

EARLY VIEW

REVIEW

Recent Advances in Understanding ALVE LTR Retrotransposons and Their Impact on Poultry Economic Traits

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Abstract

The chicken genome is a living archive, preserving the history of ancient viral encounters within its DNA. Among these genetic relics, the Avian Leukosis Virus Endogenous (ALVE) family of LTR (Long Terminal Repeats) retrotransposons stands out not as a mere fossil, but as a dynamic and influential force in modern poultry science. While often silent, these endogenous retroviruses can awaken, casting a long shadow over poultry health and profitability. This comprehensive narrative review delves into the complex duality of ALVE elements, synthesizing recent evidence that reveals their staggering diversity, with over a thousand unique insertion sites now identified across global chicken populations. We explore the multifaceted mechanisms—from insertional mutagenesis to viral protein interference and immunosuppression—by which these elements exert their effects. This review critically appraises their tangible economic toll, detailing associations with detrimental impacts on vital production traits such as egg laying and growth, while also considering the paradoxical role of endogenous retroviruses as potential contributors to genetic diversity. Finally, we confront the pressing challenges posed by novel ALVE insertions and viral recombination events, arguing that the path to sustainable, resilient poultry production lies in harnessing advanced genomics and innovative breeding strategies to navigate this intricate endogenous retroviral landscape.

Introduction

The domestic chicken (*Gallus gallus domesticus*) plays a crucial role in global food security, yet its genome reveals a narrative of ancient conflicts with retroviruses. A considerable segment of its DNA consists of Long Terminal Repeat (LTR) retrotransposons, which serve as the genomic remnants of historical retroviral infections. In some previous studies, only about 1.35% of the chicken genome was described as LTR retrotransposon sequence (ICGSC, 2004; Wicker *et al.*, 2005; Mason *et al.*, 2016).

However, in a more recent study that utilized a novel, iterative pipeline known as LocaTR (Mason *et al.*, 2016), which incorporated previously built search algorithms (McCarthy and McDonald, 2003; Ellinghaus *et al.*, 2008; Rho *et al.*, 2007; Smit *et al.*, 2013), a total of 34.8Mb of the *Gallus_gallus* 5.0 assembly was identified

as LTR retrotransposon, accounting for 2.8% of the chicken genome, although when compared to *Gallus_gallus* 4.0 assembly, it yielded 3.0% of the genomic content (Warren *et al.*, 2017). The word 'retrovirus' technically refers to any retrotransposon capable of exiting a host cell and infecting other cells through effective integration (Llorens *et al.*, 2011). It should be noted that both retroviruses and LTR retrotransposons employ reverse transcription for replication; however, they are functionally and taxonomically distinct. Retrovirus is a term that only applies to members of the Retroviridae family that have an envelope (*env*) gene. This gene allows these viruses to make infectious particles that can spread outside of cells and infect new host cells (Eickbush and Malik,

2002). On the contrary LTR retrotransposons usually do not have a functioning *env* gene. As a result, their life cycle is confined to the intracellular milieu, where they replicate through an RNA intermediate that is reverse-transcribed and reintegrated into the same host genome (Boeke and Stoye, 1997). Some retrotransposon lineages have acquired *env-like* sequences horizontally, which makes them "virus-like." However, a true retrovirus is defined biologically by its ability to spread between cells on its own (Llorens *et al.*, 2011). These retroviruses can induce varied effects on their hosts through a series of mechanisms ranging from epigenetic silencing to mutagenesis and even positively or negatively influencing gene expression. In chickens, retroviruses are generally classified by their mode of transmission into exogenous (infectious, horizontal/vertical transmission) and endogenous (heritable, integrated into the germline) categories. A notable example that demonstrates both activities is the Avian Leukosis Virus (ALV) (Borysenko *et al.*, 2008; Payne and Nair, 2012). ALV is a type of C-type retrovirus that mainly lives in birds. It belongs to the genus Alpharetrovirus. In the literature, the wider species complex is often called Avian Sarcoma Leukosis Virus (ASLV) because different viral subgroups can cause a wide range of clinical symptoms, from lymphoid leukosis to sarcomas (Weiss and Vogt, 2011). These viruses are a big problem for farmers because they affect a lot of birds in the order Galliformes, causing both neoplastic diseases and subclinical production losses (Mason *et al.*, 2018). However, the endogenous family of ALV is common only to the domestic chicken and its direct ancestor, the red jungle fowl (Frisby *et al.*, 1979; Venugopal, 1999). Avian leukosis/sarcoma viruses cause leukoses that impact the lymphoid, myeloid, and erythroid lineages of hematopoietic cells as well as various cancers and tumors (Beard, 1980; Purchase, 1987; Payne and Fadly, 1997). While the exogenous viruses in the ALV family include the subgroups A, B, C, D, J, and K, the endogenous group E is the only group that exists in all chicken breeds, but with no known pathogenicity (Adkins *et al.*, 2001; Rutherford *et al.*, 2016). The ALVE (Avian Leukosis Virus Endogenous) family serves as a direct connection to exogenous ALV. The avian genome also contains many families of endogenous retroviruses (ERVs) such as ALVE (with *ev* loci), CR1 (chicken repeat-1), EAV (endogenous avian retrovirus) and ART-CH (avian retrotransposon from chicken genome) (Crittenden, 1991; Payne, 2001). Some of these elements such as EAV and CR1 were found in line 0 chickens (which lack *ev* loci) through low-stringency hybridization (Boyce-Jacino *et al.*, 1989). While CR1 is composed of numerous repetitive retrotransposon elements, EAVs consist of moderately repeated ALV-like elements. From an evolutionary point of view, the CR1 elements seem to be the oldest, whereas the *ev* genes containing retrotransposons, the most recent (Crittenden, 1991). Moreover, the

