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TRANSPORTATION OF POULTRY AS A CRITICAL FACTOR IN THE SPREAD OF VIRAL INFECTIONS IN WATERFOWL

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Abstract – Road transportation of poultry plays a pivotal role in the dissemination of viral diseases among waterfowl populations. Movement of apparently healthy birds, combined with the potential contamination of vehicles, crates, and associated equipment, constitutes a significant epidemiological risk. This study highlights the presence of viral genetic material in clinically healthy geese and ducks transported to slaughterhouses, underlining transport as a major vector of horizontal transmission. Detected pathogens included goose parvovirus (GPV), goose hemorrhagic polyomavirus (GHPV), goose circovirus (GoCV), avian reoviruses (ARV), duck hepatitis virus (DHV), and duck viral enteritis virus (DVE). The data confirm that transportation enhances viral shedding through stress-related physiological changes in birds, while insufficient vehicle disinfection amplifies the risk of cross-flock contamination. Biosecurity failures in transportation may contribute not only to local outbreaks but also to transregional and transcontinental spread.

Key words – transport, poultry, waterfowl, viral infections, epidemiology, biosecurity

JEL Classification – Q18, R41, I18, Q13, L66

INTRODUCTION

Effective implementation of biosecurity protocols during the transportation of poultry is essential to limit the dissemination of infectious agents. Key measures include meticulous cleaning and disinfection of transport vehicles, as well as controlled access to farms. Both commercial poultry movements and interactions with wild birds serve as major reservoirs and vectors for pathogens, significantly contributing to interregional and interspecies transmission, thereby facilitating atypical outbreaks and large-scale epidemics. Transportation contributes to disease spread through several mechanisms:

1. **Movement of infected birds:** Poultry transportation-whether for sale, slaughter, or relocation-inevitably involves relocating potentially infected animals, creating a pathway for pathogens to reach previously unaffected areas.
2. **Contaminated vehicles:** Vehicles that carry birds can become reservoirs for viruses, bacteria, and other infectious agents originating from infected birds, litter, or droppings. If these vehicles are not properly sanitized, they can introduce pathogens to other farms, markets, or processing sites.
3. **Insufficient farm biosecurity:** Farms with weak biosecurity practices, including inadequate cleaning of vehicles and equipment, are particularly vulnerable to disease transmission.
4. **Human-mediated transmission:** People involved in transport and handling may act as mechanical vectors, transferring pathogens via clothing, footwear, or equipment, especially after contact with infected birds or contaminated environments [1-3].

Waterfowl are particularly vulnerable to a range of viral diseases that can be disseminated during transport, affecting both domestic and wild populations. Goose parvoviruses (GPVs) present significant epizootic and economic challenges in intensive poultry production. Three GPV variants have been identified: the classical GPV (CGPV), responsible for Derzsy's disease in geese; the novel GPV (NGPV), which specifically infects Pekin and mule ducks and causes short beak and dwarfism syndrome (SBDS); and the Muscovy duck parvovirus

(MDPV), affecting Muscovy and mule ducks. CGPV can infect not only geese, but also Muscovy ducks, swans, and swan geese (Anser cygnoides), whereas MDPV is limited to Muscovy and mule ducks [4-5, 9-12]. Despite differences in host specificity and pathology, all three viruses are classified under the species *Anseriform dependoparvovirus 1*, genus *Dependoparvovirus*, subfamily *Parvovirinae*, family *Parvoviridae* [6].

Most isolates of GPV and MDPV exhibit high virulence, particularly in young birds [7-8, 10-12]. Clinically, the disease is marked by severe lethargy, anorexia, watery diarrhea, respiratory distress, locomotor dysfunction, and feather loss. Necropsy typically reveals ascites, skeletal muscle myopathy, hepatitis, myocarditis, sciatic neuritis, and polioencephalomyelitis. Lymphoid organ atrophy, involving the bursa of Fabricius, spleen, and thymus, is also common. Surviving goslings, ducklings, and older infected birds frequently display degenerative skeletal muscle myopathy and stunted growth [15]. Transmission occurs both vertically through infected eggs and horizontally via fecal contamination and indirectly through contaminated equipment, litter, and human activity. The economic impact on waterfowl husbandry is considerable [13–14].

