Avian Leukosis Virus Subgroup J

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ABSTRACT

The avian leukosis viruses (ALV) are a class of retroviruses belonging to the avian leukosis-sarcoma group. The Avian leukosis viruses subgroups (A-E) are the most commonly occurring retroviruses associated with lymphoma-type tumor producing diseases and other production problems in poultry. These viruses cause serious economic losses in the poultry industry worldwide since infected chickens not only develop neoplasias but also have decreased production efficiency. A new subgroup of ALV named J (ALV-J) was first reported in the United Kingdom in the late 1980's and in the United States in the early 1990's, and was found to be associated with myeloid leukemia (ML) primarily in meat-type chickens. The virus has been reported to be transmitted vertically and horizontally making it more difficult to eradicate from primary breeders as had been done with the other subgroups of ALV. The disease primarily affects adult broiler breeders (BB) but recent research has reported that commercial broilers (CB) are also affected. The ALV-J infection has emerged as a serious cause of mortality and other production problems in meat type chickens and appears to be gaining in virulence.

INTRODUCTION

The incidence of avian leukosis virus-induced diseases has been significantly reduced in the last 30 years as the result of the implementation of eradication programs in most commercial poultry breeding operations. However, despite these efforts, a new strain of ALV was isolated in 1988 from meat-type chickens in the United Kingdom. This new strain was determined to represent a new subgroup, designated J (ALV-J), of which the strain HPRS-103 is the prototype (Payne et al., 1991, 1992). In contrast to other known ALV that usually cause lymphoid leukemia, ALV-J induces myeloid leukemia and a variety of less common tumors, most commonly in meat type chickens (Crittenden, 1991). The disease has been reported to primarily affect broiler breeders causing high mortality and decreased production performance. However, recent studies have shown that commercial broilers generated from infected breeding flocks are affected as well, resulting in high mortality, body weight suppression, stunting, and immunosuppression (Stedman and Brown, 1999). The disease is vertically and horizontally transmitted. The only control is prevention by eradication from primary breeders, because no vaccine has been developed at the present time. The worldwide emergence of ALV-J in the last decade is the biggest disease problem facing the poultry industry today.
**LITERATURE REVIEW**

**ALV.** ALV's are categorized into five major subgroups, A to J, based on their ability to infect chicken embryo fibroblasts (CEF), viral infectivity interference patterns with other viral subgroups, and their viral envelope glycoprotein antigens (Smith et al., 1998a, Arshad et al 1997, Payne et al., 1991;1992;1995.). The viral RNA genome of ALV has three genetic regions in the sequence 5'-gag-pol-env-3', which encode respectively the viral internal group-specific antigens, RNA dependent DNA polymerase or reverse transcriptase, and viral envelope glycoprotein (Payne, 1998a,b; Ruis et al., 1999). The gag gene encodes for the viral structural proteins such as p27 which is common to all viruses in the group and is used in diagnostic tests for detection of ALV infected birds. The envelope glycoproteins are composed of a globular surface (SU) 85 kD glycoprotein (gp 85) that mediates binding to the cellular receptor, and a transmembrane (TM) 37 kD glycoprotein subunit that mediates fusion (Young, 1998; Benson et al. 1998; Venugopalan, 1999). The subgroup specificity of chicken retroviruses is determined by sequences within the portion of the env gene that encode the surface glycoprotein gp85 (Jordan and Pattison, 1996; Bova et al., 1986; 1988). The central region of the gp85 subunit contain five clusters of variable regions designated hr1, hr2, vr1, vr2 and vr3 which are important in determining the subgroup specificity (Bova et al., 1986). The sequence of the env gen of ALV-J is highly diverged from that of other ALV, justifying its placement as a new subgroup called J (Bai et al., 1995). The SU (gp85) domain is highly conserved among ALV subgroups A-E sharing approximately 85% homology among each other. In contrast, ALV-J shares only 40% homology to the corresponding sequences of subgroups A-E. The TM (gp37) domain of ALV-J shares about 65% identity to that of subgroups A-E, which share around 93% homology among each other (Ruis et al., 1999; Venugopal, 1999).

