AN ABSTRACT OF THE THESIS OF

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Title: Comparative Electrophoretic Analysis of Plasma Protein Fractions From Deep Pectoral Myopathic, Hereditary Muscular Dystrophic, and a Normal Line of Turkeys

Abstract Approved:

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Two experiments were conducted to try and differentiate variations in the levels of total plasma protein and plasma protein fractions from three lines of turkeys, each with distinct muscle morphological characteristics--Deep Pectoral Myopathy (DPM), Hereditary Muscular Dystrophy (MDY), and a Broad Breasted Bronze control (J).

Ten day old poults from each of the three lines were grown in similar environments. Individual blood plasma samples were collected, body weights and breast width measurements determined at 8, 12 and 16 weeks of age (WOA) (Experiment 1), and at 20, 30, 40 and 50 WOA (Experiment

2).

Total plasma protein concentrations of both myopathic lines (combined sexes) were greater ($P<0.05$) than that of the control line at 12 WOA. DPM birds continued to exhibit higher ($P<0.05$) total protein levels than the control line through 16 WOA. At 20 and 40 WOA, DPM females had higher ($P<0.05$) total protein levels than the control females.

A total of 6 plasma protein fractions were commonly observed in the electrophoretograms of the three lines of turkeys. The highest levels of pre-albumin and albumin were encountered in both the DPM and MDY lines at 12 WOA, with DPM birds maintaining higher levels ($P<0.05$) of albumin also at 16 WOA. No consistent trends were observed in pre-albumin and albumin levels of males and females from the three lines from 20 to 50 WOA; however, significant differences ($P<0.05$) between sexes were observed in the two fractions when compared within each line.

DPM and MDY groups exhibited higher levels ($P<0.05$) of alpha-1 and alpha-2 globulins than the control values at 12 WOA, with MDY birds having greater levels ($P<0.05$) of beta and gamma globulins at 16 WOA. When compared to females from the control group (J), DPM females had significantly greater levels of alpha-2 and gamma globulins at 20 WOA, and higher levels ($P<0.05$) of alpha-2 and beta globulins at 40 WOA. At 40 and 50 WOA, DPM males

exhibited greater levels ($P<0.05$) of alpha-2 globulin when compared to that of the control males. Comparisons between sexes within each of the three lines showed DPM and MDY females to have significantly greater levels of alpha-1 globulin than that of the respective males at 20 and 40 WOA. At 50 WOA, DPM females exhibited lower levels of ($P<0.05$) alpha-2 globulin compared to DPM males, with both J and DPM females exhibiting significantly higher levels of beta globulin than those of males from the respective lines.

Body weight and breast width values of both myopathic lines were lower ($P<0.05$) than those of the control (J) line at 8, 12 and 16 WOA (Experiment 1). Both females and males from the myopathic lines exhibited significantly lower body weights and breast widths when compared to J females and males, respectively. Significant differences were observed consistently when sexes were compared within each of the lines, with males having greater breast widths and body weights at 20, 30, 40 and 50 WOA (Experiment 2).

Although variations were observed in both levels of total plasma protein and its fractions, no consistent differences were associated with either myopathic disorder. The results from this study indicate that plasma protein electrophoresis was not a suitable diagnostic technique for the two myopathic conditions.

Comparative Electrophoretic Analysis of Plasma Protein Fractions From Deep Pectoral Myopathic, Hereditary Muscular Dystrophic, and a Normal Line of Turkeys

by

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DEDICATION

I wish to dedicate this thesis to my loving husband, Todd. Words are inadequate to describe the love, patience, support, and encouragement that he has given me during my years in graduate school.

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CONTRIBUTION OF AUTHORS

James A. Harper, Professor Emeritus at the Department of Poultry Science, Oregon State University, conducted some of the earliest research involving Deep Pectoral Myopathy and Hereditary Muscular Dystrophy of turkeys. Subsequent extensive genetic studies performed by Professor Harper and colleagues determined the inheritance of the two myopathic disorders. The information generated from Professor Harper's exhaustive research, and his development of the two myopathic lines, laid the foundation for further research. Without Professor Harper's pioneering research, this study would not have been possible.

Dr. E. Duane Lassen is an Associate Professor of Veterinary Medicine at the College of Veterinary Medicine, Oregon State University. Dr. Lassen conducted the necessary laboratory tests which provided the total plasma protein values for this study.

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CHAPTER I

INTRODUCTION

Two myopathic conditions, both of genetic origins, have been observed in domestic turkeys. Deep pectoral myopathy (DPM), initially reported by Dickinson et al. (1968), is characterized by spontaneous necrosis and degeneration of the supracoracoideus muscle. The disorder affects primarily older female turkeys (Harper et al., 1975); however, occurrence of DPM may range from as early as 12 weeks of age to as old as 156 weeks of age in males or females (Harper and Schmitz, 1979). Several investigators have hypothesized that genetic selection for larger breast musculature of the modern turkey has gradually impaired the vascular supply to the breast muscle, causing the myopathic condition (Jones et al., 1974; Harper et al., 1975; Siller and Wight, 1978; Martindale et al., 1979; Wight and Siller, 1980; Wight et al., 1981; Siller, 1980 and 1985).

Hereditary muscular dystrophy (MDY) was first reported by Harper and Parker (1964). Typical gross changes of the condition are extensive atrophy of the pectoral and alar muscles which occur usually between 8 and 16 weeks of age (Schmitz and Harper, 1975; Harper and Schmitz, 1979).

Clinically, hereditary muscular dystrophy of the turkey is characterized by the inability of the bird to right itself after falling or when physically placed in a supine position (Harper and Parker, 1964 and 1967).

Both myopathic conditions impose major economic problems to the poultry industry. Although hereditary muscular dystrophy is grossly apparent as early as 8 weeks of age, there is great difficulty in obtaining an early diagnosis of DPM in the live intact bird. Palpation of a focal depression, present under the skin of the superficial pectoral muscle, has been the traditional method for detecting DPM; however, by this time the condition is generally in an advanced state of atrophy (Harper et al., 1969 and 1975).

Characteristic alterations of blood protein levels are often indicative of the age, nutritional, reproductive and general health status of a species. Plasma/serum protein electrophoresis is frequently conducted to aid in the diagnoses of various pathological conditions; however, normal baseline values must be established prior to this.

Although variations have been reported in electrophoretic protein profiles of avian species, numerous studies involving chickens have provided ranges of normal protein fraction levels as well as physiological information (Brandt et al., 1951; Sturkie and Newman, 1951; McKinley et al., 1953; Lush, 1963; Patterson et al.,

1967; Glick, 1968; Harris and Sweeney, 1969; Sibley and Hendrickson, 1970; Torres-Medina et al., 1971; Butler and Bell, 1971; Harduf and Alumot, 1971; Wood et al., 1971; Griminger and Sturkie, 1976; Kaneko, 1980).

Information regarding normal blood protein values of domestic turkeys is less abundant than that of chickens. Serum protein fractions in normal turkeys have been reported by Clarkson (1966) and Bierer (1969). Variations in protein fractions of turkeys due to hormonal make-up (Ranaweera and Wise, 1981), strain and seasonal conditions (Lisano and Kennamer, 1977; Al-Heeti et al., 1985) have also been recorded.

Characteristic changes of protein fraction levels have been associated with various pathological conditions of poultry. A marked decrease in albumin levels, and an elevation in the alpha-globulin fractions were observed by Sanders et al. (1944) in the electrophoretic patterns of chickens afflicted with leucosis. Lynch and Stafseth (1954) reported a distinct similarity in the degree of Pullorum disease immunization to the relative percentage of serum gamma-globulin in turkeys. A reduction in albumin concentration, and increases in total protein and the globulin fractions were observed during periods of peak mortality of chickens afflicted with Marek's disease (Samadieh et al., 1969). Tureen et al. (1965) compared the plasma protein profiles of chickens manifesting hereditary

muscular dystrophy, and birds affected with nutritional muscular dystrophy. During the course of the study, the latter group of birds exhibited a significant reduction in albumin, accompanied by an elevation in beta-globulin. The electrophoretic profiles of the genetic muscular dystrophic birds appeared normal; however, a significant reduction in total protein concentration was observed.

Therefore, with the significance of Deep Pectoral Myopathy and Hereditary Muscular Dystrophy to the poultry industry, and the clinical value of protein electrophoresis, this study was conducted. The objectives of the study were to compare the protein electrophoretic profiles of the two myopathic lines to a control line at specific age periods, and to determine if any alterations in certain protein fractions might provide a means for early detection as well as any physiological information associated with the two myopathic conditions. The influence of sex upon the protein profiles was also evaluated.

CHAPTER II

REVIEW OF LITERATURE

A. DEEP PECTORAL MYOPATHY

1. Background and Gross Pathological Description

A degenerative myopathic condition, resulting in an increased number of downgrades and/or condemnations of spent breeder turkeys, was first detected in 1967 by an Oregon poultry processing plant (Harper et al., 1975). During the period of 1967-1969, American processing plants were not exclusive in their reports of an increased number of downgraded turkey breeder hens affected with the defect. Processing plants of Western Canada and the United Kingdom also experienced economic losses as a result of the myopathic condition (Jones, 1974).

Deep pectoral myopathy (DPM), also referred to as degenerative myopathy, green muscle disease, and Oregon disease, was initially reported by Dickinson et al. (1968). These same investigators described the condition's occurrence as either unilateral or bilateral, and generally confined to the caudal two-thirds of the supracoracoideus muscle (also referred to as the deep pectoral muscle or Pectoralis profundus). Sequentially, the degenerative process is

characterized by the thickening of the superficial fascia overlying the supracoracoid muscle. At early stages of the condition, the affected area may become a red hemorrhagic color which later transforms into a encapsulated green lesion. Liquefaction and absorption may also follow. At advanced stages of atrophy, the necrotic muscle is replaced with dense fibrotic connective tissue or with adipose tissue (Dickinson et al., 1968; Harper et al., 1975). The presence of a focal depression in the skin or overlying the superficial pectoral muscle often occurs in advanced stages of atrophy, and palpation of this indentation can confirm the presence of DPM (Harper et al., 1969 and 1975). Although the extent of damage to the supracoracoideus varies, Harper et al. (1975) described advanced stages of atrophy to be characterized by the green lesion appearing dehydrated and "wood-like" in texture. The encompassing muscle tissue was observed to be paler in color at later stages of the disorder.

Degenerative myopathy, initially believed to be a unique entity of turkeys, was reported in adult meat-type chickens (Harper et al., 1971). As with the degenerative myopathic condition observed in turkeys, one or both sides of the breast muscle may be affected in broiler breeders; however, Siller (1980) reported 39% of the DPM cases to occur bilaterally in broilers. In contrast, a 4-year summary on symmetry in DPM-affected breeder turkey hens revealed damage in only the left muscle (29.3%), in only the right muscle

(29.5%) and 41.2% on both sides (Harper et al., 1975). Page and Fletcher (1975) described a myopathic condition affecting the supracoracoid and the ambiens muscles of 27-32 week old broiler breeder hens. Although feed analysis of the ration fed to the breeders indicated adequate levels of selenium and vitamin E, a clinical response was observed following oral supplementation of the two nutrients; implying a possible nutritional relationship. Richardson (1980) reported a myopathy in 7 week old broiler chickens, similar to the DPM found in turkeys. The green necrotic lesions were observed primarily in middle third portion of the supracoracoid muscle, unilaterally or bilaterally. An "acute pectoral myopathy" was reported in broiler breeders between 12 and 24 weeks of age (Randall, 1982). This myopathic condition affected primarily the superficial pectoral muscle, with lesions characterized by extensive myodegeneration.

In the early developmental stages of DPM, the condition is clinically asymptomatic. In advanced stages of the abnormality, turkeys with considerable bilateral muscular damage have been reported to exhibit extreme difficulty in righting themselves when placed in a supine position (Harper et al., 1975). This same phenomenon was described in chickens (Asmundson and Julian, 1956), and in turkeys afflicted with hereditary muscular dystrophy (Harper and Parker, 1967).

2. Hereditary Characteristics and Incidence

Extensive studies conducted by Harper and his colleagues at the Oregon Agricultural Experiment Station (OAES) provided fundamental knowledge regarding the genetic origin of DPM. Harper et al. (1969, 1971) first suggested a heritable basis for the expression of DPM after the defect was detected in progeny from parents exhibiting the anomaly. The myopathic condition has been reported to occur in several strains of turkeys; however, data provided by an anonymous Oregon processing plant (over a period of 4 years) revealed a distinctly higher incidence of DPM in white turkeys compared to a strain of Broad Breasted Bronze (Harper et al., 1975).