Hemorrhagic nephritis and enteritis of geese (HNEG), historically referred to as "young geese disease" or "late Derzsy's disease," is another critical viral condition in industrial waterfowl production [16-18]. The etiologic agent, goose hemorrhagic polyomavirus (GHPV), belongs to the genus *Gammapolyomavirus*, family *Polyomaviridae*. HNEG was first diagnosed in Hungary in 1969 and subsequently reported in Germany, France, Poland, Belgium, and more recently in Taiwan [19-23]. Ducks may act as asymptomatic carriers [24-25]. The virus exhibits endothelial tropism, primarily affecting blood vessels in the gastrointestinal and urogenital systems, leading to necrosis of renal tubules, circulatory disturbances, swelling, congestion, and renal parenchyma necrosis. In goslings, ascites and edema related to vascular injury are observed, with disease typically occurring between three and six weeks of age, though older birds can also be affected [26-27].

Goose circovirus (GoCV), a member of the *Circoviridae* family, genus *Circovirus*, poses a continuing epidemic threat to goose populations. First reported in Germany in 1999, the virus has since spread to various European and Asian countries [28]. Clinical manifestations include growth inhibition, diarrhea, and plumage abnormalities. Postmortem findings often reveal enlarged spleen, thymus, and liver, as well as petechiae in the epicardium, endocardium, lungs, and thymus. Notably, GoCV induces immunosuppression, rendering birds more susceptible to secondary infections. Experimental infections indicate that disease severity is influenced by viral dose, strain, inoculation route, co-infections, and host breed [18-21].

Duck virus enteritis (DVE), or duck plague (DP), caused by Anatid herpesvirus type 1 (family *Herpesviridae*, subfamily *Alphaherpesvirinae*), is an acute, sometimes chronic, highly contagious disease affecting ducks, geese, swans, and other waterfowl globally [12-18]. While many affected birds die without obvious clinical signs, observable symptoms may include vascular lesions, internal hemorrhage, lymphoid organ damage, digestive mucosal eruptions, diarrhea, and degenerative changes in parenchymal tissues. Additional clinical signs include partially closed eyelids with photophobia, extreme thirst, ataxia, nasal discharge, drooping plumage, and tremors [24-25]. DVE establishes latent infections in trigeminal ganglia, from which the virus can reactivate, with recovered birds shedding the virus in feces for several months [24-28].

Duck viral hepatitis (DVH) represents another significant pathogen, causing high mortality in ducklings and substantial economic losses. Three types of DVH are recognized: type I, caused by three genotypes of duck hepatitis A virus (DHAV-1, DHAV-2, DHAV-3; Picornaviridae, genus *Avihepatovirus*), with DHAV-1 being most pathogenic; type II, caused by duck astrovirus type 1 in the UK; and type III, caused by duck astrovirus type 2 in the USA, which is less virulent. DHAV primarily affects ducklings under three weeks, producing acute liver necrosis, hemorrhage, and neurological signs [14-15].

Avian reoviruses (ARVs), belonging to genus *Orthoreovirus*, family *Reoviridae*, also pose health risks to waterfowl. Classical Muscovy duck reovirus (C-MDRV), first reported in South Africa in 1950, manifests as weakness, arthritis, watery diarrhea, and stunted growth, with necrotic foci in liver and spleen [12-18]. Goose reovirus (GRV), identified in Hungary in the 1990s and later in China, presents with similar clinical symptoms, including locomotor disorders, arthritis, diarrhea, splenitis, and hepatic necrotic foci [15-19].

The primary objective of this research was to evaluate the risk of viral infections in waterfowl during transport. Transportation represents a critical step in pathogen dissemination, as clinically healthy birds may carry infections undetected. The study aimed to quantify infection risks during movement between farms and other locations and to assess the health status of commercial waterfowl flocks, focusing on viral infections in ducks and geese.

1. MATERIALS AND METHODS
SAMPLE COLLECTION AND PREPARATION

Samples were obtained from apparently healthy birds intended for slaughter, which exhibited no visible clinical signs of disease. Birds were loaded onto transport vehicles for delivery to the slaughterhouse. For analysis, tracheal swabs were collected, with cloacal swabs taken less frequently. During postmortem examinations, sections of internal organs-including liver, spleen, heart, lungs, kidneys, and intestines-were also collected. Swabs and organ tissues were suspended in Eagle's minimum essential medium (MEM, Sigma Aldrich, USA) supplemented with 1% antibiotic-antimycotic solution (Gibco, Scotland) to prevent microbial contamination. To release viral particles from host cells, homogenates underwent three cycles of freezing and thawing. Prepared samples were then centrifuged at 3000 × g for 5 minutes at 4°C, and the resulting supernatant was collected for total DNA extraction. DNA was isolated using the commercial Indispin Pathogen Kit, following the manufacturer's protocol, and stored at −20°C until PCR analysis.