functional roles of the CR1 and EAV sequences are yet to be determined completely.

Furthermore, about 11 novel families of ERVs occupying more than 21 million base pairs, nearly 2% of the chicken genome, were characterized by Huda and colleagues (2008). The identified ERVs included GGERV 10, GGERV 29, GGERV 12, GGERV 20, GGERV 28, GGERV 30, GGERV 21, GGERV 11, GGERV 23, GGERV 22, and GGERV 24, with the most recent insertions spanning 0-3 million years ago being GGERV 10 and the oldest being GGERV 24 at 136.5-143.9 million years old. According to this, GGERV 10 family can be used as a molecular marker for identifying modern chicken breeds, and although they are not retrotranspositionally inactivated in the genome, they could potentially regulate the expression of neighbouring genes because they contain 23 transcription binding sites (Lee *et al.*, 2017). Generally, even though ERVs are associated with several traits like blue shelled egg phenotype (Wang *et al.*, 2013; Wragg *et al.*, 2013), recessive white feather phenotype and slow feathering (Chang *et al.*, 2006; Elferink *et al.*, 2008; Takenouchi *et al.*, 2018), immunological and immunosuppressive roles (Chiu *et al.*, 2021; Aswad *et al.*, 2012; Mason *et al.*, 2020) and decline in several productive and economic traits (Gavora *et al.*, 1991; Iraqi *et al.*, 1994; Fulton *et al.*, 2021), they can contribute to the genetic diversity and potential adaptation of indigenous chickens thereby supporting development of conservative and breeding strategies for a more sustainable poultry production (Ishihara *et al.*, 2025).

Although numerous ALVE elements have deteriorated over time, a portion still exists in an intact state and can be activated, serving as a continuous genetic risk. This review seeks to connect recent molecular findings with practical applications in poultry science, concentrating on research that elucidates the impact of ALVE elements on economically significant traits. Grasping this connection is essential for improving the sustainability and profitability of poultry production.

Endogenous retroviruses in chickens

The principal origin of Endogenous Retroviruses (ERVs) is the integration of exogenous retroviruses (XRVs) into the host DNA. The XRV integration mechanism into the host genome comprises four essential steps, which are viral infection, reverse transcription, nuclear transport of proviral DNA, and genomic integration (Jiang *et al.*, 2025). Reverse transcriptase (RT) transcribes viral RNA into DNA, thereby facilitating genomic integration via integrase (IN) to create proviruses. ERVs have a genome structure or sequence that is comparable to those of exogenous retroviruses. They create a proviral sequence that is usually 5-10 kb long on the host's genome by reverse transcription and integration. This sequence usually has *gag*, *pro*, *pol*, and *env* genes, as well as long terminal repeats (LTRs) at both ends. The LTRs act as promoters of

proviral expression, regulatory elements, and cis-acting domains that are needed for integration (Qi *et al.*, 2025). The *gag* gene makes the structural proteins of the viral particle, such as the matrix protein, capsid protein, and nucleocapsid protein, which are very important for the viral particle stability and assembly. The *pol* gene codes for important enzymes that help viruses replicate, such as reverse transcriptase and integrase. The *env* gene encodes the envelope protein (Env) on the viral membrane, which is important for the virus to get into host cells because it helps the viral envelope fuse with the host cell membrane. A complete ERV element usually has two long terminal repeats (LTRs) at the ends of the genome, one at the 5' end and one at the 3' end (Jiang *et al.*, 2025). Subgroup E (ALVEs; historically known as *ev* genes (Crittenden, 1991) is found in the domestic chicken and its wild ancestor. The designation "*ev* genes" traditionally refers to the specific loci where endogenous avian leukosis virus sequences are inserted and integrated into the chicken (host) genome, serving as stable genetic markers that follow Mendelian inheritance. While early nomenclature focused on their role as phenotypic modifiers or viral reservoirs, recent high-resolution pangenome mapping has reclassified these elements as highly polymorphic structural variants that continue to influence host immune response and genome architecture (Edwards *et al.*, 2025).

