

An HSUS Report: Welfare Issues with Selective Breeding for Rapid Growth in Broiler Chickens and Turkeys

Abstract

Chickens and turkeys raised commercially for meat production are selectively bred to reach market weight rapidly. As consequence of unnaturally rapid growth and heavy body weight, these birds often experience skeletal and metabolic disorders that severely compromise their welfare, including lesions in the hip joint and painful leg disorders, such as angular bone deformity, tibial dyschondroplasia, and tendon rupture or slippage. Severely crippled birds may become non-ambulatory and, if unable to reach feed and water, die. Heart and circulatory disorders are also common causes of death in birds raised for meat. Selective breeding for rapid growth has inadvertently led to birds with increased appetite, and breeding stock are severely feed-restricted in order to prevent certain health and reproductive problems. As such, birds used for breeding may be chronically hungry. The prevailing mind-set in the poultry industry has been that economic considerations outweigh the welfare of individual birds. Only with a change in priorities can primary breeding companies begin to aggressively address welfare problems associated with rapid growth.

Introduction

In the United States in 2007, more than 9 billion chickens and 260 million turkeys were raised and slaughtered for human consumption.¹ Breeding programs for these commercially farmed birds aim to maximize productivity by accelerating growth while reducing feed consumption. Although primary breeding companies include bird health in their genetic selection programs,^{2,3} growth has consistently been the top selection trait, followed closely by other economically important characteristics, such as feed efficiency and processing yield.^{4,5} Selection for efficient productivity is often accompanied by correlated changes in other genetic traits, which may negatively impact the welfare of these animals.⁵

Breeding for rapid growth to unprecedented body weight has been highly successful. In 1925, it took approximately 16 weeks to raise a 1.1 kg (2.5 lb) chicken,⁶ but broiler chicken strains now reach 2.5 kg (5.5 lb) in less than 7 weeks.⁷ Daily growth rates have increased from 25 g (0.88 oz) to 100 g (3.52 oz)—an increase of more than 300%.⁸ Similarly, while male wild turkeys grow from approximately 51 g (1.8 oz) at hatch to 3.5 kg (7.7 lb) in about 4 months,⁹ selectively bred commercially reared turkey toms exceed 11.3 kg (25 lb) in the same amount of time.¹⁰ In the late 1960s, it took 220 days to raise a 35-pound turkey. In 2004, it took only 132 days.¹¹

This change in growth is a severe welfare problem as unintended genetic side effects of selection for rapid growth and increased body weight have resulted in leg disorders, including bone deformities, lameness, tibial dyschondroplasia (TD), and ruptured tendons, as well as metabolic diseases, such as ascites and sudden death syndrome.¹²⁻¹⁵ Fast-growing broiler chickens and turkeys also suffer from weakened immune systems, making them more susceptible to a variety of additional diseases.^{5,16,17} The link between selection for heavy body weight and leg abnormalities, cardiovascular problems, and immune system development is recognized by poultry breeders and thought of as a challenge that must be addressed on the "road to the biological maximum."¹⁸

Skeletal Disorders

Fast-growing muscle outpaces bone development during the early life of chickens and turkeys, stressing bone, joint, and ligament, and leading to problems with skeletal weakness. As a result, broiler chickens and turkeys often suffer from leg deformities and lameness.^{15,19-22} A scientific review published in 2003 concluded, "There is no doubt that the rapid growth rate of birds used for meat production is the fundamental cause of skeletal disorders, nor that this situation has been brought about by the commercial selection programmes used over a period of 40-50 generations."²³

Skeletal disorders are common. Studies consistently show that approximately 26-30% of broiler chickens suffer from gait defects severe enough to significantly impair walking ability,^{8,21,24} and additional research strongly suggests that birds at this level of lameness are in pain.^{25,26} For turkeys, experiments have shown that up to 3.3% of 16 week old male birds exhibit gait abnormalities severe enough to hinder walking ability.²⁷ Severe leg deformities are fatal if birds can no longer stand to reach food or water,²⁸ and 1% of broiler chickens die or are culled due to leg problems.^{29,30} For turkeys, a 5% mortality rate due to lameness may be normal in heavy toms with up to 20% mortality caused by lameness in problem flocks.³¹