A line of Medium White turkeys selected for the predisposition to DPM resulted from the combination and close inbreeding of two Medium White lines of turkeys in 1970 at the OAES. The defect was selected for over a period of seven years by Harper et al. (1975). These same investigators reported DPM to be quite variable in its penetrance and expressivity, and concluded that the disorder is a polygenic trait involving modifiers. These genetic observations led to the hypothesis that selection for rapid growth and greater musculature in the modern turkey may have altered the vascular network in the muscle such that the blood flow to the supracoracoid muscle may be adversely affected. Other reports have contributed evidence that genetic selection for

larger body size and wider breast width in poultry is a major factor that causes the occurrence of DPM (Page and Fletcher, 1975; Henrichs et al., 1979; Siller, 1985).

The incidence of DPM in turkeys and in broiler breeders is extremely variable. Time of DPM expression in turkeys, determined by breast palpation, is reported to range from 12 to 156 weeks of age, with a mean age of 48 weeks (Harper and Schmitz, 1979). Incidence levels of 10% have been reported in spent turkey breeders and broilers, although an incidence level as high as 42% was observed in groups of broiler breeders (Wight and Siller, 1980). Following 11 years of intense genetic selection for DPM in turkeys, the incidence of DPM increased from 0% to 80% in males and from 2.5% to 90% in females (Harper et al., 1983). Grunder et al. (1984) conducted similar studies regarding the incidence of DPM in broiler breeders. Their study involved 20 strains of commercial parental breeding stocks, and three experimental strains (developed at the Animal Research Center in Ottawa, Canada) resembling the smaller commercial broilers of 20 years ago. The incidence of DPM in the commercial strains varied from 0% to 22% in females and from 0% to 43% among males. None of the slower growing experimental strains exhibited the myopathic condition.

3. Histopathology

Initial histopathological examinations of the supracoracoideus from DPM turkeys revealed hyperplasia of the intimal linings of several arteries, the presence of thrombi in some of the larger arteries, and the appearance of possible muscle fiber lysis (Dickinson et al., 1968; Harper et al., 1969). These observations, particularly the thickening of the arterial linings, led to the hypothesis that ischemia or an insufficient vascular supply caused the muscle degeneration (Harper et al., 1971, 1975).

Jones et al. (1974) compared muscle tissue from DPM-affected and normal turkey breeder hens. Immediately following post-mortem examination, a pH change from 7.4 to 7.8 was measured within the "brittle" green lesion, as opposed to a pH of 6.6 determined in the same muscle location of the control birds. The pH of the green tissue remained relatively high, while the pH of the normal tissue steadily decreased as the post-mortem time increased. A high concentration of albumin was also measured within the DPM lesion. In this same study, histological and electron microscopic examinations of the affected muscle tissue revealed abnormal curvature of the muscle fibers, granular and ill-defined A-bands, with extensive degradation of the I-band and Z-line. The ultrastructure of the muscle fiber showed considerable disruption of the sarcoplasmic reticulum,

and extensive mitochondrial deterioration. These findings prompted the same investigators to conclude that the muscular damage resulted from an impaired blood supply to the supracoracoid muscle.

Micro-opaque perfusion angiography of the pectoral muscles of DPM-affected turkeys has revealed extreme variation in patterns of vasculature. Radiography showed the regions of ischemia to correspond to the "green" infarcted lesions (Henrichs et al., 1979; Martindale et al., 1979).

Upon light microscopic examination of the affected tissue, Siller and Wight (1978) reported that there was no evidence of inflammation; however, the muscle fibers were swollen and tranversely split into disc shapes, a characteristic feature which is indicative of ischemia. Ultrastructurally, the muscle fibers exhibited deterioration of the actin filaments, degradation of the Z-lines, loss of glycogen, and sparse numbers of pale nuclei (Siller and Wight, 1978; Wight and Siller, 1980; Wight et al., 1981). Siller (1980) reported pale atrophic muscle fibers, caudal to the necrotic lesion, to be characteristic of a loss in nerve supply. On the basis of Siller's findings, he hypothesized that the necrotic lesion resulted from "a circulatory failure", while the caudal region atrophied due to an interruption of nerve impulse transmission.

4. Etiology and Pathogenesis

Initial attempts to determine the etiology of DPM were inconclusive. Failure to isolate bacteria or viruses from affected muscle tissue negated the possibility of infectious causative agents (Dickinson et al., 1968; Harper and Helfer, 1972).

Several myopathies that occur in poultry have been associated with specific nutrients. Vitamin E, selenium, and sulfur amino acids were reported to be contributing factors to the incidence of nutritional muscular dystrophy (Walter and Jensen, 1964; Scott 1966) and genetic muscular dystrophy in chickens (Tureen et al., 1965). Harper and Helfer (1972) examined the possibility of a nutritional relationship to the expression of DPM in turkeys. Day old poults from a Medium White DPM line were divided equally into two separate groups. One group was fed a ration supplemented with vitamin E (44 I.U./kg.), sodium selenate (1 ppm) and DL-methionine (.10%), and the other group fed the same ration except without supplementation. Both rations were fed to the birds through 52 weeks of age. There was no difference in the incidence of DPM between the two treatments. This result suggested that the three nutrients were not related to the expression of the abnormality. A higher incidence of DPM was reported among turkey flocks in Western Canada compared to Eastern Canada. Differences in Western Canada and Eastern Canada ration

formulations motivated Grunder et al. (1979) to determine if the expression of DPM was influenced by nutritional factors. Corn (primary feedstuff of Eastern Canada), and wheat (primary feedstuff of Western Canada) -based rations were fed to groups of turkeys. Birds were necropsied on six different dates ranging from 28 to 118 weeks of age. Incidence of DPM was reported to be 0% among 125 males, and 7.9% among 139 females. No significant difference occurred between birds fed either the corn or wheat based diets.

Investigations to determine if DPM resulted from ischemia have been conducted. Orr and Riddell (1977) were unsuccessful in their attempts to produce infarction in the supracoracoid muscle of turkeys by ligating selected cranial and pectoral arteries and veins. Lesions resembling those of DPM were experimentally produced by unilateral surgical occlusion of the subclavian artery in a strain of light-weight laying hens (Siller et al., 1978). The same investigators (Wight et al., 1979) also reported that mild electrical stimulation, applied either directly to the supracoracoid muscle or to the nerve supplying it, would induce the myopathic condition in heavy meat-type chickens. Utilizing angiographic techniques, Martindale et al. (1979) demonstrated that the deep pectoral muscle remained in an ischemic state for a duration of one hour post-stimulation, with the middle third of this muscle remaining ischemic for 24 hours following stimulation.

Extended muscular activity has also been shown to induce

DPM. Siller et al. (1979) reported that 25 to 40 seconds of induced forced wing flapping was sufficient to initiate the myopathic process in both broiler-type chickens and turkeys. Forced wing flapping (forced wing exercise) is accomplished by holding the bird by the shanks and tipping it slightly backwards inducing the bird to attempt to fly back into a position of normal equilibrium. Martindale et al. (1979) reported the supracoracoid muscle mass of turkeys, broilers and layers increased in weight by approximately 20% during muscle activity. Following muscle exertion, incompartmental pressure (IC) of the supracoracoideus was reported to increase by greater than 20% in heavy meat-type birds as compared to lighter birds (Martindale et al., 1979; Siller et al., 1979). This rise in IC pressure obstructs blood flow to the muscle tissue resulting in the development of necrosis. Several researchers (Orr and Riddell, 1977; Henrichs et al., 1979; Siller et al., 1979; Wight et al., 1981) have compared DPM in turkeys and chickens to anterior tibial syndrome or "march gangrene" that Carter et al. (1949) described in humans. The affected muscles of both disorders, human and avian, are enclosed in rigid osteofascial compartments that restrict the amount of space available for expansion of the contracted muscle; hence, an increase in subfacial pressure culminating in ischemic necrosis. The routine procedure for relieving the IC pressure "build-up" in march gangrene is by surgical fasciotomy (Blandy and Fuller, 1957). Likewise,

Siller et al. (1979) reported that DPM lesions could be prevented by excising the fascia of the deep pectoral muscle prior to forced wing exercise.

Harper et al. (1983) reported that the incidence of DPM expression at 20 weeks of age in a line of turkeys subjected to forced wing exercise was similar to the incidence in unexercised DPM birds at 72 weeks of age. The DPM incidence in exercised females ranged from 30% to 50% as compared to 5% observed in the unexercised controls. Forced wing exercise did not induce the myopathic condition in lines and crosses which had no previous history of DPM. From these observations, Harper and colleagues suggested that forced wing exercise had the potential as a screening method for producing DPM lesions in birds at pre-breeding ages.

Studies have been conducted to determine if normal poultry husbandry practices such as weighing, vaccination and artificial insemination could cause sufficient stress to induce the disorder (Wight et al., 1979). Three groups of female turkeys, of varying ages, were fed identical rations but maintained in different controlled environments. Birds maintained under normal commercial conditions had the highest incidence of DPM (16.2%), followed by those birds maintained in cages (8.1%), with the lowest incidence of DPM occurring in birds maintained in extremely "quiet" and minimal stress-controlled pens (0.68%). Richardson et al. (1980) reported a 5% incidence of DPM in 7 week old broiler chickens one week after being subjected to handling stress.

5. Diagnostic Techniques

The difficulty of obtaining an early diagnosis of DPM in the live intact bird presents a major economic problem to the poultry industry, and to the scientists studying the pathogenesis of the disorder. Palpation of the focal depression, present under the skin of the superficial pectoral muscle, is a successful means for DPM detection; however, by this time the condition is generally in an advanced state of atrophy (Harper et al., 1969 and 1975). According to Hollands et al. (1978 and 1980), live bird diagnosis of DPM based upon breast palpation is unreliable due to variation in the degree of atrophy. A detection procedure used by Canadian processing plants was to incise or "slash" the breast muscles of all birds originating from any DPM-suspicious flocks (Jones et al., 1977). The obvious drawback to this method is the carcass downgrading of normal birds due to breast slashing. Harper et al. (1983) reported a 1% error in the accuracy of DPM diagnosis when palpation methods were used as opposed to breast slashing. The United States and the United Kingdom have used visual inspection as a means of DPM detection, based upon breast asymmetry due to the atrophic state (Jones et al., 1977). There are several major disadvantages to the detection methods described. Those methods that diagnose DPM in the live bird are not always consistent. The disorder can occur bilaterally, causing the

breast shape to appear symmetric. Palpation is only effective in advanced stages of atrophy.

Development of less destructive and non-invasive detection methods have been employed to reduce the economic losses from carcass downgrading. Jones et al. (1977) developed a light probe which could be inserted into the carcass cavity for the detection of muscle lesions. Siller and Wight (1978) used spectrophotometry as a tool to compare normal muscle with green DPM lesions. Absorbancy differences between normal muscle and green lesions were reported to be at 430 and 590 nm, respectively. Swatland and Lutte (1984) reported that the combination of the two optical methods was a relatively effective and non-destructive approach for detecting DPM in turkey carcasses. The investigators proposed the use of a long fiber optic probe, trifurcated such that one branch carries white light into the muscle and two branches for measuring reflected light at 480 and 550 nm. The ratio of the two wavelengths (480/550 nm), determined by a microprocessor, was suggested to be a simple diagnostic test. The mean ratio for normal muscle was reported to be 0.68. The average ratios for the green lesions ranged from 1.17 to 1.38.