Apart from the ALVE, endogenous avian retroviruses (EAVs), which are characterized by similar structural semblance, are present in all *Gallus spp.*, including chicken. However, other ALV endogenous subgroups (ALV-F and ALV-G) are also present in the Galliformes (Fandiño *et al.*, 2024). EAVs such as EAV-0, EAV-33, EAV-51, EAV-15I, EAV-HP, as well as ART-CH, which are chimeric defective ERVs comprising portions of EAV-51 and EAV-HP (Sacco and Nair, 2014), as opposed to authentic retrotransposons (Gudkov *et al.*, 1992), have been characterized. One very well-researched ERV in poultry is the EAV-HP. This retrotransposon is thought to be a source of ALV-J (also known as *ev/J*), an exogenous retrovirus from recombinations with an exogenous ALV (Sacco *et al.*, 2004). ALV subgroup J infections typically lead to suppression of the immune system, causing cancers or tumors, which can have detrimental effects on both poultry health and economics. A more recent study by Liang and colleagues (2017) on 10 chicken breeds in China demonstrated that the exogenous ALVs that infected the breeds were more closely related to the subgroup J ALV prototype strain HPRS-103 based on phylogenetic tree analysis on the *env* gene of the *ev/J* endogenous retroviral sequences. This highlights a very important aspect of endogenous retroviral research, as these retroviruses could be sources of further recombination with other exogenous ALV subgroups.

ALVE family

While exclusively exogenous ALVs (subgroups A-D, J, and K) provoke leukoid tumors in Galliformes, endogenous ALVs can also spread horizontally within a

population, albeit with a species-specific range. Subgroup E (the ALVEs) is present in red jungle fowl and domestic chickens, but not in other *Gallus* species. Subgroups F and G are found in pheasants, subgroup H in partridges, and subgroup I in quail (Frisby *et al.*, 1979; Venugopal, 1999). A recent study revealed the presence of endogenous retroviral sequences in the genomes of a minimum of 12 Galliformes (Fandiño *et al.*, 2024). A separate study analyzed whole-genome resequencing data from 407 non-commercial, indigenous chickens from Ethiopia, Iraq, and Nigeria. Using an advanced bioinformatics pipeline, obSERVer, researchers found a total of 974 different ALVE insertions, 837 of which were new (Mason *et al.*, 2020). A different investigation looked at ALVE elements in 7 Chinese domestic breeds and 4 standard breeds. It found a total of 37 ALVE insertion sites, 23 of which were new. There were some ALVE sites that were only found in certain breeds. For example, 16 out of 23 new ALVEs were only found in one breed of Chinese domestic chicken. The discovered ALVE insertions were found to be very similar to ALVE1 after being confirmed by PCR and Sanger sequencing (Wang *et al.*, 2023). Currently, the recognized diversity of ALVE encompasses over 1300 distinct integration sites. (Rutherford *et al.*, 2016; Mason *et al.*, 2018; Mason *et al.*, 2020).

ALVE insertions are both new and common because there is a lot of variation in the elements between chicken populations, the number of copies of each element is low, and the overall structure is usually strong. In fact, almost half of the twenty-three ALVEs found in different White Leghorn lines can make virions that are competent for replication (Benkel, 1998; Borisenko, 2003). The virion surface protein gp85, which is encoded by the envelope gene (*env*), defines ALV subgroups. This is because it defines the specific TV (tumor virus) cell entry receptor (Mason *et al.*, 2018). For instance, ALV-A enters through the TVA receptor, ALV-C through the TVC receptor, and subgroups B, D, and E through the TVB receptor. Researchers have found two TVB alleles (TVB*S3 and TVB*R) that make cells resistant to ALVE entry. However, these alleles are both recessive to and much less common than the wildtype TVB*S1, which is susceptible to all three subgroups (Hunt *et al.*, 2008; Yu *et al.*, 2008; Kaya, 2018). There are no recorded instances of ALVEs harboring accessory oncogenes, and their expression is two to three orders of magnitude lower than that of exogenous ALV, attributable to the deletion of enhancers in the ALVE LTR U3 domain (Coffin *et al.*, 1983; Norton and Coffin, 1987; Conklin, 1991). ALVE LTRs typically possess a singular enhancer, in contrast to the tandem enhancer cassette found in exogenous ALV (Ruddell, 1995). As a result, ALVEs infrequently provoke tumorigenesis; however, the existence of these loci within the host genome can influence the infection dynamics of exogenous ALV (Yu *et al.*, 2008; Kanda *et al.*, 2013; Justice and Beemon, 2013; Liang *et al.*, 2017; Fandiño *et al.*, 2024). Numerous previously identified ALVE elements have been detected

in both commercial and indigenous chickens (Mason *et al.*, 2020; Benkel *et al.*, 1998; Rutherford *et al.*, 2016). A number of other ALVE elements have also been reported to be involved in several other economically significant traits, particularly production traits, which will be addressed in the subsequent subheading.

