Trends in developmental anomalies in contemporary broiler chickens – part 2

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The common (and probably most intriguing) anomalies seen in our study were in the general category of deformities involving the embryo’s brain and spinal cord. Representative examples of various forms of these anomalies are shown in Fig. 4.

The commonly observed brain anomalies were characterised as partial absence of some components of the cranial bones (Fig. 4a, arrow), or total absence of the parietal, occipital and temporal bones, with the entire brain being fully exposed (Fig 4b, arrows). In some cases brain anomalies involved structures resembling multiple brains (Fig. 4c, black arrows). Many embryos showed anomalies of the spinal cord known as spina bifida (Fig. 4d, white arrows).

Deformities such as open cerebral cavity (acrania) with brain structures exposed were previously reported. However, the occurrence of anomalies such as excessive brain structures or deformity of the spinal cord has not been observed in commercial broilers prior to discovery in our studies. The pathology of Spina bifida in commercial broilers was described in detail only recently.

Anomalies in hatched chicks

The anatomical anomalies and monstrosities in embryos described above can be categorised as lethal. Interestingly, however, during the course of our studies we observed numerous instances where various anomalies were detected in newly hatched chicks, or were observed during various stages of the growth cycle in commercial operations.

Anomalies most frequently seen in newly hatched chicks are deformities such as multiple sets of legs, and involving beak and/or eyes. An example of a chick with cross beak and missing eyes that successfully hatched is presented in Fig. 5.

This chick was detected in the otherwise normal commercial broiler flock on the third day after placement. It appeared considerably smaller in comparison to normal flock mates, and was very vocal. It is obvious that because of its anatomical anomalies, this bird was not able to eat or drink, which explains its generalised poor body condition. Although some congenital defects may not be lethal during the embryonic development, the chickens with such deformities have no chance of surviving more than a few days and must be euthanised. Otherwise they would face death by starvation and dehydration.

In the case presented above, the lack of sight was the primary factor that reduced the chances of this individual for survival to practically zero. On the other hand, chickens with significant beak deformities, but no eye problems, may function relatively normally in a commercial situation as illustrated in Fig. 6.

This chicken was detected in a commercial broiler flock approaching market age. It was considerably smaller in comparison to normal flock mates, but otherwise appeared to be in good health. It is obvious that anatomical anomalies of this bird impaired its ability to eat or drink normally, which explains its poor growth relative to normal broilers from this flock.

Drastic changes

Manifestation of anatomical anomalies during embryonic development is a part of nature in animals and humans, so it is expected to see some incidence of anomalies. In this context, it was not surprising that we observed malformations in embryos, or in broilers at various stages of the production cycle.

However, our study revealed drastic changes in epidemiological trends, both in the frequency of occurrence and presentation of anomalies.

While relatively rare just a decade ago, in continued on page 13
our experience today chicks showing extra limbs (wings and legs), missing eyes, beak defects etc, can be found in practically every broiler flock. It is also noteworthy that more and more cases of anomalies are also observed by frontline personnel involved in day to day poultry husbandry (personal communications). A typical case of such observation is illustrated by the example presented in Fig. 7.

If the leg anomalies affect the ability of the chicks to move, the affected individuals may survive in the commercial flock for several days, but eventually, being unable to reach feeders and drinkers, they deteriorate. However, in many instances chicks with extra limbs may live without overt difficulties until market weight, but the deformity of legs will always create some technological troubles on the processing line.

From an epidemiological standpoint, observations made by poultry husbandry personnel provide solid evidence that anatomical anomalies have become deeply rooted in the phenotype of contemporary broiler chickens.

Undoubtedly, noticing individual chicks with deformities in the background of thousands of otherwise normal flock mates can be a considerable challenge even for highly trained personnel. So, the fact that anatomical anomalies are detected by casual perusal of the flock is remarkable, but these detected cases likely represent only a proverbial ‘tip of the iceberg’.

The level of congenital deformities in broilers has already reached alarming proportions, but based on our long term observations, it is apparent that the incidence of anatomical deformities continues to increase, and this trend is particularly disturbing.

**Epidemiological trends**

A cross-sectional study conducted during 2012 and 2013 in our laboratory showed that as much as 30-50% of broiler embryos that failed to hatch had anatomical malformations. This is in sharp contrast to the epidemiological studies done between the 1930s and 1950s where the incidence of malformations detected in eggs with dead embryos ranged from 3-9%.

So, if we assume the levels from that period as a baseline of naturally expected incidence in broiler population, it is apparent that after 60+ years of intensive genetic selection for rapid growth, the incidence of congenital anomalies in broilers has increased 5-10 fold.

