MALFORMATIONS OF THE UPPER BEAK (*campylognathia superior*) AND ADJACENT BONES WITHIN THE SPLANCHNOCRANIUM OF DOMESTIC GEESE: AN EVALUATION OF CASES THAT HAVE BEEN DESCRIBED DURING FOUR CENTURIES

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INTRODUCTION

Scientific literature contains an abundance of descriptions of various congenital and acquired malformations of bones and organs of humans but also of farmed mammals and domestic poultry (Lachmund 1673; Thompson Lowne 1872; Gurlt 1877, 1878; Meckel 1781-1733; Lambrecht 1933; Koch *et al*. 1957; Mayr 2007). Some of these malformations are considered as curiosities or monsters, some other reports underline the possible negative effect on rational economic use of affected geese. Regular uptake of food and water requires a 'normal' structure and function of the beak. In contrast, pathological deviations of the beak will inevitably result in impeded food and water uptake and as a direct consequence in poor growth and economic loss (Hilbrich 1978; Pingel 2000).

Various breeds of geese have been kept worldwide since ancient times for commercial and hobby-like purposes. In many countries, farming of geese contributes significantly to the economy of rural people. The main advantages of all farmed geese are physical properties such as production of meat, fat, feathers and down for clothes and bedding. Also, behavioural traits of geese like vigilance and aggressiveness towards alien visitors to farms are frequently employed. Geese are commonly maintained yearlong on grassland and in winter in rather simple constructed enclosures. The feed for geese consists in most cases of vegetation on available grassland and oats or maize. Only in larger farms are commercial diets provided.

REVIEW OF THE LITERATURE

Specimens of prehistoric petrified birds provide no hints on pathological structures of the skeleton (Lambrecht 1933). Authors in Ancient Egypt, in the Roman Empire (Columella Varro, Plinius and Titus all cited from Baldamus 1876) and historic Greece (Aristoteles 350 b. Chr. and Baldamus 1903) concentrated their descriptions on morphology, function and practical use of

healthy animals (Warkany 1977). All historical authors focussed in their voluminous monographs on 'normal' animals and did not mention any form of beak anomaly of birds and geese in particular. All of them stressed the necessity of permanent availability of food and water and, therefore, their paramount importance for the wellbeing of all animals. Accordingly, direct access of geese to drinking and bathing water must be provided at all times in specially designed 'drinkers' or alternatively in small creeks and ponds.

Authors in Medieval Times described in great detail monstrous human embryos, adolescent boys and girls and adult men. In contrast, farmed animals including poultry received only little if any attention. The anatomy, form and function of the body, behaviour and practical use of various birds are detailed in works of Friedrich II (1198-1250) in *De arte venandi cum avibus*, Conrad Gesner (1516-1565) in *Historia animalium*, Girolamo Fabricius de Aquapadente (1537-1619) in *De formation ovi et pulli, tractatus accuratissimus*, William Harvey (1578-1657) in *Exercitationes de generatione animalium*, and Iacobus Theodorus KLEIN in *Historiae avium prodromus* (1685-1759). Additional detailed observations of goose farming during the Baroque period were published by Meyer (1752), Hallen (1760), von Haller (1776) and Barowski et al. (1782). All these authors focussed their interest and writing on 'normal' life of mammals and birds, and obviously tried to avoid descriptions of any monstrous beings. In contrast, Ulysse Aldrovandi (1522-1605) in *Monstrorum historia cum paralipomenis historiae omnium animalium*, described and illustrated numerous crippled mammals, birds and reptiles. A detailed historical account of beliefs, facts, superstitions and interpretations of misshapen individuals was provided by Warkany (1977).

