u>Hours within T<sub>3</sub> (alcohol)</u>			
24 hrs	8 hrs	4 hrs	1 hr
<u>114.79</u>	<u>198.11</u>	<u>247.51</u>	<u>1261.68</u>

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\* Means not underlined by the same line are significantly different at  $P \leq .05$ .

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THE INFLUENCE OF PRE- AND POST-MORTEM TREATMENTS  
ON GLYCOGEN CONTENT AND TENDERNESS OF POULTRY MUSCLES

by

Bonnie Sue Emswiler

(ABSTRACT)

The effects of pre- and post-mortem treatments on the glycogen content and tenderness of broiler Pectoralis major muscles were investigated. Pre-mortem treatments of the broilers included fasting, full-feeding, subcutaneous adrenaline injection, and oral administration of ethyl alcohol. The effect of stretch-tension during rigor on muscle tenderness was also studied.

Ante- and post-mortem treatments of the broilers had significant effects upon the rigor patterns, glycogen contents, and tenderness of the poultry meat. A discussion of the biochemistry of pre- and post-mortem muscle is included.