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Animal genomics and infectious disease resistance in poultry

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Summary
Avian pathogens are responsible for major costs to society, both in terms of huge economic losses to the poultry industry and their implications for human health. The health and welfare of millions of birds is under continued threat from many infectious diseases, some of which are increasing in virulence and thus becoming harder to control, such as Marek's disease virus and avian influenza viruses. The current era in animal genomics has seen huge developments in both technologies and resources, which means that researchers have never been in a better position to investigate the genetics of disease resistance and determine the underlying genes/mutations which make birds susceptible or resistant to infection. Avian genomics has reached a point where the biological mechanisms of infectious diseases can be investigated and understood in poultry and other avian species. Knowledge of genes conferring disease resistance can be used in selective breeding programmes or to develop vaccines which help to control the effects of these pathogens, which have such a major impact on birds and humans alike.

Keywords
Animal quantitative trait locus database – Avian – Avian influenza – Candidate gene – Chicken – Disease resistance – Genomics – Infection – Marek's disease virus – Poultry – *Salmonella*.

Introduction

The chicken has long been used as a model organism for developmental and immunological studies (1), but it was not until the 1990s, when detailed genetic maps of the chicken were developed (2, 3, 4, 5), that an understanding of gene and chromosomal organisation began to advance. Publication of the chicken genome sequence in 2004 (6) proved a game-changer in chicken genetics and paved the way for the revolution in avian genomics in which we find ourselves today. More than 57 other bird genomes are now available (7), with the ultimate aim of sequencing each of the 10,476 known avian species (b10k.genomics.cn). The chicken, however, remains the best-studied avian genome and acts as the reference upon which other bird genomes are based.

Recent advances in high-throughput sequencing methodologies, genome annotation, variant discovery, gene expression, etc. have also meant that data are being produced at an unprecedented level. In addition, most of this information is available in public databases and can be viewed by genome browsers, providing a number of ways to visualise and interrogate the data. This large amount of available knowledge means that one important consequence of the genomics era is an understanding of how genes from different organisms react and interact with each other during the course of pathogenic infection. As the pressures of food security and the risks from pandemic disease increase in an ever-expanding global population, understanding the genetics of disease resistance is undoubtedly a priority. Identification and refining of quantitative trait loci regions (QTLRs), discovery of novel genes, high-density variant maps, whole-genome association studies and increasing knowledge of genetic and epigenetic gene regulation mean that researchers are now in a position to unravel the biological mechanisms of host–pathogen interactions.

This review will look at the current situation in poultry and summarise the resources available for chicken, turkey and duck species. The authors briefly report on how genomics is currently being used in the fight against viral, bacterial and parasitic avian pathogens, with a more detailed look at what is being done to understand Marek's disease virus (8, 9) and *Salmonella* infections.

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Genomic resources

Many resources are now available to avian genome researchers which are all being used to gain an understanding of how the host is affected upon infection with pathogens. These include:

1. Genetic linkage maps: chicken (5), turkey (10), and duck (11)

2. Bacterial artificial chromosome (BAC) physical maps: chicken (12), turkey (13)

3. Radiation hybrid maps: chicken (14), duck (15)

4. Single nucleotide polymorphism (SNP) maps: chicken (16, 17, 18), turkey (19), duck (20)

5. Expressed sequence tag (EST) collections: chicken (21, 22, 23), turkey (24, 25), duck (26, 27)

6. Microarrays (28): as listed in Array Express (29, 30, 31)

7. SNP arrays: chicken (32, 33)

8. Copy number variants (34): chicken (35, 36, 37), turkey (38), duck (39)

9. RNA sequencing (RNA-Seq): chicken (40, 41), turkey (42), duck (43), and PacBio data (44)

10. Genome sequences: both host and pathogen genome sequences are now publicly available

11. Genomic databases:

- Ensembl (www.ensembl.org/index.html)
- University of California Santa Cruz (genome.ucsc.edu)
- Array Express (www.ebi.ac.uk/arrayexpress)
- Gene Expression Omnibus (www.ncbi.nlm.nih.gov/geo)
- chicken section of the Animal Quantitative Trait Locus (QTL) database (QTLdb) (www.animalgenome.org/cgi-bin/QTLdb/GG/index), with a list of disease susceptibility traits as shown in Box 1
- BirdBase (birdbase.arizona.edu/birdbase)
- Avianbase (avianbase.narf.ac.uk/index.html)
- VIrus Pathogen database and analysis Resource (VIPR) (www.viprbrc.org/brc/home.spg?decorator=vipr)
- National Microbial Pathogen Database Resource (www.nmpdr.org/FIG/wiki/view.cgi/Main/WebHome).