"Twisted leg" (valgus-varus or angular bone deformity) is a common cause of leg problems. The growth deformity is characterized by bowed or knock-kneed legs. It is estimated that 0.5-2% of broiler chickens in normal flocks have bone deformities, with occasional cases where 5-25% of male broiler chickens are affected in problem flocks. The deformity may occur in five-day-old chicks, but is more often seen when the birds are 1-4 weeks old or older.³² Turkeys may also suffer from angular deformities, and some may have problems standing. In typical, high stocking density environments, fallen birds may be trampled and bruised, and some may seek refuge from being stepped upon by hiding under the feeders.³³ As body weight accumulates, deformities can lead to a slipped tendon or spontaneous fracture.^{13,32} Whether moderate or severe, affected birds have difficulty walking and may hobble, preferring to sit. More severely affected birds are unable to rise and may walk or creep on their hocks.³² These crippled birds may not be able to access feed and water.¹⁹

Tibial dyschondroplasia (TD), an abnormal mass of cartilage at the growth plate of a bone, usually the tibia, is another common cause of leg problems.³⁴ The end of the tibia may become enlarged, deformed, and weakened, and the bone may bend backward as it grows. This can cause necrosis, spontaneous fracture, severe lameness, and, in some cases, the complete inability to stand.^{13,19} Like birds suffering from severe twisted leg deformities, crippled and unable to rise, some birds with severe TD can move only by creeping on their hocks and may be unable to reach food and water.¹⁹

Sources differ in describing the prevalence of TD. In 1995, researchers estimated that, in extreme cases, the incidence of TD could reach 30-40%, especially for flocks of male birds.³⁵ Aviagen, a leading breeding company, has worked to reduce the incidence of TD, and its own figures show a reduction from approximately 8% in 1999 to a projected level of less than 2% by 2005.² However, studies published in 2001 and 2003 report elevated cases in common commercial strain broiler chickens, with a mean prevalence of approximately 57%.²¹ While TD may be relatively common in broiler chickens and turkeys raised for meat, it is rare or absent in other types of birds.¹⁹

Rupture of the gastrocnemius tendon that runs along the back of the leg is a common problem in heavy broiler chickens. It is caused by excessive weight on weak tendons, a consequence of rapid growth. If one leg is affected, the added stress may cause rupture of the tendon in the other leg. Discoloration may be seen on the back of the legs due to hemorrhage. A ruptured tendon is a chronic, debilitating, and painful condition.^{13,19}

At six weeks, broiler chickens have such difficulty supporting their abnormally heavy bodies that they spend 76-86% of their time lying down.³⁶ Combined with poor litter condition, the immobility of the birds leads to increased incidence of painful contact dermatitis—breast blisters, hock burns, and foot pad lesions.³⁷ Sheds are not cleared of litter and excrement until chickens are taken to slaughter and, may not even be cleared after each

flock depopulation, meaning the birds have no choice but to stand and lay in their own waste.^{38,39} As a result, skin sores may become infected.⁴⁰

Metabolic Disorders

Increased body weight of broiler chickens can also lead to sudden death syndrome (SDS).⁴¹ Young birds die from SDS after sudden convulsions and wing-beating, and are frequently found lying on their backs.¹³ Between approximately 1-4% of broiler chickens may die from this condition.²⁹ The syndrome is a form of acute cardiovascular collapse caused by fatal dysrhythmias, which are common in broiler chickens⁴² and have been linked to their rapid growth rate.⁴³

For turkeys, sudden death is associated with perirenal hemorrhage (SDPH). Turkeys who die of SDPH exhibit signs of acute heart failure and bleeding of the kidneys.⁴⁴ Between approximately 8-14 weeks of age, SDPH is a significant cause of mortality for otherwise healthy, rapidly growing turkey toms.^{45,46} During this peak time period, producers may lose 0.2-0.5% of the flock, due mainly to SDPH⁴⁵ with total mortality reaching up to 6% in some flocks.⁴⁶ Although rapid growth may be an important contributing factor, continuous lighting regimens, stress, crowding, and exposure to toxins may also lead to SDPH.⁴⁶

Ascites is a condition in which rapidly growing birds do not have the heart and lung capacity needed to distribute oxygen throughout the body.⁴⁷ The high metabolic demand for oxygen and relatively low capacity for blood flow through the lungs increases the workload of the heart, and is a leading cause of mortality as birds reach market weight.¹² Characteristic symptoms include accumulation of fluid in the abdominal cavity, an enlarged flaccid heart, the appearance of a shrunken liver, and heart failure.⁴⁸

In artificial selection programs for economically important traits, resources within the body are diverted toward productivity at the expense of those needed for coping with unexpected stress.⁵ Consequently, genetic selection for production characteristics, such as rapid growth, has negatively influenced immune function, compromising antibody production and overall disease resistance.^{17,49-52}

