Cervical scoliosis and torticollis: a novel skeletal anomaly in broiler chickens

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Abstract

Background: Among the most prominent health problems marring the global poultry industry for several decades are skeletal abnormalities. The aim of this study was to investigate a recent emergence of a novel form of skeletal deformity affecting cervical spine in broiler chickens. This work presents the natural history of this newly emerging skeletal anomaly along with long term observations of epidemiological trends in commercial broiler flocks, and clinical and pathological features.

Results: In distinction from other forms of skeletal deformities commonly reported in broiler chickens, this new form of cervical spine anomaly have been observed in newly hatched chicks and in fully developed embryos that died in the shell. On clinical and post mortem examination this condition presents characteristic features consistent with congenital cervical scoliosis and torticollis (CCST). The pathogenesis of CCST appears to be linked to pathological remodeling of the cervical vertebrae bone associated with excessive activity of osteoclasts. Long term observations indicate that the incidence of CCST showed increasing epidemiological trends over time. More recently CCST has been observed in newly hatched chicks with incidence ranging from 0.1 to > 1%, and in fully developed embryos that failed to hatch about 4 to 5%.

Conclusions: The increasing trends in incidence of CCST in commercial broiler flocks are of concern from an economic perspective, and also represent a very specific and important aspect of animal welfare.

Keywords: Broiler chicken, Cervical scoliosis, Osteoclast, Skeletal deformity, Torticollis

Background

For many years the commercial broiler industry has focused on genetic selection for economically important traits such as rapid gain of muscle mass, decreased time from hatch to market, and increased feed efficiency. Undoubtedly, this strategy has resulted in the development of a chicken genotype with superior growth characteristics, but history has shown that intensive genetic selection in meat type poultry for production traits will inevitably lead to occurrence of undesirable traits [1]. In particular, the poor physical fitness predisposing chickens selected for rapid growth to skeletal disorders is a prime example of negative effects of intensive genetic selection for production traits, and represents an important aspect of the health problems which the global broiler industry has been facing continually for several decades [2–5].

In the agricultural animal production sector, genetic improvement for economically important traits is an ongoing process, and the emergence of any health problems in food producing animals is always a cause for concern. In particular, an emergence of skeletal anomalies in the meat type poultry sector needs special attention because of high selection pressure for fast growth, and rapid turnover of populations subjected to selection.

The Department of Animal and Poultry Science, University of Saskatchewan, Canada has been monitoring health problems in commercial broiler flocks for more than two decades [5–10], and this has resulted in a large data base of health-related records. A recent review of our records revealed the emergence of a new form of skeletal anomaly characterized by an abnormal posture of the neck and head. Increasing trends in the incidence of this anomaly were noted over time, and in particular...
over the past decade. This anomaly attracted attention not only because of its clinical novelty, but taken together with increasing incidence, this condition also has become a growing economic and animal welfare concern. Accordingly, herein we present the natural history and pathological features of this newly emerging skeletal anomaly along with long term observations of epidemiological trends in commercial broiler flocks documented by our research group.

Methods
General
The epidemiological trends of the emerging skeletal abnormality presented in this report are described in the context of historical records collected by our research group between 1994 and 2016. The data on the prevalence were collected from day old broiler chicks destined for various experiments at the University of Saskatchewan and from small commercial flocks in Saskatchewan, Canada. Overall, during the course of our observations we screened between 5000 and 40,000 day-old broiler chicks per year (around 350,000 in total). All birds were commercial broilers supplied by local commercial hatchery. The birds were delivered to the facility in plastic crates (100 chicks per crate). On arrival, all chicks were examined for general fitness, together with observation for overt signs of cervical spine deformities. The diagnostic criteria included abnormality of the cervical spine with the neck in a bent and/or twisted position such that the head is drawn to side, upwards or downwards. In the experimental setting, the evaluation was performed by principal investigator (PI), and in commercial situation the data was collected either through personal observation by PI or personal communications with producers or caregivers. The birds showing signs of cervical spine deformity were removed and either were euthanized or some taken to the lab for further examination.

