

Metabolic Disease Symposium: Metabolic and Cardiovascular Diseases in Poultry: Nutritional and Physiological Aspects

268 Inadequate pulmonary vascular capacity and susceptibility to pulmonary hypertension syndrome in broilers. R. F. Wideman*, *University of Arkansas, Fayetteville.*

Broilers are susceptible to pulmonary hypertension syndrome (PHS, ascites) when their pulmonary vascular capacity (PVC) is anatomically or functionally inadequate to accommodate the requisite cardiac output (CO) without an excessive elevation in pulmonary arterial pressure (PAP). The consequences of an inadequate PVC include sustained pulmonary hypertension (PH), systemic hypoxemia, right ventricular hypertrophy, and right A-V valve failure leading to central venous hypertension. PVC encompasses anatomical constraints related to the compliance and effective volume of blood vessels, as well as functional limitations related to the tone (degree of constriction) maintained by the precapillary arterioles within the lungs. Anatomical constraints on PVC are evident from poor correlation between lung volume and body mass, coupled with elevated pulmonary vascular resistance (PVR) attributable to non-compliant, fully engorged vascular channels. Surgical occlusion of one pulmonary artery halves the anatomical PVC, doubles the PVR, eliminates PHS-susceptible broilers, and reveals PHS-resistant survivors whose lungs are innately capable of handling sustained increases in CO. Intravenous microparticle injections also increase the PVR and trigger PH sufficient in magnitude to eliminate susceptible broilers while identifying robust survivors to serve as progenitors of PHS-resistant commercial lines. The microparticles obstruct pulmonary arterioles and cause local tissues and responding leukocytes to release vasoactive substances. Eicosanoid vasodilators of mammalian lungs (PGI₂, PGE₂) are ineffective in broilers, whereas the vasodilator nitric oxide (NO) and vasoconstrictors TxA₂ and serotonin (5-HT) are highly effective. NO is the principal vasodilator and 5-HT is the principal vasoconstrictor involved in the PH response and ensuing mortality triggered by i.v. microparticle injections. These observations demonstrate that susceptibility to PHS is a consequence of anatomically inadequate pulmonary vascular capacity combined with the functional predominance of 5-HT mediated vasoconstriction over NO mediated vasodilation. Supported by USDA/CSREES/NRI 2003-35204-13392

Key Words: ascites

269 Patho-physiology of heart failure in broiler chickens: Structural, biochemical, and molecular characteristics. A. Olkowski*, *University of Saskatchewan, Saskatoon, SK, Canada.*

Modern strains of fast growing meat type poultry are highly susceptible to heart failure. Heart related mortalities are observed predominantly in fast growing broiler chickens, with ascites and sudden death syndrome (SDS) being the most common heart related conditions in modern broiler flocks.

This paper examines the role of structural, molecular, and biochemical factors pertinent to patho-physiology of heart failure in fast growing broilers. Evidence explaining the pathogenesis of both acute and chronic heart failure, in the context of the underlying molecular and biochemical changes in the cardiomyocytes, contractile apparatus and extra-cellular matrix in the ventricular myocardium, are critically evaluated and discussed with reference to the clinical signs associated with deterioration of heart pump function. The secondary pathophysiological effects on the cardiovascular system, resulting from

hemodynamic changes associated with failing heart pump, are also reviewed and critically discussed.

Key Words: broiler, heart failure, patho-physiology

270 The response of the heart and vasculature to hypoxia, pressure and volume. R. J. Julian*, *University of Guelph, Guelph, ON, Canada.*

