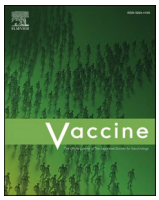
Vaccine 72 (2026) 128048

Contents lists available at ScienceDirect

Vaccine

journal homepage: www.elsevier.com/locate/vaccine



Short communication

B cells play an important role in HVT vaccine-mediated protection against Marek's disease virus

Mohammad A. Sabsabi [a,b], Ahmed Kheimar [a,b,c], Dominik von La Roche [d], Sonja Härtle [d], Dusan Kunec [a], Yulin Cong [a,b], Lisa Kossak [a,b], Theresa von Heyl [e], Benjamin Schusser [e,f,*], Benedikt B. Kaufer [a,b,*]

[a] Institut für Virologie, Freie Universität Berlin, Robert von Ostertag-Straße 7-13, 14163, Berlin, Germany
[b] Veterinary Centre for Resistance Research (TZR), Freie Universität Berlin, 14163 Berlin, Germany
[c] Department of Poultry Diseases, Faculty of Veterinary Medicine, Sohag University, 82524, Sohag, Egypt
[d] Department of Veterinary Sciences, Ludwig-Maximilians-Universität München, 82152 Planegg, Germany
[e] Reproductive Biotechnology, TUM School of Life Sciences, Technische Universität München, 85354 Freising, Germany
[f] Center for Infection Prevention (ZIP), Technische Universität München, 85354 Freising, Germany

ARTICLE INFO

Keywords:
Marek's disease virus
Turkey herpesvirus
B cells
Humoral immunity
Oncogenesis
Lymphoma

ABSTRACT

Marek's disease virus (MDV) is an alphaherpesvirus that infects chickens, causing immunosuppression, neurological symptoms, and fatal lymphoma. Vaccines are used to protect billions of chickens, but remain poorly understood. To investigate the role of B-cells in vaccine protection, we vaccinated B-cell knockout ($JH^{-/-}$) chickens with the commercial HVT vaccine and challenged them with very virulent MDV. Vaccinated $JH^{-/-}$ chickens showed significantly increased disease incidence and neurological symptoms compared to wild-type siblings, indicating that B-cells contribute to protection against clinical disease. Tumor incidence remained low and comparable to wild-type, suggesting that B-cells are dispensable for preventing tumors. Aside from the absence of B-cells, no major changes in T-cell subsets were detected. Viral genome levels were comparable in the blood and spleen, but elevated in skin and dust in $JH^{-/-}$ birds early on. These findings reveal that B cells are critical for full HVT vaccine protection and limiting early virus shedding.

1. Introduction

Marek's disease virus (MDV) is a highly oncogenic alphaherpesvirus that infects chickens and causes a high mortality in unvaccinated chickens [1]. MDV is a strictly cell-associated virus that targets various immune cells and induces severe clinical symptoms, including immunosuppression, neurological disorders and T-cell lymphomas [2]. The virus causes substantial economic losses in poultry production worldwide [2–4]. Therefore, billions of chickens are vaccinated every year. The turkey herpesvirus (HVT, Mardivirus meleagridalpha 1, MeAHV1) was the first widely used vaccine to protect against MDV [3]. In addition to MDV protection, HVT is also extensively used as a vector vaccine encoding antigens for other pathogens such as infectious bursal disease virus, Newcastle disease virus, avian influenza virus, and infectious laryngotracheitis virus [4,5]. Although HVT does not prevent MDV infection or transmission, it significantly reduces clinical symptoms and improves survival rates [1]. The HVT vaccine induces both innate and adaptive immune responses, and protects against classical disease manifestations, including neurological disorders and MDV-induced lymphomas [6,7].

MDV enters the host through the respiratory tract and subsequently spreads to lymphoid organs where it mainly infects B and T cells. The virus establishes latency primarily in CD4+ αβ T cells [7], which it can transform leading to fatal lymphomas in the viscera organs [7]. Infected lymphocytes also transport the virus to feather follicle epithelial (FFE) cells in the skin, enabling shedding and transmission to other birds [8].

Given the tropism of the virus for immune cells, understanding the role of specific immune subsets in MDV pathogenesis and immunity is critical. B cells were long thought to contribute to pathogenesis by amplifying the virus and transferring it to T cells [9]. However, recent work using B cell knockout chickens revealed that B cells are dispensable for viral replication, shedding and tumor formation [10,11], highlighting that they have no or only a very limited role in MDV pathogenesis. In the context of vaccination, B cells mediate humoral immunity

* Corresponding authors at: Reproductive Biotechnology, TUM School of Life Sciences, Technische Universität München, 85354 Freising, Germany.
E-mail addresses: benjamin.schusser@tum.de (B. Schusser), b.kaufer@fu-berlin.de (B.B. Kaufer).

https://doi.org/10.1016/j.vaccine.2025.128048
Received 17 September 2025; Received in revised form 25 September 2025; Accepted 25 November 2025
Available online 3 December 2025

by producing antibodies that not only neutralize virus particles but also opsonize targets, engage antibody-dependent cellular cytotoxicity (ADCC), and activate complement [7]. Maternal antibodies can only delay or mitigate clinical symptoms but do not prevent infection with MDV [1,6]. Antibody-mediated protection has been considered limited, as MDV spreads primarily by direct cell-to-cell contact [12]. Consequently, the protective role of B cells in vaccine-induced immunity still remains poorly understood.