Late expression of deep pectoral myopathy in genetically susceptible breeding populations prevents the breeder from eliminating those carriers from the breeding flocks at an early age. Attempts have been made to identify genetic carriers of DPM by examining various blood chemistries for

quantitative changes that may occur as the condition progresses. The enzyme creatine phosphokinase (CPK or CK) is a major component of cardiac and striated muscle cells. Stress, physical exercise, trauma, bruising and other adverse conditions to the muscle cause a transient elevation of CPK in the blood due to its release from damaged muscle cells. Therefore, measurement of CPK activity in the blood has proven to be an effective clinical procedure for the diagnosis of muscular disorders in humans and animals (Richterich, 1963; Holliday et al., 1965; Farrell et al., 1966; Wagner et al., 1971; Hollands et al., 1980; Tripp and Schmitz, 1982). Hollands et al. (1980) reported that measurement of plasma CPK activity levels could be an effective method for the detection of DPM in the live turkey. Thompson-Cowley (1981 and 1982) investigated the effect of exercise on CPK levels and clearance times in relation to the onset of deep pectoral myopathy in turkeys. Plasma activity levels of the enzyme were found to be similar to those levels observed in chickens. Hens kept in a low-stress environment exhibited consistently lower average CPK levels than did a normal medium white cross hen. Peak values of plasma CPK were observed in both DPM and control lines to occur between 24 and 48 hours post-exercise stress. Higher peak levels and longer clearance time of CPK were observed in hens from DPM lines following strenuous exercise-stress. Determination of other hemophysiological factors such as blood pressure,

plasma lipoprotein levels, and erythrocyte membrane fatty acids have been evaluated for possible relationships to the onset of DPM in turkeys (Thompson-Cowley, 1978 and 1981; Thompson-Cowley et al., 1981 and 1982). Normal males were found to have higher systolic and distolic pressures than the females of the same line and breed; however, lower pressures were measured in myopathic lines. Similar lipoprotein profiles of normal and DPM lines were observed when the sexes were compared with each other during the same time of the year; however, reproductive status was found to influence the levels. DPM-afflicted turkeys were reported to have higher erythrocyte lipid membrane weights than non-afflicted birds (of the same DPM line) at 17-19 weeks of age, and lower weights at 23 and 32-33 weeks old (Thompson-Cowley, 1981).

B. HEREDITARY MUSCULAR DYSTROPHY

1. Background and Gross Pathological Description

A hereditary muscular dystrophic condition in two Broad Breasted Bronze turkey females was initially observed in 1960 at the Oregon Agricultural Experiment Station (Harper and Parker, 1967). The first formal report of the condition was in turkeys by Harper and Parker (1964). Asmundson and Julian (1956) described an inherited muscular dystrophy of a strain

of New Hampshire chickens. Subsequent studies have been reported of hereditary muscular dystrophic conditions in other avian species such as the Pekin duck (Rigdon, 1966), and Cornish chicken (Wagner and Peterson, 1970). Sutherland (1974) inaccurately regarded hereditary muscular dystrophy and deep pectoral myopathy as the same disorder. Whereas, hereditary muscular dystrophy of turkeys affects more than one muscle (pectoral and alar muscles), deep pectoral myopathy involves spontaneous necrosis and degeneration of only the supracoracoid muscle (Harper et al., 1969 and 1975).

Although the muscular dystrophies of both the turkey and the chicken resemble each other, there are several distinct and noteworthy differences between the two disorders. Clinically, both abnormalities, the turkey (Harper and Parker, 1964 and 1967) and the chicken (Asmundson and Julian, 1956; Julian and Asmundson, 1963) are characterized by the inability of the bird to right itself after falling or when physically placed in a supine position. Typical gross changes are extensive atrophy of the pectoral and alar muscles which occur between 8 and 16 weeks of age in turkeys (Schmitz and Harper, 1975; Harper and Schmitz, 1979), and in chickens aged between one and 12 weeks old (Julian and Asmundson, 1963; Julian, 1973). Only the "white" muscles of the dystrophic turkey are affected (Schmitz and Harper, 1975). This is not always the case in dystrophic chickens, as hypertrophy is occasionally observed in two "dark" muscles: the biceps

femoris and gastrocnemius (Julian and Asmundson, 1963). Unlike the dystrophic chicken, hypertrophy of the pectoral muscles in the turkey does not precede atrophy, and bone length and growth are normal (Harper and Parker, 1967; Schmitz and Harper, 1975; Harper and Schmitz, 1979). Asmundson and Julian (1956) reported dystrophic New Hampshire chickens to have shorter bones, and wider breast widths than normal birds of the same breed. Harper and Parker (1964 and 1967) reported the mean body weights and breast widths of dystrophic and normal Broad Breasted Bronze turkeys to be very similar at 8 weeks of age. However, 23 week old dystrophic turkeys exhibited breast widths 60% narrower than the controls. The pectoral muscles of dystrophic turkeys were reported to weigh 40 to 65% less than normal (Harper and Parker, 1967; Harper and Schmitz, 1979). In contrast, the pectoral muscles of dystrophic chickens are 40% to 60% greater than normal birds (Julian and Asmundson, 1963). Harper and Parker (1964 and 1967) reported that "fertility, hatchability, livability and mobility" are not adversely affected in dystrophic turkeys. The atrophic muscle of both the turkey (Harper and Parker, 1967), and the chicken (Julian and Asmundson, 1963) lack normal tonal qualities. Harper and Parker (1967) reported atrophy of the pectoral muscles ceases following the onset of sexual maturity in dystrophic turkeys.

Both dystrophic conditions of turkeys (Harper and Schmitz, 1979) and chicken (Julian and Asmundson, 1963;

Julian, 1973; Mrak, 1985), are often compared with human muscular dystrophy, as each share certain similar features. Furthermore, both avian dystrophies are useful animal models for the human disorder.

2. Hereditary Characteristics

Genetic studies were conducted to determine the inheritance of the muscular dystrophic disorder (Harper and Parker, 1967). In 1960, two dystrophic hens (Broad Breasted Bronze) were mated to a normal Broad Breasted Bronze male of a different strain. Subsequent matings of normal non-dystrophic turkeys, normal heterozygotes, and dystrophic turkeys were performed the following year. Thereafter, the dystrophic turkeys were only mated within the abnormal line. No attempts were made to select for larger or smaller muscle size. Harper and Parker (1967) concluded that an autosomal recessive gene was responsible for the mutation found in turkeys, and designated the gene "dy". Likewise, Asmundson and Julian (1956) earlier reported that hereditary muscular dystrophy of chickens resulted from the expression of an autosomal recessive gene.

3. Histopathology

Schmitz and Harper (1975) examined the histopathology of

dystrophic muscles from turkeys at selected ages. Whereas early stages of chicken dystrophy are not pronounced (Julian and Asmundson, 1963), acute histopathological changes as early as 10 weeks of age in dystrophic turkeys have been observed. Turkey muscle fibers varied in size and morphology, accompanied by severe swelling and necrosis of individual fibers. Lesions of the 16 and 24 week old homozygous dystrophic turkeys were reported to be similar to those observed in 10 week old turkeys. A characteristic feature that was observed in 120 week old dystrophic turkeys, and to a lesser extent in birds between 24 and 37 weeks, was the replacement of endomysial sheaths and adipose tissue. In contrast to fat deposition occurring at the more advanced stages in the dystrophic turkeys, dystrophic chicken muscle at all ages may exhibit the distinctive features of endomysial proliferation and fat deposition (McMurtry et al., 1973). Vacuolization and phagocytosis of the pectoral muscle fibers, observed in dystrophic chickens as young as 6 weeks of age (McMurtry et al., 1972) were not prominent features in the early stages of turkey dystrophy (Schmitz and Harper, 1975). Microscopically, the muscle fibers of dystrophic turkey pectoral muscles were reported to have significantly fewer numbers of nuclei as compared to the control birds (Schmitz and Harper, 1975), whereas proliferation of nuclei was observed in the muscle fibers of both dystrophic and normal chickens (Julian and Asmundson, 1963; McMurtry et al.,

1972). In dystrophic turkeys, the diameters of muscle fibers from the pectoral muscle were reported to be significantly smaller than normal (Schmitz and Harper, 1975). In contrast, McMurtry and Dreumel (1972) reported muscle fiber diameters of dystrophic chickens to be substantially greater than the control birds. Ring fibers ("Ringbinden") were present in dystrophic chicken muscle (Julian and Asmundson, 1963), but absent from dystrophic turkey muscle (Schmitz and Harper, 1975).

4. Etiology and Pathogenesis

The etiology of muscular dystrophy of turkeys, as that of chickens, man and many other animals, is unknown. Biochemical, anatomical and physiological investigations have contributed to theories regarding the pathogenic nature of the defect in chickens, and to a much lesser extent, in turkeys.

Tureen et al. (1965) compared the plasma protein profiles of chickens afflicted with nutritional (antioxidant deficiency), and genetic muscular dystrophy. Nutritional muscular dystrophic birds exhibited characteristic changes in specific protein fractions. The genetic dystrophic strain had a significant reduction in total protein concentration; however, levels of protein fractions were normal. Various plasma enzymes, such as aldolase (Asmundson and Julian, 1956;

Julian and Asmundson, 1963) and glutamic oxaloacetic transaminase (Cornelius et al., 1959; Walter and Jensen, 1964) have been shown to be present at elevated activity levels in genetic dystrophic chickens. Plasma creatine phosphokinase (CPK) activity of 27-35 day old dystrophic chickens was reported to be significantly higher than that of control birds, and continued to rise up until 131-134 days of age (Holliday et al., 1965). At 10 to 16 months of age, CPK activity was observed to be lower than at 131-134 days of age; however, it was still greater than that of normal chickens. Farrell et al. (1966) compared plasma CPK changes in nutritional and hereditary muscular dystrophic chickens. Chickens afflicted with nutritional muscular dystrophy showed substantial increases in plasma CPK levels, with elevations occurring linearly between 12 and 18 weeks of age. Significant increases in plasma CPK activities were observed as early as 10 weeks of age in the genetic muscular dystrophic chickens peaking at 15 weeks of age on the average and decreasing thereafter. Tripp and Schmitz (1982) reported plasma CPK activities of normal and dystrophic turkeys to be extremely sensitive to physical exercise and stressful conditions. Plasma CPK levels of Broad Breasted Bronze dystrophic turkeys were significantly higher than those levels of Broad Breasted Bronze and Wrolstad normal turkeys (all at 10 weeks of age) exposed to minimal stress and exercise.

C. BLOOD PROTEINS

Analyses of blood proteins are frequently employed to determine the nutritional, reproductive, and general health of a species. Numerous assays of blood and its constituents are used routinely for the diagnosis of various pathological conditions; however, normal baseline values must be established prior to this.

1. Blood Protein Fractionation

Plasma or serum proteins may be fractionated by the technique of electrophoresis. The general principle of serum/plasma protein separation is based upon the fact that charged protein molecules will migrate when placed within an electric field. The rate and direction of migration is dependent upon the ionic charge and size of the protein molecule. Furthermore, the electrical current, pH, support medium and buffer are other factors which influence protein separation. In the 1930's, Tiselius carried out electrophoresis in a liquid medium. In 1933 he reported human blood serum to contain four major components: Albumin, alpha-globulin, beta-globulin and gamma-globulin (Griminger and Sturkie, 1976). Later, the use of a solid support medium was introduced by Konig in 1939 (Helena Laboratories, 1976), which utilized electrolyte saturated paper strips to

electrophoretically separate a yellow pigment originating from snake venom (Helena Laboratories, 1976). The name zone electrophoresis (as opposed to free) was used to refer to all electrophoretic methods in which a support medium was used (Kaneko, 1980).

Subsequent studies led to the use of a wide array of support media. Paper was used initially, later, agar gel, starch, cellulose acetate, and polyacrylamide gel were found to be useful materials. Certain media have been shown to provide superior resolution than others. This fact is illustrated by numerous electrophoretic studies involving avian species. Zone electrophoresis of sera from the adult male chicken, and the sexually immature pullet was reported to resolve six protein fractions: albumin; alpha-globulins 1, 2, and 3; and gamma-globulin (McKinely et al., 1953). In contrast, a comparative study of the development of the electrophoretic patterns of two hybrids of chicks resulted in the resolution of seven protein fractions, in order of decreasing relative mobilities: albumin, alpha-1, alpha-2, beta-1, beta-2, gamma-1, and gamma-2 globulins (Patterson et al., 1967). This investigation utilized cellulose acetate as the support medium. However, Bierer (1969) reported cellulose polyacetate to resolve nine protein fractions from normal turkey serum. Polyacrylamide gel, column and disc electrophoretic techniques were conducted by Harris and Sweeney (1969), who observed 7 to 10 protein fractions in

rooster serum. Torres-Medina et al. (1971) used serum from the chick in a comparative investigation of several electrophoretic methods. The media selected for the study were paper, cellulose acetate, and polyacrylamide gel which resolved 5, 8 and 16 protein fractions, respectively. Disc acrylamide gel electrophoresis of serum from a New Hampshire strain of chickens separated nineteen protein fractions (Glick, 1968). This medium was also utilized in an electrophoretic study involving serum and egg yolk from the laying hen (Harduf and Alumot, 1971). The study resulted in the resolution of 25 and 23 protein fractions from the serum and egg yolk, respectively.