Box 1

List of chicken disease susceptibility traits found on the Animal Quantitative Trait Locus database

Alternative complement activation by BRBC {ACBRBC}

Antibody response to *Brucella abortus* (BA) {ABR-BA}

Antibody response to *Escherichia coli* {ABR-E.coli}

Antibody response to KLH antigen {ABR-KLH}

Antibody response to MB antigen {ABR-MB}

Antibody response to Newcastle disease virus (NDV) {ABR-NDV}

Antibody response to *Salmonella* Enteritidis (SE) {ABR-SE}

Antibody response to sheep red blood cells (SRBC) antigen {ABR-SRBC}

Antibody titre to infectious bursal disease (IBD) {ABIBD}

Antibody titre to infectious bronchitis virus (IBV) {ABIBV}

Antibody titre to KLH antigen {ABKLH}

Antibody titre to LPS antigen {ABLPS}

Antibody titre to LTA antigen {ABLTA}

Antibody titre to SRBC antigen {ABSRBC}

Caseous caecal core {CCECC}

Caecal bacterial burden after challenge with *S.* Enteritidis {CECUM-SE}

Caecal bacterial burden after challenge with *S.* Typhimurium {CECUM-ST}

Classical complement activation by SRBC {CCSRBC}

Cloacal bacterial burden after challenge with *S.* Enteritidis {CLOAC-SE}

Cloacal bacterial burden after challenge with *S.* Typhimurium {CLOAC-ST}

Interferon-gamma level {IFNG}

Lesions {LES}

Liver bacterial burden after challenge with *S.* Enteritidis {LBACT-SE}

Marek's disease-related traits {MD}

Oocyst shedding {OOCSHD}

Viral infections

Viral diseases of poultry have a huge economic impact on the industry, with the ability to affect performance and productivity even if birds do not show overt clinical signs of disease. Many viruses also leave birds immunosuppressed, which then renders them susceptible to secondary bacterial infections.

Marek's disease

One of the most widely studied avian pathogens is Marek's disease virus (MDV) and various reviews on Marek's research

are available (e.g. 45). This highly contagious herpesvirus is responsible for losses to the poultry industry of around US$2 billion per annum (46). Signs of Marek's disease include depression, wasting, loose watery stool, paralysis, lymphomas and severe immunosuppression. Even if birds survive the disease, they are left highly susceptible to secondary infections such as *Escherichia* coli. MDV affects mainly young birds, with most clinical signs seen at around 12 to 24 weeks of age (8, 9).

The genetics of the host response to the virus have been studied for many years. There are known to be several loci involved in resistance to the disease but, to date, only a few genes have been identified as having a role. Older studies suggested that only a few major loci were involved, but it now seems clear that many loci of small effect are in play, which creates a challenge to define actual causal genes and variants. Genotyping birds known to be susceptible or resistant to the virus has resulted in the location of various QTLRs in the chicken genome which appear to be implicated in Marek's disease (MD) resistance (47, 48, 49, 50). The major histocompatibility complex (MHC) is known to play an important role in disease resistance in general (51), and in MD in particular (52). Non-MHC genes currently suggested as affecting susceptibility to MDV include *GH1* (yeast-2-hybrid assay and co-immunoprecipitation) (53), *SCYC1* (microarray and genetic mapping) (54), *SCA2* (virus–host protein interaction screen) (55), *IRG1* (microarray and SNP mapping) (56), *CD79B* (allele-specific expression) (57) and *SMOC1* and *PTPN3* (genome-wide association studies) (58), but many more remain to be determined. Genome-wide association studies have been carried out on commercial brown egg-layers, and a potential QTL for MD mortality has been identified and candidate genes suggested (59).