Table 1. Control Strains and Templates [14]

Virus	Primers	Primer sequence 5'-3'	Products size (base pirs)
GHPV	GHPV-F GHPV-R	ACCCGTGCTTCCATTCACAA CTGCTCCCCAAACCTGTCAA	397 bp
GoCV	ORFC1-F ORFC1-R	GGAAGGGGTTAAATGCGGGA ACGATACAGACGACGAAGGC	308 bp
GPV	VP3F VP3R	GTGCCGATGGAGTGGGTAAT GCGCCAGGAAGTGCTTTAT	1604 bp
ARV	967F 967R	CCCACTTTCCATTCCTTTCA GCCATCCTAGCTGGAGAGAC	967 bp
ARV nested PCR	sigmaNSF sigmaNSR	CCGAGTGGCCCTATTGACTA CAGCGACCACTTAGATGCAA	508 bp
DVE	DVE F DVE R	ATCAGGGTGATTCTAACCAG CTTATTTCTAATTTGGTCAG	300 bp
DHV-1	DHV1F DHV1R	ATCAGGGTGATTCTAACCAG CTTATTTCTAATTTGGTCAG	467 bp
MDPV	MDPV1 MDPV2	GGAGAGAAATGGCAGTGG GCTGTTGTTGTGTTTTGT	1204 bp

Positive controls for molecular diagnostics included: strain 50/15 (GenBank MG869737) for goose hemorrhagic polyomavirus (GHPV), strain 39/14 (GenBank MH138278) for goose circovirus (GoCV), strain FM (Vilmos Palya, CEVA-Phylaxia, Budapest, Hungary) for Muscovy duck parvovirus (MDPV), and the GPV H strain from the commercial Palmivax vaccine for goose parvovirus (GPV). Strain 1227 (Department of Poultry Viral Diseases, NVRI, Pulawy, Poland) served as a positive control for duck virus enteritis (DVE). Negative controls consisted of DNA extracted from non-infected goose embryo fibroblasts (GEFs).

PCR Assay Extracted DNA samples were tested for the presence of GPV, MDPV, GHPV, GoCV, DVE, and duck hepatitis virus (DHV) using specific nucleotide primers complementary to conserved viral gene sequences.

Table 1 sequences of primers used for individual viruses and the size of the expected produc.

The reaction was performed in a gradient thermocycler (Biometra, Germany) in a final reaction volume of 25 µl. The time-temperature conditions of PCR were as follows:

Table 2. Temperature-time conditions for DNA viruses [15]

Virus	Initial Denaturation	Denaturation	Primer Annealing	Extension	Number of cycles	Final Extension
GPV	95°C/5 min.	94°C/15 sec.	60°C/20 sec.	72°C/45 sec.	40	72°C/10 min.
MDPV	95°C/5 min.	94°C/15 sec.	60°C/20 sec.	72°C/45 sec.	40	72°C/10 min.
GHPV	95°C/5 min.	94°C/15 sec.	55°C/20 sec.	72°C/45 sec.	35	72°C/10 min.
GoCV	95°C/5 min.	94°C/15 sec.	54°C/20 sec.	72°C/45 sec.	35	72°C/10 min.
DVE	95°C/5 min.	94°C/15 sec.	54,5°C/1 min.	72°C/1 min.	35	72°C/10 min.
REO nested	95°C/5 min.	94°C/1 min.	60°C/1 min..	72°C/1 min..	35	72°C/10 min.

Table 3. Temperature-time conditions for RNA viruses [author's research]

Virus	Reverse transcription	Initial Denaturation	Denaturation	Primer Annealing	Extension	Number of cycles	Final Extension
DHV	50°C/30 min.	95°C/15 min.	94°C/45 sec.	52°C/1 min.	72°C/1 min.	40	72°C/10 min.
REO	50°C/30 min.	95°C/15 min.	94°C/45 sec.	57°C/1 min.	72°C/1 min.	40	72°C/10 min.

RESULTS

Transport represents a substantial route for the transmission of viral infections in poultry. Vehicles, transport crates, and associated equipment can act as fomites, becoming contaminated with pathogens and subsequently transmitting infections to previously healthy birds.

Fig. 1. Example of a poultry transport vehicle. (W. Kozdruń)

In the present study, samples from 65 flocks of geese and 42 flocks of clinically healthy ducks were analyzed for the presence of viral genetic material, including GPV, GHPV, GoCV, DHV, ARV, DVE, and MDPV, using specific oligonucleotide primers targeting conserved viral protein sequences.

