GENETIC SELECTION IN TURKEYS AND BROILERS AND THEIR IMPACT ON HEALTH CONDITIONS

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Genetic selection in wild living animals favours a variety of different traits that are important for maintenance, and reproduction. In contrast selection in domesticated animals not only favours individuals that can cope best with the modified circumstances of living under human custody but also is artificially influenced by the needs of man, e. g. for fast growth or high reproductivity, but also for behavioural traits as in dog or sheep.

The poultry industry is the largest (in terms of animal numbers) and the most highly automated, vertically integrated, and intensified of the animal production industries. The productivity of domestic livestock and poultry has almost tripled in the last 100 years through the use of both improved feeding methods and genetic selection (Grandin and Deesing, 1988). The most outstanding and visible changes in modern turkey and broiler compared to their ancestors is the rapid growth, the increased body weight, and the higher percentage of breast muscle (Havenstein *et al.*, 2003b; Havenstein *et al.*, 2003a)). Due to genetic selection, the ability of a chicken to gain weight has increased phenomenally. In 2000 it took a broiler on average 34 days to reach 1.82 kg., whereas in 1966, 60 days were needed on average to reach the same weight. The figure for 1956 was 84 days. The performance of modern market turkeys produced in 2003 was compared with that of randombred turkeys started in 1966, when grown using representive 1966 and 2003 diets. The data collected indicated that the number of days to reach the weight was halved during this period (Havenstein et al., 2004). In addition, changes in husbandry, improved nutrition, and control of diseases and parasites have contributed to the escalating growth rate of meat-type poultry.

However, there is a big concern that serious animal welfare problems have already been caused by genetic selection for rapid growth and high meat production. (Grandin and Deesing, 1988). European citizens are increasingly concerned about the health and welfare of chickens and turkeys kept for meat production. In particular, a number of animal welfare organisations have initiated campaigns calling for improved welfare standards.
Broiler chickens and turkeys have been selected to grow muscle mass very quickly, but growth of the skeleton and the internal organs has not kept up. The birds have reduced cardiopulmonary capacity in relation to their muscle mass, and cannot withstand much physical exertion (Julian et al., 1986; Broom, 1987; Broom, 1993; Julian, 1993; Norci and Montella, 2003; Havenstein et al., 2004). Selection for rapid increases in muscle mass is highly correlated with selection for increased appetite drive. Examinations on chickens showed that birds selected for egg production stop eating when their metabolic needs are met, but broiler chickens selected for meat production do not stop eating until their gut is completely full (Nir et al., 1978). The lack of synchronous growth among body components in broilers, including the heart and lungs, can contribute to pulmonary hypertension causing excess fluids in the body (ascites). An additional problem is "sudden death syndrome", the cause of which is unknown. A correlated response to the selection of turkeys for increased body weight and a broad breast is the development of deep muscle myopathy (atrophy of the inferior pectoralis muscle) caused by an inadequate blood supply to the tissues.
Both meat chickens and turkeys exhibit skeletal disorders, particularly in the bones of the pelvic limb and their associated tendons. These disorders are not necessarily associated with

body weight or conformation, but instead with the differential growth of body parts, particularly accelerated growth of muscle that is not commensurate with skeletal development. Comparison of today's broilers with a randombred control that represented broilers of the year 1957 showed only half the mortality in the 1957 strain. Most of the mortality in the modern line was due to leg-associated problems (Havenstein *et al.*, 2003b).
The most relevant health problems, which seem to be related to genetic selection, although they were described for many years are summarized in table 1.

Table 1: The most relevant health problems, which seem to be related to genetic selection in meat poultry

Affected organ systems	Disease conditions
Circulatory system	Sudden death Ascites Aortic rupture Perirenal haemorrhage
Musculoskeletal system and legdisorders	Dyschondroplasia, Deep pectoral myopathy Pododermatitis
Others	Breast blister Susceptibility to infectious diseases

Sudden death syndrome (SDS)
SDS is poorly understood. SDS is a major cause of mortality in broilers, and in Europe its reported incidence is between 0.1 and 3%. SDS is an acute heart failure disease that affects mainly male fast-growing chickens which seem to be in good condition (Newberry et al., 1987). SDS is characterised by sudden vigorous wing-flapping, muscle contractions and obvious loss of balance. In the final phase the birds fall on their back or to the side and die. The affected birds generally are of good health and well nourished when they die. At post mortem symptoms of a shock can be seen (Crespo and Shivaprasad, 2003). Birds that later died of sudden death syndrome had a higher heart rate and the incidence of cardiac arrhythmias was higher than average (Olkowski et al., 1997; Olkowski and Classen, 1998). On the other hand the heritability is low (Chambers, 1986).