Broiler Breeders

In the United States in 2007, approximately 56 million "hatching type" egg-laying hens⁵³ were used to breed approximately 9 billion broiler chickens raised for meat production.¹ These broiler breeders have the same genetic predisposition as their progeny for fast growth, lameness, and heart disease. When fed unrestricted (*ad libitum*) diets, as their offspring are, they would develop health and reproductive disorders⁵⁴⁻⁵⁶ and fewer than half of broiler breeders would survive more than one year.⁵⁷ To increase their productivity, broiler breeders are given as little as one-fifth of the amount of food they would eat *ad libitum* and weigh only half as much.^{54,58}

In many parts of the world, including the United States, broiler breeders may be fed on a "skip-a-day" regimen in which the animals are fed every other day^{55,59-61}—though this practice has been outlawed in several European countries.⁵⁵ In some cases, water may also be restricted in order to reduce litter moisture.⁵⁵

Selection for increased body weight is associated with a concomitant increase in appetite.⁶² Feed restriction causes stress,⁶³ behavior patterns characteristic of chronic hunger, undernourishment, specific nutritional deficiency, and frustration.⁶⁴ Indeed, their motivation to eat is as high after their daily meal as before it,⁵⁴ indicating that breeding birds are rarely satiated. As a result, broiler breeders demonstrate increased injurious pecking and other stress-induced stereotypic behaviors, such as repetitive pecking and pacing.⁵⁸

Broiler breeders are kept alive substantially longer than those raised solely for slaughter—16 months versus 6-7 weeks, respectively. As a result, even with restricted diets, the parent stock are vulnerable to a higher incidence of skeletal disorders. One study found that 92% of male breeders had pelvic limb lesions, 85% had total or partial rupture of ligaments or tendons, 54% had total ligament or tendon failure at one or more skeletal sites,

and 16% had total detachment of the femoral head.⁶⁵ In two other experiments, 40-47% of male broiler breeders were killed because of lameness or infertility and 63-83% of those culled had musculoskeletal lesions.⁶⁶

It has been shown that the level of feed restriction among broiler breeder females can be reduced by using females from recessive gene dwarf strains.⁶⁷ This measure does not, however, positively influence the poor welfare of breeder males or their offspring.

Turkey Breeders

Parent flocks are also used to breed turkeys who are used for meat production. As with broiler breeders and chickens, turkey breeders have the same genetic predisposition as non-breeding turkeys for fast growth, skeletal disorders, and heart disease. If fed unrestricted diets, few would survive to sexual maturity, and most would suffer from reproductive disorders, decreasing egg production.⁶⁸ Hence, turkey breeders are given as little as half of the amount of food they would eat *ad libitum*. Feed restriction is stressful for turkeys, as demonstrated by the measurable rise in the stress hormone, corticosterone.⁶⁹

Because breeders are kept alive longer than turkeys raised exclusively for slaughter, their skeletal problems are more severe. At termination of breeding, at least 75% of breeders suffer from abnormal gait or lameness.⁷⁰ One study of turkey breeders found that, by the time of slaughter, 25-50% of feed restricted, commercial line birds suffered destructive cartilage loss in the hip joint, while none of the turkeys in the comparison groups of traditional line birds suffered from the disease.⁷¹ Another study found that all male breeders had extensive hip joint degeneration, and results strongly suggest that turkeys experience chronic pain from hip problems,⁷² though one study found no evidence of pain associated with destructive cartilage loss of the hip joint.⁷³

Male turkeys have been bred for such heavy body weight that natural mating has become physically impossible. Artificial insemination is now the standard practice.⁵ Male breeding turkeys are "milked" for semen collection, and females are then inseminated by tube or syringe.

Improving the Welfare of Birds Genetically Selected for Rapid Growth

By modifying the birds' environment and diet, growth can be slowed and welfare improved. Growth rates can be reduced by altering the artificial lighting regime and dietary adjustments can prevent certain disorders.⁷⁴ When they have sufficient daylight hours, birds cease to feed in darkness; therefore, longer, uninterrupted dark periods when young slow growth rates and subsequently reduce leg problems, sudden death syndrome, and mortality.^{8,75-78} Departing from the near-continuous lighting schedules common within the commercial industry offers other welfare benefits, as well, such as a reduction in the eye disorders glaucoma and buphthalmia.⁷⁶ However, the problems associated with fast growth are principally genetic in origin, thereby requiring genetic solutions.

