

Review Article

SELECTION METHODS FOR DISEASE RESISTANCE IN POULTRY – AN OVERVIEW

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Abstract: Animal production is often disrupted by diseases costing livestock producers and consumers crores of rupees each year due to mortality, subclinical losses due to poor production efficiency, increased veterinary costs and product loss. Disease also impairs genetic improvement in production traits because efficiency of selection is reduced. Methods to control disease include various biosecurity measures such as vaccination, medication, sanitation, and isolation of animals from pathogens and eradication of certain diseases. However, these approaches are not always effective. A clear understanding of disease and the animal's defence systems is required for alternative approaches to disease control. The onset of disease is often the result of the interaction between an individual animal's genotype and the environment to which the animal is exposed. If an animal has a genetic predisposition for acquiring a disease, then environmental conditions, including standard disease-prevention methods may be only partly effective in preventing disease. An often-overlooked alternative approach to standard disease control methods would be selective breeding to increase disease resistance. Genetic resistance to disease involves many facets of the body's defence system and their interactions and is extremely complex.

DISEASE RESISTANCE - INTRODUCTION

The presence of some diseases may result from strictly genetic control, whereas others may be caused by a combination of genetic predisposition and exposure to pathogens. Disease resistance research has included measurement of genetic control of disease losses, estimation of heritability, and characterization of breed or strain differences. Genetic control of certain diseases may be the result of the presence or absence of receptors that are inherited simply. Resistance to specific subgroups of leukosis virus in chickens seems to be inherited simply (Crittenden, 1975) and may be the result of not having the receptor for the virus.

SELECTION FOR DISEASE RESISTANCE USING DIRECT APPROCHES

Depending on the species, breeders routinely select animals on the basis of from one to perhaps as many as 20 traits. This selection is practiced under challenge environments that may increase the incidence of disease if management is poor. Many researchers have

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examined approaches to selection for disease resistance. The simplest method would be to observe and select breeding stock for disease resistance under normal production conditions. This would have no negative effects on production of breeding stock but probably would not be very informative, because, without disease (under good hygiene and management), expression of disease resistance would be questionable. Challenging breeding stock, progeny, or sibs would be costly, depending on the severity of the disease challenge, and production could be adversely affected. This method would be of limited value unless sufficient numbers of progeny or sibs are tested. Thanks to the new opportunities animal cloning offers, one alternative possibility to testing progeny or sibs would be to obtain a large number of clones of embryos from planned matings of breeding stock. Once raised, one set of these animals (clones) could be challenged with a specific disease or diseases. Selection of their clones (other animals) could then occur on the basis of results from the cloned animals. Because the animals tested were clones, accuracy would be equal to testing the individuals themselves.

Direct approaches to selection for genetic resistance to disease				
Type of selection	Method	Effects on production of breeders	Expression of disease resistance	Cost
Direct	1. Observe breeding stock	0	Questionable	0
	2. Challenge breeding stock	Negative	Good	Low-high
	3. Challenge sibs or progeny of breeding stock	0	Good	Low-high
	4. Challenge clones	0	Excellent	Moderate-high

Source: Adapted from Gavora and Spencer (1983) and Rothschild (1985).

SELECTION FOR DISEASE RESISTANCE USING INDIRECT APPROACHES

a. Immune response

Given the difficulties in selecting for disease resistance under challenging environments, alternatives to those methods have been proposed. Immune responsiveness has been suggested as an indirect indicator of disease resistance (Rothschild, 1989; Warner et al., 1987). Early studies (Biozzi et al., 1980) have revealed that genetic control of antibody response to sheep red blood cells was moderately heritable and that selection for humoral immune response for one antigen may improve humoral immune response for other antigens.

They also investigated genetic control of cell-mediated responses. Selection for increased humoral response to sheep red blood cells did not improve cell-mediated response, suggesting independence of these traits.