Ascites
Ascites affects fast growing chickens when the right side of their hearts becomes enlarged in response to increased workload during the bird's rapid growth. The bird has to breathe more rapidly and its lungs become congested. The liver function is affected and the abdomen becomes swollen with fluid increasing the risk of heart failure.
Ascites and sudden death syndrome (SDS) are relatively common and are likely to be due to the fact that the broilers' fast growth requires high levels of oxygen to support metabolic demands. All their energy is spent on growth and efficient feed conversion, leaving them short of oxygen for their other bodily needs so that their hearts have to work much harder (Maxwell and Robertson, 2000).

Aortic rupture

Aortic rupture or dissecting aneurysm is characterized by sudden death in growing turkeys due to internal haemorrhage. Mortality in the past has been reported to reach 50%, but losses in affected flocks at present reach usually only 1-2%. The condition occurs in male turkeys with a peak between 12 and 16 weeks of age.

Affected birds die suddenly. Gross and microscopic lesions have been described by McSherry et al. (1945). The abdominal aorta is affected, where a longitudinal slit can be seen. Blood clots are present in the body cavity and the birds may bleed from the mouth. Several causes are suspected (Fig. 1). Amongst the possible causes high blood pressure and copper deficiency are a contributing factor (Crespo and Shivaprasad, 2003), but paradoxically, the administration of diethylstilbestrol decreased blood pressure and increased the incidence of aortic aneurysm (Krista *et al.*, 1965a; Krista *et al.*, 1965b). Diets containing high levels of protein and fat may increase the incidence of aortic rupture (Pritchard et al., 1958). Low copper levels were found in the livers of turkeys from field outbreaks of aortic rupture (Graham, 1977) and coronary artery aneurysm (Shivaprasad and Crespo, 1997). As in today's large white turkeys systemic blood pressure is doubled as high than in wild turkeys these lines also are predisposed for aortic rupture (Krista *et al.*, 1965a). According to Peckham (1978) broad-breasted bronze turkeys are more susceptible to this syndrome than Beltsville-white turkeys.

Fig. 1 : Some possible causes of *Aortic rupture*

Non infectious	Infectious
	Not known
Genetic	
Rapid growth	
High blood pressure of some birds	
Nutrition	
High protein content	
High fat content	
Copper deficiency	
Management	
Stress	
Stocking density	
Disquietness in the animal house	

Sudden death in turkeys associated with perirenal haemorrhage

Sudden death in turkeys associated with perirenal hemorrhage (SDPH) has been recognized as a significant cause of mortality in male turkeys between 8 and 14 weeks of age. The dead turkeys are in good condition, with food in their crops and the remainder of the gastrointestinal tract. They have congested and oedematous lungs, splenomegaly, congested livers and digestive tracts, and clotted blood surrounding a portion or the whole of the kidneys (Crespo and Shivaprasad, 2003). It often is associated with perirenal hemorrhage and hypertrophic cardiomyopathy affecting the left ventricle and the intraventricular septum (Larochelle et al., 1992). The renal hemorrhage may result from severe passive congestion, which may be compounded in part by closure of the renal valve in the renal portal circulation (Larochelle et al., 1992). Fast weight gain, continuous lighting programs, crowding, and hyperactivity have been suggested as factors that may influence the incidence of SDPH (Mutalib and Hanson, 1990). Increased room temperature, toe clipping, step up/step down lighting, and dietary reserpine reduced the incidence of SDPH (Frank et al., 1990). Dietary

aspirin had no effect on the incidence of SDPH (Boulianne and Hunter, 1990; Frank et al., 1990).