The incidence of skeletal disease is markedly reduced in slower growing broiler chicken strains. In France, breeds such as the ISA 657 with a lower growth rate have been used to produce "Label Rouge" chickens for more than 40 years and may comprise the majority of broilers purchased in that country.⁷⁹ These chickens reach slaughter weight in 12 weeks, compared with an average of 48 days for U.S. broiler chickens, and suffer substantially lower incidences of leg disease⁸⁰ and very low mortality rates—in one study, zero—despite having a grow-out period that is nearly twice as long as conventional broiler chickens.⁸¹ In the United Kingdom, several slow-growing breeds of chicken are promoted by organic standards, which require a minimum slaughter age of 70 days. These breeds include Sasso, Poulet Bronze, Poulet Grey, Sherwood Gold, and Hubbard ISA 657, ISA 257, and PAC57.^{82,83} In the United States, there is an increasing interest in slow growing strains and heirloom breeds of chickens and turkeys for sustainable pasture-based production.⁸⁰

Conclusion

The intensive, commercial poultry industry has a long history of failure to recognize the severity of problems associated with rapid growth and the welfare implications of breeding chronically unhealthy animals. Economic considerations have trumped concern for the well-being of individual animals, as demonstrated by two prominent poultry researchers who offer the following analysis:

Two decades ago the goal of every grower was to ensure that the flock grew as rapidly as possible. However, the industry has developed a broiler that, if grown as rapidly as possible, will achieve a body mass that cannot be supported by the bird's heart, respiratory system or skeleton.

The situation has forced growers to make a choice. Is it more profitable to grow the biggest bird possible and have increased mortality due to heart attacks, ascites, and leg problems, or should birds be grown slower so that birds are smaller, but have fewer heart, lung and skeletal problems?...A large portion of growers' pay is based on the pound of saleable meat produced, so simple calculations suggest that it is better to get the weight and ignore the mortality."⁸⁴

Further analysis has been offered by Scott Beyer, a Kansas State University poultry scientist, who stated "Although a small percentage of birds may be predisposed to leg problems, use of highly selected fast-growing strains is recommended because savings in feed costs and time far out-weigh the loss of a few birds."⁸⁵ This interest in reaching market weight in the shortest period of time without due regard to the welfare of individual birds has been the prevailing mind-set over the past several decades. In a survey published in 1990, some farmers ironically regarded sudden death syndrome of turkeys as a sign of "good flock health and fast growth rate."⁴⁶

However, leading scientists and animal advocates have worked to bring attention to the issue, and, if primary breeding companies address these concerns, change could occur. Dr. John Webster of the University of Bristol strongly asserts that the chronic pain associated with leg problems experienced by heavy strain broiler chickens and turkeys "must constitute, in both magnitude and severity, the single most severe, systematic example of man's inhumanity to another sentient animal."⁸⁶ In the United States, broiler chickens and turkeys now come from strains produced by only four and three primary breeding companies, respectively.^{87,88} If these seven companies refocused genetic selection programs to prioritize health and well-being above all else, they could dramatically improve the welfare of fast growing broiler chickens and turkeys, and the long term solution is in their hands.⁸⁹

References

- 1. U.S. Department of Agriculture National Agricultural Statistics Service. 2008. Poultry slaughter: 2007 annual summary. <u>http://usda.mannlib.cornell.edu/usda/current/PoulSlauSu/PoulSlauSu-02-28-2008.pdf</u>. Accessed July 10, 2008.
- 2. Aviagen. 2001. Leg health in broilers. Ross Tech 01/40. <u>www.aviagen.com/docs/ross tech leg.pdf</u>. Accessed July 10, 2008.
- 3. Hardiman J. 1993. Producing a quality broiler: a primary breeder's perspective. Technical News 1(3):1-6. www.cobb-vantress.com/Publications/documents/TN-Fall-93.pdf. Accessed July 10, 2008
- 4. Arthur JA and Albers GAA. 2003. Industrial perspective on problems and issues associated with poultry breeding. In: Muir WM and Aggrey SE (eds.), Poultry Genetics, Breeding and Biotechnology (Wallingford, U.K.: CABI Publishing, pp. 1-12).
- 5. Rauw WM, Kanis E, Noordhuizen-Stassen EN, and Grommers FJ. 1998. Undesirable side effects of selection for high production efficiency in farm animals: a review. Livestock Production Science 56(1):15-33.
- 6. Aho PW. 2002. Introduction to the U.S. chicken meat industry. In: Bell DD and Weaver WD Jr (eds.), Commercial Chicken Meat and Egg Production, 5th Edition (Norwell, MA: Kluwer Academic Publishers, pp. 801-18).