Heart muscle is very sensitive to hypoxic damage. Mild generalized lack of oxygen or other pathology may result in death of individual cardiac myocytes. As the heart wall muscle thins the ventricle wall dilates, the myocytes lengthen and the chamber enlarges. This heart structural response is described as dilatory cardiomyopathy. This response occurs in spontaneous turkey cardiomyopathy and in furazolidone toxicity. The heart muscle response to increased pulmonary or systemic blood pressure (increased workload) is hypertrophy of cardiac myocytes by adding sarcomeres in parallel to make myocytes and the ventricular wall thicker. This response can be seen in the right ventricular wall in pulmonary hypertension syndrome in broiler chickens and in the left ventricle in sudden death in turkeys. The response to reduced pressure is atrophy with the individual myocytes and the ventricular wall become thinner as occurs in the left ventricular wall in broilers in chronic right ventricular failure. The response to increased blood volume is enlargement of the heart because of enlargement of the chambers by myocytes becoming longer as sarcomeres are added in sequence. This may have a physiologic or pathologic etiology. The pulmonary arterioles are not sensitive to local hypoxia but air capillary hypoxia causes immediate contraction of pulmonary arterioles that may result in pulmonary hypertension and increased pressure in the pulmonary arteries and right ventricle. Increased blood volume through the pulmonary arteries and blood capillaries may also cause pulmonary hypertension as may high blood viscosity, or lung pathology reducing vascular space. The response of pulmonary arterioles to increased pressure is hypertrophy of the myocytes of the arteriolar wall with thickening of the wall and a reduction in lumen size. This structural change further increases pulmonary arteriolar pressure resulting in increased pressure in the right ventricle which if sustained results in right ventricular failure. Hypertension in other arteries may cause fibromuscular dysplasia of the artery wall or necrosis with rupture of the artery. These responses will be described and illustrated.

Key Words: heart, vasculature, pathophysiology

271 Metabolic and cardiovascular diseases in poultry: Role of dietary fat. G. Cherian*, *Oregon State University, Corvallis.*

Due to progress and advances in genetic selection, the modern day commercial broiler chicken has a fast growth rate, high feed conversion ratio and metabolic rate. These features also predispose broiler birds to metabolic diseases such as pulmonary hypertension syndrome, cardiac arrhythmias and sudden death, with a mortality rate over 5%. Despite the high economic loss to the industry, very little research effort has been directed toward investigating the role of dietary fatty acids in minimizing the risk of metabolic diseases in broiler chickens. In the US, supplemental dietary fat provided as an animal-vegetable blend

contributes a major portion of the calories in high-energy rations for broiler birds. These fats are rich in linoleic (n-6) fatty acids and are low in n-3 fatty acids. Long chain (>20-carbon) n-6 and n-3 fatty acids such as arachidonic acid (20:4n-6) and eicosapentaenoic acid (20:5n-3), derived from linoleic (18:2n-6) and alpha-linolenic (18:3n-3) acids, respectively, are the precursors for eicosanoids. Eicosanoids derived from n-3 fatty acids are less proinflammatory than those derived from n-6 fatty acids. The author's previous research demonstrated a total depletion of long chain n-3 fatty acids in cardiac and liver tissues and an increase in arachidonic acid in hepatic and cardiac triglycerides of birds succumbing to sudden death when compared to control birds. Developing dietary strategies in poultry that enhance the n-3 fatty acid content of tissues during growth may lead to better health by reducing inflammatory disorders and metabolic disease-related pathologies. However, increasing long chain n-3 fatty acids in broiler chickens is also associated with lipid oxidation and issues of muscle product quality. Therefore, alternative strategies for enhancing tissue n-3 fatty

acid content without affecting growth and product quality must be devised. The role of maternal (yolk) fatty acids in modulating the long chain n-3 fatty acid content of vital tissues and eicosanoid production in growing chickens fed a diet lacking in long chain n-3 fatty acids was investigated. Up to day 35 of growth, the cardiac and hepatic tissues of chicks hatched from hens fed a high n-3 diet retained higher levels of docosahexaenoic acid (22:6n-3) and total n-3 fatty acids than those of chicks hatched from hens fed a low n-3 diet. The effect of maternal diet on eicosanoid production was evident up to day 14 of growth. Chicks hatched from hens fed a high n-3 diet produced less proinflammatory eicosanoids than chicks hatched from hens fed a low n-3 diet. Modulating maternal dietary n-3 fatty acids enhances tissue retention of n-3 fatty acids during growth and reduces proinflammatory eicosanoid production in chicks, which could lead to fewer metabolic and inflammatory-related disorders in poultry.