Current literature contains reports that deal with various aspects of healthy geese (Crawford 1990; Frosch 1993; Romanoff 1999; Pingel 2000; Mayr 2007) but omitted skeletal abnormalities that are similar to those described by authors of the Medieval period. The lack of recent publications on bone pathology of geese might be due to the fact that such malformations are rarely seen or are not worthwhile to publish. The aetiology and pathogenesis of malformations of head, wings and feet were not thoroughly investigated (Gurlt 1878). Any proven cause(s) of these alterations remain obscure as was discussed at the First World Waterfowl Conference in Taichung, ROC (Cheng-Taung Wang 1999).

Around the world, various breeds of geese exist that have mostly local significance. With only a few exceptions, sophisticated breeding programs are not applied to geese farming (Crawford 1990; Pingel 2000). Consequently, inbreeding at undetermined levels may be widespread in smallholder farming. So far, the complete genome of the domestic goose remains unknown

(Crawford 1990; Fujihara and Xi 1999). Koch et al. (1957) described a *brachygnathia superior* in White Leghorn chickens. Their paring experiments, using again White Leghorn chickens, provided strong evidence for a single recessive autosomal gene as the causative factor for the deformed upper beak. Asmundson (1936) studied an 'abnormal upper mandible' in domestic fowl and considered a new lethal mutation as likely cause of this defect.

Goose embryos carrying a crooked beak are not able to pick the eggshell and cannot hatch. A few other goslings that hatched with assistance, suffer from inability of food and water uptake (Reinhardt 1925). Inbreeding, poor farming conditions and errors in appropriate maintenance that cause beak malformations raise serious questions of animal welfare (Pingel 2000; Dayen and Petermann 2012). Depending on the degree of beak lesions, most authors differentiate between variation, anomaly and malformation (Gurlt 1878).

A few reports, mainly speculative in nature, associate the development of malformations with the presence of adverse environmental conditions, such as arbitrary exposure to X-rays, rays emitted from mobile phones, electro-smog, radioactive rays from damaged nuclear reactors or even superstition (Dayen and Petermann 2012). In contrast to these opinions, Heiss (2015, personal communication) links the development of beak abnormalities of growing goslings to mechanical trauma, such as accidental fixation of head and neck on obstacles like wired fences and broken structures within the enclosures and to subsequent vain attempts to escape. Such trauma is associated with soft tissue trauma, fissures, luxations or even fractures of the upper beak and other bones in the head.

Deficiencies in regular supply of vitamins and minerals may result in rickets and under-developed bone structures (Pines and Reshef 2015). In contemporary times, certain infectious diseases (Egyed et al. 1974; Warkany 1977; Hilbrich 1978; Smyth et al. 2005; Handel et al. 2010; Guo et al. 2011) are associated with pathological lesions.

Our review of the scientific literature on skeletal malformations in geese provides information on single cases over a range of four centuries. To our knowledge, the first report on a malformation in a goose was published by Schwenckfeld (1603) in his *Theriotropheum (θηριοτροφειον) Silesiae in quo animalium, hoc est, quadrupedum, reptilium, avium, piscium, insectorum natura, vis et usus sex libris perstringuntur concinnatum et elaboratum.* The medical doctor, Caspar Schwenckfeld, was born on 14.8.1568 in Greiffenstein, Silesia and died on 9.6.1609 in Görlitz, Saxony. As far as avian species are concerned, he mentioned an '*Anser monstrosus*'. Schwenckfeld (1603)

examined a rare case of a goose with four legs on its back and reports that this goose succumbed after a short life.

Lachmund (1673), also a medical doctor working at that time in Hildesheim, Germany, confirmed Schwenckfelds's statement on the low frequency of malformations in geese in his textbook under the heading 'De Cygni, Lingua, Ossea' [On swans, the tongue, the bones]. He stated that of all birds, geese exist that may display various monsters. Lachmund (1673) had seen a *cranio anseris monstroso* already in the year 1664 and provided a pen-and-ink drawing of this malformed head including a bent beak of a goose (Fig. 2 A). To facilitate a direct comparison of Lachmund's head to a recently described pathological head by Lingnau (2016) is shown in Fig. 2.