Periodical cross-sectional studies to evaluate incidence of the cervical spine deformity in full term, unhatched chicks were conducted between 2013 and 2017 in the province of Saskatchewan, Canada (North American arena) and eastern Poland (European arena) in collaboration with local commercial hatcheries (one per location). A total of 2730 unhatched eggs were examined in 2013 and 2014 in Canada, and 2360 in Poland. The unhatched eggs were examined within 2 to 6 h after the completion of hatching process. The chicks were carefully extracted from the shell and subjected to visual evaluation for cervical spine deformity.

Gross pathology and light microscopy
Following clinical evaluation, representative samples of affected birds and normal flock mates were euthanized using T-61 Euthanasia Solution (Intervet, ON, Canada) and subjected to post mortem evaluation. Specimens from five affected and five normal broilers were processed for detailed examination of gross and microscopic changes in cervical vertebrae and cervical spinal cord. For microscopic examination sections of the neck were fixed in phosphate buffered 10% formalin solution. Following fixation, segments of spinal cord were removed, and the bone specimens were decalcified in 20% aqueous formic acid solution. Blocks of respective tissue sections were embedded in paraaffin. Longitudinal and transverse sections (5 μm in thickness) were processed for light microscopy and stained with hematoxylin and eosin (H&E) or phosphotungstic acid hematoxylin (PTAH) stains.

Results
General clinical observation and epidemiological trends
Retrospective analysis of the records collected by our research group over the last 25 years revealed emergence of a skeletal deformity affecting the cervical spine in commercial broiler flocks. Chicks affected by this condition typically show the neck in a bent and twisted position such that the head is drawn to one side, upwards or downwards. Typical examples of this skeletal deformity in newly hatched chicks and broilers at various stages of growth are shown in Fig. 1. The observed pathological features are consistent with cervical scoliosis and torticollis, and since these changes are clearly discernible in newly hatched chicks, this anomaly can be characterized as congenital cervical scoliosis and torticollis (CCST) syndrome.

Based on our records, the CCST syndrome in commercial broiler flocks was not observed in the past until sporadic cases were noted in the late 1990s. Since early 2000s an increase in incidence was noted over time, and in particular over the last decade an increase in the incidence of this condition has been observed in newly hatched chicks with the frequency of occurrence ranging between 0.1 and >1%. Cervical spine scoliosis and torticollis was also observed in many full term, unhatched, dead in the shell chicks (example shown in Fig. 2). Further examination of large populations of unhatched eggs (n=5090) revealed that approximately 4 to 5% of well-developed, otherwise normal chicks that died in the shell showed features characteristic of CCST (Table 1).

Gross pathology and histopathology
All cases of CCST showed similar general gross pathological features, only differing in the neck and head deformity orientation (head drawn towards left or right side, bent or twisted upwards or downward), and magnitude of the changes. Gross pathological changes of the neck of commercial broilers showing clinical signs of
severe cervical spine deformity are shown in Fig. 3. Noteworthy are skeletal changes in the specimen with intact muscular anatomy shown in topical view (Fig. 3a) and cervical spine following removal of soft tissue (Fig. 3b) characteristic of scoliosis with helical twist characteristic of torticollis.

### Table 1 Incidence of congenital deformity of cervical spine in apparently normal, dead in the shell, full term chick embryos observed in commercial hatcheries

| Item                      | Unhatched eggs examined | Dead in shell embryos showing signs of cervical scoliosis and torticollis |
|---------------------------|------------------------|--------------------------------------------------------------------------|
| North American studya     | 2730                   | 132 (4.8)                                                                |
| European studyb           | 2360                   | 93 (4.0)                                                                  |

Numbers in parentheses indicate % examined

a Study conducted in the province of Saskatchewan, Canada

b Study conducted around Siedlce district, Eastern Poland
In more severe cases, the deformity of the cervical spine (Fig. 4a) also affected the morphology of the spinal cord (Fig. 4b). Microscopic examination of sections from the affected area of the spinal cord shown in Fig. 4b revealed multifocal and locally extensive areas of myelin loss and localized rarefication of glial fibers (Fig. 5). Interestingly however, there were no apparent changes in motor neurons in the affected areas of the spinal cord. There were no apparent gross or microscopic changes in the muscles of the cervical spine.