Table 4. Flocks of clinically healthy geese tested in 2018-2025 [author's research]

Year	Number of goose flocks examined	Number of infected flocks GPV	Number of infected flocks GHPV	Number of infected flocks GoCV	Number of infected flocks ARV
2018	21	3	7	12	8
2019	8	3	1	8	6
2020	13	3	7	7	4
2021	3	1	0	2	1
2022	6	0	2	4	3
2023	3	0	2	3	3
2024	23	0	8	15	7
2025	9	0	1	4	2
Total	**86**	**10** (**11,62%**)	**28** (**32,55%**)	**55** (**63,95%**)	**34** (**39,53%**)

Table 5. Flocks of clinically healthy ducks tested in 2018-2025 [author's research]

Year	Number of ducks flocks examined	Number of infected flocks GPV	Number of infected flocks GHPV	Number of infected flocks GoCV	Number of infected flocks ARV	Number of infected flocks DVE	Number of infected flocks DHV	Number of infected flocks MDPV
2018	15	1	7	9	6	1	2	1
2019	9	3	1	6	3	1	2	3
2020	3	0	0	1	2	0	0	0
2021	2	0	2	2	1	0	1	0
2022	2	0	0	0	1	0	1	0
2023	0	0	0	0	0	0	0	0
2024	2	0	1	1	1	0	0	0
2025	7	0	2	4	2	0	1	0
Total	**40**	**4** (**10%**)	**13** (**32.5%**)	**23** (**57,5%**)	**16** (**40%**)	**2** (**5%**)	**7** (**17.5%**)	**4** (**10%**)

Overall, GPV genetic material was detected in 10 goose flocks (11.62%) and 4 duck flocks (10%). GHPV was detected in 28 goose flocks (32.55%) and 13 duck flocks (32.5%). GoCV was identified in 55 goose flocks (63.95%) and 23 duck flocks (57.5%). ARV genetic material was present in 34 goose flocks (39.53%) and 16 duck flocks (40%). In duck samples, DHV was detected in 7 samples (17.5%), DVE in 2 samples (5%), and MDPV in 4 samples (10%).

Sample collection included tracheal swabs from 49 goose flocks, cloacal swabs from one goose flock, and internal organ samples from 36 goose flocks. Yearly details were as follows: in 2018, tracheal swabs from 19 flocks, cloacal swabs from 1 flock, and internal organs from 11 flocks; 2019, tracheal swabs from 4 flocks and internal organs from 4 flocks; 2020, tracheal swabs from 7 flocks and internal organs from 6 flocks; 2021, internal organs from 3 flocks; 2022, tracheal swabs from 2 flocks and internal organs from 4 flocks; 2023, tracheal swabs from 1 flock and internal organs from 2 flocks; 2024, tracheal swabs from 19 flocks and internal organs from 4 flocks; 2025, tracheal swabs from 7 flocks and internal organs from 2 flocks.

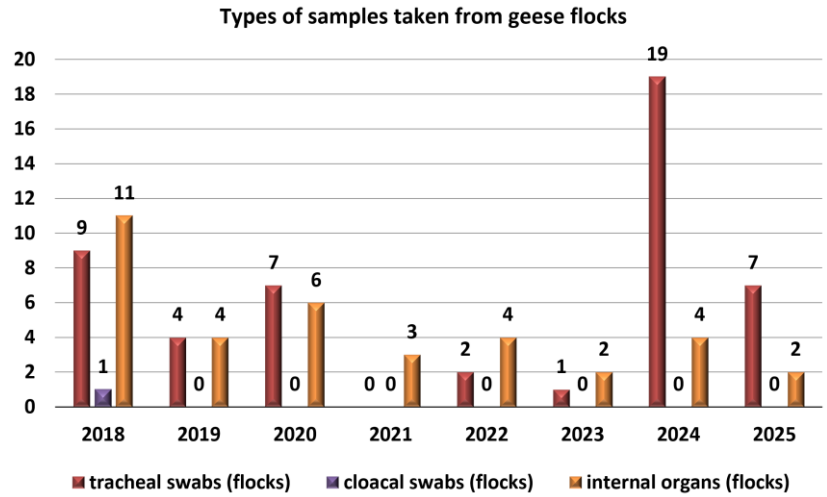


Fig. 2. Types of samples collected from clinically healthy goose flocks, 2018–2025. [14]

For ducks, tracheal swabs and internal organs were collected from 20 flocks each; cloacal swabs were not collected. Yearly distribution: 2018, tracheal swabs from 7 flocks and internal organs from 8 flocks; 2019, tracheal swabs from 4 flocks and internal organs from 5 flocks; 2020, internal organs from 3 flocks; 2021, internal organs from 2 flocks; 2022, internal organs from 2 flocks; 2023, no samples collected; 2024, internal organs from 4 flocks; 2025, tracheal swabs from 7 flocks.