GURLT (1831), professor of veterinary pathology at the university of Berlin, introduced the term *campylognathia superior* for a malformed upper beak. However, Zumpe (1929) pointed out that not only the upper beak but also additional bones of the splanchnocraium appeared altered and he consequently suggests that terms like 'campylorhinus' or 'Schädelskoliose' fit better to describe the observed pathological deformation. Gurlt (1877, 1878) provided information on two geese, one of these had a beak that was bent to the left and the other goose displayed its beak to the right side. This author proposed the name *loxia curvirosta* for these malformations, and for an aberrant shorter beak the name *brachyrrhynchos*. In a detailed publication, Gurlt (1878), summarizes current literature and cites a number of previously published studies on malformations (now re-named 'campylorhinus') in domestic avian and mammalian species. He mentioned different malformations of bones and viscera in horses, calves, piglets, goats, sheep and cites various pathologies also in a frog and domestic chickens, pigeons, ducklings and goslings.

Joest (1926), focused in his review on pathological anatomy of the head of geese. He describes in detail a diverticulum in the lower beak with accumulation of feed. Lambrecht (1933), described in his handbook on Palaeornithology his own observations and those of other authors on deformations of the skeleton of birds. Koch *et al.* (1957), analysed a large number of malformed heads of chickens, turkeys, ducks and pigeons but not of geese.

This contribution places its focus on malformations of the bony structures of the head, the splanchnocranium, and in particular of the upper beak of domestic geese that were published in historical and contemporaneous contributions. The frequency and severity of such lesions, their consequences for food and water

uptake, are considered as welfare-associated questions. Also, the most likely aetiology and pathogenesis of beak abnormalities will be discussed.

MATERIALS AND METHODS

Skull of Lachmund (1673): Lachmund termed his specimen *cranio anseris monstroso*. He published a rather short note in Latin and provided a pen-and-ink-drawing that depicts a monstrous head (Fig. 2 A). The same drawing was subsequently reproduced by Lambrecht (1933) in his textbook 'Palaeornithologie'.

A pathological skull (Lingnau 2016): This specimen was obtained as a complete head from a goose, euthanized in 2013, from a smallholder of domestic geese (Fig. 1 B, D, F). Specimens of 'normal' skulls served as controls (Fig. 1 A, C, E). The pathological head originates from an approximately six months old goose that lived in a small group of geese of the same age under traditional small-scale farming conditions. No accidents or other deleterious events were noted by the owner that could explain the abnormal structure of the beak. Due to poor body condition and any unlikely improvement by surgery or other means, the goose was euthanized and the head was submitted for detailed examination (www.skulls-and-more.de).

Skeletons of several heads of geese are exhibited at the Institute of Veterinary Anatomy, Fachbereich Veterinärmedizin, Freie Universität Berlin, Catalogue No. 4, 1862. Photographs of pathological heads of geese are accessible now using <http://www.vetmed.fu-Berlin.de/-einrichtungen/institute/we01/gurltsche Sammlung>.

Examination and terminology of the bones of heads

The macroscopic normal and patho-anatomical structures and their terminology as described by Vollmerhaus (1992) form the baseline.

RESULTS

Comparison of a normal and a pathological skull LINGNAU (2016)

The skull of an arbitrarily selected healthy adult domestic goose (Figs. 1 A, C, E) serves as a detailed comparison to the pathological skull Lingnau (2016) (Figs. 1 B, D, F). Most obvious is the upper beak in Figs. 1 B, D, F that is bent to the right side at an angle of approximately 90 degrees. The bending initiates at the Zona elastica craniofacialis at the distal end of the Os maxillare and the onset of the Os praemaxillare. On the tip of the Os praemaxillare in