**ALV Strains.** The HPRS-103 and ADOL-Hc1 strains of ALV are the prototypes of the ALV-J isolated from meat type chickens in the United Kingdom and the United States, respectively (Payne et al., 1991; Venugopalan et al., 1997; Fadly and Smith, 1999). Both HPRS-103 and ADOL-Hc1 strains of ALV-J are antigenically different from ALV subgroups A-E. Recent research has reported that antibodies to ADOL-Hc1 neutralize HPRS-103 virus, while antibody to HPRS-103 strain fails to neutralize ADOL-Hc1 strains of ALV-J. These findings suggest that both strains are antigenically related but not identical, and confirms the existence of antigenic variations among different strains of ALV-J (Fadly and Smith, 1999).

**ALV Replication.** Avian leukemia virus infection of cells occurs through binding of the envelope glycoprotein to specific receptors on the cell membrane of the host (Young, 1998). Single-stranded viral RNA is released into the cytoplasm and serves as the template for the synthesis of a double-stranded viral DNA by the action of reverse transcriptase. The viral DNA migrates to the nucleus and integrates into chromosomal DNA of the host making a provirus. Once the viral DNA is integrated into the host, it controls transcription of proviral DNA to viral mRNA and RNA. Translation of the viral mRNA results in the production of viral enzymes and proteins of the gag, pol and env.
genes which form a viron. The virions migrate and bind to the cellular membrane of the host, which eventually bud off from the cell (Kim et al., 1998; Jordan and Pattison, 1996).

**Diagnostic Tests.** ALV-J infection in poultry flocks can be diagnosed by conventional tests such as the detection of viral group specific antigen (Gag p27), virus isolation and specific neutralization tests, and also by polymerase chain reaction (RT-PCR) tests (Smith et al., 1998a,b). Carrier birds can be identified by testing vaginal swabs, egg albumen and serology using p27 antigen capture ELISA, RT-PCR and virus antibody neutralization tests (Eleazer, 1999a; Venugopal et al. 1997; 1998). PCR is increasingly being used in the identification of ALV-J infected birds using a set of primers that amplify unique regions of the env gene within the ALV-J genome (Smith et al., 1998a,b). Arshad et al. (1999) developed an in situ hybridization procedure for detecting viral nucleic acids in tissues collected from chickens infected with ALV-J.

**Transmission.** The virus is transmitted vertically, from parent to offspring, but also spreads horizontally, from infected to non-infected poultry. Vertically infected birds may not develop antibodies but shed the virus throughout their lives increasing the horizontal transmission of the virus. Fadly and Smith (1999) reported that contact transmission of ALV-J is highly efficient, increasing from 4% to 25% from hatch to 4 weeks of age and from 25% to 100% from hatch to 8 weeks in chickens hatched from BB naturally infected with AVL-J. The great susceptibility of meat-type chickens to horizontal infection is of concern because it impairs ALV-J eradication programs.

**Susceptibility.** ALV-J has been found primarily in meat type chicken strains. The disease affects BB and has been isolated from great grandparent (GGP), grandparent (GP), and parent (P) BB flocks as early as 4 weeks of age. Commercial broilers have been diagnosed positive for AVL-J at 6 weeks of age (Fadly and Smith, 1999). In contrast to other subgroups of ALV no evidence of genetic resistance has been reported in chickens (Zavala, 1998; Payne, 1998a,b). Fadly and Smith (1999) reported that the incidence of ALV-J infection from flocks of four different primary breeders ranged from 11% to 87%, and the incidence of antibody to ALV-J varied from 30% to 87%.