To address this knowledge gap, we used genetically modified B cells knockout chicken line, which represent the optimal model to investigate the role of B cells in vaccine-mediated protection against MDV [11]. These B-cell knockout chickens have a deletion of the immunoglobulin heavy-chain joining (JH) region, abolishing Ig heavy-chain recombination and thus the development of mature peripheral B cells in the bursa of Fabricius as published previously [11]. We vaccinated B cell knockout chickens and their wild-type siblings with HVT and challenged them with very virulent MDV five days post-vaccination. Unexpectedly, the absence of B cells resulted in more severe clinical symptoms, although protection against tumors remained unaffected. Our data revealed an unappreciated role of B cells in the HVT vaccine-mediated protection against MDV.

2. Methods

2.1. Ethics statement

All animal experiments were performed according to the international and national guidelines for the humane use of animals in research. The permission to conduct these experiments was granted by the Landesamt für Gesundheit und Soziales (LAGeSo) in Berlin, Germany (approval number: G 0050/23).

2.2. Cells and viruses

Chicken embryo cells (CECs) were generated from specific-pathogen-free (SPF) Valo embryos (ValoBioMedia) as described previously [13]. The HVT vaccine FC126 and the very virulent MDV-1 (RB-1B strain) were propagated on CECs, stocks were frozen and titrated prior to their use [14]. The cells were cultured in MEM (Pan-Biotech, Aidenbach, Germany) supplemented with 1–10 % FCS and 1 % penicillin/streptomycin in a humidified incubator at 37 °C under a 5 % CO2 atmosphere.

2.3. Animal experiments

To investigate the role of B cells in vaccine-induced protection against MDV-1, we used B cell-knockout chickens that are lacking mature and peripheral B cells and were thoroughly characterized previously [10,11]. Upon hatching, the chickens were genotyped by PCR as described previously (Table 1) [11]. Wild-type (WT; $n = 24$) and B cell knockout ($JH^{-/-}$; $n = 21$) animals were vaccinated subcutaneously with 2000 PFU of the HVT vaccine. Five days post-vaccination, the chickens were infected subcutaneously with 2000 PFU of the RB-1B strain. To ensure that the animals obtained the correct dose, the vaccine and challenging virus inoculum was backtitrated. During the experiment, the two groups were housed in separate isolation rooms with free access to food and water for 90 days. During the first weeks of life, the chicks in both groups had a mild diarrhea and we detected *Clostridium perfringens* in the feces. The symptoms resolved shortly afterward, and no related abnormalities were found at necropsy. To assess the RB-1B replication in the animals, peripheral blood samples were collected at 7, 10, 14, 21, and 28 days post-vaccination (dpv). Feather and dust samples were taken at 21, 28, 35, and 42 dpv to measure viral genome copies in the skin of infected animals and MDV-1 shedding.

Throughout the experiment, chickens were observed twice daily for MDV-clinical symptoms such as ataxia, paralysis of the legs, wings, or neck, somnolence and torticollis. Chickens that developed severe symptoms or at final termination were humanely euthanized, checked for gross tumors and spleen samples taken to measure the viral load.

Table 1
PCR and qPCR primers and probes used in this study.

Construct region	Direction[a]	Primer or probe[b] sequence (5′ → 3′)
WT (PCR)	For	ATGGGGCCACGGGACCGAA
	Rev	GCCCAAAATGGCCCCAAAAC
$JH^{-/-}$ (PCR)	For	AGTGACAACGTCGAGCACAGCT
	Rev	GCCCAAAATGGCCCCAAAAC
HVT SORF1 (qPCR)	For	GGCAGACACCGCGTTGTAT
	Rev	TGTCCACGCTCGAGACTATCC
	Probe	AACCCGGGCTTGTGGACGTCTTC
RB-1B ICP4 (qPCR)	For	CGTGTTTTCCGGCATGTG
	Rev	TCCCATACCAATCCTCATCCA
	Probe	FAM-CCCCCACCAGGTGCAGGCA-TAM
Chicken iNOS (qPCR)	For	GAGTGGTTTAAGGAGTTGGATCTGA
	Rev	TTCCAGACCTCCCACCTCAA
	Probe	FAM-CTCTGCCTGCTGTTGCCAACATGC-TAM

[a] For, forward primer; Rev., reverse primer.
[b] FAM, 6-carboxyfluorescein; TAM, TAMRA.