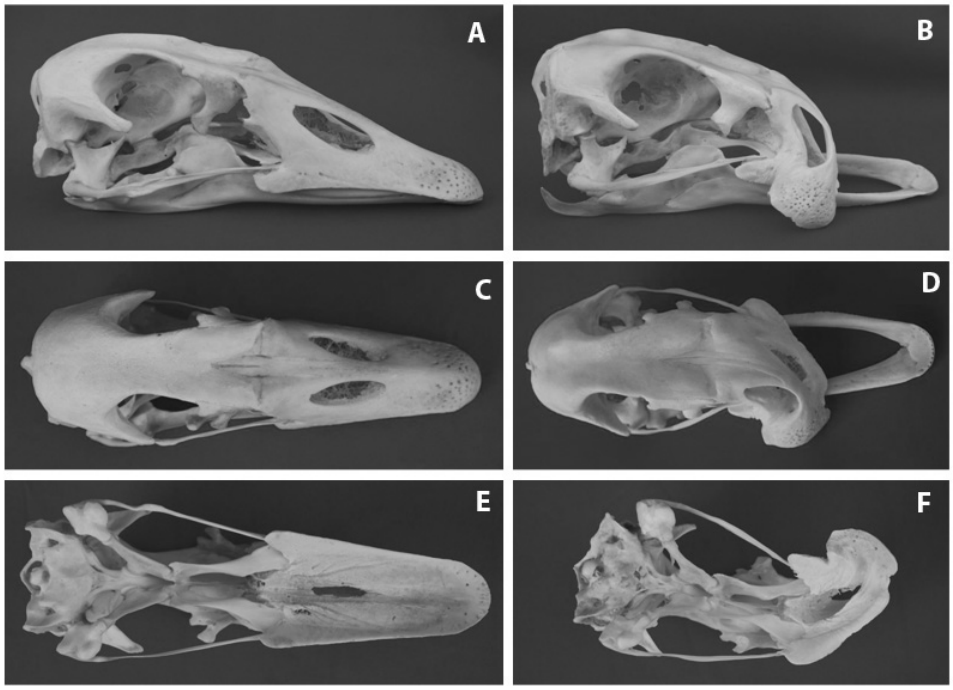

Fig. 1. Lateral, Dorsal and Ventral Views of a 'Normal' Goose Skull (A, C, E) and a Pathological Goose Skull (B, D, F)

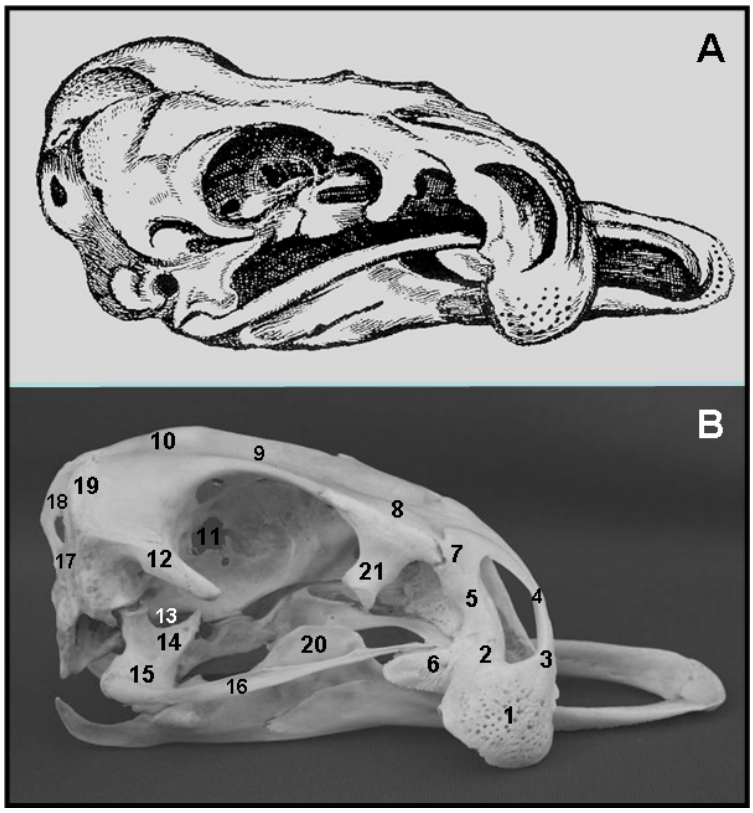

Fig. 2. Comparison of the Pathological Skull (A) Published by Lachmund (1673) and a Pathological Skull (B) by Lingnau (2016) – Numbers Indicate Different Areas of the Skull