Among livestock, the most extensive research with genetic control of immune response has been in poultry (Van der Zijpp, 1983a; Lamont and Dietert, 1990). Genetic control of immune responsiveness to sheep red blood cells has been thoroughly investigated (Siegal and Gross, 1980; van der Zijpp and Leenstra, 1980). Heritability estimates have ranged from 0.28 to 0.38, suggesting that this trait is under moderate genetic control. Results suggested that immunization procedures, dosage and site of immunization all affect measurement and extent of genetic control (Van der Zijpp, 1983b). Such details may make the use of immune response as an indicator of disease resistance more difficult. Other experiments have demonstrated that response to vaccination with Newcastle disease, *Salmonella pullorum*, and *E. coli* are under moderate genetic control and that selection for high and low antibody response following vaccination is effective (Lamont and Dietert, 1990).

b. *In vitro* methods

A second indirect approach would be to consider *in vitro* methods as indicators of disease resistance. These methods include, for instance, phagocytic and bactericidal actions of peripheral blood monocytes against disease agents such as *Salmonella typhimurium* and *Staphylococcus aureus* (Lacey et al., 1990). Other such methods include neutrophil metabolic and phagocytic activity and lymphocyte blastogenesis in response to antigens. Use of mitogens as an indicator of cell-mediated response has revealed that genetic differences exist in poultry for the T cell mitogens phytohaemagglutinin and concanavalin. As with poultry, response to mitogen stimulation has also been demonstrated in swine for reactivity to phytohaemagglutinin and poke-week mitogen. Efforts to create an immunocompetence index in swine have been only moderately successful (Buschmann et al., 1985).

Indirect approaches to selection for genetic resistance to disease				
Type of selection	Method	Effects on production of breeders	Expression of disease resistance	Cost
Indirect	1. Vaccine challenge	0	Good	Low
	2. <i>In vitro</i> tests	0	Good	Low
	3. Genetic markers	0	Good	Low
Molecular Genetics	Construct resistant genotypes	0	Good	High

Source: Adapted from Gavora and Spender (1983) and Rothschild (1985).

c. **Marker assisted selection**

A third method would be to locate genetic markers associated with disease resistance or the actual genes themselves. This approach was generalized first by those suggesting use of RFLP analyses in genetic improvement programs (Soller and Beckmann, 1983). Given the complexity of immune response and its relationship with disease resistance, the search for marker genes associated with these traits was enormous. Modern immunology, however, has revealed that a group of genes, called the major histocompatibility complex (MHC) genes, seem to be intimately associated with both disease resistance and immune responsiveness. All higher life forms are known to possess a MHC that codes for the predominant cell-surface proteins on cells and tissues of each individual species. These antigens are markers of "self" and are unique for animals other than identical twins or clones. Three classes of protein molecules, class I, class II and class III, are encoded for by the MHC. The class I antigens act as restricting elements in T cell recognition of virally infected target cells and, thus, are necessary to generate an immune response. The class II genes control the interaction of T cells, B cells, and macrophages in the generation of the humoral immune response and participate, as well, in aspects of cellular immunity. The class III genes are intimately involved with the complement cascade, which ends with the lysis of the cell or virus particle to which antibody has bound. The structure and function of the MHC in pigs seems to be similar to that of humans and mice (Warner et al., 1988; Warner and Rothschild, 1991).

The B complex, the MHC in chickens, has been extensively studied and shown to be involved with both immune response and disease resistance. More specifically, the B complex has been shown to be associated with immune response to synthetic antigens, bovine serum albumen, *Salmonella pullorum* bacterium, total IgG levels, and cell-mediated

responses (Lamont and Dietert, 1990). Resistance to Marek's disease, Rous Sarcoma virus, fowl cholera, and lymphoid leukosis viruses has also been demonstrated to be associated with the chicken MHC (van der Zijpp, 1983a; Lamont, 1989). In retrospect, it seems that poultry-breeding companies have, in fact, indirectly selected for certain MHC genotypes by eliminating lines or strains that were susceptible to certain diseases. More recent molecular genetic approaches have included using gene maps and quantitative trait loci (QTL) scans to find genes associated with disease resistance and immune response.

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