Economic impact of ERV elements

It is well known through research that endogenous retroviruses generally negatively affect production traits, especially through their interaction with exogenous ALV (Liang *et al.*, 2017; Mason *et al.*, 2018), impact the expression of certain genes (Ishihara *et al.*, 2025), and also directly infect the host if expressed in its pathogenic form (Gavora *et al.*, 1991). ALVE has been associated with performance traits in layers. Fulton *et al.* tested the association of ALVE with 18 different layer performance traits in their 2021 study on 8 Hyline elite layer lines. Out of these traits, 15 exhibited significant associations with at least one trait in at least one line. There were instances where a single ALVE type had varying impacts on the same trait in different lines. Conversely, the same ALVE insertions could have a positive effect on a particular trait in one line but an opposite effect in another (Fulton *et al.*, 2021). For instance, ALVE 15 is noted to have a negative effect on albumen height in one line of White Leghorn but not in another. ALVE can also associate significantly with multiple traits (Fulton *et al.*, 2021). These outcomes evince the complex variability of ALVE in associating with various traits while eliciting several effects on production traits even within the same line. One of the economically important production traits for poultry farmers is egg production. ALVE 3 is shown to detrimentally impact this trait (Gavora *et al.*, 1991). However, in one White Leghorn line (WL3), a positive impact was realized. (Fulton *et al.*, 2021). Meanwhile, consistent with previous studies on the detrimental effects of ALVEs, about six ALVE types in Fulton *et al.*'s experiment had an exclusively negative impact on egg production traits. Of the known ALVEs, ALVE 21 is the most common among breeds that are of economic importance. ALVE 21 is situated approximately 650 base pairs downstream of a CR1 (Chicken Repeats 1) element within a substantially duplicated genomic segment (Iraqi and Smith, 1995). ALVE21/Slow feathering complex has been characterized as a replication-competent provirus linked to sex-linked slow feathering in the K locus (Tixier-Boichard *et al.*, 1997; Elferink *et al.*, 2008; Bu *et al.*, 2013). Apart from ALVE, other endogenous retroviruses are of economic importance, especially *ev/J*, which by itself is non-infectious but can recombine with exogenous ALVs to form the more oncogenic ALVJ retrovirus. ALVJ can cause leucosis in different bird species (Nikzad *et al.*, 2025), and symptoms could include paleness, weight loss, and even reduced egg production in laying hens, which are economically important traits (Liang *et al.*, 2017; Eid *et al.*, 2019).

ALVK, also a newly discovered low-pathogenic retrovirus (Cui *et al.*, 2014), is said to be endemic in domestic chickens in China (Dong *et al.*, 2015), and recombination of this retrovirus with other ALV subgroups has also been shown to increase its pathogenicity (Lv *et al.*, 2019; Li *et al.*, 2022). This poses a risk for the emergence of new recombinant ALVs and challenges control efforts towards eradicating ALVs from the chicken genome (Zhang *et al.*, 2024). In recent research, the ability of endogenous retrovirus insertions in influencing the expression of nearby genes was investigated in Japanese indigenous chickens (Ishihara *et al.*, 2025). 172 ERV loci were identified in their experiment, with a focus on genes located close to these loci. It was observed that out of 75 genes located near these ERV loci, there were significant differences in the expression of GTP/ATP binding carboxypeptidase 1 (AGTPBP-1), N-acetylated alpha-linked acidic dipeptidase 2 (NAALAD2), and phosphoribosylaminoimidazolesuccinocarboxamide synthase (PAICS). While PAICS expression was increased, NAALAD2 had decreased expression. These results support previous postulations that ERV insertions within or near genes affect their expression.

Although ALVE-free lines have been successfully introduced (Astrin *et al.*, 1979; Zhang *et al.*, 2008), in recent times, novel insertions and recombinations seem to dampen efforts to control and eradicate ALVs, especially in the non-commercial poultry industry (Li *et al.*, 2025). With the call to and rise of sustainable livestock production, especially with the use of local breeds, efforts could be seriously hampered by these retroviral elements. Therefore, in order to compensate for these novel additions, new detection analysis techniques, vaccines, and breeding strategies need to be developed.

Conclusion

The story of ALVE elements in the chicken genome is one of enduring conflict and unexpected complexity. Far from being inert passengers, these endogenous retroviruses are active participants in the biology of the host, capable of undermining economic gains through their direct and indirect effects on health and productivity. The discovery of vast ALVE diversity in both commercial and indigenous flocks, coupled with their potential for recombination with exogenous viruses, underscores a persistent and evolving threat.

However, this challenge is not insurmountable. The same genomic tools that have illuminated the scale of the problem also light the path toward solutions. The development of ALVE-free lines was a landmark achievement, yet the dynamic nature of these elements demands a more nuanced and continuous approach. The future of ALVE management lies in the utilization of the pan-genome-wide association studies, including integration of high-resolution pan-genome maps, advanced diagnostic assays, and precise breeding

selecting against detrimental insertions without eroding the valuable genetic diversity found in local breeds. Also, the possible use of CRISPR/Cas9 to cut out proviral sequences could lead to a precision genome editing method for managing ALVE. This would allow highly productive lines to be cleansed of viral reservoirs without the generational lag or linkage drag that comes with traditional backcrossing. There are still technical and legal problems with off-target effects and GMO laws, but the ability to remove viral reservoirs without the generational lag of backcrossing is a big step forward for elite line conservation. As the industry moves towards more sustainable, enhanced resilience, and robust production systems, a deep and proactive understanding of the endogenous retrovirus nexus will be paramount. By moving from observation to intervention, we can begin to rewrite the narrative, transforming these ancient viral foes from economic liabilities into manageable elements of the chicken's rich genetic heritage.