The availability of high-density SNP chips, used in conjunction with large populations of birds with known resistance phenotypes, will allow for the refining of known QTLRs, thus reducing the number of potential resistance candidate genes. Continuing improvement of genome annotation (R. Kuo *et al.*, Roslin Institute, unpublished data), particularly in identifying non-coding RNAs, enables the role of these molecules in disease resistance to be elucidated. Micro RNAs (miRNAs) have been seen to be important in transcriptional regulation (60, 61, 62, 63), as has methylation (64, 65, 66), and these potentially have a crucial role in directing gene expression during the host response to infection. RNA-Seq methodologies can also now enable the determination of variants in candidate genes which show allele-specific expression between susceptible and resistant birds (57, 67, 68).

Avian influenza

Avian influenza (AI) (69) poses a very serious threat, not only to poultry, but also to humans, as the possibility of a zoonotic pandemic increases. Wild birds such as ducks act as carriers for the disease, with chickens and turkeys succumbing to infection. Depending on their ability to cause disease, viruses are classified as being of 'high pathogenicity' or 'low pathogenicity'. The current AI crisis in the United States, where more than 48 million birds have been killed or culled due to infection with highly pathogenic H5N2/H5N8, spotlights the need to understand the mechanisms of resistance to influenza (70). Literature searches provide access to many reviews of different AI strains in different species and their pathogenic potential (71 and many recent publications).

Although a few QTL for AI resistance have been reported in mice, with some candidate genes postulated (72), none have been reported in avian species as yet. Transcriptomic studies are getting under way, however, with Wang *et al.* (73) studying gene expression in resistant Fayoumi chickens and susceptible Leghorn birds. Studies have also compared the host responses of ducks and chickens after infection with both low- (74) and high-pathogenicity viruses (75, 76). Smith *et al*. (77) report differing responses of the interferon inducible transmembrane (IFITM) genes between chickens and ducks and hypothesise that this is one mechanism by which ducks can tolerate AI infection and chickens cannot. The lack of the viral sensor retinoic acid-inducible gene I (*RIG-I*) in chickens has also been postulated as a reason for varying susceptibilities to AI among species (78). Polymorphisms in the *MX1* gene are also associated with differing susceptibilities to avian influenza in different species (79). A recent genome-wide association study for immune-related traits in Beijing-You chickens has identified candidate SNP for involvement in the chicken immune response, including the response to AI virus (80).

Newcastle disease virus

Newcastle disease virus (NDV) is highly contagious and has a wide host range (81). Virulent strains of the virus are responsible for high mortality in chickens, although the effects are milder in turkeys, with the main problem being that of reduced production in breeder flocks (82). Clinical signs include depression, ruffled feathers, open mouth breathing, hyperthermia, anorexia, listlessness and hypothermia before death (83). In 2011, it was reported that NDV was responsible for the fourth greatest loss to the poultry industry after highly pathogenic avian influenza (HPAI), infectious bronchitis virus (IBV) and low-pathogenicity avian influenza (LPAI) (84). The current defence strategy is to use vaccination, although the focus has recently turned to genetics to address the control problem. Ten alleles have been identified within various markers (MHC-B locus, LEI0070, ADL0146, LEI0104, ADL0320, ADL0304), which show a favourable response to antibody titre against NDV in native Cameroon chickens (85).

A genome-wide association study has also been conducted, which found two SNPs in and around the *ROBO2* gene as being involved in modulating antibody response (86).

Infectious bursal disease virus

Infectious bursal disease virus (IBDV), which is responsible for infectious bursal disease (IBD) or 'Gumboro' disease (named after the place where it was first identified in 1962), is an increasingly serious problem for the poultry industry (87). It is highly infectious in young chicks and targets the lymphoid organs, and primarily the Bursa of Fabricius. Almost a bigger problem than the disease itself is the immunosuppression that results from IBDV infection. Current control measures centre on vaccination with both immune complex vaccines and the Marek's herpesvirus of turkeys (HVT) vaccine. However, more and more virulent forms of the virus continue to emerge and it is becoming obvious that an understanding of the host–pathogen interaction and underlying molecular mechanisms of the disease is required (88). Different chicken lines have been shown to exhibit differing susceptibility to IBDV infection (89), and two of the most extreme lines have since been used in gene expression studies to try to define the genetic basis of resistance. Bursa and spleen from lines 61 and BrL have been used in microarray experiments to identify genes differentially expressed between susceptible and resistant birds (90). Recently, RNA-Seq has also been used on IBDV-infected chicken embryo fibroblasts to look at the very early responses to infection. Genes involved in cell membrane fluidity and anti-apoptotic mechanisms are highlighted (91).