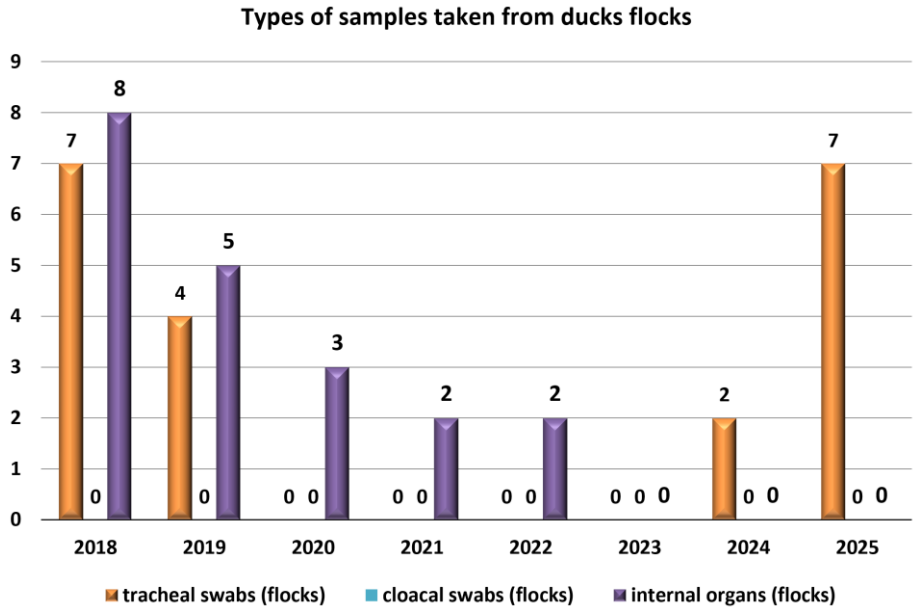


Fig. 3. Types of samples collected from clinically healthy duck flocks, 2018–2025. [20]

Tables 6 and 7 summarize the number of positive and negative samples for each virus from tracheal swabs, cloacal swabs, and internal organs of geese and ducks between 2018 and 2025. Positive samples (P) indicate the presence of viral genetic material, while negative samples (N) indicate absence.

Table 6. A compilation of samples positive and negative for a given virus from swabs from the trachea, cloaca and internal organs of clinically healthy goose in the years 2018-2025 [14]

Virus	Year / Samples	2018	2019	2020	2021	2022	2023	2024	2025
GPV	Tracheal swabs	1P*/8N**	2P/2N	0P/5N	-	0P/2N	0P/1N	0P/19N	0P/7N
	Cloacal swabs	0P/1N	-	-	-	-	-	-	-
	Internal organs	2P/9N	1P/2N	3P/3N	1P/2N	0P/4N	0P/2N	0P/4N	0P/2N
GHPV	Tracheal swabs	3P/6N	0P/4N	3P/4N	-	1P/1N	1P/0N	5P/14N	0P/7N
	Cloacal swabs	1P/1N	-	-	-	-	-	-	-
	Internal organs	3P/8N	1P/3N	4P/2N	0P/3N	1P/3N	1P/1N	3P/1N	1P/1N
GoCV	Tracheal swabs	6P/3N	4P/0N	4P/3N	-	2P/0N	1P/0N	13P/2N	3P/1N
	Cloacal swabs	1P/1N	-	-	-	-	-	-	-
	Internal organs	5P/6N	4P/0N	3P/3N	2P/1N	2P/2N	2P/0N	2P/2N	1P/1N
ARV	Tracheal swabs	3P/6N	4P/0N	1P/6N	-	2P/0N	1P/0N	5P/15N	1P/1N
	Cloacal swabs	0P/1N	-	-	-	-	-	-	-
	Internal organs	5P/6N	2P/2N	3P/3N	1P/3N	1P/3N	2P/0N	2P/2N	1P/1N

Table 7. A compilation of samples positive and negative for a given virus from swabs from the trachea, cloaca and internal organs of clinically healthy ducks in the years 2018-2025 [15]

