

CONGENITAL TRANSMISSION OF THE GROUP-SPECIFIC ANTIGEN OF AVIAN LYMPHOID LEUKOSIS VIRUS IN COMMERCIAL STOCKS IN BRAZIL¹

CHERYL A. ROWE², CARLOS H. ROMERO², M. WANDA DOS SANTOS³, PAULO G. OLIVEIRA DIAS³ E OSVALDO A. RESENDE³

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Albuminas de ovos de galinhas, pertencentes a nove linhagens comerciais de postura e uma de corte, obtidas de cinco granjas de matrizes, foram testadas pela presença do antígeno específico de grupo (gs) dos vírus da leucose linfóide aviária (LLV). As taxas de transmissão congênita nas linhagens de postura variaram entre 6,7 e 24,7 por cento, enquanto que a taxa na linhagem de corte testada foi de 1,3 por cento. O significado destes achados em relação à erradicação dos LLV é discutido.

TERMOS DE INDEXAÇÃO: Vírus da leucose aviária, transmissão congênita, antígeno específico de grupo.

ABSTRACT.— Egg albumens from hens belonging to nine commercial laying stocks and one broiler stock, obtained from five poultry breeders, were tested for the presence of the group-specific (gs) antigen of lymphoid leukosis viruses (LLV). The congenital transmission rates in the laying stocks ranged from 6.7 to 24.7 percent, while the rate in the broiler stock tested was found to be 1.3 percent. The significance of these findings in relation to the eradication of LLV is discussed.

INDEX TERMS: Avian leukosis virus, congenital transmission, group-specific antigen.

Lymphoid leukosis (LL) is induced by C-type RNA tumor viruses that are currently classified in the leukosis/sarcoma group of viruses and share a common group-specific (gs) antigen (Sarma et al. 1964). The exogenous viruses of this group, that naturally infect the chicken, are known to be responsible for the LL losses in the field (Calnek 1968) and are perpetuated from generation to generation by vertical (congenital) transmission from dams to their offspring (Cottral et al. 1954).

The demonstration of C-type viral particles in the magnum of mature, egg-laying, LLV congenitally infected hens (DiStefano & Dougherty 1965) led to the discovery of LLV and their gs antigen in the albumen of unincubated fresh eggs (Spencer et al. 1976, Romero 1977). The systematic identification and immediate elimination of these infected dams leads to the eradication of the source of infection for the next generation of chicks.

We report here on the congenital transmission rates of the gs antigen of LLV found in commercial stocks representative

of some of the major poultry breeders operating in Brazil and briefly discuss possible economic benefits in relation to eradication.

MATERIALS AND METHODS

Birds and housing. One-day old female chicks, representative of 10 different lines (A–J), obtained from five poultry breeders, were reared on the floor in conventional commercial pens until they were 16 weeks old. At this age, the pullets were housed either in individual cages or at the rate of two pullets per cage. Lines A through I were commercial egg-type hens, while the hens of line J were of the broiler-type. All birds were vaccinated against Marek's disease, Newcastle disease and fowl pox.

Sample processing. Eggs were collected twice a day and promptly cooled to 4°C. On the same day, approximately two ml of albumen from each egg were aspirated with a Pasteur pipette, through a small hole in the shell over the air cell, placed in a vial and frozen at –20°C. Albumens were usually maintained frozen up to two weeks before they were tested for the presence of gs antigen by a microcomplement fixation test (Spencer et al. 1976, Romero 1977).

Reference reagents. Reference antiserum, prepared in rabbits by the inoculation of purified avian myeloblastosis virus (Smith 1977) was kindly supplied by Dr. Eugene J. Smith of the USDA Regional Poultry Research Laboratory, East Lansing, Michigan. Reference antigens, obtained from the same laboratory were (1) an extract made from chicken embryo fibroblasts, infected with the RPL 12 strain of LLV, (2) gs antigen-positive albumen harvested from eggs obtained from shedder-hens infected with the RAV-1 strain of LLV (Romero 1977) and (3) gs antigen-negative albumen harvested from LLV-free SPF hens. Albumens were routinely tested at the 1:2 and 1:4 dilutions.

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² Unidade de Pesquisa de Patologia Animal, EMBRAPA, 23460 Seropédica, Rio de Janeiro, Brazil.

³ PESAGRO–Rio, 23460 Seropédica, RJ.