Microscopic evaluation of cervical vertebrae bone typically showed increased numbers of pathologically altered osteoclasts (Fig. 6). These features were not seen in specimens from normal chickens. Therefore, the increased activity of osteoclasts in the deformed areas of cervical vertebrae appears to be associated with the excessive resorption of bone (Fig. 6a). Interestingly, bone resorption appears to commence as soon as the new bone is formed, where it is clearly discernible in the areas of primary ossification centers and endochondral bone formation (Fig. 6b). Noteworthy are the activities of osteoclasts in the cartilage zone of proliferation, hypertrophic zone, and zone of calcification, as well as in areas where new bone was already formed.
Discussion

Over the entire history of intensive genetic selection for economically important targets, the broiler industry has witnessed a plethora of health problems. Undoubtedly, among the most prominent health problems mar ring the global poultry industry for several decades are skeletal abnormalities [2, 3, 5]. The deformity affecting the cervical spine described in the present report exemplifies a new category of skeletal disorders in broiler chickens.

Idiopathic deformities of the spine in chickens described as scoliosis were reported previously [11], but in these cases the deformity involved the thoracic vertebrae. However, idiopathic cases of torticollis were not reported previously, and reported cases of torticollis were always associated with some causative factor including bacterial infections [12, 13], yeast infection [14], parasitic infections [15], viral diseases including Marek's disease [16], Newcastle disease [17], avian paramyxovirus [18], enteric reovirus strains [19], and avian influenza virus [20, 21]. Torticollis was also observed in broilers fed soybean based diets [22] and lupine based diets [23]. Furthermore, the cases of torticollis reported in avian medicine literature have been described primarily in older birds. Notably, in contrast to previous reports, torticollis in the present study was observed in newly hatched chicks, and had no apparent association with any known foregoing etiology.

To the best of our knowledge, idiopathic cervical scoliosis in combination with torticollis has not been described previously in chickens. At present, the pathogenesis of this novel form of scoliosis/torticollis is not clear. However, it is noteworthy that the studies on etiology of various forms of thoracic scoliosis in chickens have shown that its etiology includes a significant genetic component [24]. Given that the deformity of the cervical spine observed in our studies has attributes of a congenital condition, the genetic predisposition to this anomaly warrants further investigation.

The potential pathophysiological factor that merits consideration is the pineal gland inadequacy, as several studies have shown that in broiler chickens scoliosis can be induced experimentally by removal of the pineal gland [25–27]. In the context of increased activity of osteoclasts observed in the vertebral bone of the affected chicks, of particular interest is the work of Yoshihara et al. [27] where the authors noted that vertebral deformity induced by pinealectomy was associated with increased activity of osteoclasts, and the authors attributed this activity to the development of spinal column deformity through the changes in bone modeling. Since the pineal gland is the primary
source of melatonin, it is possible the melatonin insufficiency may be involved in the etiology of cervical bone changes during embryonic development. Interestingly, it has been shown that melatonin inhibits osteoclast formation and activation [28], whereas lack of melatonin promotes osteoclast proliferation [27]. Notably, in the present study, in comparison to normal chickens, osteoclasts seen in the cervical vertebral bone of the affected broilers were increased in numbers. They are also enlarged and contain a large number of nuclei, which are attributes of osteoclasts pathology (for review see [29, 30]). These features were absent in specimens obtained from normal chickens of similar age. Furthermore, unlike in normal chickens, cervical vertebrae from chickens affected with CCST showed osteoclast activity in the cartilage zone of proliferation, hypertrophic zone, and zone of calcification, as well as in areas where new bone was formed. Taken together, these findings suggest that the cervical vertebrae bone metabolism in the affected broilers involves both impaired bone formation and increased resorption activity. Such changes, which are particularly prominent in the zones where new bone is formed, indicate pathological bone development and remodeling, which is likely associated with activation of osteoclasts. Of note, our observations on increased activity of osteoclasts in cervical vertebral bone from affected broilers bear a similarity with the findings from studies in broiler chickens where scoliosis was induced experimentally by pinealectomy [27, 31]. Hence, the possibility that cervical spine deformity observed in our study may be linked to genetic defect of pineal gland metabolism warrants further investigation.