References

- Adkins, H.B., Blacklow, S.C., & Young, J.A. (2001). Two functionally distinct forms of a retroviral receptor explain the nonreciprocal receptor interference among subgroups B, D, and E avian leukosis viruses. *Journal of Virology*, 75(8), 3520–3526. <https://doi.org/10.1128/JVI.75.8.3520-3526.2001>.
- Astrin, S.M., Buss, E.G., & Hayward W.S. (1979). Endogenous viral genes are non-essential in the chicken. *Nature*, 282(5736), 339–341. <https://doi.org/10.1038/282339a0>.
- Aswad, A., & Katzourakis, A. (2012). Paleovirology and virally derived immunity. *Trends in Ecology & Evolution*, 27(11), 627–636. <https://doi.org/10.1016/j.tree.2012.07.007>.
- Beard, J.W. (1980). Biology of avian oncornaviruses. In G. Klein (Ed.), *Viral Oncology*, 55–87. Raven Press.
- Benkel, B.F. (1998). Locus-specific diagnostic tests for endogenous avian leukosis-type viral loci in chickens. *Poultry Science*, 77(7), 1027–1035. <https://doi.org/10.1093/ps/77.7.1027>
- Borisenko, L. (2003). Avian endogenous retroviruses. *Folia Biologica*, 49(5), 177–182.
- Borysenko, L., Stepanets, V., & Rynditch, A.V. (2008). Molecular characterization of full-length MLV-related endogenous retrovirus ChiRV1 from the chicken, *Gallus gallus*. *Virology*, 376(1), 199–204. <https://doi.org/10.1016/j.virol.2008.03.017>
- Boyce-Jacino, M.T., Resnick R., & Faras A.J. (1989). Structural and functional characterization of the unusually short long terminal repeats and their adjacent regions of a novel endogenous avian retrovirus, *Virology*, 173:157.
- Bu, G., Huang G., Fu, H., Li, J., Huang, S., & Wang Y. (2013). Characterization of the novel duplicated PRLR gene at the late-feathering K locus in Lohmann chickens. *Journal of Molecular Endocrinology*, 51(2), 261–276. <https://doi.org/10.1530/JME-13-0068>
- Chang, C.M., Coville, J.L., Coquerelle, G., Gourichon, D., Oulmouden, A., & Tixier-Boichard, M. (2006). Complete association between a retroviral insertion in the tyrosinase gene and the recessive white mutation in chickens. *BMC Genomics*, 7, 19. <https://doi.org/10.1186/1471-2164-7-19>.
- Chiu, E.S., & Vandewoude, S. (2021). Endogenous retroviruses drive resistance and promotion of exogenous retroviral homologs. *Annual Review of Animal Biosciences*, 9, 225–248. <https://doi.org/10.1146/annurev-animal-061220-023215>.
- Coffin, J.M., Tschlis, P.N., Conklin, K.F., Senior, A., & Robinson, H.L. (1983). Genomes of endogenous and exogenous avian retroviruses. *Virology*, 126(1), 51–72. [https://doi.org/10.1016/0042-6822\(83\)90461-0](https://doi.org/10.1016/0042-6822(83)90461-0)
- Conklin, K.F. (1991). Activation of an endogenous retrovirus enhancer by insertion into a heterologous context. *Journal of Virology*, 65(5), 2525–2532. <https://doi.org/10.1128/JVI.65.5.2525-2532.1991>
- Crittenden, L.B. (1991). Retroviral elements in the genome of the chickens: implications for poultry genetics and breeding. *Critical Reviews in Poultry Biology*, 3, 73–109.
- Cui, N., Su, S., Chen, Z.M., Zhao, X.M., & Cui, Z.Z. (2014). Genomic sequence analysis and biological characteristics of a rescued clone of avian leukosis virus strain JS11C1, isolated from indigenous chickens. *Journal of General Virology*, 95(11), 2512–2522. <https://doi.org/10.1099/vir.0.068106-0>.
- Dong, X., Zhao, P., Li, W., Chang, S., Li, J., Li, Y., Ju, S., Sun, P., Meng, F., Liu, J., & Cui, Z. (2015). Diagnosis and sequence analysis of avian leukosis virus subgroup J isolated from Chinese Partridge Shank chickens. *Poultry Science*, 94(4), 668–672. <https://doi.org/10.3382/ps/pev040>.
- Eid, A.E., Abd-Ellatieff, H.A., Ellakany, H.F., Abou-Rawash, A.-R. A., & Abdel-Hamid, H.S. (2019). Studies on tumor disease viruses in chickens in Egypt. *Alexandria Journal of Veterinary Sciences*, 60(1), 184–195.
- Elferink, M.G., Vallee, A.A.A., Jungerius, A.P., Crooijmans, R.P.M.A., & Groenen M.A.M. (2008). Partial duplication of the PRLR and SPEF2 genes at the late feathering locus in chicken. *BMC Genomics*, 9, 391. <https://doi.org/10.1186/1471-2164-9-391>.
- Ellinghaus, D., Kurtz, S., & Willhoeft, U. (2008). LTR harvest, an efficient and flexible software for de novo detection of LTR retrotransposons. *BMC Bioinformatics*, 9, 18. <https://doi.org/10.1186/1471-2105-9-18>.
- Fandiño, S., Gomez-Lucia, E., Benítez, L., & Doménech, A. (2024). Comparison of endogenous alpharetroviruses (ALV-like) across galliform species: New distant proviruses. *Microorganisms*, 12(1), 86. <https://doi.org/10.3390/microorganisms12010086>
- Frisby, D.P., Weiss, R.A., Roussel, M., & Stehelin, D. (1979). The distribution of endogenous chicken retrovirus sequences in the DNA of galliform birds does not coincide with avian phylogenetic relationships. *Cell*, 17(3), 623–634. [https://doi.org/10.1016/0092-8674\(79\)90270-8](https://doi.org/10.1016/0092-8674(79)90270-8)
- Fulton, J.E., Mason, A.S., Wolc, A., Arango, J., Settar, P., Lund, A.R., & Burt, D.W. (2021). The impact of endogenous Avian Leukosis Viruses (ALVE) on production traits in elite layer lines. *Poultry Science*, 100(6), 101121. <https://doi.org/10.1016/j.psj.2021.101121>.
- Gavora, J.S., Kuhnlein, U., Crittenden, L.B., Spencer, J.L., & Sabour, M.P. (1991). Endogenous viral genes: Association with reduced egg production rate and egg size in White Leghorns. *Poultry Science*, 70(3), 618–623. <https://doi.org/10.3382/ps.0700618>.
- Gudkov, A.V., Komarova, E.A., & Zaitsevskaya, T.E. (1992) – ART-CH, a new chicken retroviruslike element. *Journal of Virology*, 66(3), 1726–1736. <https://doi.org/10.1128/JVI.66.3.1726-1736.1992>