Virus	Year / Samples	2018	2019	2020	2021	2022	2023	2024	2025
GPV	Tracheal swabs	0P*/7N**	1P/3N	-	-	-	-	0P/2N	0P/7N
	Internal organs	1P/7N	2P/3N	0P/3N	0P/2N	0P/2N	-	-	-
MDPV	Tracheal swabs	0P/7N	1P/3N	-	-	-	-	0P/2N	0P/7N
	Internal organs	1P/7N	2P/3N	0P/3N	0P/2N	0P/2N	-	-	-
GHPV	Tracheal swabs	3P/4N	0P/4N	-	-	-	-	1P/1N	2P/5N
	Internal organs	4P/4N	1P/4N	0P/3N	2P/2N	2P/2N	-	-	-
GoCV	Tracheal swabs	4P/3N	3P/1N	-	-	-	-	1P/1N	4P/3N
	Internal organs	5P/3N	3P/2N	1P/2N	2P/0N	0P/2N	-	-	-
ARV	Tracheal swabs	3P/4N	2P/2N	-	-	-	-	1P/1N	2P/5N
	Internal organs	3P/5N	1P/4N	2P/3N	1P/1N	1P/1N	-	-	-
DHV	Tracheal swabs	1P/6N	0P/4N	-	-	-	-	0P/2N	1P/6N
	Internal organs	1P/7N	2P/3N	0P/3N	1P/1N	1P/1N	-	-	-
DVE	Tracheal swabs	1P/6N	0P/4N	-	-	-	-	0P/2N	0P/7N
	Internal organs	1P/7N	1P/4N	0P/3N	0P/2N	0P/2N	-	-	-

*P - positive sample (sample in which the presence of genetic material of the examined virus was detected)

**N - negative sample (sample in which no genetic material of the examined virus was detected)

Across all years, GPV was detected sporadically in both species, with the highest frequency in geese in 2018–2020. MDPV was only observed in 2018–2019 duck flocks. GHPV was widespread, particularly in geese, with several positive detections in duck flocks in 2018, 2019, 2021, 2024, and 2025. GoCV was consistently detected in both species, while ARV was identified in multiple goose and duck flocks, with similar frequency patterns. DHV and DVE were detected only in ducks and in limited cases.

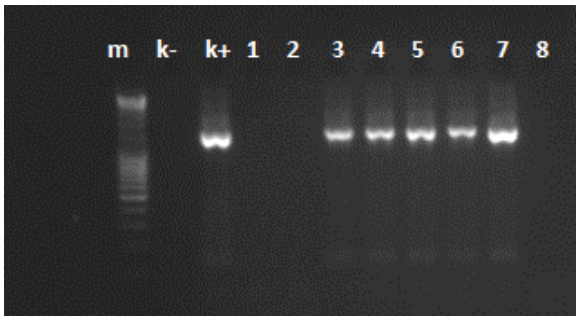


Fig. 4. Agarose gel electrophoresis of GPV detection. (**M - molecular marker, K - negative control, K+ - positive control; lanes 1-8 - VP3 amplicons**)

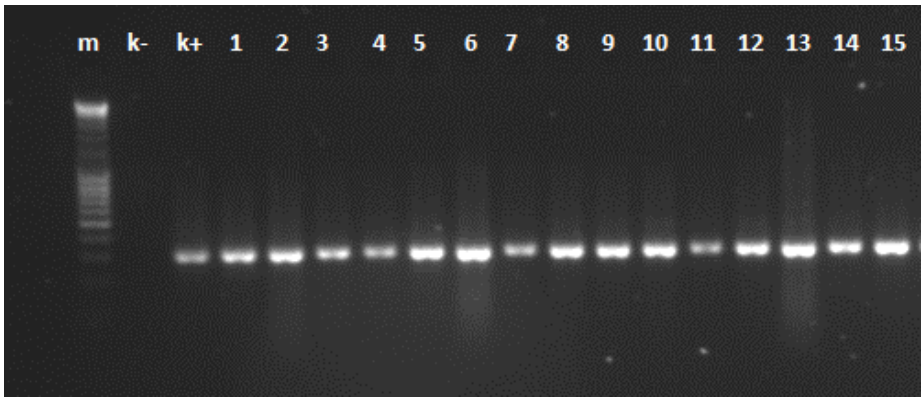
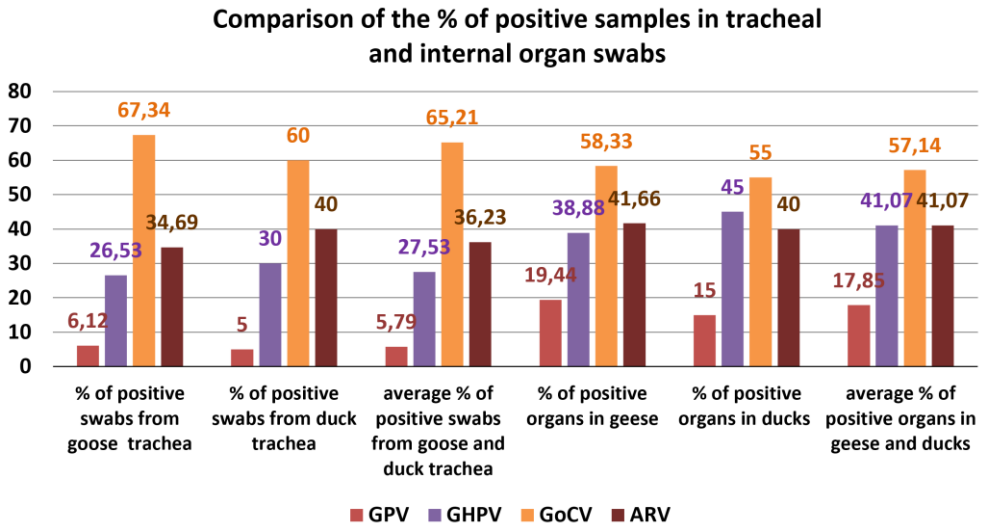