**Clinical Signs in BB.** Clinical signs seen with ML subgroup J virus infection in BB are variable depending on management conditions, breed, and age of the flock (Zavala, 1998). Symptoms associated with ALV-J disease include reduced egg production (25 - 30 fewer eggs per hen housed in a single lay cycle), reduced hatchability, increased production of small eggs, peritonitis, severe reactions to live fowl cholera vaccines, bacterial infections, and an increase in mortality of 0.5% to 1.5% per week (Hunton, 1999; Eleazer, 1999c; Fadly and Smith, 1999; Stedman and Brown, 1999; Zavala, 1998). Venugopal (1998) reported that in the US and some other countries, increased involvement of the respiratory tract, with gross tumors in the trachea have been observed.
**Clinical Signs in CB.** Reported field symptoms associated with ALV-J infection in broilers are variable but include: lower body weights (up to 25% lower), poor feed conversion ratios and uniformity, higher 2 week and overall mortality (up to 25%), weak chicks, poorly closed navels, enlarged proventriculus, petechial hemorrhages in muscle, brittleness in bones, spleen tumors, head crown brain tumors, poor feathering, severe stunting, and immunosuppression. (Stedman and Brown, 1999; Eleazer, 1999b,c).

**Oncogenicity of HPRS-103 ALV-J.** Payne et al. (1998a) investigated the oncogenicity of the HPRS-103 strain of ALV-J. In this study, 11 day old chicken embryos were inoculated with HPRS-103 strain in three commercial lines of BB meat-type chickens and reared until 42 weeks of age. A high incidence of ML and renal tumors was observed. Mortality was elevated at 9 weeks of age and the median age at death with ML was 20 weeks of age. The ML lesions were characterized by moderate to severe enlargement of the liver, and skeletal myelocytomas in the sternum, ribs, vertebrae and synsacrum in 88% of the chickens. Tumors were observed in the spleen, thymus, gonads and kidneys. Sexes were equally affected. Findings were consistent with the pathogenic expression of ALV-J infected chickens in the UK. Field infections with ALV-J were reported to affect adult meat-type chickens of both sexes and were characterized by enlarged liver, spleen and kidneys, skeletal tumors and tumors in other internal organs.

**Body Weight Suppression in CB with Congenital ALV-J Infection.** Stedman and Brown (1999) studied the effects on performance traits in CB naturally infected with ALV-J. Broilers from five consecutive hatches of breeders positive and negative for ALV-J were raised until 8 weeks of age and the effects of ALV-J infection on body weight and growth were investigated. Blood samples were taken at hatch and tested by p37 antigen-capture ELISA and RT-PCR for ALV-J infection. Broilers were grouped according to their ALV-J status and raised in isolation units to avoid horizontal transmission. Body weight at hatch was similar for ALV-J negative and ALV-J positive broilers. However, body weight of ALV-J positive broilers were significantly reduced (P<0.005) by week of age and remained consistently lower up to 8 weeks of age when compared to the body weights of ALV-J negative broilers. Body weights of broilers ALV-J positive were consistently between 52.2% and 66% of the weight of ALV-negative broilers throughout the entire rearing phase. ALV-J positive broilers exhibited severe body weight suppression, delayed maturation and feather development, and stunting.

**Prevention and Control.** No vaccine is available at the present time, therefore biosecurity and eradication of ALV-J from breeders are the only present means of control (Zavala, 1998; Payne, 1998a,b; Venugopal, 1999). Payne (1998a,b) reported that the application of eradication protocols in commercial operations have been successful in reducing the prevalence of infection of ALV-J. The eradication protocol involves testing pedigree birds of both sexes at 20 weeks for Gag by ELISA in the cloacal/vaginal swabs, at 22 weeks of age for serum antibody and viremia, at 23 weeks for Gag by ELISA of the first two eggs per hen, at 26 weeks for Gag by ELISA in the
meconium of the hatched chicks, and at 40 weeks for Gag by ELISA in the albumen of the two first eggs per hen. Birds positive for virus or Gag by ELISA in cloacal swabs or albumen and meconium are culled immediately.

CONCLUSION

ALV-J infection is widely spread in primary breeder meat-type chicken flocks resulting in high mortality and decreased egg production. Commercial broiler performance is severely affected by ALV-J infection resulting in severe body weight suppression. Until eradication of ALV-J is achieved at the primary breeder level or effective vaccines are developed, management practices along with biosecurity can help to minimize the transmission and expression of ALV-J infection. Horizontal transmission should not be facilitated, especially during the first weeks of life to maintain low levels of infection and disease expression.

REFERENCES


