INTERNATIONAL JOURNAL OF INNOVATIVE RESEARCH & DEVELOPMENT

ISSN: 2278 – 0211 (Online)

Fast Growth and its Demerits Associated with Broilers

Patil Ajay Dadaji

Research Scholar, Shramsafalya, Samartha Nagar,

New Sanghvi, Pune, Maharashtra, India

Dr. Sakharam Balu Patil

Head Dept. of Zoology,Hutatma Rajguru Mahavidyalaya,
Rajgurunagar, Tal. Khed, Dist. Pune., Maharashtra. India

Abstract :

Over the last few decades, genetic choice for ascension and improved feed potency has been very effective in meat-type poultry. Combined with changes within the feed that have inflated both the nutritionary and physical density to encourage a high nutrient intake, rate of growth has quite doubled. The impact of genetic choice for high muscle to bone ratio and high calorie intake of a ration that provides all nutritionary needs causes vital mortality from cardiovascular disease. in the chicken, sudden death syndrome (flip-over) and pulmonary cardiovascular disease syndrome leading to ascites are the foremost vital.

Ruptured artery, spontaneous turkey, cardiomyopathy (round heart) and cardiomyopathy inflicting sudden death turn out high mortality in turkeys. Ascension induced by high nutrient intake alone will cause severe lameness, bone defects, and deformity, as these issues are seen in animals that haven't been chosen for fast growth: dogs, horses, pigs, ratites and wild birds kept in zoology gardens.

In meat-type poultry, growth-related illness are often reduced or eliminated by reducing feed intake without touching final weight. Ascension alone might not be the infective mechanism that leads to cardiovascular or musculoskeletal defects. Metabolic imbalance induced by high nutrient intake could cause some of the conditions. These metabolic issues may well be corrected without reducing rate of growth.

1.Introduction

Fast development and heavy body weight, chiefly related with a small skeletal frame, have been drawn in musculoskeletal and cardiovascular disease in meat-type poultry (Riddell, 1992; Julian, 1993; Lilburn, 1994). Research on these conditions has regularly produced contradictory results. Though many of the problems can be abridged or eradicated by slowing growth rate, fast development and weight do not essentially effect in disease. Possibly we should deem whether some of these problems are the result of metabolic imbalances associated with fast development and should be more appropriately called metabolic disease.

2.Ascites

Ascites caused by valvular insufficiency and right ventricular failure (RVF) following right ventricular dilation and hypertrophy from pulmonary high blood pressure (PH) has become a prominent reason behind sickness, death, and condemnation in meat-type chickens. Ascension in a bird with insufficient pulmonary vascular capability is the primary reason behind pH, and when it's associated with ascension it's been known as pulmonary hypertension syndrome (PHS) (Huchzermeyer and de Ruyck, 1986; Julian, 1993). There are ranges of additional or secondary causes, which will increase the incidence of PHS.

Additive factors that increase the incidence of ascites could result from higher blood flow as a result of higher rate (cold, heat, certain nutrients or chemicals, etc.) or larger resistance to flow that is the result of hypoxemia increasing blood viscousness (high altitude, rickets, disease, reduced oxygen transfer), red blood cell rigidity, or reduced vascular capability within the lung (Julian and Goryo, 1990; Julian and Squires, 1994; Mirsalimi et al., 1992, 1993).

3.The Anatomy And Physiology

The anatomy and physiology of the avian respiratory system are necessary within the susceptibility of meat-type chickens to PHS. The tiny stature of the modern meat type chicken, the large, heavy breast mass, the pressure from abdominal contents on air sacs, and the small lung volume might all be concerned within the increased incidence of PHS. The lungs of birds are smaller as a proportion of weight than those of mammals, and the lungs of meat-type chickens are smaller than those of Leghorns (Julian, 1989). The lungs of birds are also firm and stuck within the thoracic cavity. They do not expand and contract with every breath as mammalian lungs do. The blood and air capillaries form a network that enables the tiny blood capillaries of the lung to dilate solely very little to accommodate increased blood flow. Increased blood flow to provide the oxygen needed for metabolism in aggressive meat-type chickens causes a rise within the blood pressure needed to push the blood through the blood capillaries within the lung (pulmonary hypertension).

This increase in employment for the correct facet of the center leads to sporadic cases of RVF and ascites. Secondary factors that increase the oxygen requirement of the bird, scale back oxygen pick-up or transfer within the lung or release in the tissue, scale back oxygen carrying capability of the blood, or increase blood volume or interfere with blood flow through the lung (polycythemia with increased blood viscousness, increased red blood cell rigidity, lung pathology narrowing or occluding capillaries) might end in flock outbreaks of ascites caused by PHS (Monge and Leo´n-Velarde, 1991; Julian, 1993; Diaz et al. 1994a).