Fig. 5. Agarose gel electrophoresis of GoCV detection. (**M - molecular marker, K - negative control, K+ - positive control; lanes 1-15 - ORFC1 amplicons**) [author's research]



Summary of the percentage of positive samples by virus and species in tracheal and internal organ swabs. [author's research]

Tracheal swabs were collected from 49 goose and 20 duck flocks, and internal organs from 36 goose and 20 duck flocks. The percentage of positive tracheal swabs in geese was: GPV 6.12%, GHPV 26.53%, GoCV 66.34%, ARV 34.69%; in ducks: GPV 5%, GHPV 30%, GoCV 60%, ARV 40%. When considering the total number of tracheal swabs across both species, positive detection rates were GPV 5.79%, GHPV 27.53%, GoCV 65.21%, and ARV 36.23%.

For internal organs, detection in geese was: GPV 19.44%, GHPV 38.88%, GoCV 58.33%, ARV 41.66%; in ducks: GPV 15%, GHPV 45%, GoCV 55%, ARV 40%. Across both species, positive internal organ samples were GPV 17.85%, GHPV 41.07%, GoCV 57.14%, and ARV 41.07%. These results indicate that both tracheal swabs and internal organ samples provide comparable efficiency in detecting viral genetic material, supporting their suitability for molecular monitoring of waterfowl viruses.

DISCUSSION

Transport of waterfowl represents a critical pathway for viral dissemination, yet its role as a reservoir for pathogens in clinically healthy birds remains insufficiently understood. Despite the repeated use of transport vehicles and crates within and between farms, empirical evidence quantifying their contribution to viral spread is limited. Moreover, it is unclear whether tracheal swabs provide comparable diagnostic value to internal organs for detecting major waterfowl viruses under field conditions.

In this context, we hypothesized that clinically healthy geese and ducks transported under commercial conditions carry detectable viral genetic material, and that tracheal swabs are as effective as internal organ samples for identifying the presence of Goose Parvovirus (GPV), Goose Haemorrhagic Polyomavirus (GHPV), Goose Circovirus (GoCV), Avian Orthoreoviruses (ARV), Duck Hepatitis Virus (DHV), and Duck Viral Enteritis (DVE). We further hypothesized that multiple viruses may co-circulate within flocks, increasing the potential for environmental dissemination during transport.

Standard cleaning and disinfection of vehicles and transport crates are often insufficient, and repeated use of crates within a single day facilitates cross-farm transmission of infectious agents. Even clinically healthy birds can shed viruses, particularly under the stress of transportation, which enhances pathogen dissemination via feces and secretions.

GPV has been reported across Europe, the Americas, and Asia since 1956 and is highly pathogenic to geese and ducks, resulting in considerable economic losses. It exhibits remarkable environmental stability, persisting on inadequately disinfected surfaces and in farm facilities, thereby infecting successive flocks. In our study, GPV genetic material was detected in both tracheal swabs and internal organs of clinically healthy waterfowl, despite a relatively low prevalence in Poland (12.04% in geese, 10% in ducks). Road transport emerges as a significant horizontal route for GPV spread, as vehicles may serve as long-term reservoirs of the virus. Furthermore, environmental persistence allows potential infection of free-living birds, which can transmit the virus over long distances.