Infectious bronchitis virus

Infectious bronchitis virus is a highly contagious gammacoronavirus (γ-coronavirus) which has serious consequences for chicken flocks and the poultry economy. It infects the upper respiratory tract and the reproductive tract (with serious implications for egg production) and some strains also cause nephritis. There are many serotypes of the virus and most are not cross-protective (92). A novel duck coronavirus has also recently been identified (93). Again, vaccination is the current method of attempted control, with the major viral attachment protein, the protein spike, being the target for vaccine development. It is known to be involved in tissue binding, cell tropism and pathogenesis, and so is an obvious choice in vaccine research (94). As with many infections, the MHC is known to confer resistance to birds against IBV (95). However, very few studies have been undertaken to clarify the genetics of disease resistance. Genes expressed in the lung upon IBV infection have been examined in a small microarray (1,191 genes) experiment (96), with a larger whole-genome array highlighting expression differences between susceptible (line 15I) and resistant (line N) birds (97). Further work involving RNA-Seq and genome-wide association study technologies would obviously help further research into this problematic pathogen.

Bacterial infections

Bacterial infections of poultry not only cause major losses for poultry breeders but also pose a very real threat to human health through the consumption of infected meat.

Salmonella

Salmonellosis, caused by the Gram-negative enteric bacteria *Salmonella*, is a frequently occurring disease in poultry stocks. While certain serotypes of *Salmonella*, such as *S.* Pullorum and *S.* Gallinarum, are host-specific and the major cause of salmonellosis in poultry, other serotypes, e.g. *S.* Typhimurium and *S.* Enteritidis, can infect humans after the consumption of contaminated poultry meat and eggs (98). Asymptomatic carriage of the pathogen by chickens is the principal cause of contamination of poultry products as it is difficult to identify and isolate the carriers, thus *Salmonella* is a serious public health concern.

Prophylactic measures, including vaccination and the use of antibiotics, are often insufficient to eradicate the disease fully in poultry. Selective breeding, therefore, has been considered a valuable alternative (99). Early selective breeding efforts and related studies focused on reducing the incidence of the disease in poultry production systems. Later studies, however, gave more emphasis to selection for resistance to the carrier state ability or bacterial colonisation of the birds, to reduce asymptomatic propagation of the pathogen (98, 100).

To dissect the genetic basis of *Salmonella* resistance, a large number of studies have been conducted on different aspects of the disease. These include assessments of variations in the susceptibility of different chicken lines (101, 102); estimations of heritability (83, 103); analyses of QTL (104, 105, 106, 107, 108); and assessments of candidate genes (109, 110, 111). These studies have been conducted on widely varying models, using chickens of different ages (either young chicks or adult laying hens) and from different genetic backgrounds for experimental infection. Resistance has been measured based on a range of traits, such as survival rate, bacterial load in various organs at different time points post infection, innate or adaptive immune responses, and antibody responses towards vaccination, gene expression, etc. (98, 100).

The use of different models in different studies has elicited variable results, proving that the genetic control underlying *Salmonella* resistance is complex. Many QTL and candidate

genes – located in 16 of the 38 autosomes in chickens – have been identified as having an association with resistance to salmonellosis and/or the carrier state, with these associations varying according to the genetic background of the chicken, the age of the birds, and the traits assessed (98, 100). A major QTL called *SAL1*, associated with salmonellosis resistance in chickens, has been detected on chromosome 5 (105). Fine mapping of the QTL using advanced back-crossed lines and a high-density SNP panel refined its position to between 54.0 to 54.8 megabases (Mb) on chromosome 5 (112). This QTL is possibly involved in bacterial clearance by macrophages in resistant birds. The QTL covers 14 genes, including the two most likely candidates, i.e. CD27-binding protein (SIVA) and the RAC-alpha serine/threonine protein kinase homolog, *AKT1* (protein kinase B, *PKB*). A recent publication on genome-wide QTL analysis using a dense marker panel has reported four QTL associated with *Salmonella* colonisation, including one genome-wide significant QTL on chromosome 2 and three additional QTL on chromosomes 3, 12 and 25, significant at the chromosome-wide level (108).