- Huda, A., Polavarapu, N., Jordan, I.K., & McDonald, J.F. (2008). Endogenous retroviruses of the chicken genome. *Biology Direct*, 3, 9. <https://doi.org/10.1186/1745-6150-3-9>.
- Hunt, H.D., Fadly, A.M., Silva, R.F., & Zhang, H. (2008). Survey of endogenous virus and TVB* receptor status of commercial chicken stocks supplying specific-pathogen-free eggs. *Avian Diseases*, 52(3), 433–440. <https://doi.org/10.1637/8183-112907-Reg.1>
- Iraqi, F., Smith, E.J. (1995). Organization of the sex-linked late-feathering haplotype in chickens. *Animal Genetics*, 26(3), 141–146. <https://doi.org/10.1111/j.1365-2052.1995.tb03153.x>
- Iraqi, F., Darvasi, A., Zeitlin, G., Beckmann, J.S., & Soller, M. (1994). Nonlinear effects of chicken endogenous viruses on body weight may be responsible for maintaining these elements in a stable genetic polymorphism. *Poultry Science*, 73(11), 1625–1632. <https://doi.org/10.3382/ps.0731625>
- Ishihara, S., Shiraiishi, J.I., Shimamoto, S., & Ijiri D. (2025). Endogenous retrovirus loci and induced changes in gene expression in Japanese indigenous chickens. *Scientific Reports*, 15(1), 12290. <https://doi.org/10.1038/s41598-025-96881-z>
- Jiang, R., Zhou, J., Liu, Y., Zhou, G., Fan, D., Xiang, L., Chen, Y., & Shao, J. (2025). Endogenous retroviruses in host-virus coevolution: From genomic domestication to functional innovation. *Genes*, 16(8), 964. <https://doi.org/10.3390/genes16080964>.
- Justice, J. Iv., & Beemon, K.L. (2013). Avian retroviral replication. *Current Opinion in Virology*, 3(6), 664–669. <https://doi.org/10.1016/j.coviro.2013.08.008>
- Kanda, R.K., Tristem, M., & Coulson, T. (2013). Exploring the effects of immunity and life history on the dynamics of an endogenous retrovirus. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 368(1626), 20120505. <https://doi.org/10.1098/rstb.2012.0505>
- Kaya, M. (2018). Determination of the tumor virus B locus in Turkish native chicken breeds. *Kafkas Universitesi Veteriner Fakültesi Dergisi*, 24 (2), 239-242, 2018. Doi: 10.9775/kvfd.2017.18769
- Lee, J., Mun, S., Kim, D.H., Cho, C.S., Oh, D.Y., & Han, K. (2017). Chicken (*Gallus gallus*) endogenous retrovirus generates genomic variations in the chicken genome. *Mobile DNA*, 8, 2. <https://doi.org/10.1186/s13100-016-0085-5>
- Li, T., Li, J., Wang, Z., Wu, J., Ma, L., Wang, S., Wan, Z., Xie, Q., Shao, H., Qin, A., & Ye, J. (2025). Promising strategies for accelerating the eradication of avian leukosis in China. *Animals and Zoonoses*, <https://doi.org/10.1016/j.azn.2025.03.003>
- Li, Y., Liu, Y., Lin, Z., Cui, S., Chang, S., Cui, Z., & Zhao, P. (2022). Role of env gene and LTR sequence in the pathogenesis of subgroup K avian leukosis virus. *Journal of General Virology*, 103(2), 001719. <https://doi.org/10.1099/jgv.0.001719>
- Liang, X., Liu, L., Fang, C., Gu, Y., Li, T., & Yang Y. (2018). Identified novel deletions in the genomes of avian endogenous retroviruses ev/J in chicken breeds in China. *Kafkas Universitesi Veteriner Fakültesi Dergisi*, 24(3), 409–414. <https://doi.org/10.9775/kvfd.2017.19079>
- Liu, Q., Ji, S., Ren, T., He, L., Ding, K., Yu, Z., & Chen, J. (2025). A double-edged sword: The multiple roles of endogenous retroviruses in chickens. *Poultry Science*, 104,12. <https://doi.org/10.1016/j.psj.2025.105907>