2. Functions of Avian Plasma Proteins

In general, plasma proteins are characterized as carrier molecules for hormones, vitamins, immuno-components, and many additional vital elements.

Pre-albumins have been found to function as carrier proteins. Retinol is transported by a specific carrier protein which is bound to pre-albumin 2 in chicken plasma (Heller, 1976; Kopelman et al., 1976; Bhat and Cama, 1978, 1979). Approximately 15% of thyroxine is bound to a pre-albumin of chicken plasma, in contrast to the 75% bound to albumin (Bhat and Cama, 1978, 1979).

Albumin serves as a major protein storage reserve and

provides a source of proteins during subnormal intake (Griminger and Sturkie, 1976). Another important function of albumin is as a binder and transporter of amino acids, fatty acids, including essential vitamins and minerals. Butler (1971) concluded that the plasma albumin of the fowl is unique as it is a carrier of thyroxine (T4) and triiodothyronine (T3). Albumin accounts for about 75% of the osmotic activity of plasma due to its abundance and small size (Kaneko, 1980).

The alpha fraction is comprised of two major components; the alpha-1 (faster migrating) and alpha-2 (slower migrating) globulins. Several important proteins with specific functions migrate within each of the two alpha fractions. Transcortin, found in both chicken and mammalian plasma, is a carrier protein of the majority of circulating corticosterone (Butler, 1971). Haptoglobin (which binds hemaglobin) and copper-binding ceruloplasmin are two of the more prominent proteins which migrate in the alpha-2 region (Sibley and Hendrickson, 1970).

The beta-globulins follow the alpha-globulins and also migrate fast (beta-1) and slow (beta-2) fractions in most animal species excluding ruminants (Kaneko, 1980). Transferrin, also referred to as siderophilin, serves as a binding site for iron (Sibley and Hendrickson, 1970). Williams (1962b) demonstrated that the protein moeity of the transferrin molecule is identical to that of conalbumin

(found in egg white) and that the only difference between the two was in the prosthetic carbohydrate groups. Two beta-globulins, detected in fowl plasma, are responsible for the transportation of cholecalciferol and 25-hydroxycholecalciferol (Butler and Bell, 1971). In mammalian blood plasma; however, the two calciferols are transported by alpha-globulin or albumin. Another protein which slightly trails the beta-globulins is fibrinogen. Fibrinogen functions as an essential protein required for the blood clotting mechanism; the transformation of fibrinogen into the insoluble fibrin. It is also an important indicator of acute inflammatory disease (Kaneko, 1980).

As with the alpha and beta fractions,the gamma fraction is observed as two fractions, a gamma-1 (fast) and a gamma-2 (slow). Important proteins of this fraction are the immunoglobulins; IgA, IgM and IgE are observed usually in the gamma-1 region, with IgG found within the gamma-2 fraction (Kaneko, 1980). In avian species, the B lymphocytes of the Bursa of Fabricius respond to antigenic stimuli with the proliferation of plasma cells, which produce immunoglobin. The gamma-globulins are characterized as having the slowest electrophoretic mobilities due in part to their higher molecular weights and lower isoelectric points (at a pH of 8.0 or greater; Kaneko, 1980).

3. Factors Influencing Levels of Protein Components

Age, sex, breed, species, season, environment, reproductive and health status of the bird are just a few of the many factors that influence one or more of the protein fractions. In addition, the electrophoretic method used also contributes to the resolution of the various protein fractions.

The influence of age and sexual maturity on the serum proteins of the chicken have been the focus of several studies. Total protein concentration, alpha and gamma globulin levels have been reported to increase with the bird's maturity (Brandt et al., 1951; Amin, 1961; Tureen et al., 1966; Patterson et al., 1967). In a study conducted by Brandt et al. (1951), total serum protein levels for the 4-7 week old chick were observed to be 3.36 $\pm$ 0.25 g/dl, whereas levels of 5.40 $\pm$ 0.71 g/dl were reported in laying hen sera. Gamma-globulin levels of 0.41 g/dl and 1.78 g/dl were reported for the 4-7 week old chick and laying hens, respectively. Cockerels and pullets (both at 4 months of age) exhibited a marked similarity in total protein, alpha and gamma-globulin levels. Little differences in the levels of albumin and beta-globulin were thought by Brandt and colleagues to correspond to the age of the bird. In contrast, Morgan and Glick (1972) measured total serum protein levels of 2.68 g/dl for the day old chick and 4.63 g/dl for 12 week

old birds.

Tureen et al. (1966) identified twelve distinct protein fractions in chickens between 1 to 210 days of age. Distinct pre-albumin components were reported to diminish after 12 days of age, and were not observed after 20 days of age. Additional gamma-globulin fractions have been detected in birds older than 12 days of age.

Specific embryonic protein fractions have also been observed to be present at various stages of development but are absent in the serum of mature chickens and in the undeveloped egg. Carcini and Grossi (1973) first detected a specific serum protein component present on the 18th day of embryonic development. The presence of an "embryonic" or second albumin, of a greater mobility, was observed in an experiment conducted by Siegel and Gould (1976). The investigators also reported a significantly higher plasma protein concentration during the first four to six days of embryonic development. Weller (1976) reviewed a number of investigations regarding the ontogeny of chicken serum protein components. The following generalizations were made based upon the review: 1) During the early stages of embryonic development, the protein profile is extremely simple; 2) As development continues, complexity arises as the number of protein components increases; 3) Complexity declines post-hatching and continues to do so as the chicken matures (Sanders and Kline, 1977).

The influence of sex and reproductive status upon the bird's blood protein profile has been the objective of numerous studies. Sturkie and Newman (1951) examined the effects of laying, ovulation, blood plasma volume and number of blood samples collected upon the plasma protein components of the laying hen. Blood samples were collected on the days which the hens: a) laid egg(s); b) ovulated; c) laid eggs and ovulated; d) neither laid eggs nor ovulated. The results of Sturkie's study indicated that the time of ovulation, time of egg laying, and position of the egg in the oviduct had no significant effect upon levels of plasma albumin (A) or globulins (G). Data indicated that the time and frequency of sampling, time of ovulation or oviposition, and changes in blood volume did not significantly effect the albumin/globulin (A/G) ratios of the laying hens. Differences in protein fraction levels have been reported to occur between males and females. Levels of plasma proteins from roosters were observed to be significantly lower than the hens. Ranaweera and Wise (1981) reported androgens to decrease serum protein levels in turkeys while estrogens were shown to increase serum protein levels in chickens (Sturkie and Newman, 1951).

Wood et al. (1971) showed no significant differences to exist in the total serum protein concentrations of dwarf and non-dwarf White Leghorn and White Rock strains of chickens. Dwarf White Leghorn females were reported to have greater A/G

ratios compared to non-dwarf hens of the same breed. In contrast, no difference in A/G ratios was observed between the two White Rock strains.

Al-Heeti et al. (1985) reported total serum protein levels of three turkey strains ("Red", "Black", and "White") to be greatest under moderate seasonal conditions and lowest under higher temperatures at lower humidity. Total protein was also determined to be significantly ($P<.05$) influenced by sex. Albumin levels in this study were the highest in the Black and Red strains with a significant interaction existing between strain and season ($P<.05$). In addition, the strain was found to have a significant effect upon the alpha 1-globulin fraction, but not on the remaining globulins. Season was shown to cause significant variation ($P<.05$) in the globulin fractions with maximum levels occurring under moderate temperatures and relative humidity.

Polymorphism in 5 of 14 plasma or serum protein fractions was demonstrated in Brown Leghorn hens forced out of lay initially and then induced back into lay by manipulation of day length (Lush, 1963). Kristjannson et al. (1963) observed the presence of three pre-albumins (PaA, PaB, and PaC) in the serum of the laying hen. Minor to no differences were found between individual birds with respect to pre-albumins A and C; however, extreme differences in the quantity of pre-albumin B were noted between individuals. Studies of the variation in levels PaB revealed greatest concentrations to

occur during 4-10 hours post-ovulation, with the lowest concentration prior to or immediately following ovulation. Because PaB concentrations coincided with the first six hours when an egg was present in the uterus, the investigators suggested a possible relationship of the component with the early stages of egg shell formation. PaB was not observed in the sera of pullets, male chickens, and hens which were not producing eggs.

Harduf and Alumot (1971) described a distinct difference in specific serum protein fractions from the laying hen and the rooster. Bands, designated #4 and #11, were present in sera from laying hens only; bands #6 and #7 were absent from rooster serum, but present in egg yolk and serum from the laying hen. Band #27 (pre-albumin), absent from the serum of the hen, was reported to have the same electrophoretic mobility as free phosphovitin which had been previously isolated from egg yolk by Wallace et al. in 1966.

4. Taxonomic and Comparative Studies of Avian Proteins

The possibility of utilizing the technique of serum protein electrophoresis in the area of avian taxonomy was examined by Sibley and Johnsgard (1959). The electrophoretic patterns from twelve avian species were evaluated including several breeds within seven of the species. Data obtained from the Ring-necked pheasant (Phasianus colchicus), Domestic

Fowl (Gallus domesticus), Mallard (Anas platyrhynchos), Black Duck (Anas rubripes), Pintail (Anas acuta), Redhead Duck (Aythya americana) indicated that age, sex, and reproductive condition significantly influence the variation in serum protein levels. Later, Sibley and Hendrickson (1970) conducted a comparative electrophoretic study of the plasma proteins of 450 different species of birds. Although a common or "typical" pattern was observed among the different species, variation was frequently observed between individual birds. Much of the individual variation observed was suggested to be due to age, sex, season, health status, and polymorphism. On the basis of their observations, Sibley and Hendrickson (1970) concluded that one-dimensional electrophoretic patterns did not provide sufficient information regarding the relationship of higher avian taxonomic categories, but may have potential in the evaluation of lower category relationships.

Electrophoretic patterns of egg white proteins from 37 avian species revealed similarity in configuration between species considered to be closely related, taxonomically. However, in other cases, the patterns exhibited a more distant relationship than indicated on the taxonomic level. Although many of the electrophoretic patterns were similar, each species exhibited a distinct pattern of its own (McCabe and Deutsch, 1952).

Classification of avian hybrids based on traditional

methods positions the chicken, pheasant and quail in the Phasianidae family and the turkey in the Meleagridiadae family. Sarvella and Morris (1976) challenged this classification when they reported similar electrophoretic mobilities of serum albumins from the turkey, pheasant, and quail. The albumin from chicken serum, however, was observed to have a greater mobility. A mixture of different parental plasma samples (chicken x quail; chicken x Ring-necked pheasant; Ring-necked pheasant x quail; and turkey x Ring-necked pheasant) confirmed the mobility results. Based on these results, the investigators suggested that the turkey should be included within the Phasianidae family.

The pheasant (Phasianus colchicus) was determined to be closely related to the domestic fowl (Gallus domesticus) due to the occurrence of similar protein variants. Because of the similarity in protein variants, extrapolation from pedigree chickens to non-pedigree pheasants was permissible (Baker and Manwell, 1962; Ogden et al., 1962; Lush, 1963; Baker et al., 1966a,b).

The determination of biochemical structures of various protein components has provided useful information for comparative studies. Peters et al. (1958) identified a single aspartic acid residue to occupy the N-terminal position of serum albumin from the chicken, turkey and duck. Alanine was found to be present in the C-terminal position of chicken and duck albumin while a valine residue occupied this position in turkey albumin.

5. Polymorphisms of Avian Serum Proteins

Polymorphisms of serum components have been observed in numerous electrophoretic studies. As previously mentioned, three pre-albumin fractions have been observed in the sera of laying hens (Kristjannson *et al.*, 1963). Polymorphism of pheasant (Baker *et al.*, 1966a) and duck (Przytulski and Csuka, 1979) pre-albumins have also been reported.