A large number of studies have investigated candidate genes for their association with *Salmonella* resistance in chickens (98, 100). Most of these genes are known to have other effects on immunity and some have been found to be associated with *Salmonella* resistance in other species, such as mice. For instance, *SLC11A1* was found to be associated with survival rate and bacterial load in the spleen, liver and caeca of chickens after their infection with *S.* Typhimurium. The *TLR4* gene was linked to resistance to *S.* Typhimurium infection. Several other genes, namely *CD28*, *IAP1*, *TGF-β2, 3, 4*, *GAL11, 12, 13*, *TRAIL*, *IL–2, IL–10*, *PSAP*, *IGL*, *CASP1*, *iNOS*, *PIGR*, and *MAPKAPK12*, were found to be associated with a variation in caecal load of *S.* Enteritidis. The association of *SLC11A1* and *TLR4* has been evaluated in several experimental populations. None of these genes, however, were found to have major effects and in many cases the effects were not stable across populations. Microarray-based gene-expression analyses have also identified many genes differentially expressed in different chicken lines upon challenge trials or between control and infected birds. These are also candidate genes that might have a direct or indirect involvement in *Salmonella* resistance.

The wealth of genetic studies and the new data that are being generated by high-throughput technologies, such as microarrays and RNA-Seq, are fast increasing our understanding of the genetic mechanism of *Salmonella* resistance in chickens. Although traditional selective breeding has, so far, been applied in relation to *Salmonella* resistance, results from QTL and candidate gene analyses could be applied in marker-assisted selection (MAS). However, most of the QTL identified thus far are for small effects with large confidence intervals and MAS is not very effective for these. Under the circumstances, genomic selection offers a more efficient approach, as this does not require the identification of major QTL but only that genome-wide SNP markers tag all the genes with small or large effects, to be incorporated in the estimation of the genetic merit of breeding candidates. With the availability of high-density genotyping arrays (33), and millions of SNP variants from the chicken genome (113), this is soon going to be a reality.

Campylobacter jejuni

Although a harmless gut commensal in chickens, *Campylobacter jejuni* is the major cause of food poisoning in the United Kingdom, with 80% of cases caused by contaminated poultry (114). Efforts to elucidate resistance mechanisms have centred around bacterial colonisation as opposed to actual resistance, since birds which themselves show immunity can still go on to spread infection. Levels of colonisation are strain-dependent and have been shown to be heritable (115). Research into *Campylobacter* colonisation has often gone hand-in-hand with that of *Salmonella*, with the view that colonisation by each pathogen would be orchestrated by similar biological mechanisms (100, 116). However QTL for resistance to colonisation were found to be located in different regions of the genome for each species, although it is assumed that there are probably shared resistance factors. The *Campylobacter* resistance QTL included one genome-wide significant QTL on chromosome 11 and three QTL on chromosomes 7, 12 and 27, which were significant at the chromosome-wide level (117). A gene expression analysis of colonisation-susceptible and colonisation-resistant birds showed a significantly higher expression of genes involved in the innate immune response, cytokine signalling, B-cell and T-cell activation and immunoglobulin production in resistant birds (99). A recent genome-wide association study also identified SNPs in Barred Rock chickens that are significantly associated with *C. jejuni* colonisation – one in the *CDH13* gene and one upstream of *CALM1* (118).