- Llorens, C., Muñoz-Pomer, A., Bernard, L., Botella, H., & Moya, A. (2011). The Gypsy Database (GyDB) of mobile genetic elements: Release 2.0. *Nucleic Acids Research* 39(Database issue), D70–D74. <https://doi.org/10.1093/nar/gkq1061>
- Lv, L., Li, T., Hu, M., Deng, J., Liu, Y., Xie, Q., & Zhang, Y. (2019). A recombination efficiently increases the pathogenesis of the novel K subgroup of avian leukosis virus. *Veterinary Microbiology*, 231, 214–217. <https://doi.org/10.1016/j.vetmic.2019.03.020>
- Mason, A.S. (2018). The abundance and diversity of endogenous retroviruses in the chicken genome [Doctoral dissertation, University of Edinburgh].
- Mason, A.S., Fulton, J.E., Hocking, P.M., & Burt, D.W. (2016). A new look at the LTR retrotransposon content of the chicken genome. *BMC Genomics*, 17, 688. <https://doi.org/10.1186/s12864-016-3043-1>
- Mason, A.S., Miedzinska, K., Kebede, A., Bamidele, O., Al-Jumaili, A.S., Dessie, T., Hanotte, O., & Smith, J. (2020). Diversity of endogenous avian leukosis virus subgroup E (ALVE) insertions in indigenous chickens. *Genetics Selection Evolution*, 52, 29. <https://doi.org/10.1186/s12711-020-00548-4>.
- Mccarthy, E.M., & McDonald, J.F. (2003). LTR_STRUC: A novel search and identification program for LTR retrotransposons. *Bioinformatics*, 19(3), 362–367. <https://doi.org/10.1093/bioinformatics/btg005>
- Nikzad, M., & Bozorgmehrifard, M. H. (2025). Novel insights into immunity, diagnosis, and vaccination of avian leukosis. *Journal of Poultry Sciences and Avian Diseases*, 3(4), 59–72. <http://dx.doi.org/10.61838/kman.jpSad.3.4.7>
- Norton, P.A., & Coffin, J.M. (1987). Characterization of Rous sarcoma virus sequences essential for viral gene expression. *Journal of Virology*, 61(4), 1171–1179. <https://doi.org/10.1128/JVI.61.4.1171-1179.1987>
- Payne, L.N., (2001). Avian leukosis virus – new mutations: a threat for the upcoming century. *World's Poultry Science Journal*, 57(3), 265–274. doi:10.1079/WPS20010019
- Payne, L.N., & Fadly A.M. (1997). Leukosis/sarcoma group. In B. W. Calnek, H. J. Barnes, C. W. Beard, L. R. McDougald, & Y. M. Saif (Eds.), *Diseases of poultry* (10th ed., pp. 414-466). Iowa State University Press.
- Payne, L.N., & Nair, V. (2012). The long view: 40 years of avian leukosis research. *Avian Pathology*, 41(1), 11–19. <https://doi.org/10.1080/03079457.2011.646238>
- Purchase, H.G. (1987). The pathogenesis and pathology of neoplasms caused by avian leukosis viruses. In G. F. de Boer (Ed.), *Avian leukosis*, (pp. 171–196). Martinus Nijhoff Publishing.
- Rho, M., Choi, J.H., Kim, S., Lynch, M., & Tang, H. (2007). De novo identification of LTR retrotransposons in eukaryotic genomes. *BMC Genomics*, 8, 90. <https://doi.org/10.1186/1471-2164-8-90>
- Ruddell, A. (1995). Transcription regulatory elements of the avian retroviral long terminal repeat. *Virology*, 206(1), 1-7. [https://doi.org/10.1016/s0042-6822\(95\)80013-1](https://doi.org/10.1016/s0042-6822(95)80013-1)
- Rutherford, K., Meehan, C.J., Langille, M.G.I., Tyack, S.G., Mckay, J.C., Mclean, N.L., Benkel, K., Beiko, R.G., & Benkel, B. (2016). Discovery of an expanded set of avian leukosis subgroup E proviruses in chickens using Vermillion, a novel sequence capture and analysis pipeline. *Poultry Science*, 95(10), 2250–2258. <https://doi.org/10.3382/ps/pew194>