Figs. 1 A, B, C, D numerous Foramina neurovasculare are visible that completely penetrate this bone and appear on the oral cavity as clearly seen in Figs. E and F. The Fig. 1 D depicts in comparison to Fig. 1 C more clearly the beginning and extent of the bending of the Os praemaxillare to the right side. The nares in Figs. 1 B and D assumed a more oval shape as compared to the more biconvex form in Figs. 1 A and C which is possibly due to the limited space or to compression due to bending of the tip of the beak.

The ventral view of the skull in Fig. 1 F shows in comparison to Fig. 1 E the asymmetric shape of the Processus praemaxillare. The curved (instead of straight) form of the Os zygomaticum and the compression and slight dislocation of the Processus maxillare palatini are visible. The Os quadratum is also dislocated.

Comparison of the pathological skull Lachmund (1673) *and the skull* Lingnau (2016)

A striking similarity (but not completely identical) exists if the skulls in Fig. 2 A and B are compared. Signs of healing of a previous fracture are not detectable. Distinct ossification is visible in all bones. The neurocranium in Fig. A shows signs of a hydrocephalus. The straight forward directed mandibula in specimens Fig. 2 A and B seems to be of normal size and strength. Only the tip of the mandibula is more rounded in the drawing Fig. 2 A.

The degree of bending of the upper beak appears to be undistinguishable between both specimens. It is obvious that neighbouring bones are affected as a result of the bending of the upper beak. These bones include the distal part of the Os nasale with its Processus orbitale and the Os jugale that combines the Os quadratum with the Os maxillare. The Processus lacrimalis is slightly curved in the drawing of Lachmund (Fig. 2 A) but straight in Fig. 2 B provided by Lingnau (2016). The Os quadratum in Fig. 2 A carries a longer and slightly curved Processus orbitalis. Not visible but most likely affected, is the Concha nasalis. A detailed comparison of shape and size of the bones of heads is presented in Table 1. The terminology used was derived from a healthy skull as described by Vollmerhaus (1992).

DISCUSSION

A distinction has to be made between pathological alterations that already developed during embryonic life and those that were later seen in juvenile birds. A bent beak in un-hatched embryos prevents picking as well as opening of the eggshell which results in death-in-shell embryos. The few embryos that do

Name of bones of the head	Shape and size of bones according to descriptions by	
	Lachmund, 1673	Lingnau, 2016
Os intermaxillare	Distal of zona elastica craniofacialis the tip of beak is bended to right and down	As for Lachmund
Processus frontalis of Os intermaxillare	Bended downwards and to the right side	As for Lachmund
Processus maxillaris of Os premaxillaris	Strongly thicker and bended to the right side	As for Lachmund
Processus orbitalis of Os temporale	to ventral bended	As for Lachmund
Processus lacrimalis	Slender, blunt end	Slender, blunt end
Septum nasale	partially not visible	not visible
Conchae nasales	Not visible	Not visible
Septum nasale	Missing	Missing
Os zygomaticum	Strong, sinus-curve like bended	Slim, straight
Os quadratum	Extended, both Processus dorsalis thicker	As for Lachmund, but larger as Articulatio Ossis zygomaticus
Mandibula	Processus retroangularis only slightly dorsally bended	Slightly shorter as in Lachmund

Table 1. Comparison of Bones of the Splanchnocranium of Pathologically Deformed Skulls Published by Lachmund (1673) and Lingnau (2016). Scientific Names were Derived from Vollmerhaus (1992).