RESULTS AND DISCUSSION

A total of 1004 egg albumens, corresponding to 1004 hens of 10 different lines, were tested for the presence of the gs antigen of avian LLV by a microcomplement fixation test. It has been shown that congenital transmission of infectious LLV is detected less frequently than the transmission of gs antigen (Romero 1977, Payne et al. 1979, DeBoer et al. 1980), most likely due to thermal inactivation of the virus in the oviduct. Shedder hens were identified in all commercial lines tested. The percentages of shedder hens per line ranged from 6.7 to 24.7 for the laying stocks and 1.3 for the broiler stock (Table 1). Lines F and G have been kept as closed flocks for over 30 years and these lines and their crosses (H and I) had the highest infection rates of all the laying stocks tested. The lowest rate of infection was found in hens belonging to the broiler stock tested.

Table 1. *Congenital transmission of the group-specific antigen of avian lymphoid leukosis virus in commercial stocks in Brazil*

Line	Stock	$\frac{\text{N}^\circ \text{ of eggs positive}^{(a)}}{\text{N}^\circ \text{ of eggs tested}^{(b)}}$	Shedders %
A	Layer	7/98	7.1
B	"	11/84	13.1
C	"	6/89	6.7
D	"	12/100	12.0
E	"	14/81	17.3
F	"	22/99	22.2
G	"	23/96	24.0
H	"	24/100	24.0
I	"	24/97	24.7
J	Broiler	2/160	1.3

(a) Albumens positive for the presence of gs antigen.

(b) One egg was tested for every hen comprising each line.

Flocks genetically susceptible to both the infection and the tumorigenic effect of LLV can experience overt economic losses, mainly due to mortality, especially when infection rates of the maternal stocks are high (Crittenden 1975). Moreover, in the absence of overt disease, the infection with LLV can

acquire economic significance due to their general depressive effect on parameters of productivity such as fertility, hatchability, non-specific mortality, growth and laying (Romero et al. 1979). Thus, the eradication of the infection from reproductive stocks may result in increased performance and better quality products free of oncogenic viruses.

REFERENCES

- Calnek B.W. 1968. Lymphoid leukosis virus: A survey of commercial breeding flocks for genetic resistance and incidence of embryo infection. *Avian Dis.* 12:104-111.
- Cottral G.E., Burmester B.R. & Waters N.F. 1954. Egg transmission of avian lymphoid leukosis. *Poultry Sci.* 33:1174-1184.
- Crittenden L.B. 1975. Two levels of genetic resistance to lymphoid leukosis. *Avian Dis.* 19:281-292.
- DeBoer G.F., Van Vloten J. & Hartog L. 1980. Comparison of complement fixation and phenotypic mixing tests for the detection of lymphoid leukosis virus in egg albumen and embryos of individual eggs. *Avian Pathol.* 9:207-218.
- DiStefano H.S. & Dougherty R.M. 1965. Virus multiplication in the oviduct of hens infected with an avian leukosis virus. *Virology* 26:156-159.
- Payne L.N., Holmes A., Howes K., Pattison M. & Walters D.E. 1979. Studies on the association between natural infection of hens, cocks and their progeny with lymphoid leukosis virus. *Avian Pathol.* 8:411-424.
- Romero C.H. 1977. The prevention of avian lymphoid leukosis tumors with the androgen analog mibolerone: Pathological, virological and immunological studies. Ph.D. dissertation, Michigan State University.
- Romero C.H., Oliveira Dias P.G., Resende O.A., Santos M.W. & Rowe C.A. 1979. Decreased productivity as a consequence of inapparent infection with lymphoid leukosis viruses. 6th Latin American Congress of Poultry Science, Lima, Peru, Oct. 10-13.
- Sarma P.S. Turner H.C. & Huebner R.J. 1964. An avian leukosis group-specific complement fixation reaction. Application for the detection and assay of non-cytopathogenic leukosis viruses. *Virology* 23:313-321.
- Smith E.J. 1977. Preparation of antisera to group-specific antigens of avian leukosis-sarcoma viruses: An alternate approach. *Avian Dis.* 21:290-299.
- Spencer J.L., Crittenden L.B., Burmester B.R., Romero C.H. & Witter R.L. 1976. Lymphoid leukosis viruses and gs antigen in unincubated chicken eggs. *Avian Pathol.* 5:221-226.