Avian pathogenic *Escherichia coli*

Escherichia coli strains causing systemic disease in poultry are termed avian pathogenic *E. coli* (APEC) and the diverse array of disease manifestations caused by APEC is collectively called avian colibacillosis (119, 120). Although *E. coli* is a normal member of the intestinal microflora, APEC spread into other internal organs and cause colibacillosis. This is a major disease affecting poultry of all ages and causing significant economic losses worldwide. A predisposition to other infections or environmental stress increases the risk of the disease (120). APEC are also a major concern for public health as pathogenic strains may be transmitted to humans through consumption of contaminated products.

A major challenge in controlling avian colibacillosis is the difficulty in identifying the causative strains, as the

disease can be caused by a diverse range of serogroups. The available vaccines are not very effective as they provide protection only against homologous strains. To develop broad-spectrum vaccines, a thorough understanding of APEC pathogenicity is essential and this is an area in which genetic and genomic studies have proven crucial. Genetic and comparative genomic approaches have been used to identify APEC virulence factors that can be evaluated as vaccine candidates (119). The availability of complete genome sequences of APEC strains O1 (121) and O78 (122), in particular, has been a turning point in the use of genetic methods to gain insight into APEC pathogenicity. Genotyping virulence genes with multiplex polymerase chain reaction has been used to diagnose pathogenic strains and establish a phylogenetic link between avian and human extra-intestinal pathogenic *E. coli* (exPEC) strains (123, 124). A recent study has identified four different associations of virulence genes (by characterising a large number of *E. coli* isolates and applying a statistical analysis based on the tree-modelling method) that enabled identification of over 70% of the pathogenic strains (125). Comparative genomic analyses between the APEC and human exPEC strains have revealed striking similarities in genomic islands, virulence genes, overlapping serogroups and phylogeny, suggesting that poultry may serve as a vehicle for human exPEC strains and may even transfer virulence-associated genes to exPEC strains (119). The microarray-based gene expression response to APEC infection has been studied in an effort to identify the genes and networks associated with resistance (126).

Although a great deal has been learned, there is still a lack of detailed information on the combination(s) of genes essential for inducing APEC infection as the results are highly variable. This could indicate the existence of subpathotypes or different pathotypes within the present APEC group, with different virulence mechanisms employed by the different subpathotypes. Some researchers have, therefore, suggested revising the current definition of APEC (127).

Parasitic infections

Genetic research into parasitic infections of poultry is, unfortunately, lagging behind that of viral and bacterial disease, even though infections caused by pathogens such as *Eimeria*, *Ascaridia* and *Histomonas* continue to have a major impact on the poultry industry.

Eimeria

Eimeria tenella is one of the main causative agents of coccidiosis in chickens (128), while six other species are known to infect turkeys: *E. adenoeides, E. dispersa, E. gallopavonis, E. innocua, E. meleagridis* and *E. meleagrimitis*. Coccidiosis causes severe growth impairment in infected birds, caecal lesions and sometimes death. Anticoccidial drugs are the main measure against the disease, with ongoing research into improving commercially available vaccines (128). Genetic variability for resistance has previously been shown in birds (129), as well as a strain-specific protective immune response associated with *E. maxima* (130). This has allowed investigation into the genes involved in disease resistance, which may ultimately enable the marker-assisted selection of resistant birds. *MLF2* has been identified as a potential candidate gene for resistance to *Eimeria* (131). More recently, a medium-density SNP panel has been used to identify 31 QTLRs for *Eimeria* resistance, with several potential candidate genes being highlighted (132).

Ascaridia

Parasitic nematodes of the genus *Ascaridia* also infect the intestines of birds. *Ascaridia galli* is the main infective agent of chickens and turkeys, with *A. dissimilis* also being found in turkeys. Signs include anorexia, diarrhoea, stunted growth and enteritis. Antihelminthic drugs are currently used to try to control the disease, although present research is now focusing on a genetic approach to attempt to identify candidate genes for resistance, due to concerns over antihelminthics (133). Studies have shown that breeding for resistance to *A. galli* is possible (134). An SNP association study has characterised *IFNG* as a gene with a potential role in helminth resistance (135). Proteomic studies have also identified 16 proteins that are immunoreactive in naturally and experimentally infected hens (136) and immune gene expression has been analysed in experimentally infected birds (137).