Diseases of the Musculoskeletal system and Leg disorders

Diseases of the musculoskeletal system and leg disorders, currently, receive a lot of attention as a cause of concern from the animal welfare point of view and are mostly accompanied with economic losses for turkey meat producers due to mortality, predisposition to cannibalism, retardation of growth, increase of the condemnation rate and downgrading at the processing plant. Skeletal disorders, in particular different forms of leg weakness can impair the birds' welfare by reducing walking ability, in severe cases linked with pain and discomfort. Bone weakness and deformations may result in bone fractures during catching and at the slaughter process. The causes of leg disorders can be infectious and non-infectious. Beside genetic reasons, management and environment are two further important factors that can influence leg disorders (Fig. 2).

The incidence of leg problems are significantly higher in stuffy and poor ventilated houses and houses with poor litter condition, high dust concentration, and inappropriate adjusteded feeders and drinkers. Further environmental factors such as light intensity, light duration, feed restriction, floor type, lack of exercise, high brooding temperature, poor sanitation and general stress are shown to be involved in leg disorders in meat turkeys (Julian, 1984; Julian, 1985; Cummings, 1987; Schuhwerk, 1989; Heim, 1990; Ferket, 1992; Thorp, 1994; Hafez, 1996; Hafez, 1999).

Attempts to treat leg weakness are unrewarding. Affected birds should be separated from the healthy animals (animal welfare). Addition of vitamin D3 in drinking water and/or calcium-phosphorus- preparation in the feed can improve the condition. The incidence of skeletal disease can also be reduced by providing suitable litter and flooring, by providing adequate bird exercise and good husbandry techniques. Genetic selection to reach stable skeletal and body balance must be progressed.

The most common problems of the musculoskeletal system that are related to fast growth are dyschondroplasia, footpad dermatitis and deep pectoral myopathy.

Fig. 2 : Some possible causes of leg disorders

Non infectious	**infectious**
Genetic	***Viral agents***
Rapid growth	Reovirus
Nutrition	***Bacterial agents***
	Mycoplasma
Management	E. coli
Litter quality	P. multocida
Stocking density	Streptococci, Staphylococci
Lighting programme	ORT
Exercise	
Deviation in hatching	

Dyschondroplasia is a common defect associated with the growth plates of meat-type poultry. It is characterized by an avascular plug of abnormal cartilage in the growth plate of long bones. It is most commonly recognized in the proximal tibiotarsus, and, hence, the condition is often described as tibial dyschondroplasia **(TD)** (Poulos, 1978). The incidence and severity of TD can be influenced by nutrition and genetic selection (Farquharson and Jefferies, 2000).

TD is a specific form of growth plate abnormality and at post-mortem is visible as an abnormal mass of cartilage under the growth plate. Lameness only occurs if there is deformity or enlargement with loss of bone strength that results in weakness, fracture, or necrosis. Its incidence in broilers partly is hereditary as the selection of strains with low and high incidence of TD respectively show, but can be influenced by the diet (Riddell, 1976).

Numerous investigators have shown that the occurrence of TD is susceptible to genetic selection. On the other hand other investigations showed that, although reduction of the growth rate in broiler was accompanied by a decreased incidence of TD, there was no direct correlation between the growth of individual birds and the incidence of tibial dyschondroplasia (Riddell, 1981; Kuhlers and McDaniel, 1996). Hafez et al. (2004) investigated five commercial meat turkey lines (Kelly Bronze, Nicholas 300, BUT 9, Nicholas 700, and BUT-Big 6) regarding leg disorders. Under commercial rearing and feeding conditions these turkey lines are characterised by different body weight development. The obtained results revealed that there was no correlation between turkey lines and the incidence of tibial dyschondroplasia, fractures, and arthritis.