Albumin polymorphisms have been reported to be present in several species. Earl (1959) and Gitlan *et al.* (1961) both observed a double albumin fraction in the sera from humans that was genetically controlled. The two albumins differed by a specific peptide.The serum/plasma of the domestic fowl has been reported to contain two genetic variants of albumin (McIndoe, 1962; Fried and Cun, 1971). Three albumin phenotypes were observed in sera from the Domestic turkey (*M. gallopavo*), the Ocellated turkey (*M. ocellata*) and in the descendants from a Domestic turkey x Ocellated turkey cross (Quinteros *et al.*, 1964). Other species such as the Muscovy and Peking ducks (Przytulski and Csuka, 1979), and the Golden pheasant (Baker, 1965) have been reported to exhibit albumin polymorphism.

Polymorphisms of other avian serum protein have been observed such as with the transferrins of the chicken (Ogden *et al.*, 1962) and duck (Przytulski and Csuka, 1979).

6. Protein Fractions and Avian Pathological Conditions

Characteristic changes of protein fraction levels are often associated with various pathological conditions of avian and mammalian systems. Sanders et al. (1944) described a new protein component, designated the "L" component, which represented 10% of the total serum protein from leukosis-affected chickens. Electrophoretic patterns where the L component was absent were found to exhibit high concentrations of gamma-globulin. The same investigators postulated the existence of an interrelationship between the new component and the gamma-globulin fraction. A marked decrease in albumin levels, and an elevation in the alpha-globulin fractions also were observed in the electrophoretic patterns of birds afflicted with leukosis.

Several investigations have associated the gamma-globulin fraction to the level of circulating antibodies. A distinct similarity was shown in the degree of Pullorum disease immunization to the relative percentage of serum gamma-globulin in turkeys (Lynch and Stafseth, 1954). In all instances where high agglutination titers existed, a high relative percentage of the gamma fraction was observed. As antibody titers decreased, there was also a reduction in the gamma-globulin levels.

Specific alterations in the concentrations of plasma proteins have been related to several nutritional

deficiencies and disorders. Serum protein profiles from chickens afflicted with exudative diathesis (Goldstein and Scott, 1959; Bieri and Pollard, 1959) and vitamin E deficient encephalomalacia (Tureen et al., 1964) illustrated a consistent elevation in the beta-globulin fraction, and a decline in the A/G ratio.

Tureen et al. (1965) compared the plasma protein profiles of chickens manifesting hereditary muscular dystrophy, and birds with a nutritional muscular dystrophy. A significant reduction in the albumin fraction and A/G ratio, accompanied by an elevation in the beta-globulin were typical changes observed during the course of nutritional muscular dystrophy birds. The genetic dystrophy strain of chickens failed to show such alterations and exhibited plasma protein profiles comparable to those of the controls. However, when the genetic dystrophy birds were fed an antioxidant and sulfur amino acid deficient diet growth was depressed and severe paralytic state ensued. Although the electrophoretic pattern appeared normal for the genetic dystrophy birds, Tureen and workers observed the profile to be characterized by a significant reduction in the total protein concentration.

Specific progressive changes in serum proteins have been correlated with the pathology of Histomoniasis in turkeys (Clarkson, 1966). Birds used were intra-rectally infected with Histomonas meleagridis. Two birds were sacrificed daily, the development of caecal and liver lesions monitored, and a

serum protein analysis was performed. Serum albumin concentration was observed to decline from 1.60 g/dl to 0.80 g/dl between the 4th and 5th days post-infection, and to about 0.30 g/dl on the 14th day. The sudden decline in albumin coincided with acute inflammation of the caecal mucosa, and the presence of large quantities of serum protein in the caecal contents. Clarkson also reported gamma-globulin concentrations to increase from 0.25 g/dl on the 8th day to 1.00 g/dl on the 14th day of infection (3 times the level of control birds). This change in the gamma-globulin fraction was explained as an immunological response to tissue damage.

Several electrophoretic studies have been conducted involving Marek's Disease (MD). Serum samples from healthy chickens and birds affected with MD were examined using paper electrophoresis. A reduction in albumin concentration, and an increase in the total protein and globulin fractions were observed during the periods of peak mortality associated with the MD inoculated birds. A sharp decrease in the albumin fraction, and a distinct elevation in the beta and gamma globulins was reported to occur with a group of birds that had survived the MD inoculation and then re-exposed to an MD infected environment (Samadieh et al., 1969).

Cellulose acetate electrophoresis, was conducted to compare the plasma protein profiles of MD-resistant and susceptible chickens (Washburn and Eidson, 1970). Those birds exposed to the GA isolate of MD virus exhibited significant

increases in fibrinogen and gamma-globulin fractions; however, the albumin fraction was observed to be significantly depressed. Levels of alpha-globulins-1 and 2, and beta fractions showed no significant difference between the control group and the MD-exposed birds. A unidentified component with the same electrophoretic mobility of fibrinogen was observed in both plasma and serum samples. Blood samples from MD-exposed chickens exhibited extremely high levels of the component.

Augustine (1985) reported the separation of five major protein fractions from the serum of Eimeria-infected turkeys. Significant decreases in albumin levels and significant increases in the alpha-2 and gamma-globulins were observed in the infected birds as compared to the control birds. Levels of alpha-1 and beta-globulins did not differ between the Eimeria-infected birds and controls.

CHAPTER III

COMPARATIVE ELECTROPHORETIC ANALYSIS OF PLASMA PROTEIN FRACTIONS FROM DEEP PECTORAL MYOPATHIC, HEREDITARY MUSCULAR DYSTROPHIC, AND A NORMAL LINE OF TURKEYS[1]

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ABSTRACT

Two experiments were conducted to try and differentiate variations in the levels of total plasma protein and plasma protein fractions from three lines of turkeys, each with a distinct muscle morphological characteristic--Deep Pectoral Myopathy (DPM), Hereditary Muscular Dystrophy (MDY), and a Broad Breasted Bronze control (J). Ten day-old poults from each of the three lines were grown in similar environments. Individual blood plasma samples were collected, body weights and breast width measurements determined at 8, 12 and 16 weeks of age (WOA) (Experiment 1), and at 20, 30, 40 and 50 WOA (Experiment 2).

Six plasma protein fractions were generally observed in the electrophoretograms of the three lines of turkeys at the various ages studied. Body weight and breast width values of both myopathic lines were lower ($P<0.05$) than those of the control (J) line at 8, 12 and 16 WOA (Experiment 1). Both females and males from the myopathic lines exhibited significantly lower body weights and breast widths when compared to J females and males, respectively at 20, 30, 40 and 50 WOA (Experiment 2).

Although variations were observed in both levels of total plasma protein and its fractions, no consistent differences were associated with either myopathic

disorder. The results of this study indicate that plasma protein electrophoresis does not appear to be a suitable diagnostic technique for the two myopathic conditions.

INTRODUCTION

Two myopathic conditions, both of genetic origins, have been observed in domestic turkeys. Deep pectoral myopathy (DPM), initially reported by Dickinson et al. (1968), is characterized by spontaneous necrosis and degeneration of the supracoracoideus muscle. The disorder affects primarily older female turkeys (Harper et al., 1975); however, occurrence of DPM may range from as early as 12 weeks of age to as old as 156 weeks of age in both sexes (Harper and Schmitz, 1979). Several investigators have suggested that genetic selection for larger breast musculature of the modern turkey has gradually impaired the vascular supply to the breast muscle, causing the myopathic condition (Jones et al., 1974; Harper et al., 1975; Siller and Wight, 1978; Martindale et al., 1979; Wight and Siller, 1980; Wight et al., 1981; Siller, 1980 and 1985).

Hereditary muscular dystrophy (MDY) was first reported by Harper and Parker (1964) and is characterized by extensive atrophy of the pectoral and alar muscles usually occurring between 8 and 16 weeks of age (Schmitz and Harper, 1975; Harper and Schmitz, 1979). Clinically, hereditary muscular dystrophy of the turkey is characterized by the inability of the bird to right itself after falling or when physically placed in a supine

position (Harper and Parker, 1964 and 1967).

Both myopathic conditions impose major economic problems to the poultry industry. Although hereditary muscular dystrophy is grossly apparent as early as 8 weeks of age, there is great difficulty in obtaining an early diagnosis of DPM in the live bird. Palpation of a focal depression, present under the skin overlying the superficial pectoral muscle, has been the traditional method for detecting DPM; however, by this time the condition is generally in an advanced state of atrophy (Harper et al., 1969 and 1975).

Characteristic alterations of blood protein levels are often indicative of the age, nutritional, reproductive and general health status of a species. Plasma/serum protein electrophoresis is frequently conducted to aid in the diagnoses of various pathological conditions.

The objectives of this study were to compare total plasma protein and protein fraction levels of the two myopathic lines to a control at specific ages, and to determine if plasma protein electrophoresis provides physiological and prognostic information. The influence of sex was also investigated.

MATERIALS AND METHODS

Three genetically different lines of medium weight turkeys, each with different breast muscle characteristics, deep pectoral myopathy (DPM) (Harper *et al.*, 1970), hereditary muscular dystrophy (MDY) (Harper and Parker, 1964) and a Broad Breasted Bronze strain (J) as a control group, were obtained at one day of age for this study.

Day-old unsexed poults from the three lines, obtained from the same hatch source, were wingbanded for identification and brooded separately in electrically heated batteries until two weeks of age (WOA). Ten poults from each line were then selected at random and transferred to separate but adjacent floor pens (3.05 m x 3.05 m) containing wood shavings. The birds were provided water and feed *ad libitum*, and grown with an 8 hour light:16 hour dark daily lighting program to 26 WOA. The feeding program is detailed in Table III.1. At 20 WOA, five birds of each sex (with the exception of only four DPM females) were selected from each line, and maintained in floor pens (3.05 m x 3.05 m) containing wood shavings. Prior to 26 WOA, males from each line were separated from the females, and housed in separate floor pens (4.87 m x 4.87 m) with wood shavings to 50 WOA. All three lines were offered water and feed (Table III.1) *ad libitum* for the

duration of the study, with different dietary protein levels between sexes.

Individual body weight and breast width measurements were determined at 8, 12 and 16 WOA (Experiment 1), and at 20, 30, 40 and 50 WOA (Experiment 2). Breast width was measured with a specialized caliper according to the procedure of Harper et al. (1975). Hereditary muscular dystrophy was visually apparent between 8 and 16 WOA; however, the presence of myopathic lesions of the breast in the DPM birds was determined by palpation (Harper et al., 1975), and confirmed by post mortem examination at 50 WOA.

Blood samples were obtained at 8, 12 and 16 WOA (Experiment 1), and at 20, 30, 40 and 50 WOA (Experiment 2). Each sampling period, of both experiments, was conducted during the same hour of the morning. Approximately 3 milliliters of blood was collected by brachial venipuncture into evacuated heparinized tubes, and immediately placed in an ice bath. The samples were centrifuged at 1500 x g for 10 minutes, then the plasma was separated and stored in plastic tubes at -12C until analyses. Plasma samples were analyzed for total protein concentrations using a modified Biuret[1] method (Gornall et al., 1949). Since the influence of physical handling or

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[1]Gilford Diagnostics, Cleveland, Ohio.

stress upon blood analyses was undetermined, body weight and breast width measurements were performed on the day following each blood collection period.

Agarose-gel electrophoresis was conducted using three microliter samples of plasma, with cathodic applications 2.5 cm from the top edge of the 8.0 cm x 9.7 cm agarose plates. Electrophoretic separations were performed at 250 volts using a Barbital buffer[2] for 20 minutes. The electrophoresed proteins were subsequently visualized with Coomassie brilliant blue dye according to the manufacturer[2].

Densitometric quantitation of the electrophoresed protein fractions were accomplished using a Helena Cliniscan[2] densitometer set at a wavelength of 520 nm. Tentative identification of protein fractions was achieved by comparison to a human serum standard[3], electrophoresed simultaneously with the turkey samples on each agarose-gel plate. Quantitative values of the protein fractions were determined by multiplying the total protein value (g/dl) by the relative percentage of each fraction achieved by densitometry.

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[3]Allied Corporation, Fisher Scientific, Orangeburg, NY 10962.

Experiment 1. Ten poults of each of the three lines were selected at random (with the exception of 9 MDY poults) identified, and with the various body measurements and protein analyses conducted on the same birds at 8, 12 and 16 WOA.

Experiment 2. Five birds of each sex (with the exception of only four DPM females) were selected from each line, and the methods previously outlined performed at 20, 30, 40 and 50 WOA.

