AAAP Tumor Virus Committee Report on Relationship of Slow Feathering Gene with incidence of Lymphoid Leukosis and Field Results with CVI988 Clone C Marek's Disease Vaccine.

The members of the Tumor Virus Committee identified the above two topics for this report because of current industry interest. The report was prepared by two subcommittees: the portion on slow feathering was prepared by Drs. Lyman Crittenden (Chairperson) and Greg Stewart and the portion on CVI988C by Drs. Bill Chase (Chairperson) and Bob Pitts. The authors reviewed relevant published literature and obtained data and verbal input from industry personnel to prepare this report. The participation by the industry personnel is gratefully acknowledged. This report should not be viewed as a scientifically complete document; our purpose is to share with the AAAP membership general information and current knowledge on the two topics.

Relationship of Slow Feathering Gene with Incidence of Lymphoid Leukosis

About one-half of the white-egg layers in the United States are rapid-feathering progeny of slow-feathering dams. The incentive for using the gene for sex-linked slow-feathering to sex commercial layers is to save the three to four cents per pullet chick that must be spent for vent-sexing. With the reduced availability of vent-sexors world-wide, there is increasing pressure for feather-sexing breeding stock. Even though some breeders are successfully marketing these layers, other breeders have not sold feather-sexed layers because they often have higher mortality and lower productivity than comparable vent-sexed stocks. Industry experience indicates that feather-sexed broilers and brown-egg layers do not exhibit this problem.

It has been recognized for several years that slow-feathering White Leghorn lines may be more likely to have high rates of infection with avian leukosis virus (ALV) although there are differences among lines and that this infection may be more difficult to reduce in slow-feathering lines than in conventional lines. Probably poor performance is due to the comparatively high rates of ALV infection in these lines and in their fast-feathering daughters used as commercial layers. We now know that the endogenous ALV gene, ev21, is tightly linked and probably inseparable from the slow-feathering gene. ev21 codes for a complete subgroup E endogenous ALV, that can infect embryos by genetic or congenital transmission and induce immunological tolerance to antigens shared with exogenous avian leukosis virus. Such chickens cannot develop a good antibody response to ALV.

Two approaches to the control of this effect of the subgroup E virus encoded by ev21 seem reasonable: 1. Eradicate ALV from the slow-feathering parent stock. Eradication may be facilitated by immunizing the breeding flock with ALV to protect the progeny for the first few weeks with maternal antibody. While eradication is feasible and currently underway, thus far, we have no apathogenic live ALV or killed product to use as a vaccine. 2. Increase the frequency of the gene for resistance to subgroup E virus infection in the female parent stock and thus reduce the titer of the ev21 encoded virus in the dams and decrease congenital transmission to their rapid-feathering daughters. This approach would require the fixation of the recessive gene for resistance in both lines that are crossed to produce female parent stock.

These approaches are presently under investigation and further research is needed before definitive recommendations can be made.

Field results with CVI988 Clone C Marek's disease vaccine

This vaccine consists of serotype 1 Marek's disease virus attenuated by serial cell culture passage. The vaccine was developed in Holland and was introduced to the U.S. market in early 1987. The parent virus for this vaccine has been used with good results in Europe for a number of years.

The following data were obtained by contacting some of the U.S. companies that have used the new vaccine under field conditions.

Broiler Trials:

Trial No.	Housing Environment	Vaccine used	% Condemnation
1	Recent clean-out, well ventilated	HVT-SB1	0.25
		CVI988C	0.20
2	Delmarva, built-up litter, winter brooding	HVT-SB1	0.33
		CVI988C	4.00
3	Delmarva, built-up litter,winter brooding	HVT-SB1	0.33
		HVT-CVI988C	2.50

The above data represent over two million broilers. All vaccines were given at comparable dilutions. If the "Control" vaccine was given undiluted, so was the CVI988C. In no case was the dilution greater than 1:2.

Commercial Leghorn Trials:

Only preliminary data are available. Very few layer flocks have been vaccinated with CVI988C and observations on these birds are continuing. Clean brooding environment of most test flocks resulting in low challenge is likely to make comparisons difficult. In most flocks, the HVT-SB1 vaccine was compared with HVT-CVI988C with no apparent differences in mortality to date. In one trial involving 140,000 birds, HVT-SB1 was compared with CVI988C. Mortality was about equal but low in both test flocks.

Respectfully submitted,
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