Breeders reflect on genetics and disease resistance at Merial Avian Forum

At the recent Merial Avian Forum the major breeders gave presentations on the correlation of immune foundation and health status with genetic expression.

William Stanley of Aviagen highlighted that health status is an important component of broiler breeding and that optimal genetic expression of production traits is correlated with immunity and health status. Within Aviagen's breeding programme, selection for traits to improve health, immunity and animal welfare include a targeted focus on improving leg health.

Selections to reduce skeletal leg deformities have been in place since the early 1970s and since then, many other health and welfare traits, including tibial dyschondroplasia, contact dermatitis (foot pad dermatitis and hock burn), heart and lung function and liveability have been successfully introduced into their broiler breeding programme.

Range of welfare issues

Various studies have shown that leg disorders are associated with a range of welfare issues, including pain, reduced activity, and decreased feed intake due to pain, and in Europe the levels of contact dermatitis at post-mortem inspections are used as an indicator of poor welfare conditions.

There is substantial variation among broiler strains in the prevalence of leg disorders, the majority of which is due to differences in stockmanship, housing, nutrition and health. The genetic component of leg health traits is typically low, ranging from 10-25%. In the selection process, any bird with skeletal leg deformities is discarded for breeding and culled using a 'zero tolerance' policy. This has led to a tremendous improvement of leg health over the last 25 years. Furthermore, fully pedigreed populations allow the identification and rejection of birds originating from families with higher incidences of leg disorders.

Since 1989 Aviagen has used an X-ray device (Lixiscope) for the identification of tibial dyschondroplasia at clinical and subclinical levels. The combination of culling of affected birds and identification of superior families has resulted in a decrease of tibial dyschondroplasia by 1% per year.

From 1990 onwards all selection candidates were assessed for hock burn and in 2006 pododermatitis was added to the selection process.

To improve the robustness of the birds, Aviagen introduced a 'commercial sibling test' in 2000, in which brothers and sisters of selection candidates are grown in a non-biosecure commercial environment assessing gut health, digestive and immune function along with liveability, growth and uniformity, as well as leg health. High genetic correlations seen between the biosecure pedigree environment and the commercial sibling test environment suggest that a reduction in an undesirable trait, such as the predisposition to develop foot pad dermatitis, can be achieved through direct selection in the first environment and confirmation of a response from recording in the second environment.

The same principles outlined for leg health apply to heart and lung function and liveability, where individuals are selected from families of superior quality. In 1991 Aviagen implemented pulse oximetry to measure the oxygen saturation level of the blood, which is an important indicator of susceptibility to develop ascites and sudden death syndromes.

Only birds with a family index above the average are selected, thereby reducing the incidence of ascites and sudden death at commercial level. This has resulted in significant improvements in liveability and heart and lung function through the years and continues to do so.

Table 1. Focal duodenal necrosis (FDN) was found in 28 of 228 birds necropsied for a prevalence of 12.3%.

Age (wks)	FDN	Birds	%	Av. age
10-19	4	22	18.2	16.7
20-29	16	35	45.7	22.5
30-39	3	41	7.3	35.0
40-49	1	26	3.8	45.3
50-59	1	26	3.8	53.8
60-69	2	36	5.6	64.8
70-79	1	42	2.4	72.0
Total	**28**	**228**	**12.3**	**44.3**

Innate immunity

In his presentation, Igal Pevzner of Cobb Vantress highlighted research carried out at the USDA ARS Center at College Station, Texas on innate immunity which is a general defence mechanism that protects the animal against a wide spectrum of pathogens. The presentation centred on identifying novel tools that Cobb could use to select chickens for improved disease resistance to a variety of pathogens and not necessarily to only a single disease.

It was demonstrated that genetic differences for innate immunity and disease resistance in Cobb lines were associated with expression levels of several pro-inflammatory cytokines and chemokines. A divergent selection for high and low expression levels of a variety of key cytokines and chemokines resulted in 'High' and 'Low' experimental chicken lines.

These High and Low lines were challenged throughout the selection experiment with various pathogens and the results indicated that innate immunity is closely associated with genetic control of health and disease resistance.

It was shown that in response to challenge with several bacterial pathogens and coccidiosis the high-immunity birds were more resistant than their low-immunity counterparts. A challenge study with necrotic enteritis indicated significantly higher resistance in the High compared to the Low lines. However, no differences could be detected in response to challenges with the avian influenza and Marek's disease viruses.

Chickens with higher levels of innate immunity had a low degree of organ invasion and mortality compared to their

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low-immunity counterparts in response to salmonella infection. The High line also had lower campylobacter colonisation, indicating again the general nature of disease resistance associated with innate immunity. Although not yet proven it is thought similar findings for necrotic enteritis would soon be proven.

Development of pathogen-resistant birds provides the poultry industry with more robust birds and a final product that is safer and healthier for the consumer.

Ian Rubinoff from Hy-Line discussed focal duodenal necrosis (FDN) in a European context. This new disease was first recognised in the USA in 1997. The aetiological agent is thought to be Clostridium colinum with a possible role of Clostridium perfringens, although this has not yet been proven. FDN has been found across all commercial layer breeds and in a variety of different housing systems.