Since dietary protein levels and physiological status of the birds varied with increasing age, statistical analyses of the data were conducted using a one-way analysis of variance among the lines within each age period. The measured parameters of each myopathic line were compared separately to the control line values, and where applicable, Fisher's Significant Difference Tests ($P<0.05$) performed. Additional statistical analyses were conducted for Experiment 2 in order to compare values from males and females of each myopathic line with respective control values, and between sexes within each line.

RESULTS

A total of six plasma protein fractions were generally observed in individual electrophoretograms from the three lines of turkeys (J, DPM and MDY). Protein bands (designated 1 through 6) correspond to the pre-albumin, albumin, alpha 1, 2-, beta-, and gamma-globulins of human serum (Figure III.1).

Experiment 1: Levels of total plasma protein and protein fractions from each myopathic line (DPM and MDY) were compared to the control group (J) and are summarized in Table III.2. Total protein concentrations of all three lines were observed to increase with the highest levels occurring at 16 weeks of age (WOA). There were not differences in mean levels of total plasma protein concentrations at 8 WOA; however, 12 and 16 week plasma protein values of the DPM and MDY groups were significantly higher ($P<0.05$) than those of the control group (J).

The mean level of fraction 1 (pre-albumin) for the DPM line was significantly greater ($P<0.05$) than that of the control line (J) at 8 WOA. At 12 WOA mean fraction 1 levels of the DPM and MDY lines were higher ($P<0.05$) than those of the J line. No differences were observed between the three groups at 16 WOA.

Fraction 2 (albumin) levels of DPM and MDY lines were higher ($P<0.05$) than the J line at 12 WOA, and only the level of the DPM group remained higher through 16 WOA.

Levels of fractions 3, 4, 5, and 6 (corresponding to alpha-1, alpha-2, beta-, and gamma globulins, respectively) fluctuated among the lines and at different ages showing no consistent trends.

Mean body weights and breast widths of the myopathic lines (DPM and MDY) were compared separately to the J line values and are presented in Tables III.7 and III.8. Body weight and breast width values of all three lines were observed to increase with age. The DPM and MDY lines exhibited lower ($P<0.05$) body weights and breast widths than those of line J at 8, 12 and 16 WOA.

Experiment 2: Comparisons of total plasma protein and protein fractions levels between males and females within each line are summarized in Tables III.3 and III.4. Total protein and protein fraction levels of females and males from each myopathic line are compared separately to those values of the J females and males (Tables III.5 and III.6). No differences ($P>0.05$) in mean total protein levels were observed between sexes within the J and MDY lines at 20, 30 and 40 WOA. DPM females; however, had higher ($P<0.05$) total protein values than those of DPM males at 20 and 40 WOA. At 50 WOA, females of

lines J and DPM exhibited greater ($P<0.05$) mean total plasma protein levels than J and DPM males. Comparison of mean total protein concentrations of DPM and MDY males to J males revealed no differences at the various ages examined. DPM females exhibited greater ($P<0.05$) total protein values than those of the J females at only 20 and 40 WOA.

J and DPM males exhibited significantly higher levels of pre-albumin (fractional) ($P<0.05$) than those of J and DPM females at 40 WOA. In contrast, 50 week old J and MDY females exhibited greater ($P<0.05$) pre-albumin levels than J and MDY males. No differences were detected in pre-albumin values of the DPM and MDY males when each was compared separately to those of the J males at 20, 30, 40 and 50 WOA. In contrast, DPM females at 30 WOA had greater ($P<0.05$) pre-albumin levels than the J females.

No differences were observed in albumin (fraction 2) levels between sexes within lines at 20 and 30 WOA. At 40 WOA, DPM and MDY females exhibited higher ($P<0.05$) albumin levels than the males. DPM and J females had greater ($P<0.05$) levels of albumin than either DPM or J males. Comparisons of albumin levels by sexes between lines revealed no differences or trends between DPM or MDY males compared with J males at the ages examined. Both DPM and MDY females had greater ($P<0.05$) levels at 50 WOA than that of the control (J) females.

Levels of the globulins (fractions 3, 4, 5 and 6) compared between sexes within each line revealed DPM and MDY females to exhibit higher ($P<0.05$) mean values of fraction 3 (alpha-1) than DPM and MDY males at 20 and 40 WOA. DPM males had higher ($P<0.05$) values of fraction 4 (alpha-2) compared to the J males at 40 and 50 WOA. In contrast, DPM females had greater levels ($P<0.05$) of fraction 4 than those of the J females at 20 and 40 WOA.

At 50 WOA an unidentified fraction (designated fraction *), located between fractions 4 and 5, was observed in the protein profiles of all three lines. Females of each line had greater ($P<0.05$) concentrations of this fraction than the males of each line.

Post mortem examination of the DPM line at 50 WOA revealed six birds to exhibit DPM lesions varying from mild to acute. Plasma protein fraction levels (PPFL) of individual DPM birds were examined. The variations in PPFL observed could not be associated with the presence or absence of the disorder.

Body weights and breast widths of males and females from each myopathic line were compared (separately) to the control males and females and are presented in Tables III.9 and III.10. Males consistently exhibited greater body weights and breast widths when compared to females within each line; however, no differences in breast widths were observed between the MDY males and females at 20, 40

or 50 WOA. Both DPM and MDY females exhibited lower ($P<0.05$) body weights and breast widths than the J females for the duration of the experiment. Likewise, DPM and MDY males had lower ($P<0.05$) body weights and breast widths when compared to the J males.

DISCUSSION

Plasma protein levels may be influenced by several factors such as the age, strain, sex or reproductive status, and the environmental condition of a bird. The electrophoretic technique or method utilized also contributes to the resolution of the various protein components. Often fluctuations in one protein fraction will affect the level of another protein fraction (Sibley and Hendrickson, 1970). Therefore, these factors should not be ignored when evaluating the results from this study, and variations in plasma protein fractions may not be attributed to the myopathic conditions exclusively.

Total Protein

Total protein concentrations of all three lines (Experiments 1 and 2) increased with age. This was in agreement with Brandt et al. (1951), Amin (1961), Tureen et al. (1966), Patterson et al. (1967), and Lisano and Kennamer (1977), all who noted that total protein levels increased as the birds matured. Al-Heeti et al. (1985) reported strain differences influenced total protein levels in turkey serum. In this study, both the J and MDY lines were varieties of Broad Breasted Bronze, while the DPM line was a Medium White variety. Although results from Experiment 2 revealed total plasma protein levels of the

DPM females to be significantly higher than the control females, it is difficult to ascertain whether or not these variations are due to strain differences alone.

Sex and reproductive status of the bird may have influenced protein levels. In Experiment 1, due to the young ages of the birds, the sexes were combined and reproductive status was not considered. In Experiment 2, females of all three lines exhibited higher total protein values than males of the respective lines, which was most likely due to hormonal differences. Estrogens may increase total serum protein concentrations in the chicken (Sturkie and Newman, 1951), while androgens have been reported to cause a decrease in serum levels of male turkeys (Ranaweera and Wise, 1981). Similarily, total serum protein concentrations of turkeys have been determined to be significantly influenced by sex (Al-Heeti et al., 1985).

The environmental conditions present during the course of this study could also have contributed to the variation in total plasma protein levels. All three lines of birds were reared under identical environmental conditions for the duration of Experiment 1, and to 30 weeks of age in Experiment 2, at which time males and females from each line were separated and maintained on different dietary rations. Stress, dehydration, increased adrenal activity and protein turnover generally result in a decrease in total protein concentration (Kaneko, 1980). Lisano and

Kennamer (1977) and Al-Heeti et al. (1985) observed variations in the total serum protein of the turkey was also due to seasonal conditions. The latter investigators reported that the highest total protein levels occurred during moderate seasonal conditions while the lowest levels were observed under high temperatures and low humidity. In the present study, the total protein levels of the three lines were greater at 8 and 12 WOA (moderate months of May and June), while a decrease was observed in those levels at the 16 week old sampling period (warmer month of July). Likewise, total protein values for Experiment 2 were recorded to be lower during the winter months of October and December at 30 and 40 WOA, with peak values occurring during moderate seasonal conditions (March) at 50 WOA.

Serum protein levels may also be responsive to nutritional influences (Kaneko, 1980). Leveille and Sauberlich (1961) reported that serum protein concentrations of growing chicks increased as the dietary protein level was increased. In contrast, total serum protein values of laying pullets fed 17% dietary protein were observed to decrease by 12.5% when a ration containing 9% crude protein was fed to the birds (Waldroup et al., 1965). In this study, total plasma protein levels increased even though the crude protein levels were gradually being reduced.

Protein Fractions

In both Experiments 1 and 2, pre-albumin was present in both sexes of turkeys (all three lines). Kristjannson et al. (1963) observed 3 pre-albumins ("PaA", "PaB", and "PaC") in the sera of chickens, with PaB absent from the sera of males, immature females, and mature hens that were out of production. The pre-albumin type observed in the turkeys of this study could be either PaA or PaC as described by Krisjannson et al. (1963).

The higher levels of albumin in the plasma of the two myopathic lines (Experiment 1) may be associated with metabolic or strain differences. Higher serum albumin levels have been observed in dwarf White Leghorns compared to those of non-dwarfs (Wood et al., 1971), while lower serum albumin levels were reported in a White strain of turkeys when compared to two other strains (Al-Heeti et al., 1985).

Al-Heeti et al. (1985) found that sex did not influence serum albumin of turkeys. Although no difference was observed in albumin (fraction 2) levels between sexes within each of the lines at 20 and 30 WOA, differences were detected at 40 and 50 WOA.

Because of the 10 week time intervals between sampling periods (Experiment 2), numerous factors in addition to or in place of reproductive status could be attributed to the results observed. Higher serum albumin values have been

reported in chickens adapted to certain seasonal conditions (Wood et al., 1971) whereas Al-Heeti et al. (1985) did not find season to significantly influence those levels in turkeys. However, the latter investigators did report a significant strain x season interaction with respect to albumin levels.

As with total protein concentration, albumin levels have been reported to increase in the growing chick as the level of dietary protein was increased (Leveille and Sauberlich, 1961). Contrary to this, in the present study as the levels of dietary protein were reduced the albumin levels did not decrease. This may indicate that the changes in albumin levels of the turkeys are not related to dietary protein.

A marked decrease in the blood albumin level has been associated with several avian diseases such as with leucosis (Sanders et al., 1944) and Marek's Disease (Samadieh et al., 1969; Washburn and Eidson, 1970) of chickens, and with Histomoniasis (Clarkson, 1966) and Eimeria infection (Augustine, 1985) of turkeys. Both deep pectoral myopathy and hereditary muscular dystrophy are of genetic origin, and not to be considered infectious diseases.

Alpha and gamma globulins have been reported to increase with age, while beta globulin levels are not related with the age of chickens (Brandt et al., 1951).

Likewise, Medway and Kare (1959) observed levels of gamma globulins to increase as the chicken matures. In the present study, alpha and beta globulin levels of the two myopathic line were greater at 12 WOA as compared to 8 WOA; however, levels did not continue to increase consistently thereafter during the course of the study.

Comparisons between sexes within each of the lines between 20 and 50 WOA revealed considerable variation in globulin levels, the DPM and MDY females exhibited significantly greater levels of alpha-1 globulin than respective males of the two lines at 20 and 40 weeks of age. Al-Heeti et al. (1985) also reported sex to influence serum globulins of turkeys.

The electrophoretograms of pullets fed low dietary protein rations illustrated significant reductions in the alpha and beta globulin fractions which may have accounted for the decline in total protein levels (Waldroup et al., 1965). The birds of Experiment 1 did exhibit higher alpha and beta globulin levels when compared with those levels of the birds of Experiment 2. This may be attributed to the fact that between 8 and 16 WOA, birds were fed rations containing higher protein levels than between 20 and 50 WOA.

The unidentified fraction (fraction *) could possibly be alpha-3 globulin or Beta-1 globulin due to its location between fractions 4 and 5; however, why it was present

only at 50 WOA is difficult to explain.

Certain pathological conditions have been demonstrated to cause variations in globulin levels. A five-fold increase in serum gamma globulins of turkeys afflicted with Histomoniasis was suggested to result from the birds' immunological response to tissue damage (Clarkson, 1966). In the present study, DPM females at 20 weeks of age, and DPM males at 30 weeks of age exhibited significantly greater levels of gamma globulin when compared to the control group (J). Postmortem examination of the supracoracoid muscles of the DPM line revealed six birds to exhibit DPM lesions varying from mild to very acute. Although the supracoracoid muscle of DPM birds becomes necrotic and deteriorates in advanced stages of the condition, it is difficult to explain why gamma globulin levels were greater only at 20 and 30 weeks of age.