- National Chicken Council. 2006. U.S. broiler performance. <u>www.nationalchickencouncil.com/statistics/stat_detail.cfm?id=2</u>. Accessed July 10, 2008.
- 8. Knowles TG, Kestin SC, Haslam SM, et al. 2008. Leg disorders in broiler chickens: prevalence, risk factors and prevention. PLoS ONE 3(2):e1545. doi:10.1371/journal.pone.0001545.
- 9. Healy WM. 1992. Behavior. In: Dickson JG (ed.), The Wild Turkey: Biology and Management (Harrisburg, PA: Stackpole Books, pp. 46-65).
- Hulet RM, Clauer PJ, Greaser GL, Harper JK, and Kime LF. 2004. Small-flock turkey production. The Pennsylvania State University Agricultural Research and Cooperative Extension. <u>http://agalternatives.aers.psu.edu/livestock/turkey/SmallflockTurkeys.pdf</u>. Accessed July 10, 2008.
- 11. Ferket PS. 2004. Tom weights up seven percent. WATT PoultryUSA, July, pp. 32-42.
- 12. Boersma S. 2001. Managing rapid growth rate in broilers. World Poultry 17(8):20-1.
- 13. Julian RJ. 2004. Evaluating the impact of metabolic disorders on the welfare of broilers. In: Weeks CA and Butterworth A (eds.), Measuring and Auditing Broiler Welfare (Wallingford, U.K.: CABI Publishing, pp. 51-9).
- 14. Havenstein GB, Ferket PR, Scheideler SE, and Larson BT. 1994. Growth, livability, and feed conversion of 1957 vs 1991 broilers when fed "typical" 1957 and 1991 broiler diets. Poultry Science 73(12):1785-94.
- 15. Bessei W. 2006. Welfare of broilers: a review. World's Poultry Science Journal 62(3):455-66.
- Li Z, Nestor KE, and Saif YM. 2001. A summary of the effect of selection for increased body weight in turkeys on the immune system. In: Eastridge ML (ed.), Research and Reviews: Poultry 2001 (Wooster, OH: The Ohio State University Agricultural Research and Development Center, pp. 21-8). <u>http://ohioline.osu.edu/sc184/index.html</u>. Accessed July 10, 2008.
- 17. Qureshi MA and Havenstein GB. 1994. A comparison of the immune performance of a 1991 commercial broiler with a 1957 randombred strain when fed "typical" 1957 and 1991 broiler diets. Poultry Science 73(12):1805-12.
- Buddiger N and Albers G. 2004. Future trends in turkey breeding. Hybrid Turkeys. <u>www.hybridturkeys.com/Media/PDF_files/Management/Mng_future_trds_lbs.pdf</u>. Accessed July 10, 2008.
- 19. Julian RJ. 1998. Rapid growth problems: ascites and skeletal deformities in broilers. Poultry Science 77:1773-80.
- 20. Mench JA. 2004. Lameness. In: Weeks CA and Butterworth A (eds.), Measuring and Auditing Broiler Welfare (Wallingford, U.K.: CABI Publishing, pp. 3-17).
- Sanotra GS, Lund JD, Ersboll AK, Petersen JS, and Vestergaard KS. 2001. Monitoring leg problems in broilers: a survey of commercial broiler production in Denmark. World's Poultry Science Journal 57(1):55-69.
- 22. Scientific Committee on Animal Health and Animal Welfare. 2000. The welfare of chickens kept for meat production (broilers). European Commission. <u>http://ec.europa.eu/food/fs/sc/scah/out39_en.pdf</u>. Accessed July 10, 2008.
- 23. Whitehead CC, Fleming RH, Julian RJ, and Sørensen P. 2003. Skeletal problems associated with selection for increased production. In: Muir WM and Aggrey SE (eds.), Poultry Genetics, Breeding and Biotechnology (Wallingford, U.K.: CABI Publishing, pp. 29-52).
- 24. Kestin SC, Knowles TG, Tinch AE, and Gregory NG. 1992. Prevalence of leg weakness in broiler chickens and its relationship with genotype. Veterinary Record 131(9):190-4.
- 25. Danbury TC, Weeks CA, Chambers JP, Waterman-Pearson AE, and Kestin SC. 2000. Self-selection of the analgesic drug carprofen by lame broiler chickens. Veterinary Record 146(11):307-11.
- 26. McGeown D, Danbury TC, Waterman-Pearson AE, and Kestin SC. 1999. Effect of carprofen on lameness in broiler chickens. Veterinary Record 144:668-71.
- 27. Martrenchar A, Huonnic D, Cotte JP, Boilletot E, and Morisse JP. 1999. Influence of stocking density on behavioural, health and productivity traits of turkeys in large flocks. British Poultry Science 40(3):323-31.
- 28. Sørensen P, Su G, and Kestin SC. 1999. The effect of photoperiod: scotoperiod on leg weakness in broiler chickens. Poultry Science 78(3):336-42.
- 29. Riddell C and Springer R. 1985. An epizootiological study of acute death syndrome and leg weakness in broiler chickens in Western Canada. Avian Diseases 29(1):90-102.
- 30. Morris MP. 1993. National survey of leg problems. Broiler Industry, May, pp. 20-4.