Typically, there are no recognisable clinical signs to be seen in live birds. Affected flocks usually have low egg weights, decreased egg production, failure to gain or maintain body weights, and they often also have calcium deficiency.

Diagnosis involves euthanising healthy birds and assessing their ascending and descending duodenal loops for evidence of focal and multi-focal to coalescing plaques and lesions. Due to normal post mortem liquefaction, these lesions often can not be identified by necropsy of mortality that is more than a couple of hours old (Table 1).

Routine necropsy of birds on a poultry farm is the most effective way to monitor flocks for enteric diseases or other health and nutritional issues. Early observation will ensure that appropriate treatments can be utilised prior to excessive mortality or production issues.

Treatments typically involve using a zero day egg withdrawal antibiotic, although alternative treatments are being explored.

Paul Grignon Dumoulin of ISA-Hendrix looked at the correlation of immune foundation and health status with genetic expression. Optimisation of the immune system starts from early age and is completed by about five weeks of age. Good bodyweight development during brooding is necessary to get a good immune system for the whole life of the bird. This means that feed and feeding method for young chicks has a big impact. Feed components and anti-oxidative properties can also modulate the immune response.

Table 2 shows the impact of feed supplementation on some growth parameters and IBD titers. Good antioxidant status of the birds allows better vaccinal response and better resistance to disease challenge.

Some diseases can also be responsible for immunodepression of birds (Marek's disease, CAV, reovirus, IBD and others). The control of such diseases is necessary to prevent increased sensitivity to field disease challenge and, as a consequence, better expression of the genetic potential.

On top of those immunosuppressive diseases, farmers try to improve antibody titers by vaccinating more frequently during rearing period, but also during the production period. The main problem of such repetitive vaccinations is that, when using low attenuated vaccines, this may also be responsible for production drops.

A good immune system and good vaccination practices are key to getting the best protection out of layers and as a consequence a good expression of their genetic potential.

The fact that layer lifecycle is long (and will be longer in the coming years) imposes long

	Daily growth (g)	FCR	IBD titers (log10)
Flavonoid + zinc (at 2nd IBD vaccination)	97.8	1.78	2.0
Control	99.7	1.76	1.5

Table 2. Effect of feed supplementation on vaccinal response (20-35 days).

lasting protection on the birds, that is frequent vaccination, including during production period. New vector vaccines can also be a solution to improve resistance of the birds and control immunosuppressive diseases.

Genetic potential

Matthias Vos of Lohmann Tierzucht then emphasised that the genetic potential of layers in poultry breeding today can only be achieved if biosecurity is combined with optimal immunisation strategies. In addition, for some disease complexes, genetic selection could be used to overcome or reduce disease associated clinical problems or mortalities.

Peak production persistency of lay and egg quality in the second half of the laying cycle has been the major selection criteria for commercial layers in recent years. In the past a normal laying cycle was 72 weeks but today commercial layers can easily be kept for 90 weeks or even longer.

This results in the need for farmers, veterinarians but also vaccine producers to evaluate the vaccination strategies before and during production in order to provide sufficient protection against diseases like Newcastle, infectious bronchitis and others.

For many years Marek's disease has been the best example of genetic selection for disease resistance. Peripheral neuropathy (PN), a disease condition described in White Leghorn with similar clinical signs to those of the nervous form of Marek's, is another example.

With PN, 1-3% affected birds during week 6-8 of life could be seen. Clinical symptoms are paralysis and at post mortem examination swollen nerves but no tumours can be seen. No Marek's disease virus is involved in the condition and there is also no protection from various methods of Marek's vaccination. PN seems to be a kind of autoimmune disease with increased susceptibility of certain MHC/B blood groups.

The primary cause is a demyelination of peripheral nerves which is associated with the blood group B19, whereas birds with blood group B21 show a higher resistance against the condition. Genetic selection by increasing the population of B21 carrying birds has clearly reduced the problem in the field.

Despite a moderate heritability for E. coli resistance, in contrast to PN, it is difficult to create genetic progress in E. coli resistance using conventional genetic tools because recording of phenotypes is difficult and due to animal welfare concerns can not be applied to high numbers of hens.

As seen in Table s, which is based on results of a challenge trial with 353 pure line white layer hens from 20 families, there is no clear difference between resistant and susceptible hens within a line.

Identification of genes that influence E. coli resistance and genomic selection would have the advantage that no more challenge tests are necessary and the results are available already at young age of the selection candidates. However, preliminary results indicate that there is low to medium genetic variation for E. coli susceptibility. ■

Trait		Resistant	Susceptible
Rate of lay (%)	Period 1-2	61.2	60.6
	Period 3-7	96.2	96.2
	Period 9-12	90.0	91.0
Egg weight (g)		58.7	58.7
Breaking strength (N)		42.9	43.2
Daily feed intake (g)		102.4	102.8
Body weight (g)		1601	1595

Table 3. Challenge trial results.