Footpad dermatitis

Footpad dermatitis, also known as plantar pododermatitis is a condition characterized by lesions on the ventral footpad of poultry. The lesions vary from hyperkeratosis to severe erosions and ulceration. Generally, the plantar area of the foot and the weight bearing metatarsal pads are mostly affected. Several predisposing factors such as genetic line, rapid growth, feed, stocking density, and the litter quality have been discussed. Field outbreaks of contact dermatitis have been associated with poor litter conditions (Martland, 1984; Green et al., 1985), high stocking density and increased age (McIlroy et al., 2005). Irritation from faeces or litter causes a thickening of the foot-pad epidermis (acanthosis and hyperkeratosis). If faeces stick to the foot it may cause ischemic necrosis and ulceration that is accompanied by suffering and pain (Julian and Gazdzinski, 1999). In addition, deficiency of biotin has been suggested as a possible cause of pododermatitis in experimental birds ((Harms and Simpson, 1975). Other investigations showed that supplemental biotin reduced the pododermatitis only in poults maintained on dry litter, but not on wet litter (Harms et al., 1977). Hafez et al. (2004) investigated five commercial meat turkey lines (Kelly Bronze, Nicholas 300, BUT 9, Nicholas 700, and BUT-Big 6) regarding leg disorders. Under commercial rearing and feeding conditions these turkey lines are characterised by different body weight development. The obtained results showed that the turkey lines had a statistically significant effect upon the distribution of foot-pad statuses (grades 0-3). There seems to be a correlation between body weight development and the severity of foot-pad dermatitis. The observed incidence of grade 3 lesions in turkey line Big 6 (37%) was clearly higher than in other lines and statistically significant. However, several risk factors that are involved with foot-pad dermatitis, such as feed composition, litter condition and stocking density were similar for all groups in the described investigations. Preliminary results of another investigation show that the elevation of energy content in the feed and supplementation with biotin seem to have a positive effect upon the foot-pad health (Hafez et al., 2004).

Deep pectoral myopathy can only be diagnosed post mortem and does not lead to clinical signs; however it leads to the condemnation of the affected muscles at slaughtering. Gross lesions are swollen, in the beginning pale, later on greenish supracoracoid muscles. It is caused by ischemia in the supracoracoid muscle following exercise when lactic acid is not carried away at a sufficient rate (Harper et al., 1975). Heavily muscled meat-type turkeys and chickens causes are more susceptible. Some evidence has been produced for a hereditary predisposition (Harper et al., 1975). This predisposition may be related to inadequate vasculature in muscles of meat-type birds (Wight and Siller, 1979). No specific nutritional factors are known to influence the condition (Harper and Helfer, 1972; Grunder et al., 1979), but food restriction may reduce the incidence (Wight and Siller, 1979).

Breast-blisters

Breast-blisters are an encapsulated inflammation of the bursa praesternalis. The condition was firstly observed by Hodgson and Gutteridge (1941) and is mostly accompanied by an increase of the condemnation and downgrading at the processing plant. The course of the disease is variable and influenced by different factors such as management, poor litter quality, high stocking density, poor feathering of the breast, leg disorders that lead to increased lying down, lack of exercise as well as the involved microorganisms (Bergmann and Scheer, 1977). Infectious and non-infectious causes were incriminated with the septic sternal bursitis (Fig. 3). Management procedures to optimise the litter quality and to increase the activity of the birds (Exercise) are important. In addition, genetic selection toward fast feather development can improve the situation.

Fig. 3 : Some possible causes of breast blisters

Non infectious	Infectious
Genetic	***Bacterial agents***
Rapid growing	Mycoplasma synoviae
Slow feather development	Staph. aureus.
Management	
Litter quality	
Stocking density	
Mechanical- traumatic injuries	
Humidity	
Exercise	

Susceptibility to infectious diseases

Little is known about diseases with hereditary aetiology that are not connected to the body weight. It is known however that turkey lines that are selected for high body weight are more susceptible to different diseases than a randombred control started in 1966. The selected turkey lines had a higher mortality and a lower antibody titer after experimental infection with Pasteurella multocida (Sharaf et al., 1988; Nestor et al., 1996). Similar effects were seen after natural infection with Erysipelothrix rusiopathiae (Saif et al., 1984) and experimental infection with Newcastle disease virus (Tsai et al., 1992).

In broilers a decreased relative weight of a variety of organs, including bursa, spleen and cecal tonsils has been shown. Today's broilers seem to have a decreased adaptive arm of the immune response but an increase in the cell-mediated and inflammatory responses (Cheema et al., 2003). It is not known however if this has consequences for the effectiveness of the immunresponse against infective agents.

Taken together the genetic progress can accompanied by several health and welfare related problems. Those can not be solved solely by improved management as optimal litter, an optimized diet, or vaccines for preventing infectious diseases. From an animal-welfare point of view it is necessary to select not only for the economic parameters but also for improvement of health conditions of the birds.

References

BERGMANN, V. and SCHEER, J. (1977). Beitrag zur Differenzialdiagnose der Bewegungsstörungen beim Junghuhn. 5. Mitt. Erkrankungen des knöchernen Skeletts beim Geflügel. *Mh. Vet. Med.* **32**: 141-148.