hatch carry various deviations of size and form of the beak. According to anecdotal reports, such birds are usually sacrificed during the early growing period. In maintained goslings, most of the malformations were described as single events within larger numbers of healthy offspring. It is, therefore, reasonable to anticipate that yet unknown factors during the ontogeny of an individual are responsible for malformations in young goslings. Gurlt (1877), argues that it remains enigmatical why deformations of the beak develop in a few but never in many birds of an offspring within a clutch of incubated eggs. He has no acceptable reason to belief that anything went wrong during the phylogeny. He concluded that any cause of malformation must be associated to errors during the ontogeny of birds.

Low humidity during incubation of eggs results in loss of embryonic fluids which in turn could foster adherence of the head of embryos to egg membranes (Crawford 1990; Dodgson 2015). However, Gurlt (1877) states that a mechanical cause like firm adherence of the head of a developing embryo to egg membranes was never proven.

Commercial incubators are set at an average temperature of 37.5 to 38.0 degrees Celsius (Frosch 1993). Minor deviations from these values are general practice in respect to species of birds, their age, the time and temperature during storage of eggs prior to onset of incubation and the size of eggs. Natural incubation of eggs by parents or by foster parents leads to temperatures that are influenced by the body temperature of the incubating goose and environmental temperature conditions. A breeding bird may suffer from fever during egg incubation, which could result in higher transfer of warmth from the breeding bird to the surface of eggs. Such events may cause poor development of embryos including malformations or even deaths.

The most likely cause of a developing malformation in embryos seems to be associated with a slightly elevated incubation temperature (Redmann *et al*. 2012). The temperature in an egg incubator is set at a well determined level, and is constantly monitored and adjusted if necessary at short time intervals. However, it is technically highly demanding to establish and to maintain the desired temperature in all compartments of a large mechanical incubator during the total incubation period of several weeks (Romanoff 1999). Consequently, in some parts of an incubator the temperature might be too high which causes either embryo death or – if less high – various degrees of malformation (Scholtyssek 1987). An elevated incubation temperature (0.2 to 0.5 °C) during the first week of incubation of chicken eggs induced malformed chicks (EFK, personal experience). Reliable data on correct and deviating incubation temperatures of geese eggs and there effects on embryo pathology are not available.

Bones are formed either directly (desmodal osteogenesis) or by intermediate cartilaginous mesenchymal tissue (chondral osteogenesis). A direct desmodal osteogenesial development is characteristic for the Os frontale and most of the bones of the splanchnocranium. All other bones of the skeleton follow a chondral osteogenesis (Sajonski 1969; Denbow 2015). Already during embryonic development, some mesenchymal cells develop into osteoblasts. By enlargement and incorporation of calcium compounds in osteoblasts, the osteoids are formed which develop further to osteocytes. These osteocytes merge during late embryonic and early post-hatch life to initially small islets and subsequently to solid bones (Hansen and Obel 1969).

Considering this pathway of bone development, any abnormal bone formation must be initiated during embryonic life but might be expressed without obvious alteration of bones during early life. This does not preclude that an aberrant bone structure is noted at a later time by an attending person. If an early event is postulated that is responsible for later pathological bone development, we need

to address this question by using a convincing reason for the bone pathology. Currently, we do not have any sound explanation.

Closer examination of pathological skulls did not provide any indication of a previous fracture or any other lesion of the beak bone which could be associated with later deformations of the beak. Also, a blunt trauma to the head directed to the Arteria facialis or to its Ramus nasalis can cause a obliteration of these blood vessels which results in reduced or even total prevention of growth and consequently in bending of the beak to the side of the injury. Therefore, during examination of pathological beak formation, any trauma as a plausible cause of beak deviation has to be excluded.