3. Quantification of the virus genomic

DNA was extracted from blood, feathers, dust, and organs using standard protocols and commercial kits as described previously [15]. Successful vaccination was confirmed by detecting the HVT vaccine (SORF1 gene) in the blood at 7 dpv using PCR (Table 1). MDV-1 genome copies were quantified by qPCR detecting the ICP4 gene (Table 1). Virus genome copies were normalized against the chicken-induced nitric oxide synthase (iNOS) gene as described previously [8].

3.1. Flow cytometry

Absolute cell counts were performed on the key immune cell populations, including B cells, CD8- αβ, CD8+ αβ and γδ T cells and thrombocytes in the blood as described previously [16]. The samples were assessed with a FACSCanto II (Becton Dickinson, Heidelberg, Germany), and data analyzed using the FACSDiva (Becton Dickinson, Heidelberg, Germany) and FlowJo_v10.10.0 (FlowJo LLC, Oregon, USA) software.

3.2. Statistical analysis

Statistical analyses were performed using Graph-Pad Prism v10 (San Diego, CA, USA). Details of the used statistical tests are provided in the respective figure legends.

4. Result

Disease incidence is increased in vaccinated chickens in the absence of B cells.

To investigate the role of B cells in the vaccine protection against MDV-1, we performed vaccine/challenge experiments using genetically modified B cell knockout chickens that lack mature and peripheral B cells. In these experiments, successful vaccination was confirmed by qPCR, with all animals tested (8/8 per group) being positive for HVT in the blood at 7 dpv. Upon challenge, significantly more B cell knockout chickens developed disease over the course of the experiment (Fig. 1A). The disease incidence reached 71 % in the absence of B cells, while only 17 % of their wild type siblings developed disease. Notably, classical neurological symptoms including paralysis, torticollis, and somnolence were significantly increased in B cell knockout chickens compared to their WT siblings (Fig. 1B). Intriguingly, the tumor incidence was very low in both B cell knockout and WT chickens (Fig. 1C). Consistently, the dissemination of tumors within animals was also not altered (Fig. 1D).

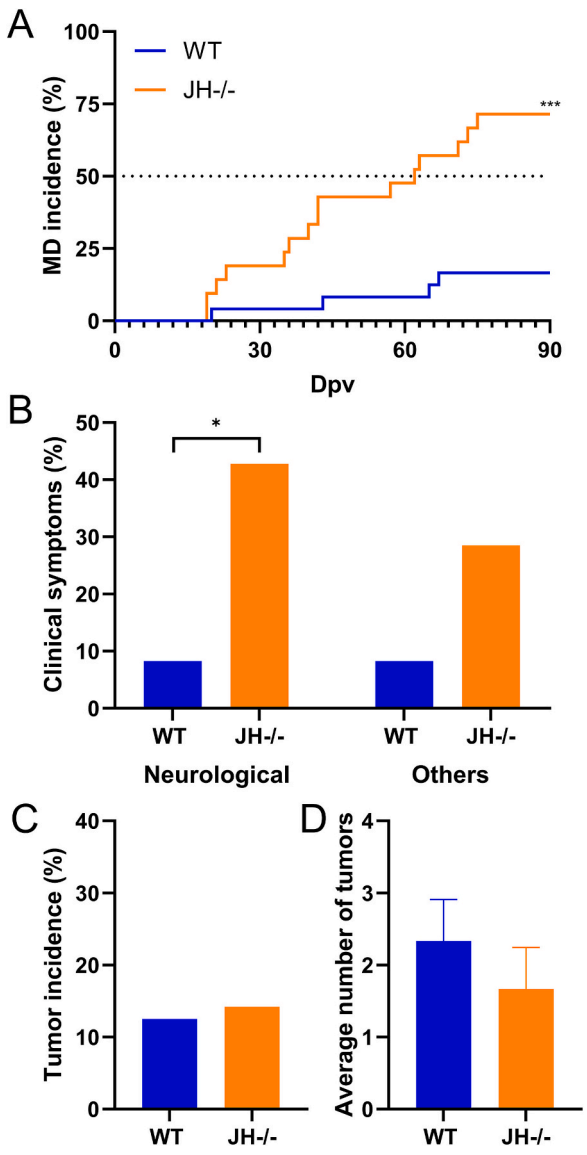


Fig. 1. B cell knockout chickens displayed a rapid disease onset in a vaccine/challenge.