Key Words: n-3 fatty acids, metabolic diseases, eicosanoids

Poultry Welfare Symposium: Poultry Welfare Symposium: Realistic Views Concerning Poultry Welfare

272 Historical perspective on the development of poultry welfare. A. J. Pescatore*, *University of Kentucky, Lexington.*

Domestic animals are dependent on man for their existence and their well being. Well being is the satisfaction of basic physical needs and the encouragement of necessary behaviors. Societal involvement in welfare began in the 1860's as local societies for the prevention of cruelty to animals were being created to prevent the abuse of horses, this continued with the passage of the 28-hour law in 1873 which required animals to have feed, water and rest every 28 hours. In 1926 the transportation of poultry was addressed when shippers agreed to uniform coops and standards. This was an era that centered on protection of the birds from the elements. This was also a time for documenting many disease conditions. Pullorum Disease and protozoan diseases were major concerns in the 1930's. Many great nutritional discoveries were made at this time. The post WWII era saw the movement of birds into poultry houses. Space allocation, heat stress and cannibalism were welfare topics. The 1950's saw the refinement of the housing systems. Social aggression was a concern with numerous prevention strategies including tranquilizers. In 1958 the Humane Slaughter Act took effect and even though it did not apply to poultry it encouraged research on the humane slaughter of poultry. The understanding of the interaction of stress and disease began in the 1960's. This was the time that pharmaceuticals had a role in poultry welfare. The next decade introduced social indexes and multi focus studies. Blood parameters became a common measure of well being. The late 1970's saw the emergence of metabolic diseases as welfare issues. In the 1980's there was more emphasis on social rank and stressors and less emphasis on productivity. Measures of welfare involved the interaction of behavior with physiology and immunity. The late 1980's and 1990's enter an era of increase emphasis on behavior especially the fear response. Alternative systems for housing and processing were topics of concern. Cellular immunity was used as an indicator of stress. In the current decade consumer pressure is the driving force for poultry welfare. Consumer perception and independent audits characterize current trends.

Key Words: poultry welfare, animal well-being

273 Animal care guidelines and future directions. A. B. Webster*, *The University of Georgia, Athens.*

Two views are broadly accepted in developed societies which have made animal care guidelines inevitable. These are that domestic animals are sentient, and that humans are responsible to ensure the proper care of domestic animals. Despite these common views, people, having differing moral understandings of the human-animal relationship, are sharply divided over how these views should be applied to domestic animal care. Animal care guidelines have been developed in different nations at several organizational levels to represent a compromise that is acceptable to a majority of people. These organizational levels include individual poultry companies, national poultry associations, individual customers of the poultry industry, national associations of customer companies, national governments, and international organizations. Animal care guideline development has typically included input from producers and scientists, and depending on the sponsoring organization, animal advocates and government representatives as well. Animal advocacy groups have also sought to influence domestic animal care by campaigning against animal production practices, or by offering their preferred guidelines for poultry companies to adopt in the hope that the endorsement of the welfare group would add value to the product. Originally, animal care guidelines were only recommended, with little or no requirement for compliance. In recent years, the need for retail companies to assure certain welfare standards has led to animal welfare auditing of poultry facilities. Animal care guidelines primarily have sought to establish standards for handling and husbandry in existing production systems. Future guidelines may call for adoption of alternative management practices or housing systems based on the combined assessment of welfare improvement, feasibility and market access. International animal care guidelines are now being developed on two levels, i.e., among national governments to create a common standard for trade in animal products, and within international retail companies to create company-wide animal care standards. These initiatives may eventually unify animal care standards world-wide.

Key Words: animal care guidelines, poultry welfare