When pH happens, the proper ventricle responds very quickly to the increased workload, as muscle will, by enlargement (hypertrophy). If pH continues, the proper ventricle needs to pump against that pressure, and also the muscle wall continues to thicken and enlarge, increasing the pressure within the respiratory organ. The correct heart valve may be a muscle flap made up primarily of fibers from the correct ventricle. Because the ventricle thickens (hypertrophies) and dilates, the correct heart valve additionally thickens and becomes stiff till it no longer covers the gap back to the body, at which time valvular insufficiency happens.

This leads to a volume overload and causes dilation and right ventricular failure. The augmented blood pressure within the veins, liver, and abdominal vessels forces plasma fluid (edema) out of the vessels, notably the fenestrated sinusoids of the liver, into the peritoneal areas, wherever it's referred to as ascitic fluid. The condition is termed ascites (waterbelly) (Wilson et al., 1988). There's unremarkably a small amount of fluid within the eight peritoneal areas that drains back to the blood stream by the lymph channels. However, the augmented pressure in the vena cava in broilers with valvular insufficiency interferes with lymph return, and therefore the fluid accumulates in the peritoneal areas (Julian, 1993). Most of the fluid is found within the ventral hepato-peritoneal areas and in the pericardial space. There is also fluid in the very slim pleural areas and in the abdominal cavity. Fluid is not found in the air sacs. It's the pressure from this fluid on the air sacs that causes the respiratory signs and death.

If the capillaries are the resistance vessels in pH in broiler chickens, right ventricular hypertrophy would lead to increased pressure in the commonly low pressure animal tissue of the lung and would cause interstitial and air capillary edema, which might increase the thickness of the respiratory membrane as represented by Maxwell et al. (1986) and will lead to hypoxemia. This augmented pressure would additionally decrease the dimensions of the already small blood capillaries and will further increase the resistance to blood-flow. Severe pulmonary edema because the result of pH would lead to hypoxic respiratory failure and death and would justify the increasing incidence of death without ascites in PHS.

These broilers show marked right ventricular hypertrophy and lung edema and die from lung edema. The unexpected boost in PHS in meat-type chickens in the 1980s was connected with a quick increase in rate of growth and feed conversion. These will increase were a result of a mixture of genetic selection for fast-growing, significant broilers with low feed conversion and a more dense, high caloric, pelleted feed that provided all the nutrients needed for rapid growth and inspired a high nutrient intake (Havenstein et al., 1994). In meat type chickens, PHS is typically primary pulmonary hypertension; there's no proof of prior heart or lung disease that would account for the rise in blood flow or resistance to flow that leads to the multiplied pressure within the pulmonary arteries.

Ascites caused by pH is a production-related disease at low altitude. It will be prevented simply by proscribing rate of growth (Arce et al., 1992; Julian et al., 1995). It's potential that some meat-type chickens of the phenotype we have created have reached the limit of blood flow through their lungs, and future enhancements in rate of growth can solely be attainable if the lung and abdominal cavity capacities are enlarged. Ascites at moderate (above 800 m) and high altitude may be a far more severe drawback owing to the polycythemia evoked by hypoxia, however proscribing rate of growth will reduce it.

Research on oxygen hemoglobin saturation in meat type chickens indicates that fast-growing broilers have lower O hemoglobin saturation than slow-growing broilers (Julian and Mirsalimi, 1992). These results counsel that some meat-type chickens aren't totally oxygenating their hemoglobin even at low altitude.

4.Skeletal Deformities

Skeletal deformities may be caused in a variety of ways. Organic process deficiencies may result in skeletal disease in all birds. Quickly growing birds have a better need for specific nutrients, and plenty of skeletal defects in broiler and roaster chickens are rare or absent in slower growing strains (Havenstein et al., 1994).

Mechanically elicited or trauma-associated issues are way more frequent in fast-growing broilers. These issues might have additional to do with immatureness and weight than ascension as a result of tissue becomes stronger and additional resilient with age. This age-related result is especially true of bone, tendon, and ligament.

Toxins in feed or water will cause skeletal deformities. Toxin effects aren't sometimes related to ascension, though quickly growing birds would consume a lot of the offending product. Genetic faults can also end in skeletal defects, and aren't sometimes growth connected.

If the hypothesis that ascension leads to musculoskeletal deformity is valid, maybe we should contemplate how specific defects might be related to ascension. 1) The defect could be associated with high weight. 2) The defect might occur, as a result of tissues (bones, ligaments, tendons, and muscles) is immature. The production of sturdy tissue, remodeling and alignment of bone needs more time than ascension permits. 3) The defect might be associated with high specific nutrient, enzyme, hormone, or oxygen demand by specialized cells (proliferating chondrocytes). The defect might be associated with metabolic by-products (lactic acid, carbon dioxide) that are raised by ascension. 5) Young speedily multiplying cells might be additional vulnerable to toxic or metabolic injury.