- Sacco, M.A., Howes, K., Smith, L.P., & Nair, V.K. (2004). Assessing the roles of endogenous retrovirus EAV-HP in avian leukosis virus subgroup J emergence and tolerance. *Journal of Virology*, 78(19), 10525-10535. <https://doi.org/10.1128/JVI.78.19.10525-10535.2004>
- Sacco, M.A., & Nair V.K. (2014). Prototype endogenous avian retroviruses of the genus Gallus. *Journal of General Virology*, 95(9), 2060–2070. <https://doi.org/10.1099/vir.0.066852-0>
- Smit, A., Hubley, R., & Green P. (2013). RepeatMasker Open-4.0 [Computer software]. <http://www.repeatmasker.org>.
- Takenouchi, A., Toshishige, M., Ito, N., & Tsudzuki, M. (2018). Endogenous viral gene ev21 is not responsible for the expression of late feathering in chickens. *Poultry Science*, 97(2), 403–411. <https://doi.org/10.3382/ps/pex324>
- Tixier-Boichard, M., Boulliou-Robic, A., Morisson, M., Coquerelle, G., Horst, P., & Benkel, B. (1997). A deleted retroviral insertion at the ev21-K complex locus in Indonesian chickens. *Poultry Science*, 76(5), 733–742. <https://doi.org/10.1093/ps/76.5.733>
- Venugopal, K. (1999). Avian leukosis virus subgroup J: A rapidly evolving group of oncogenic retroviruses. *Research in Veterinary Science*, 67(2), 113–119. <https://doi.org/10.1053/rvsc.1998.0287>
- Wang, Z., Qu, L., Yao, J., Yang, X., Li, G., Zhang, Y., Li, J., Wang, X., Bai, J., Xu, G., Deng, X., Yang, N., & Wu, C. (2013). An EAV-HP insertion in 5' flanking region of SLCO1B3 causes blue eggshell in the chicken. *PLoS Genetics*, 9(9), e1003183. <https://doi.org/10.1371/journal.pgen.1003183>.
- Wang, Z., Yuan, Y., Zheng, G., Sun, M., Wang, Q., Wu, J., Li, J., Sun, C., Wang, Y., Yang, N., & Lian, L. (2023). Short communication: Diversity of endogenous avian leukosis virus subgroup E elements in 11 chicken breeds. *Journal of Animal Science*, 101, skad081. <https://doi.org/10.1093/jas/skad081>
- Warren, W.C., Hillier, L.W., Tomlinson, C., Minx, P., Kremitzki, M., Graves, T., Markovic, C., Bouk, N., Pruitt, K.D., Thibaud-Nissen, F., Schneider, V., Mansour, T.A., Brown, C.T., Zimin, A., Hawken, R., Abrahamsen, M., Pyrkosz, A.B., Morisson, M., Fillon, V., ... & Cheng, H.H. (2017). A new chicken genome assembly provides insight into avian genome structure. *G3: Genes, Genomes, Genetics*, 7(1), 109–117. <https://doi.org/10.1534/g3.116.035923>
- Wragg, D., Mwacharo, J.M., Alcalde, J.A., Weng, C., Han, J.-L., Gongora, J., Gourichon, D., Tixier-Boichard, M., & Hanotte, O. (2013). Endogenous retrovirus EAV-HP linked to blue egg phenotype in Mapuche fowl. *PLoS ONE*, 8(8), e71393. <https://doi.org/10.1371/journal.pone.0071393>
- Yu, Y., Zhang, H., Tian, F., Bacon, L., Zhang, Y., Zhang, W., & Song, J. (2008). Quantitative evaluation of DNA methylation patterns for ALVE and TVB genes in a neoplastic disease susceptible and resistant chicken model. *PLoS ONE*, 3(3), e1731. <https://doi.org/10.1371/journal.pone.0001731>
- Zhang, F., Li, H., Lin, C., Wei, Y., Zhang, W., Wu, Y., & Kang, Z. (2024). Detection and genetic diversity of subgroup K avian leukosis virus in local chicken breeds in Jiangxi from 2021 to 2023. *Frontiers in Microbiology*, 15, 1341201. <https://doi.org/10.3389/fmicb.2024.1341201>
- Zhang, H., Bacon, L.D., & Fadly, A.M. (2008). Development of an endogenous virus-free line of chickens susceptible to all subgroups of avian leukosis virus. *Avian Diseases*, 52(1), 41–46. <https://doi.org/10.1637/8180-112707-Reg>