Conclusion
Congenital cervical scoliosis and torticollis syndrome represents a new form of skeletal disorder in broiler chickens. The increasing trends in the incidence noted in commercial broiler flocks indicate that this new skeletal anomaly should be viewed as an emerging concern. Further work should be directed towards the investigation of possible genetic and/or pathophysiological factors involved in the pathogenesis.

Prior publication
Preliminary observations was presented at Poultry Science Association annual meeting, Louisville, Kentucky, July, 2015. An abstract has been published in the Proceedings of the Poult. Sci. 94 (E-Suppl. 1, abstract 294).

Authors’ contributions
AO (PI) did the field observations and epidemiological data collection, pathology and histopathology, hatchery and clinical data collection in Canada; CW did the pathology and histopathology; BO performed the clinical and hatchery observations in Poland, BL managed the project and prepared the manuscript. All authors read and approved the final manuscript.

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Availability of data and materials
The datasets are available from the corresponding author on reasonable request.

Ethics approval and consent to participate
Experimental protocols were approved by the University Animal Care Committee and procedures were performed in accordance with the requirements of the Guide to the Care and Use of Experimental Animals (Canadian Council on Animal Care, 1993). In the case of the European study all procedures were approved by a Committee for Animal Experimentation according to the principles of the European Union and Polish Law on Animal Protection (Directive 2010/63/EU of the European Parliament and of the Council; Regulation of the Minister of Agriculture and Rural Development: Dz. U. 2008 No. 50 item 368).

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

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References
1. Hocking PM. Unexpected consequences of genetic selection in broilers and turkeys: problems and solutions. Br Poult Sci. 2014;55:1–12.
2. Wise DR. Skeletal abnormalities in table poultry—a review. Avian Pathol. 1975;4:1–10.
3. Thorp BH. Skeletal disorders in the fowl: a review. Avian Pathol. 1994;23:203–36.
4. Julian RJ. Production and growth related disorders and other metabolic diseases of poultry—a review. Vet J. 2005;69:350–69.
5. Olkowski AA, Laarveld B, Wojnarowicz C, Chirino-Trejo M, Chapman D, Wysokinski TW, Quarloni L. Biochemical and physiological weaknesses associated with the pathogenesis of femoral bone degeneration in broiler chickens. Avian Pathol. 2011;40:639–50.
6. Olkowski AA, Kumar L, Classen HL. Changing epidemiology of asciates in broiler chickens. Can J Anim Sci. 1996;76:135–40.
7. Olkowski AA, Wojnarowicz C, Rathgeber BM, Abbott JA, Classen HL. Lesions of the pericardium and their significance in the aetiology of heart failure in broiler chickens. Res Vet Sci. 2003;74:203–11.
8. Olkowski AA, Laarveld B, Wojnarowicz C. Trends in developmental anomalies in contemporary broiler chickens. Int Hatch Pract. 2013;28:1–2.
9. Olkowski AA, Laarveld B, Olkowski BI, Wojnarowicz C. Congenital torticollis in broiler chickens: a recently emerging severely debilitating condition. Poult Sci. 2015;94:Suppl 1:101.
10. Olkowski AA, Nair S, Laarveld B, Wojnarowicz C. Changes in eggshell structure and predisposition of broilers to health problems: is there a common pathophysiology? Br Poult Sci. 2015;56:267–74.
11. Rigdon RH, Mack J. Spontaneous occurrence of scoliosis in the chicken. Avian Dis. 1968;12:530–43.
12. Abe Y, Nakamura K, Yamada M, Yamamoto Y. Encephalomalacia with *Enterococcus durans* infection in the brain stem and cerebral hemisphere in chicks in Japan. Avian Dis. 2006;50:139–41. https://doi.org/10.1637/7419-080805R.1.