At the tip of the beak bone and on the Mandibula are large numbers of small pores (Foveae corpusculorum nervosorum) that can be interpreted as natural openings for blood vessels and peripheral nerves (Salomon 1993). The nerve endings of the apical part of the Os premaxillare and the Mandibula are essential for sensation of touch in normal beaks. In contrast, a bent tip of the beak cannot function appropriately for this purpose which deprives a bird of regular sensation of food and water and explains the poor development of geese that carry bent beaks.

Pathological malformations of the head but also of other parts of the body have been described for different domestic avian species (Schwenckfeld 1603; Lachmund 1673; Meyer 1752; Hallen 1760; Barowski et al. 1782; Gurlt 1877/78; Schultka & Göbbel 2005).

Hallen (1760) states ‘no other bird species generates as much malformations as the goose’. However, he did not provide exact data on frequencies and types of malformations. Malformations of osseous structures of beaks, wings and legs are easily detectable and described in conjunction with reduced health and poor physical development (Lachmund 1673; Joest 1926; Lambrecht 1933; Mayr 2007). Therefore, such changes may gain economic significance in commercial geese production and interfere with the interests of hobby breeders. According to information obtained from observations of commercially raised geese, beak abnormalities in growing geese seem to be rare events (Heiss personal communication), but are probably underreported in scientific literature. Consequently, little or no information is available on the frequency and magnitude of such pathological changes within a flock of domestic geese.

We were not yet able to determine the causative factor(s) of these malformations. It is obvious from the cited literature, that similar if not identical malformations were seen and described in historical literature. None of the

reports contain any information on specific cause(s) of these lesions. Therefore, we can only speculate. One explanation could be a direct blunt trauma on the tip of the beak and subsequent poor repair reactions, which resulted in healing of the beak in an oblique position (Heiss, pers. communication). In contrast to this theory, the absence of any callus formation at the location of an assumed fracture has to be mentioned. Another interpretation might be a wrong position of the embryo inside of a rather small egg and faulty development of the beak during the final growth period *in ovo*. However, no information is currently available on the effect of size and shape of eggs, quantitative results of hatching and further development of geese embryos.

A number of investigators see connections between various beak deformities (but different from bent beaks) and external factors that are mentioned here as likely differential diagnoses. The oral uptake of mycotoxins (for references see Dodgson 2015) by eating toxic plants like bishop's weed (*Ammi majus* syn. *visnaga*) cause foreshortening of the beak which is covered with inflammatory scab lesions and extensive scabbing of the dorsal surface of the footweb (Egyed et al. 1974). Natural infections by goose circovirus (Soike et al. 1999; Smyth et al. 2005; Guo et al. 2011) have been discussed to be associated with beak deformation and reduced growth. The goose parvovirus (Derzsy 1967) causes reduced weight gain and abnormal growth of feathers and the corneous layers of the skin including the beak. Other authors failed to detect any causative agent for these types of pathologies (Handel et al. 2010). None of the cited authors mentioned pronounced deviations of the beak in any direction.

Nutritional factors such as insufficient supplementation of the feed with minerals, vitamins, proteins, carbohydrates and fat, respectively, can be excluded as a causative factor for malformations (Heiss 2015, personal communication). In addition, toxic components in the feed or drinking water seem to be rather unlikely due to the fact that all other geese of the flock – except one – developed as expected. Poisoning following oral uptake of some plants, e.g. leafs of the wallflower (*Erysimum crepidifolium*) or the black seeds of the garden nightshade or hound's berry (*Solanum nigrum*), might happen if geese are kept on pastures and fields (Pingel 2000).

A genetic defect which caused beak deformation (Crawford 1990; Dodgson 2015; BAI et al. 2014) can also not be excluded. Developmental abnormalities in beak formation, as we describe here, may be contributed by deregulated expression of genes involved in beak formation as described by BAI et al. (2014) in chickens. This study warrants further investigation into the expression and genetics of such genes. Further detailed studies are essential to elucidate any genetic involvement in the development of pathological bone formation.

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