5.Angular Bone Deformity And Valgus-Varus Deformity

Angular bone deformity is the most frequent skeletal defect in broilers on a nutritionally adequate ratio (Julian, 1984). it should be seen as early as 6 to 8 d, or not become outstanding till 3 to 4 wk. it's sometimes progressive, and when young broilers are affected they typically become crippled and can't get to the feed and water. If just one leg is affected, the bird might not go down. Pain related to the deformity reduces activity and restricts feeding.

These broilers are also condemned for emaciation at process or there is also disuse atrophy of the muscle of the affected leg (Figure 1). Angular bone deformity seems to be specifically associated with ascension with inadequate time for correct alignment and re-modeling of the bones of the distal tibio-tarsus. It is reduced by slowing growth within the initial 10 to 21 d and by extended daily rest (long, dark) periods (Classen and Riddell, 1989; Fontana et al., 1992; Yu and Robinson, 1992; Gordon, 1994).

6.Acute Pain

Acute pain usually has to do with the trauma of catching and handling. Enhanced methods of catching and moving broilers to processing must be formulated to lessen traumatic injury to bones, joints, and muscles.

7.Cripples

Many of the skeletal deformities in broilers result in birds those are down and incapable to walk. There may be unceasing pain as well as anxiety coupled with aggression from other birds and with the intricacy of getting to feed and water. Many of these broilers lie on the floor until they die of dehydration. Poultry workers should be trained to remove and euthanatize crippled broilers every day.

8.References

1. 1.Arce, J., M. Berger, and C. Lopez-Coello, 1992. Control of ascites syndrome by feed restriction techniques. J. Appl. Poult. Res. 1:1–5.
2. 2.Classen, H. L., and C. Riddell, 1989. Photoperiodic effects on performance and leg abnormalities in broiler chickens. Poultry Sci. 68:873–879.
3. 3.Cook, M. E., and Y. Bai, 1994. Factors influencing growth plate cartilage turnover. Poultry Sci. 73:889–896.
4. 4.Diaz, G. J., R. J. Julian, and E. J. Squires, 1994a. Cobalt-induced polycythaemia causing right ventricular hypertrophy and ascites in meat-type chickens. Avian Pathol. 23:91–104.
5. 5.Diaz, G. J., R. J. Julian, and E. J. Squires, 1994b. Lesions in broiler chickens following experimental intoxication with cobalt. Avian Dis. 38:308–316.
6. 6.Edwards, H. M., Jr., 1984. Studies on the etiology of tibial dyschondroplasia in chickens. J. Nutr. 114:1001–1013.
7. 7.Edwards, H. M., Jr., 1987. Effects of thiuram, disulfiram and a trace element mixture on the incidence of tibial dyschondroplasia in chickens. J. Nutr. 117:964–969.
8. 8.Fontana, E. A., W. D. Weaver, B. A. Watkins, and D. M. Denbow, 1992. Effect of early feed restriction on growth, feed conversion, and mortality in broiler chickens. Poultry Sci. 71:1296–1305.
9. 9.Gordon, S. H., 1994. Effects of day length and increasing day length programmes on broiler welfare and performance. World's Poult. Sci. J. 50:269–282.
10. 10.Havenstein, G. B., P. R. Ferket, S. E. Scheideler, and B. T. Larson, 1994. Growth, livability, and feed conversion of 1957 vs 1991 broilers when fed "typical" 1957 and 1991 broiler diets. Poultry Sci. 73:1785–1794.
11. 11.Haynes, J. S., M. M. Walser, and E. M. Lawler, 1985. Morphogenesis of Fusarium sp.-induced tibial dyschondroplasia in chickens. Vet. Pathol. 22:629–636.
12. 12.Hester, P. Y., 1994. The role of environment and management on leg abnormalities in meat-type fowl. Poultry Sci. 73:904–915.
13. 13.Julian, R. J., 1989. Lung volume of meat-type chickens. Avian Dis. 33:174–176.
14. 14.Leach, R. M., and W. O. Twal, 1994. Autocrine, paracrine and hormonal signals involved in growth plate chondrocyte differentiation. Poultry Sci. 73:883–888.
15. 15.Lee, S. R., W. M. Britton and G. N. Rowland, 1980. Magnesium toxicity: bone lesions. Poultry Sci. 59:2403–2411.
16. 16.Mirsalimi, S. M., R. J. Julian, and E. J. Squires, 1993. Effect of hypobaric hypoxia on slow- and fast-growing chickens fed diets with high and low protein levels. Avian Dis. 37: 660–667.
17. 17.Monge, C. and F. Leo´n-Velarde, 1991. Physiological adaptation to high altitude: oxygen transport in mammals and

birds. Physiol. Rev. 71:1136–1171.

18. 18.Rennie, J. S., C. C. Whitehead, and B. H. Thorp, 1993. The effect of dietary 1, 25-dehydroxycholecalciferal in preventing tibial dyschondroplasia in broilers fed on diets imbalanced in calcium and phosphorus. Br. J. Nutr. 69:809–816.