BOULIANNE, M. and HUNTER, D. B. (1990). *Aspirin: A treatment for sudden death syndrome in turkeys?* Proceedings of the 39th Western Disease Conference: 89-90.

BROOM, D. M. (1987). Applications of neurobiological studies to farm animal welfare. *Curr. Top. Vet. Med. Anim. Sci.* **42**: 101-110.

BROOM, D. M. (1993). A usable definition of animal welfare. *J. Agric. Environ. Ethics* **6**: 6.

CHAMBERS, J. R. (1986). Heritability of crippling and acute death syndrome in sire and dam strains of broiler chickens. *Poult Sci* **65**: 164.

CHEEMA, M. A., QURESHI, M. A. and HAVENSTEIN, G. B. (2003). A comparison of the immune response of a 2001 commercial broiler with a 1957 randombred broiler strain when fed representative 1957 and 2001 broiler diets. *Poultry Science* **82**: 1519-1529.

CRESPO, R. and SHIVAPRASAD, H. L. (2003). Developmental, metabolic, and other noninfectious disorders. in: *Diseases of Poultry*. Y. M. SAIF, H. J. BARNES, J. R. GLISSON, A. M. FADLY, L. R. MCDOUGALD and D. E. SWAYNE. Ames, Iowa, Iowa State Press. 1055-1102.

CUMMINGS, T. (1987). Turkey leg weakness: Present problem economically important cause largely undetermined treatment unrewarding. *Turkey World*: 16-17.

FARQUHARSON, C. and JEFFERIES, D. (2000). Chondrocytes and longitudinal bone growth: the development of tibial dyschondroplasia. *Poult Sci* **79**: 994-1004.

FERKET, P. R. (1992). Leg problem in turkey toms: Influence of nutrition and Management. *Turkeys*: 23-26.

FRANK, R. K., NOLL, S. L., EL HALAWANI, M., NEWMAN, J. A., HALVORSON, D. A. and RUTH, G. R. (1990). Perirenal hemorrhage syndrome in market turkey toms: effect of management factors. *Avian Dis* **34**: 833-842.

GRAHAM, C. L. (1977). Copper levels in livers of turkeys with naturally occurring aortic rupture. *Avian Diseases* **21**: 113-116.

GRANDIN, T. and DEESING, M. J. (1988). Genetics and Animal Welfare. in: *Genetics and the Behaviour of Domestic Animals*. T. GRANDIN. San Diego, California, Academic Press. 319-341.

GREEN, J. A., MCCRACKEN, R. M. and EVAN, S. R. T. (1985). A contact dermatitis of broilers - clinical and pathological findings. *Avian Pathology* **14**: 23-38.

GRUNDER, A. A., HOLLANDS, K. G. and GAVORA, J. S. (1979). Incidence of degenerative myopathy among turkeys fed corn or wheat based rations. *Poult Sci* **58**: 1321-1324.

HAFEZ, H. M. (1996). Übersicht über Probleme der haltungs- und zuchtbedingten Erkrankungen bei Mastputen. *Arch. Geflügelk.* **60**: 249-256.

HAFEZ, H. M. (1999). Gesundheitsstörungen bei Puten im Hinblick auf die tierschutzrelevanten und wirtschaftlichen Gesichtspunkte. *Arch. Geflügelk.* **63**: 73 -76.

HAFEZ, H. M., WÄSE, K., HAASE, S., HOFFMANN, T., SIMON, O. and BERGMANN, V. (2004). *Leg disorders in various lines of commercial turkeys with especial attention to pododermatitis*. Proceedings of the 5th International Symposium on Turkey Diseases, Berlin, (ISBN: 3-938026-15-4): 11-19.

HARMS, R. H. and SIMPSON, C. F. (1975). Biotin deficiency as a possible cause of swelling and ulceration of foot pads. *Poult Sci* **54**: 1711-1713.

HARMS, R. H., DAMRON, B. L. and SIMPSON, C. F. (1977). Effect of wet litter and supplemental biotin and/or whey on the production of foot pad dermatitis in broilers. *Poult Sci* **56**: 291-296.