An HSUS Report: Welfare Issues with Selective Breeding for Rapid Growth in Broiler Chickens and Turkeys

- 31. Julian RJ. 1984. Tendon avulsion as a cause of lameness in turkeys. Avian Diseases 28(1):244-9.
- 32. Julian RJ. 1984. Valgus-varus deformity of the intertarsal joint in broiler chickens. Canadian Veterinary Journal 25(6):254-8.
- Smith R. 1991. Cutting edge poultry researchers doing what birds tell them to do. Feedstuffs, September 9 63(37):1, 22.
- 34. Sanotra GS, Berg C, and Lund JD. 2003. A comparison between leg problems in Danish and Swedish broiler production. Animal Welfare 12(4):677-83.
- 35. Leeson S, Diaz G, and Summers JD. 1995. Poultry Metabolic Disorders and Mycotoxins (Guelph, Canada: University Books, p. 140).
- 36. Weeks CA, Danbury TD, Davies HC, Hunt P, and Kestin SC. 2000. The behaviour of broiler chickens and its modification by lameness. Applied Animal Behaviour Science 67:111-25.
- 37. Estevez I. 2002. Poultry welfare issues. Poultry Digest Online 3(2):1-12.
- Dozier WA, Lacy MP, and Vest LR. 2001. Broiler production and management. The University of Georgia College of Agricultural and Environmental Sciences Cooperative Extension Service. <u>http://pubs.caes.uga.edu/caespubs/pubcd/B1197.htm</u>. Accessed July 10, 2008.
- 39. Lacy MP. 2002. Broiler management. In: Bell DD and Weaver WD Jr (eds.), Commercial Chicken Meat and Egg Production, 5th Edition (Norwell, MA: Kluwer Academic Publishers, pp. 829-68).
- 40. Skeeles JK. 1991. Staphylococcosis. In: Calnek BW, Barnes HJ, Beard CW, McDougald LR, and Saif YM (eds.), Diseases of Poultry, 9th Edition (Ames, IA: Iowa State University Press, pp. 293-9).
- 41. Gardiner EE, Hunt JR, Newberry RC, and Hall JW. 1988. Relationships between age, body weight, and season of the year and the incidence of sudden death syndrome in male broiler chickens. Poultry Science 67(9):1243-9.
- 42. Olkowski AA and Classen HL. 1997. Malignant ventricular dysrhythmia in broiler chickens dying of sudden death syndrome. Veterinary Record 140:177-9.
- 43. Greenlees KJ, Eyre P, Lee JC, and Larsen CT. 1989. Effect of age and growth rate on myocardial irritability in broiler chickens. Proceedings of the Society for Experimental Biology and Medicine 190(3):282-5.
- 44. Crespo R and Shivaprasad HL. 2003. Developmental, metabolic, and other noninfectious disorders. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, and Swayne DE (eds.), Diseases of Poultry, 11th Edition (Ames, IA: Iowa State Press, pp. 1055-102).
- 45. Frank RK, Newman JA, Noll SL, and Ruth GR. 1990. The incidence of perirenal hemorrhage syndrome in six flocks of market turkey toms. Avian Diseases 34:824-32.
- 46. Mutalib AA and Hanson JA. 1990. Sudden death in turkeys with perirenal hemorrhage: field and laboratory findings. Canadian Veterinary Journal 31(9):637-42.
- 47. Duncan IJH. 2001. Animal welfare issues in the poultry industry: is there a lesson to be learned? Journal of Applied Animal Welfare Science 4(3):207-21.
- 48. Balog JM. 2003. Ascites syndrome (pulmonary hypertension syndrome) in broiler chickens: are we seeing the light at the end of the tunnel? Avian and Poultry Biology Reviews 14(3):99-126.
- 49. Miller LL, Siegel PB, and Dunnington EA. 1992. Inheritance of antibody response to sheep erythrocytes in lines of chickens divergently selected for fifty-six-day body weight and their crosses. Poultry Science 71(1):47-52.
- 50. Nestor KE, Saif YM, Zhu J, and Noble DO. 1996. Influence of growth selection in turkeys on resistance to Pasteurella multocida. Poultry Science 75(10):1161-3.
- 51. Nestor KE, Noble DO, Zhu J, and Moritsu Y. 1996. Direct and correlated responses to long-term selection for increased body weight and egg production in turkeys. Poultry Science 75:1180-91.
- 52. Bayyari GR, Huff WE, Rath NC, et al. 1997. Effect of the genetic selection of turkeys for increased body weight and egg production on immune and physiological responses. Poultry Science 76(2):289-96.
- U.S. Department of Agriculture National Agricultural Statistics Service. 2008. Chickens and eggs: 2007 summary. <u>http://usda.mannlib.cornell.edu/usda/current/ChickEgg/ChickEgg-02-28-2008.pdf</u>. Accessed July 10, 2008.
- 54. Savory CJ, Maros K, and Rutter SM. 1993. Assessment of hunger in growing broiler breeders in relation to a commercial restricted feeding programme. Animal Welfare 2(2):131-52.
- 55. Hocking PM. 2004. Measuring and auditing the welfare of broiler breeders. In: Weeks C and Butterworth