Although variations were observed in levels of total plasma protein and its fractions, no consistent differences were associated with either myopathic disorder. The results from this study indicate that plasma protein electrophoresis does not appear to be a suitable diagnostic technique for the two myopathic conditions.

Table III.1. Composition of turkey rations from 1 day of age to 50 weeks of age (Exp. 1 and 2).

Ingredients	Starter (0-8 wks)	Grower I (8-12 wks)	Grower II (12-18 wks)	Grower III (18-30 wks)	Breeder Hens (30-50 wks)	Breeder Toms (30-50 wks)
			%			
Corn, yellow	43.25	62.60	73.82	81.30	73.65	81.54
Soybean meal, solv., 47.5% CP	44.00	17.50	12.25	8.75	11.00	13.00
Meat and bone meal, 50% CP	5.00	10.00	7.00	5.00	5.00	-
Whey, dried	2.50	2.50	1.75	1.25	2.50	-
Alfalfa meal, dehy. 17% CP	2.50	5.00	3.50	2.50	2.50	2.50
Limestone flour	1.00	1.00	0.70	0.50	3.82	0.60
Dical. Phosphate (21% Ca;18% P)	0.75	0.50	0.35	0.25	-	-
Defluorinated phosphate (32% Ca;18% P)	-	-	-	-	0.56	1.36
Salt (iodized)	0.50	0.50	0.35	0.25	0.40	0.50
Vitamin premix[1]	0.30	0.30	0.21	0.15	0.35	0.35
Trace mineral premix[2]	0.05	0.05	0.04	0.03	0.10	0.05
D, L methionine, 98%	0.10	0.05	0.04	0.03	0.02	-
Amprolium[3]	0.05	-	-	-	-	-
Selenium	-	-	-	-	0.05	-
Biotin (Rovamix-H)[4]	-	-	-	-	0.05	-
Calculated analyses:						
Crude protein, %	28.10	20.10	16.80	14.50	15.10	13.90
ME, kcal/kg	2713	2867	3036	3148	2963	3139
Calcium, %	1.33	1.82	1.28	0.92	2.34	0.77
Avail. phosphorus, %	0.58	0.79	0.58	0.44	0.51	0.35
Lysine, %	1.65	1.10	0.81	0.64	0.71	0.58
Methionine, %	0.54	0.37	0.32	0.28	0.28	0.24
Met. + Cyst., %	1.01	0.71	0.61	0.54	0.55	0.50

[1]Supplies per kilogram of feed: Vitamin A, 4950 IU; Vitamin D_3, 1650 ICU; riboflavin, 5.0 mg; d-pantothenic acid, 8.3 mg; niacin, 33 mg; choline, 286 mg; Vitamin B_{12}, 8.3 mcg; Vitamin E, 1.7 IU; Vitamin K, 0.83 mg; folacin, 0.22 mg.

[2]Supplies per kilogram of feed: Calcium, 97.5 mg; manganese, 60 mg; iron, 20 mg; iodine, 1.2 mg; zinc, 27.5 mg; cobalt, .02 mg; copper, 2 mg.

[3]Provided gratuitously by Merck and Company, Rahway, NJ.

[4]Provided gratuitously by Hoffmann-LaRoche, Inc., Nutley, NJ.

CP = Crude protein

Table III.2. Total plasma protein and plasma protein fraction levels (g/dl)[1] of selected lines of turkeys at varying ages (Exp. 1).

Age/Line	No. of birds	Total Protein	Plasma Protein Fractions					
			fr1	fr2	fr3	fr4	fr5	fr6
8 WOA								
J	10	3.86±.08[a]	.064±.005[a]	1.16±.03[a]	.29±.009[a]	.51±.02[a]	.72±.02[a]	1.11±.03[a]
DPM	10	3.75±.07[a]	.099±.006[b]	1.28±.05[a]	.32±.01[a]	.51±.007[a]	.73±.02[a]	0.82±.02[b]
MDY	9	4.00±.13[a]	.156±.09[a]	1.20±.03[a]	.26±.02[a]	.55±.01[a]	.77±.03[a]	1.20±.04[a]
12 WOA								
J	10	3.88±.07[b]	.053±.004[b]	1.27±.03[b]	.23±.01[b]	.50±.02[b]	.78±.03[b]	1.03±.03[a]
DPM	10	4.54±.09[a]	.078±.003[a]	1.56±.09[a]	.29±.01[a]	.55±.02[a]	1.00±.03[a]	1.06±.06[a]
MDY	9	4.39±.08[a]	.081±.006[a]	1.49±.04[a]	.23±.02[b]	.54±.02[a]	.97±.03[a]	1.10±.04[a]
16 WOA								
J	10	4.16±.10[b]	.070±.006[a]	1.45±.06[b]	.27±.02[a]	.47±.02[a]	.79±.02[b]	1.04±.03[b]
DPM	10	4.47±.09[a]	.083±.01[a]	1.75±.08[a]	.27±.02[a]	.50±.02[a]	.88±.04[b]	.992±.05[b]
MDY	9	4.41±.07[a]	.059±.005[a]	1.56±.07[b]	.25±.03[a]	.46±.03[a]	.92±.03[a]	1.17±.03[a]

[1]Values represent means ± SE.
WOA: Weeks of Age.
Lines: J = Broad Breasted Bronze control; DPM = Deep Pectoral Myopathy; MDY = Hereditary Muscular Dystrophy
Column means with different lettered superscripts within an age are significantly different (P<.05).
DPM and MDY lines compared separately to line J at each age.

Table III.3. Total plasma protein and plasma protein fraction levels (g/dl)[1] of selected lines of turkeys at 20-30 WOA. Comparisons between sexes within each line (Exp. 2).

Age/Line	Sex	No. of Birds	Total Protein	Plasma Protein Fractions						
				fr1	fr2	fr3	fr4	fr*	fr5	fr6
20 WOA										
J	F	5	3.78±.11[a]	.04±.003[a]	1.33±.06[a]	.31±.02[a]	.28±.02[a]		.86±.07[a]	.95±.03[a]
J	M	5	3.88±.18[a]	.05±.006[a]	1.20±.08[a]	.28±.04[a]	.33±.03[a]		.94±.03[a]	1.09±.07[a]
DPM	F	4	4.50±.16[a]	.04±.007[a]	1.48±.09[a]	.39±.06[a]	.39±.02[a]		1.05±.08[a]	1.15±.06[a]
DPM	M	5	4.04±.02[b]	.04±.01[a]	1.42±.05[a]	.20±.01[b]	.37±.01[a]		1.03±.06[a]	.98±.03[b]
MDY	F	5	4.24±.19[a]	.05±.009[a]	1.41±.06[a]	.28±.03[a]	.35±.03[a]		.99±.04[a]	1.15±.11[a]
MDY	M	5	4.0±.09[a]	.05±.007[a]	1.44±.06[a]	.17±.01[b]	.37±.02[a]		.95±.04[a]	1.03±.03[a]
30 WOA										
J	F	5	4.04±.27[a]	.05±.005[a]	1.39±.08[a]	.37±.05[a]	.22±.03[a]		.81±.07[a]	1.22±.09[a]
J	M	5	4.24±.25[a]	.06±.007[a]	1.34±.06[a]	.35±.09[a]	.22±.03[a]		.96±.10[a]	1.31±.07[a]
DPM	F	4	4.1±.21[a]	.06±.004[a]	1.48±.07[a]	.26±.03[a]	.28±.02[a]		.93±.06[a]	1.06±.11[a]
DPM	M	5	3.98±.15[a]	.16±.10[a]	1.40±.09[a]	.26±.02[a]	.27±.01[a]		1.00±.04[a]	1.06±.03[a]
MDY	F	5	4.24±.12[a]	.06±.004[a]	1.57±.07[a]	.35±.03[a]	.24±.01[a]		.91±.04[a]	1.12±.10[a]
MDY	M	5	4.46±.39[a]	.06±.004[a]	1.47±.06[a]	.30±.05[a]	.26±.03[a]		1.14±.10[a]	1.23±.17[a]

[1]Values represent means ± SE.
WOA: Weeks of Age.
Lines: J = Broad Breasted Bronze control; DPM = Deep Pectoral Myopathy; MDY = Hereditary Muscular Dystrophy
Column means with different lettered superscripts are significantly different (P<.05).
* = unidentified fraction.
DPM and MDY lines compared separately to line J at each age.

Table III.4. Total plasma protein and plasma protein fraction levels (g/dl)[1] of selected lines of turkeys at 40-50 WOA. Comparisons between sexes within each line (Exp. 2).

Age/Line	Sex	No. of Birds	Total Protein	Plasma Protein Fractions						
				fr1	fr2	fr3	fr4	fr*	fr5	fr6
40 WOA										
J	F	5	4.00±.28^{a}	.04±.005^{b}	1.34±.07^{a}	.33±.06^{a}	.22±.01^{a}		.89±.06^{a}	1.17±.11^{a}
J	M	5	4.16±.16^{a}	.06±.005^{a}	1.39±.04^{a}	.28±.04^{a}	.25±.02^{a}		.96±.05^{a}	1.23±.07^{a}
DPM	F	4	5.03±.17^{a}	.04±.006^{a}	1.84±.10^{a}	.40±.04^{a}	.30±.03^{a}		1.12±.05^{a}	1.36±.08^{a}
DPM	M	5	4.38±.16^{b}	.06±.005^{a}	1.44±.03^{b}	.23±.03^{b}	.34±.02^{a}		1.06±.05^{a}	1.24±.08^{b}
MDY	F	5	4.80±.21^{a}	.05±.009^{a}	1.77±.08^{a}	.30±.02^{a}	.27±.02^{a}		1.04±.04^{a}	1.34±.08^{a}
MDY	M	5	4.20±.26^{a}	.07±.007^{a}	1.42±.11^{a}	.18±.01^{b}	.27±.02^{a}		.93±.07^{a}	1.33±.10^{a}
50 WOA										
J	F	5	6.03±.05^{a}	.06±.01^{a}	1.85±.16^{a}	.34±.03^{a}	.37±.04^{a}	.35±.03^{a}	1.64±.01^{a}	1.42±.14^{a}
J	M	5	4.56±.25^{a}	.02±.007^{b}	1.43±.06^{b}	.26±.03^{a}	.34±.02^{a}	.012±.01^{b}	1.21±.08^{a}	1.27±.10^{a}
DPM	F	4	6.30±.18^{a}	.02±.02^{a}	2.05±.06^{a}	.33±.06^{a}	.37±.05^{a}	.221±.09^{a}	1.80±.16^{a}	1.48±.04^{a}
DPM	M	5	5.02±.27^{a}	.04±.007^{a}	1.54±.08^{b}	.29±.03^{b}	.50±.03^{a}	0 ± 0^{b}	1.32±.06^{b}	1.32±.10^{a}
MDY	F	5	5.92±.30^{a}	.06±.008^{a}	1.77±.19^{a}	.26±.03^{a}	.39±.03^{a}	.244±.08^{a}	1.59±.10^{a}	1.62±.16^{a}
MDY	M	5	4.83±.50^{a}	.03±.01^{b}	1.47±.11^{a}	.28±.07^{a}	.38±.07^{a}	0 ± 0^{b}	1.26±.11^{a}	1.41±.19^{a}

[1]Values represent means ± SE.
WOA: Weeks of Age.
Lines: J = Broad Breasted Bronze control; DPM = Deep Pectoral Myopathy; MDY = Hereditary Muscular Dystrophy
Column means with different lettered superscripts within an age and sex are significantly different (P<.05).
DPM and MDY lines compared separately to line J at each age.
* = unidentified fraction.

Table III.5. Total plasma protein and plasma protein fraction levels (g/dl)[1] of selected lines of turkeys from 20-50 WOA. Females of myopathic lines compared separately to control females (Exp. 2).