GHPV causes acute and often fatal Hemorrhagic Nephritis and Enteritis of Geese (HNEG) and can also infect Muscovy and mule ducks. While it can be transmitted both vertically and horizontally, horizontal transmission is the primary driver of farm-level outbreaks. Persistent detection of identical viral strains across different herds indicates environmental persistence and highlights the role of transport vehicles in moving viruses between locations. Ducks may act as subclinical reservoirs, shedding high levels of GHPV without exhibiting clinical signs. In our study, GHPV prevalence reached 33.7% in geese and 32.5% in ducks, emphasizing that asymptomatic birds can disseminate the virus during transport and contaminate the environment.

GoCV, although exhibiting low direct lethality and often subclinical infections, represents a significant epidemiological concern. Horizontal transmission via feces and feather dust is predominant, while vertical transmission remains a possibility. In our study, GoCV genetic material was detected in 63.95% of geese and 57.5% of ducks. Tracheal swabs proved as effective as internal organ samples in detecting the virus. Stressful conditions during transport may enhance viral shedding, increasing the risk of infecting other flocks and contaminating transport vehicles.

ARVs, associated with viral arthritis and tenosynovitis in chickens and turkeys, are increasingly detected in other domestic and wild avian species. ARVs transmit horizontally and vertically, with fecal-oral and respiratory routes playing major roles. In our study, 40% of clinically healthy geese and duck flocks carried ARV genetic material. Tracheal swabs confirmed the importance of the respiratory route in virus shedding. Transport conditions, which bring birds into close contact with each other and with contaminated equipment, can facilitate transmission to free-living birds, potentially turning them into reservoirs.

DHV and DVE can also be transmitted during transport. DHV was detected in 17.5% of flocks and DVE in 5%. Both viruses can persist throughout the bird's life and be shed in secretions. In combination with insufficient vehicle disinfection and inadequate biosecurity measures, transport creates an environment conducive to viral spread.

Overall, transport amplifies viral dissemination through several mechanisms:

- Contaminated vehicles moving between farms and slaughterhouses,
- Transport crates that retain pathogens despite routine cleaning,
- Direct contact among live birds,
- Indirect transmission via contaminated equipment, footwear, or clothing of handlers.

Previous studies have shown that the level of disinfection of cages and vehicles is low, even for microbial contaminants. Given the environmental resilience of these viruses and their shedding by clinically healthy birds, the risk of spreading infection during transport remains high. These findings underscore the necessity of enhanced biosecurity, optimized vehicle disinfection protocols, and monitoring of asymptomatic carriers to mitigate the role of transportation in viral dissemination among waterfowl.

TRANSPORT DROBIU JAKO CZYNNIK KRYTYCZNY W ROZPRZESTRZENIANIU SIĘ INFEKCJI WIRUSOWYCH U PTACTWA WODNEGO

Transport drogowy drobiu odgrywa kluczową rolę w rozprzestrzenianiu się chorób wirusowych wśród populacji ptactwa wodnego. Przemieszczanie pozornie zdrowych ptaków, w połączeniu z potencjalnym skażeniem pojazdów, skrzynek i sprzętu, stanowi istotne ryzyko epidemiologiczne. Niniejsze badanie podkreśla obecność materiału genetycznego wirusa u klinicznie zdrowych gęsi i kaczek transportowanych do rzeźni, podkreślając rolę transportu jako głównego wektora transmisji poziomej. Wykryte patogeny obejmowały parwowirus gęsi (GPV), poliomawirus krwotoczny gęsi (GHPV), cirkowirus gęsi (GoCV), reowirusy ptasie (ARV), wirus zapalenia wątroby kaczek (DHV) oraz wirus wirusowego zapalenia jelit kaczek (DVE). Dane potwierdzają, że transport nasila wydalanie wirusa poprzez zmiany fizjologiczne związane ze stresem u ptaków, a niewystarczająca dezynfekcja pojazdów zwiększa ryzyko zakażenia krzyżowego stada. Nieprzestrzeganie zasad bezpieczeństwa biologicznego w transporcie może przyczyniać się nie tylko do lokalnych ognisk chorób, ale także do rozprzestrzeniania się transregionalnego i transkontynentalnego.

Słowa kluczowe: transport, drób, ptactwo wodne, zakażenia wirusowe, epidemiologia, bezpieczeństwo biologiczne.

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