An HSUS Report: Welfare Issues with Selective Breeding for Rapid Growth in Broiler Chickens and Turkeys

A (eds.), Measuring and Auditing Broiler Welfare (Wallingford, U.K.: CABI Publishing, pp. 19-35).

- 56. Renema RA and Robinson FE. 2004. Defining normal: comparison of feed restriction and full feeding of female broiler breeders. World's Poultry Science Journal 60(4):508-22.
- 57. Katanbaf MN, Dunnington EA, and Siegel PB. 1989. Restricted feeding in early and late feathering chickens. 1. Growth and physiological responses. Poultry Science 68:344-51.
- 58. Kjaer JB and Mench JA. 2003. Behaviour problems associated with selection for increased production. In: Muir WM and Aggrey SE (eds.), Poultry Genetics, Breeding and Biotechnology (Wallingford, U.K.: CABI Publishing, pp. 67-82).
- 59. Shane SM. 2007. Progress in refining standards, audits. WATT PoultryUSA, October, pp. 34-7.
- 60. Coon CN. 2002. Feeding broiler breeders. In: Bell DD and Weaver WD (eds.), Commercial Chicken Meat and Egg Production, 5th Edition (Norwell, MA: Kluwer Academic Publishers, pp. 329-69).
- 61. Mench JA. 1993. Problems associated with broiler breeder management. In: Savory CJ and Hughes BO (eds.), Proceedings of the 4th European Symposium on Poultry Welfare (Edinburgh, Scotland: Universities Federation for Animal Welfare, pp. 195-207).
- 62. Siegel PB and Wisman EL. 1966. Selection for body weight at eight weeks of age. 6. Changes in appetite and feed utilization. Poultry Science 45:1391-7.
- 63. Mench JA. 1991. Research note: Feed restriction in broiler breeders causes a persistent elevation in corticosterone secretion that is modulated by dietary tryptophan. Poultry Science 70(12):2547-50.
- 64. Savory CJ and Maros K. 1993. Influence of degree of food restriction, age and time of day on behaviour of broiler breeder chickens. Behavioural Processes 29(3):179-90.
- 65. Duff SRI and Hocking PM. 1986. Chronic orthopaedic disease in adult male broiler breeding fowls. Research in Veterinary Science 41:340-8.
- 66. Hocking PM and Duff SRI. 1989. Musculo-skeletal lesions in adult male broiler breeder fowls and their relationships with body weight and fertility at 60 weeks of age. British Poultry Science 30:777-84.
- 67. Jones EKM, Zaczek V, MacLeod M, and Hocking PM. 2004. Genotype, dietary manipulation and food allocation affect indices of welfare in broiler breeders. British Poultry Science 45(6):725-37.
- 68. Nestor KE, Bacon WL, and Renner PA. 1980. The influence of genetic changes in total egg production, clutch length, broodiness, and body weight on ovarian follicular development in turkeys. Poultry Science 59:1694-9.
- 69. Hocking PM, Maxwell MH, and Mitchell MA. 1999. Welfare of food restricted male and female turkeys. British Poultry Science 40(1):19-29.
- Hocking PM. 1992. Musculo-skeletal disease in heavy breeding birds. In: Whitehead CC (ed.), Bone Biology and Skeletal Disorders in Poultry. Poultry Science Symposium No. 23 (Abingdon, England: Carfax Publishing Co., pp. 297-309).
- 71. Hocking PM, Bernard R, and Wess TJ. 1998. Comparative development of antitrochanteric disease in male and female turkeys of a traditional line and a contemporary sire-line fed ad libitum or with restricted quantities of food. Research in Veterinary Science 65(1):29-32.
- 72. Duncan IJH, Beatty ER, Hocking PM, and Duff SRI. 1991. Assessment of pain associated with degenerative hip disorders in adult male turkeys. Research in Veterinary Science 50(2):200-3.
- 73. Hocking PM, Bernard R, and Maxwell MH. 1999. Assessment of pain during locomotion and the welfare of adult male turkeys with destructive cartilage loss of the hip joint. British Poultry Science 40(1):30-4.
- 74. Bradshaw RH, Kirkden RD, and Broom DM. 2002. A review of the aetiology and pathology of leg weakness in broilers in relation to welfare. Avian and Poultry Biology Reviews 13(2):45-103.
- 75. Gordon SH. 1994. Effects of daylength and increasing daylength programmes on broiler welfare and performance. World's Poultry Science Journal 50(3):269-82.
- 76. Prescott NB, Kristensen HH, and Wathes CM. 2004. Light. In: Weeks CA and Butterworth A (eds.), Measuring and Auditing Broiler Welfare (Wallingford, U.K.: CABI Publishing, pp. 101-16).
- 77. Lewis P and Morris T. 2006. Poultry lighting: the theory and practice (Hampshire, U.K.: Northcot, p. 39), citing: Lewis PD. 2001. Lighting regimes for broiler and egg production. In: Proceedings of XVII Latin American Poultry Congress, pp. 326-35.
- 78. Classen HL and Riddell C. 1989. Photoperiodic effects on performance and leg abnormalities in broiler chickens. Poultry Science 68(7):873-9.
- 79. Westgren RE. 1999. Delivering food safety, food quality, and sustainable production practices: the Label