Histomonas

The protozoan *Histomonas meleagridis* causes histomoniasis, or blackhead disease, primarily in turkeys (up to 100% mortality in flocks) but also affecting chickens (10–20% mortality) (138). The disease was formerly controlled by the use of nitroinidazole drugs. However, the banning of such drugs in Europe and the United States means that there has been an upsurge in infections (139). Currently, antibiotics and plant substances are used to try to control the disease. Vaccination is not successful as birds do not seem to become resistant to reinfection (140). Researchers acknowledge that the way forward now is via investigation of the molecular and genomic mechanisms of the disease (139).

Conclusions

The technologies and resources now available to avian genomics researchers mean that diseases like the ones examined in this review which blight the poultry industry

and, in some cases, have serious implications for human health can now be tackled head on. The genetic basis of infection and host response can be clarified and the mechanisms of resistance resolved, thus leading to new methods of disease control, whether through selective breeding measures or the development of effective vaccines.

La génomique animale et la résistance aux maladies infectieuses chez les volailles

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Résumé

Les agents pathogènes affectant les espèces aviaires représentent un coût majeur pour la société du fait des pertes économiques colossales qu'ils font subir à la filière avicole et de leurs effets sur la santé publique. Un certain nombre de maladies infectieuses font peser une menace permanente sur la santé et le bien-être de millions d'oiseaux ; parmi les agents pathogènes en cause, certains gagnent en virulence et deviennent donc de plus en plus difficiles à contrôler ; c'est le cas par exemple du virus de la maladie de Marek et des virus de la grippe aviaire. L'ère actuelle de la génomique animale se caractérise par des avancées considérables au plan technologique et par des ressources accrues, les chercheurs bénéficiant aujourd'hui d'atouts sans précédent pour élucider la génétique de la résistance aux maladies et pour déterminer les gènes et les mutations régissant la sensibilité ou la résistance des oiseaux à une infection. La génomique aviaire a atteint un niveau permettant d'étudier et de comprendre les mécanismes biologiques des maladies infectieuses chez les volailles et d'autres espèces aviaires. La connaissance des gènes codant pour la résistance aux maladies permet de concevoir des programmes de sélection et de mettre au point des vaccins destinés à contrôler les effets induits par des agents pathogènes à fort impact sur les oiseaux ou l'être humain.

Mots-clés

Aviaire – Base de données des locus de caractères quantitatifs chez les animaux – Gène candidat – Génomique – Influenza aviaire – Maladie infectieuse – Poulets – Résistance aux maladies – *Salmonella* – Virus de la maladie de Marek – Volailles.

Genómica animal y resistencia a las enfermedades infecciosas en las aves de corral

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Resumen

Los patógenos aviares entrañan importantes costos para la sociedad, tanto por las enormes pérdidas económicas que infligen al sector avícola como por sus efectos sobre la salud humana. La salud y el bienestar de millones de aves se encuentran bajo la amenaza constante de muchas enfermedades infecciosas, algunos de cuyos agentes cobran cada vez mayor virulencia y resultan por ello

cada vez más difíciles de combatir, como ocurre con los virus de la enfermedad de Marek o de la influenza aviar. La genómica animal conoce ahora mismo un auge extraordinario, desde el doble punto de vista de la tecnología y de los recursos, lo que significa que los investigadores nunca han estado en mejor posición para estudiar los mecanismos genéticos de la resistencia a las enfermedades y determinar los genes y/o mutaciones que subyacen a la sensibilidad o la resistencia de las aves a una infección. La genómica aviar ha alcanzado un punto en el que ya es posible investigar y comprender los mecanismos biológicos de las enfermedades infecciosas de aves de corral y otras especies aviares. Ahora cabe utilizar el conocimiento de los genes que confieren resistencia como parte de programas de selección reproductiva o para obtener vacunas que ayuden a combatir los efectos de esos patógenos, que tan perjudiciales resultan para aves y personas por un igual.

Palabras clave
Aves de corral – Aviar – Base de datos de loci de caracteres cuantitativos en animales – Gen candidato – Genómica – Infección – Influenza aviar – Pollo – Resistencia a enfermedades – *Salmonella* – Virus de la enfermedad de Marek.

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