HARPER, J. A. and HELFER, D. H. (1972). The effect of vitamin E, methionine and selenium on degenerative myopathy in turkeys. *Poult Sci* **51**: 1757-1759.

HARPER, J. A., BERNIER, P. E., HELFER, D. H. and SCHMITZ, J. A. (1975). Degenerative myopathy of the deep pectoral muscle in the turkey. *Journal of heredity* **66**: 362-366.

HAVENSTEIN, G. B., FERKET, P. R. and QURESHI, M. A. (2003a). Carcass composition and yield of 1957 versus 2001 broilers when fed representative 1957 and 2001 broiler diets. *Poultry Science* **82**: 1509-1518.

HAVENSTEIN, G. B., FERKET, P. R. and QURESHI, M. A. (2003b). Growth, livability, and feed conversion of 1957 versus 2001 broilers when fed representative 1957 and 2001 broiler diets. *Poultry Science* **82**: 1500-1508.

HAVENSTEIN, G. B., FERKET, P. R., GRIMES, J. R., QURESHI, M. A. and NESTOR, K. E. (2004). *Changes in the Performance of Turkeys 1966 - 2003*. Proceedings of the 27th Technical Turkey Conference: 11-18.

HEIM, G. (1990) Beinschwäche-Syndrom bei Mastputen: Einflüsse von verschiedenen Vitamin D-Metaboliten und von Vitamin C. Dr.-vet. med. Thesis Munich, Germany

HODGSEN, G.C. and GUTTERIDGE, H. S. (1941): A progress report from Canada of research in breast blisters. U. S. *Egg and Poultry Magazine*, **47:** 150.

JULIAN, R. J. (1984). Tendon avulsion as a cause of lameness in turkeys. *Avian Dis* **28**: 244-249.

JULIAN, R. J. (1985). Osteochondrosis, dyschondroplasia, and osteomyelitis causing femoral head necrosis in turkeys. *Avian Dis* **29**: 854-866.

JULIAN, R. J. (1993). Ascites in poultry. *Avian Pathology* **22**: 419-454.

JULIAN, R. J. and GAZDZINSKI, P. (1999). *Lameness and leg problems in turkeys*. Proceedings of the 2nd International Symposium on Turkey Diseases, Berlin, (ISBN: 3-930511-75-4): 18-29.

JULIAN, R. J., FRIARS, G. W., FRENCH, H. and MCQUINTON, M. (1986). The relationship of right ventricular hypertrophy, right ventricular failure and ascites to weight gain in broiler and rooster chickens. *Avian Diseases* **31**: 130-135.

KRISTA, L. M., WAIBEL, P. E. and BURGER, R. E. (1965a). Influence of Dietary Alterations Hormones and Blood Pressure on Incidence of Dissecting Aneurysms in Turkey. *Poultry Science* **44**: 15-&.

KRISTA, L. M., WAIBEL, P. E., SHOFFNER, R. N. and SAUTTER, J. H. (1965b). Natural dissecting aneurysm (aortic rupture) and blood pressure in the turkey. *Nature* **214**: 1162-1163.

KUHLERS, D. L. and MCDANIEL, G. R. (1996). Estimates of heritabilities and genetic correlations between tibial dyschondroplasia expression and body weight at two ages in broilers. *Poult Sci* **75**: 959-961.

LAROCHELLE, D., MORIN, M. and BERNIER, G. (1992). Sudden-Death in Turkeys with Perirenal Hemorrhage - Pathological Observations and Possible Pathogenesis of the Disease. *Avian Diseases* **36**: 114-124.

MARTLAND, M. F. (1984). Wet litter as a cause of plantar Pododermatitis, leading to foot ulceration and lameness in fattening turkeys. *Avian Pathology* **13**: 241-252.

MAXWELL, M. and ROBERTSON, G. (2000). *Cardiovascular disease in poultry: epidemiology - current trends and correlates*. Proceedings of the XXI World's Poultry Congress, Montreal, Canada:

MCILROY, S. G., GOODALL, E. A. and MCMURRAY, C. H. (2005). A contact dermatitis of broilers - epidemiological findings. *Avian Pathology* **16**: 93-105.

MCSHERRY, B. J., FERGUSON, A. E. and BALLANTYNE, J. (1945). A dissecting aneurism in internal haemorrhage in turkeys. *J. Am. Vet. Med. Assoc.* **124**: 279-283.