An HSUS Report: Welfare Issues with Selective Breeding for Rapid Growth in Broiler Chickens and Turkeys

Rouge poultry system in France. American Journal of Agricultural Economics 81(5):1107-11.

- Fanatico A and Born H. 2002. Label Rouge: pasture-based poultry production in France. ATTRA, National Center for Appropriate Technology. <u>http://attra.ncat.org/attra-pub/PDF/labelrouge.pdf</u>. Accessed July 10, 2008.
- Lewis PD, Perry GC, Farmer LJ, and Patterson RLS. 1997. Responses of two genotypes of chicken to the diets and stocking densities typical of UK and 'Label Rouge' production systems: I. performance, behaviour and carcass composition. Meat Science 45(4):501-16.
- 82. Animal Health and Welfare in Organic Farming. Poultry breeds. Reading University. <u>www.organic-vet.reading.ac.uk/Poultryweb/health/breeds.htm</u>. Accessed July 10, 2008.
- 83. Gordon SH. 2002. Effect of breed suitability, system design and management on welfare and performance of traditional and organic table birds. Department for Environment, Food and Rural Affairs.
- 84. Tabler GT and Mendenhall AM. 2003. Broiler nutrition, feed intake and grower economics. Avian Advice 5(4):8-10.
- 85. Beyer RS. 2002. Leg problems in broilers and turkeys. Kansas State University Agricultural Experiment Station and Cooperative Extension Service, June. <u>www.oznet.ksu.edu/library/lvstk2/ep113.pdf</u>. Accessed July 10, 2008.
- Webster J. 1995. Animal Welfare: A Cool Eye Towards Eden (Cambridge, MA: Blackwell Science, p. 156).
- 87. Thornton G and O'Keefe T. 2003. Breeding counts. WATT PoultryUSA, October, pp. 28-31.
- Faure JM, Bessei W, and Jones RB. 2003. Direct selection for improvement of animal well-being. In: Muir WM and Aggrey SE (eds.), Poultry Genetics, Breeding and Biotechnology (Wallingford, U.K.: CABI Publishing, pp. 221-45).
- Duncan IJH. 2004. Foreword. In: Weeks CA and Butterworth A (eds.), Measuring and Auditing Broiler Welfare (Cambridge, MA: CABI Publishing, p. xi-xii).

The Humane Society of the United States is the nation's largest animal protection organization backed by 10 million Americans, or one of every 30. For more than a half-century, The HSUS has been fighting for the protection of all animals through advocacy, education, and hands-on programs. Celebrating animals and confronting cruelty. On the Web at humanesociety.org.