13. Kurazono M, Nakamura K, Yamada M, Yonemaru T, Sakoda T. Pathology of listerial encephalitis in chickens in Japan. Avian Dis. 2003;47:1496–502.

14. Wyatt RD, Simmons DG, Hamilton PB. Induced systemic candidiasis in young broiler chickens. Avian Dis. 1975;19:533–43.

15. Bianchini F, Rondini C, Grelloni V, Frescura T. Avian toxoplasmosis: experimental infection of chicken and pigeon. Comp Immunol Microbiol Infect Dis. 1986;9:337–46.

16. Ikezawa M, Goryo M, Sasaki J, Hairy M, Okada K. Late Marek’s disease in adult chickens inoculated with virulent Marek’s disease virus. J Vet Med Sci. 2010;72:1539–45.

17. Oladele SB, Nok AJ, Eisevo KA, Abdu PM, Useh N. Haemagglutination inhibition antibodies rectal temperature and total protein of chickens infected with a local Nigerian isolate of velogenic Newcastle disease virus. Vet Res Commun. 2005;29:71–9.

18. Jung A, Grund C, Müller J, Rautenschlein S. Avian paramyxovirus serotype 3 infection in *Neopsephotus cyanoramphus* and *Neophema* species. J Avian Med Surg. 2009;23:205–8.

19. Van de Zande S, Kuhn EM. Central nervous system signs in chickens caused by a new avianreovirus strain: a pathogenesis study. Vet Microbiol. 2007;120:42–9.

20. Mansour SM, Elbakrey RM, Ali H, Knudsen DE, Eid AA. Natural infection with highly pathogenic avian influenza virus H5N1 in domestic pigeons (*Columba livia*) in Egypt. Avian Pathol. 2014;43:319–24.

21. Smietanka K, Minta Z, Reichert M, Olszewski M, Wyrostek K, Jóźwiak M, van den Berg T. Experimental infection of juvenile domestic and Canada geese with two different clades of H5N1 high pathogenicity avian influenza virus. Vet Microbiol. 2013;163:235–41.

22. Olkowski B, Charuta A, Radziñski R, Bienko M, Toczko R. Skeletal response to diet with soya bean seeds used as primary source of protein in growing broiler chickens. J Anim Physiol Anim Nutr. 2016;100:731–7.

23. Olkowski AA, Olkowski B, Amarowicz R, Classen HL. Adverse effects of dietary lupine in broiler chickens. Poult Sci. 2001;80:621–5.

24. McCarrey JR, Abbott UK, Benson DR, Riggins RS. Genetics of scoliosis in chickens. J Hered. 1981;72:6–10.

25. Kanemura T, Kawakami N, Deguchi M, Mimatsumi K, Iwata H. Natural course of experimental scoliosis in pinealectomised chickens. Spine J. 1997;15:1563–7.

26. Machida M, Dubouset J, Imamura Y, Iwaya T, Yamada T, Kimura J. Role of melatonin deficiency in the development of scoliosis in pinealectomised chickens. J Bone Joint Surg Br. 1995;77:134–8.

27. Yoshihara H, Kawakami N, Matsuyma Y, Inoh H, Magana S, Ishiguro N. A histomorphologic study of scoliosis in pinealectomized chickens. Spine. 2003;30:2244–51.

28. Koyama H, Nakade O, Takada Y, Kaku T, Lau KH. Melatonin at pharmacologic doses increases bone mass by suppressing resorption through down-regulation of the RANKL-mediated osteoclast formation and activation. J Bone Miner Res. 2002;17:1219–29.

29. Ralston SH. Pathogenesis of Paget’s disease of bone. Bone. 2008;43:819–25.

30. Reddy SV. Etiology of Paget’s disease and osteoclast abnormalities. J Cell Biochem. 2004;93:688–96.

31. Aota Y, Terayama H, Saito T, Inoh H. Pineallectomy in a broiler chicken model impairs endochondral ossification and induces rapid cancellous bone loss. Spine. 2013;13:1607–16.

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