Interestingly, however, a good deal of the trends in the incidence of anomalies in broilers appears to be a relatively recent episode. Our data collected between 2003 and 2006 revealed that approximately 15-30% of embryos showed malformations, which is roughly half of the incidence seen in 2012 and 2013.

The increasing trends in the incidence of spina bifida in contemporary broiler embryos likely symbolise a more serious and widespread problem, and therefore the entire issue of escalating trends in congenital anomalies observed in contemporary broilers warrants thorough analysis.

The high rate of embryo deformities seen in commercial broilers is definitively a sign indicative of increased susceptibility of modern broiler strains to teratogenic factors.

Interestingly however, we also noted that high incidence of anatomical anomalies detected at hatch in some batches tends to be correlated with increased frequency of occurrence of several common systemic conditions such as enteritis, cellulitis, ascites, cyanosis, and leg problems in broilers flocks.

This suggests that the factors triggering developmental changes and those predisposing broilers to overall health problems are similar.

**Putative causes and factors**

Some monstrosities observed in our studies undoubtedly arise only from genetic causes, but it is most likely that a much greater number are triggered by various risk factors associated with nutrition, environment, feed and water contaminants, and air pollution.

In this context, we focused on the notion that health problems in broilers may be associated with the underlying patho-physiology of breeders. Indisputably, environmental and metabolic status of the hen has a significant impact on the development of the embryo as well as health and performance of the offspring.

No doubt, intensive selection of broilers for rapid growth characteristics inevitably led to narrowing of the genetic pool, and since typically lethal monstrosities are most likely to be associated with inbreeding, it is apparent that the predisposition to some anomalies is deeply rooted in the broiler genome.

On the other hand, the genes that contribute to non-lethal congenital deformities or health problems may be difficult to identify, because they may typically exert their effects by increasing susceptibility to risk factors.

The magnitude of the genetic predisposition effects is most likely greatly augmented by environmental and nutritional factors. So, the high incidence of some anomalies seen in our studies indicates that nutritional and environmental factors are involved.

Notably, practically all monstrosities observed in our study can be experimentally induced by subjecting the developing embryo to the influence of various gases, abnormal temperatures, restricted or excessive amounts of oxygen, excessive levels of carbon dioxide, fumigants, drugs, chemical factors (pollutants such as PBC, dioxins etc) and nutritional factors (deficiency or excess of some vitamins, amino acids, minerals etc).
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Of note, deformities of brain and spinal cord observed typically in our study, as well as various eye defects have been linked to nutritional factors such as deficiencies of vitamins A, E, B12, B6, and folic acid.

On the other hand, anomalies can also be associated with over-supplementation of some common nutrients.

For instance, excessive maternal dietary intake of vitamins A, D, and E, or selenium can induce anatomical deformities in the embryos.

In particular, vitamin A and selenium can cause high rates of developmental abnormalities (teratogenicity) in birds.

Taken together, with regard to nutritional factors, both deficiency and toxicity may be associated with anomalies that were prominent in our studies. In the commercial situation, over-supplementation of some essential nutrient in the breeder diet may be a very realistic risk factor.

**Environmental pollutants**

The high risk of anatomical anomalies has also been associated with maternal exposure to common environmental pollutants, chlorination disinfection by-products in drinking water, and electromagnetic fields.

All these factors have become enduringly entrenched in our environment.

In particular, exposure to common pollutants such as PCBs, dioxins, heavy metals, hydrocarbons, pesticides and others through atmospheric pollution, water sources, and contaminated feed stuffs is a growing concern.

**Conclusions**

So, most likely all prominent monstrosities documented in our studies can be triggered by environmental and/or nutritional factors. Selection for rapid growth traits may be associated with higher genetic susceptibility to nutritional and environmental factors.

However, the causes of the anomalies and health problems observed in our studies may be the toughest to crack, because these are most likely complex and multifactorial conditions and, as such, they cannot be ascribed to mutations in a single gene. Rather they arise from the interactions of combined genetic, environmental, management and nutritional risk factors.

The high incidence, and more so increasing trends in the incidence of broiler embryo deformities, is a sign of a profound problem in the poultry industry, but it is noteworthy that increasing trends in the incidence of embryonic anomalies has been also noted in recent years in wildlife and humans.

So, environmental factors undoubtedly play a major role in the epidemiological trends of anatomical anomalies seen in domestic animals, wildlife, and human populations.

The very high incidence of anomalies observed in broiler embryos should be viewed as a proverbial ‘canary in the coal mine’ and the findings from broilers should serve as a notice of serious changes.

In this context these issues warrant immediate urgent attention in order to contain this rapidly developing problem.

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Fig. 7. Anomaly involving leg deformity and an extra set of legs observed in a commercial broiler chicken.

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