Age/Line	Sex	No. of Birds	Total Protein	Plasma Protein Fractions						
				fr1	fr2	fr3	fr4	fr*	fr5	fr6
20 WOA										
J	F	5	3.78±.11^{b}	.04±.003^{b}	1.33±.06^{a}	.31±.02^{a}	.28±.02^{b}		.86±.07^{a}	.95±.03^{b}
DPM	F	4	4.50±.16^{a}	.04±.007^{a}	1.48±.09^{a}	.39±.06^{a}	.39±.02^{a}		1.05±.08^{a}	1.15±.06^{a}
MDY	F	5	4.24±.19^{b}	.05±.009^{a}	1.41±.06^{a}	.28±.03^{a}	.35±.03^{b}		.99±.04^{a}	1.15±.11^{b}
30 WOA										
J	F	5	4.04±.27^{a}	.05±.005^{b}	1.39±.08^{a}	.37±.05^{a}	.22±.03^{a}		.81±.07^{a}	1.22±.09^{a}
DPM	F	4	4.10±.21^{a}	.06±.004^{a}	1.48±.07^{a}	.26±.03^{a}	.28±.02^{a}		.93±.06^{a}	1.06±.11^{a}
MDY	F	5	4.24±.12^{a}	.06±.004^{b}	1.57±.07^{a}	.35±.03^{a}	.24±.01^{a}		.91±.04^{a}	1.12±.10^{a}
40 WOA										
J	F	5	4.00±.28^{b}	.04±.005^{a}	1.34±.07^{b}	.33±.06^{b}	.22±.01^{b}		.89±.06^{b}	1.17±.11^{a}
DPM	F	4	5.03±.17^{a}	.04±.006^{a}	1.84±.10^{a}	.40±.04^{a}	.30±.03^{a}		1.12±.05^{a}	1.36±.08^{a}
MDY	F	5	4.80±.21^{b}	.05±.009^{a}	1.77±.08^{a}	.30±.02^{b}	.27±.02^{a}		1.04±.04^{b}	1.34±.08^{a}
50 WOA										
J	F	4	6.03±.05^{a}	.06±.01^{a}	1.85±.16^{a}	.34±.03^{a}	.37±.04^{a}	.35±.03^{a}	1.64±.01^{a}	1.42±.14^{a}
DPM	F	4	6.30±.18^{a}	.02±.02^{a}	2.05±.06^{a}	.33±.06^{a}	.37±.05^{a}	.22±.09^{a}	1.80±.16^{a}	1.48±.04^{a}
MDY	F	5	5.92±.30^{a}	.06±.008^{a}	1.77±.19^{a}	.26±.03^{a}	.39±.03^{a}	.24±.08^{a}	1.59±.10^{a}	1.62±.16^{a}

[1]Values represent means ± SE.
WOA: Weeks of Age.
Lines: J = Broad Breasted Bronze control; DPM = Deep Pectoral Myopathy; MDY = Hereditary Muscular Dystrophy
Column means with different lettered superscripts within an age are significantly different (P<.05).
DPM and MDY lines compared separately to line J at each age.
* = unidentified fraction.

Table III.6. Total plasma protein and plasma protein fraction levels (g/dl)[1] of selected lines of turkeys from 20-50 WOA. Males of myopathic lines compared separately to control males (Exp. 2).

Age/Line	Sex	No. of Birds	Total Protein	Plasma Protein Fractions						
				fr1	fr2	fr3	fr4	fr*	fr5	fr6
20 WOA										
J	M	5	3.88±.18[a]	.05±.006[a]	1.20±.08[a]	.28±.04[a]	.33±.03[a]		.94±.03[a]	1.09±.07[a]
DPM	M	5	4.04±.02[a]	.04±.01[a]	1.42±.05[a]	.20±.01[a]	.37±.01[a]		1.03±.06[a]	.98±.03[a]
MDY	M	5	4.00±.09[a]	.05±.007[a]	1.44±.06[a]	.17±.01[b]	.37±.02[a]		.95±.04[a]	1.03±.03[a]
30 WOA										
J	M	5	4.24±.25[a]	.06±.007[a]	1.34±.06[a]	.35±.09[a]	.22±.03[a]		.96±.10[a]	1.31±.07[a]
DPM	M	5	3.98±.15[a]	.16±.10[a]	1.40±.09[a]	.26±.02[a]	.27±.01[a]		1.00±.04[a]	1.06±.03[b]
MDY	M	5	4.46±.39[a]	.06±.004[b]	1.47±.06[a]	.30±.05[a]	.26±.03[a]		1.14±.10[a]	1.23±.17[a]
40 WOA										
J	M	5	4.16±.16[a]	.06±.005[a]	1.39±.04[a]	.28±.04[a]	.22±.02[b]		.96±.05[a]	1.23±.07[a]
DPM	M	5	4.38±.16[a]	.06±.005[a]	1.44±.03[a]	.23±.03[a]	.34±.02[a]		1.06±.05[a]	1.24±.08[a]
MDY	M	4	4.20±.26[a]	.07±.007[a]	1.42±.11[a]	.18±.01[a]	.27±.02[b]		.93±.07[a]	1.33±.10[a]
50 WOA										
J	M	5	4.56±.25[a]	.02±.007[a]	1.43±.06[a]	.26±.03[a]	.34±.02[a]	.012±.01	1.21±.08[a]	1.27±.10[a]
DPM	M	5	5.02±.27[a]	.04±.007[a]	1.54±.08[a]	.29±.03[a]	.50±.03[a]		1.32±.06[a]	1.32±.10[a]
MDY	M	4	4.83±.50[a]	.03±.01[a]	1.47±.11[a]	.28±.07[a]	.38±.07[a]		1.26±.11[a]	1.41±.19[a]

[1]Values represent means ± SE.
WOA: Weeks of Age.
Lines: J = Broad Breasted Bronze control; DPM = Deep Pectoral Myopathy; MDY = Hereditary Muscular Dystrophy
Column means with different lettered superscripts within an age are significantly different (P<.05).
DPM and MDY lines compared separately to line J at each age.
* = unidentified fraction.

Table III.7 Mean body weight values (kg)[1] of selected lines of turkeys at varying ages (Exp. 1).

Line	Age in Weeks		
	8	12	16
J	1.90±.21[a] (10)	3.83±.51[a] (10)	5.98±.77[a] (10)
DPM	1.27±.10[b] (10)	2.52±.21[b] (10)	4.14±.43[b] (10)
MDY	1.40±.15[b] (9)	2.82±.23[b] (9)	4.29±.39[b] (9)

[1]Values represent means ± SE.
( ) = number of birds.
Lines: J = Broad Breasted Bronze control; DPM = Deep Pectoral Myopathy; MDY = Hereditary Muscular Dystrophy
Column means with different lettered superscripts within an age are significantly different (P<.05).
DPM and MDY lines compared separately to line J at each age.

Table III.8 Mean breast width values (cm)[1] of selected lines of turkeys at varying ages (Exp. 1).

Line	Age in Weeks		
	8	12	16
J	4.39±.09[a] (10)	8.23±.09[a] (10)	9.91±.10[a] (10)
DPM	4.04±.05[a] (10)	6.81±.05[b] (10)	8.46±.08[b] (10)
MDY	2.59±.06[b] (9)	3.84±.08[b] (9)	6.15±.11[b] (9)

[1]Values represent means ± SE.
( ) = number of birds.
Lines: J = Broad Breasted Bronze control; DPM = Deep Pectoral Myopathy; MDY = Hereditary Muscular Dystrophy
Column means with different lettered superscripts with an age are significantly different (P<.05).
DPM and MDY lines compared separately to line J at each age.

Table III.9 Mean body weight values (kg)[1] of selected lines of turkeys. Separate comparisons of myopathic lines to control (Exp. 2).

Line	Sex	Age in Weeks 20	30	40	50
J	F	5.69±.51[a] (5)	7.58±1.1[a] (5)	9.60±1.6[a] (5)	9.00±1.2a (4)
DPM	F	3.47±.47[b] (4)	4.70±.47[b] (4)	5.51±.48[b] (4)	5.28±.40[b] (4)
MDY	F	4.55±.29[b] (5)	6.17±.51[b] (5)	7.17±.50[b] (5)	6.80±.55[b] (5)
J	M	8.26±.66[a] (5)	11.75±.94[a] (5)	15.94±1.1[a] (5)	16.59±.65[a] (5)
DPM	M	6.07±.22[b] (5)	9.05±.27[b] (5)	11.05±.61[b] (5)	11.31±.85[b] (5)
MDY	M	4.77±2.6[b] (5)	8.60±.58[b] (5)	10.25±1.3[b] (3)	11.46±1.8[b] (4)

[1]Values represent mean ± SE.
( ) = number of birds.
Lines: J = Broad Breasted Bronze control; DPM = Deep Pectoral Myopathy; MDY = Hereditary Muscular Dystrophy
Column means with different lettered superscripts within a sex are significantly different ($P<0.05$).
DPM and MDY lines compared separately to line J at each age.

Table III.10 Mean breast width values (cm)[1] of selected lines of turkeys. Separate comparisons of myopathic lines to control (Exp. 2).

Line	Sex	Age in Weeks 20	30	40	50
J	F	9.22±.10[a] (5)	8.89±.08[a] (5)	10.62±.39[a] (5)	11.18±.08a (4)
DPM	F	7.32±.13[b] (4)	7.47±.19[b] (4)	8.74±.08[b] (4)	7.32±.17[b] (4)
MDY	F	5.54±.06[b] (5)	5.54±.05[b] (5)	5.08±.07[b] (5)	4.83±.16[b] (5)
J	M	10.49±.08[a] (5)	10.67±.09[a] (5)	13.18±.08[a] (5)	14.55±.08[a] (5)
DPM	M	9.27±.09[b] (5)	9.27±.13[b] (5)	11.76±.10[b] (5)	11.76±.18[b] (5)
MDY	M	6.05±.07[b] (5)	5.84±.08[b] (5)	5.77±.10[b] (3)	5.49±.26[b] (4)

[1]Values represent mean ± SE.
( ) = number of birds.
Lines: J = Broad Breasted Bronze control; DPM = Deep Pectoral Myopathy; MDY = Hereditary Muscular Dystrophy
Column means with different lettered superscripts within a sex are significantly different ($P<0.05$).
DPM and MDY lines compared separately to line J at each age.

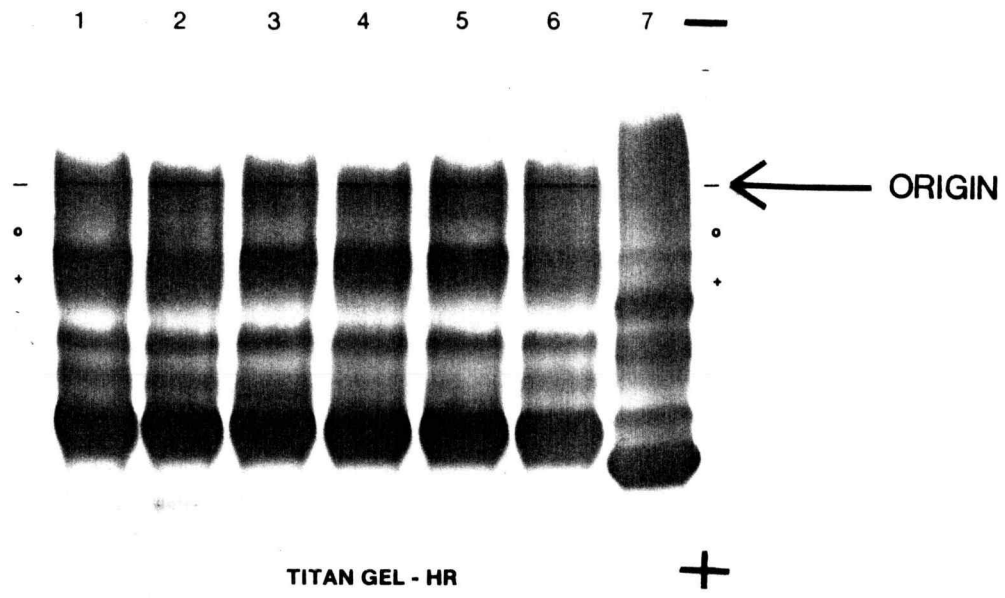

Figure III.1. Plasma protein fractions of Deep Pectoral Myopathic (DPM), Hereditary Muscular Dystrophic (MDY), and normal Broad Breasted Bronze lines of turkeys at 20 WOA. Columns: 1, 6 represent normal line; 3, 4, 5 represent DPM line; 2 represents MDY line; 7 represents Human Standard.

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CHAPTER IV

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