MUTALIB, A. A. and HANSON, J. A. (1990). Sudden death in turkeys with perirenal hemorrhage: Field and laboratory findings. *Can Vet J* **31**: 637-642.

NESTOR, K. E., SAIF, Y. M., ZHU, J. and NOBLE, D. O. (1996). Influence of growth selection in turkeys on resistance to Pasteurella multocida. *Poultry Science* **75**: 1161-1163.

NEWBERRY, R. C., GARDINER, E. E. and HUNT, J. R. (1987). Behavior of Chickens Prior to Death from Sudden-Death Syndrome. *Poultry Science* **66**: 1446-1450.

NIR, I., NITSAN, Z., DROR, Y. and SHAPIRA, N. (1978). Influence of overfeeding on growth, obesity, and intestinal tract in young chicks of light and heavy breeds. *Br. J. Nutr.* **39**: 27-35.

NORCI, C. and MONTELLA, L. (2003). Turkey welfare: Is it only a management problem? in: *Turkey Production: Balance act between consumer protection, animal welfare and economic aspects*. (ed. H. M. HAFEZ), Ulmer. 143-148.

OLKOWSKI, A. A. and CLASSEN, H. L. (1998). High incidence of cardiac arrhythmias in broiler chickens. *Journal of Veterinary Medicine Series a-Physiology Pathology Clinical Medicine* **45**: 83-91.

OLKOWSKI, A. A., CLASSEN, H. L., RIDDELL, C. and BENNETT, C. D. (1997). A study of electrocardiographic patterns in a population of commercial broiler chickens. *Veterinary Research Communications* **21**: 51-62.

PECKHAM, M. C. (1978). Vices and miscellaneous diseases conditions. in: *Diseases of poultry*. M. S. HOFSTAD, B. W. CALNEK, C. F. HELMBOLDT, W. M. REID and H. W. YODER. Ames, Iowa, Iowa State Univ. Press. 847 -893.

POULOS, P. W. (1978). Tibial Dyschondroplasia (osteochondrosis) in the turkey. *Acta Radiol. Suppl.* **358**: 197-227.

PRITCHARD, W. R., HENDERSON, W. and BEAL, C. W. (1958). Experimental production of dissecting aneurysms in turkeys. *Am J Vet Res* **19**: 696-705.

RIDDELL, C. (1976). Selection of Broiler-Chickens for a High and Low Incidence of Tibial Dyschondroplasia with Observations on Spondylolisthesis and Twisted Legs (Perosis). *Poultry Science* **55**: 145-151.

RIDDELL, C. (1981). Skeletal deformities in poultry. *Adv. Vet. Sci. Comp. Med* **25**: 277-310.

SAIF, Y. M., NESTOR, K. E., DEARTH, R. N. and RENNER, P. A. (1984). Possible Genetic-Variation in Resistance of Turkeys to Erysipelas and Fowl Cholera. *Avian Diseases* **28**: 770-773.

SCHUHWERK, K. H. (1989) Therapie Versuche zur Beeinflussung der "Beinschwäche" beim Truthahn der Big-6-Linie mit Vitamin D-Metaboliten. Dr.-vet. med. Thesis München

SHARAF, M. M., NESTOR, K. E., SAIF, Y. M., SACCO, R. E. and HAVENSTEIN, G. B. (1988). Antibody-Response to Newcastle-Disease Virus and Pasteurella-Multocida of 2 Strains of Turkeys. *Poultry Science* **67**: 1372-1377.

SHIVAPRASAD, H. L. and CRESPO, R. (1997). *High mortality due to coronary artery aneurysm in male turkeys*. Proceedings of the 40th Annual Meeting of the American Association of Veterinary Laboratory Diagnostician: 19.

THORP, B. H. (1994). Skeletal disorder in the fowl: a review. *Avian Pathology* **23**.

TSAI, H. J., SAIF, Y. M., NESTOR, K. E., EMMERSON, D. A. and PATTERSON, R. A. (1992). Genetic variation in resistance of turkeys to experimental infection with Newcastle disease virus. *Avian Dis* **36**: 561-565.

WIGHT, P. A. L. and SILLER, W. G. (1979). The induction by muscle stimulation of a deep pectoral myopathy in the fowl. *Avian Pathology* **8**: 115-121.