(A) Marek's disease (MD) incidence in HVT vaccinated and RB-1B challenged WT ($n = 24$) and $JH^{-/-}$ ($n = 21$) chickens. The percentage of chickens with clear clinical signs of Marek's disease, including paralysis, torticollis, somnolence, and tumors is shown throughout the experiment (Mantel-Cox analysis; $***p < 0.001$). (B) The percentage of chickens with classical clinical symptoms of MDV are shown. They were categorized into two groups: neurological symptoms (ataxia, paralysis, dyspnea) and other symptoms (lethargy, weight loss, sudden death). Disease severity was significantly higher in $JH^{-/-}$ chickens compared to WT controls (Fisher's exact test; $*p \leq 0.05$). (C) Tumor incidence is shown as the percentage of chickens with gross tumors ($P > 0.05$, Fisher's exact test). (D) The average number of visceral organs with gross tumors is shown for tumor-bearing animal ($P > 0.05$, Fisher's exact test).

Our data revealed that B cells play an important role in vaccine-mediated protection against certain classical symptoms (e.g. neurological symptoms) but are not required to prevent tumorigenesis.

Immune cell profiles in the blood.

Next, we determined if the absence of B cells affected other immune cell subsets in our vaccine/challenge experiment at 14 and 28 dpv. As expected, all B cell knockout animals completely lacked B cells (Fig. 2A), while normal levels were detected in WT animals. Only minor changes were observed in CD8-, CD8+ αβ T cells and γδ T cells that were not significantly different compared to their WT siblings (Fig. 2B-D). These data highlights that the absence of B cells does not significantly affect these immune cell subsets, which could have influenced the observed phenotype.

Effect on the virus load in the absence of B cells in vaccinated chickens.

To investigate if the absence of B cells influences MDV-1 replication in vaccinated chickens, we quantified the virus load in the blood, spleen, feathers and dust. qPCR revealed that the viral load was comparable in the peripheral blood of the animals (Fig. 3A). In addition, the virus genome copies were comparable in the spleen collected from all animals post mortem (Fig. 3B). These results indicate that mature and peripheral B cells do not significantly restrict MDV-1 replication in the blood and spleens of vaccinated chickens. Next, we investigated if transport of the virus to the skin and shedding is affected by quantifying virus load in feather shafts and dust. Intriguingly, MDV-1 load in the skin was up to 20-fold higher in vaccinated chickens in the absence of B cells compared to WT chickens until 35 dpv (Fig. 3C). Consistently, higher MDV-1 genome copies were detected in the dust of chickens that lack B cells at 21, 28, and 35 dpv (Fig. 3D). These findings indicate that mature and peripheral B lymphocytes do not inhibit MDV-1 replication in the blood and spleen of vaccinated chickens, but they delay/reduce virus replication in the skin and shedding at early time points.

5. Discussion

Vaccines are crucial for protecting chickens against a deadly MDV infection; however, the immune responses to the vaccines remain poorly understood. Several vaccines are commercially used against MDV. The first widely used vaccine was HVT, which is also commonly applied as a vaccine vector to protect against MDV and several other pathogens. We therefore chose HVT, even though it does not provide a perfect protection against very virulent MDV strains [17]. To better understand the importance of B cells in HVT vaccine protection against MDV infection, we used genetically modified chickens lacking B cells, representing an optimal model to assess the contribution of these cells to vaccine-mediated immunity. The B cell knockout chickens and their WT siblings were vaccinated with the widely used HVT FC126 vaccine strain, then challenged with the very virulent RB-1B strain. Strikingly, our experiment revealed significant differences in the disease incidence between the two groups (Fig. 1A). Most B cell knockout chickens developed clinical symptoms with a high frequency of paralysis and ataxia (Fig. 1B). These symptoms are common in unvaccinated chickens, in which MDV-1 causes inflammation and nerve damage, leading to these severe neurological signs [18]. The observed increase in neurological symptoms indicates that B cells and/or antibodies can inhibit the dissemination of the virus into the central nervous system or reduce the local inflammation. HVT vaccination has been previously shown to induce MDV specific antibodies, which can reduce the frequency of clinical signs but cannot prevent infection [6,7]. In this study, we used B cell knockout chickens that lack mature and peripheral B cells (Fig. 2A) and do not produce antibodies [10,11]. The high disease incidence observed in the absence of B cells suggests that the lack of B cells and/or antibody production compromise vaccine efficacy, leading to more severe clinical outcome.

In contrast, the tumor incidence between both groups was comparable, highlighting that the cellular immunity induced by vaccination efficiently prevented tumorigenesis. Consistently, tumor dissemination was also not affected. This highlights that the protection against tumors is (mostly) dependent on the cell mediated immunity induced by HVT.

Early studies in bursectomized chickens showed that B-cell depletion abrogated HVT-mediated protection [19], whereas vaccination with an attenuated MDV-1 strain still provided protection [20], supporting potential differences between the vaccine serotypes. A recent study assessed the role of B cells in the vaccine protection provided by CVI988 [21], an attenuated MDV strain that provides a better protection against very virulent strains than HVT [7]. They surgically removed the bursa of

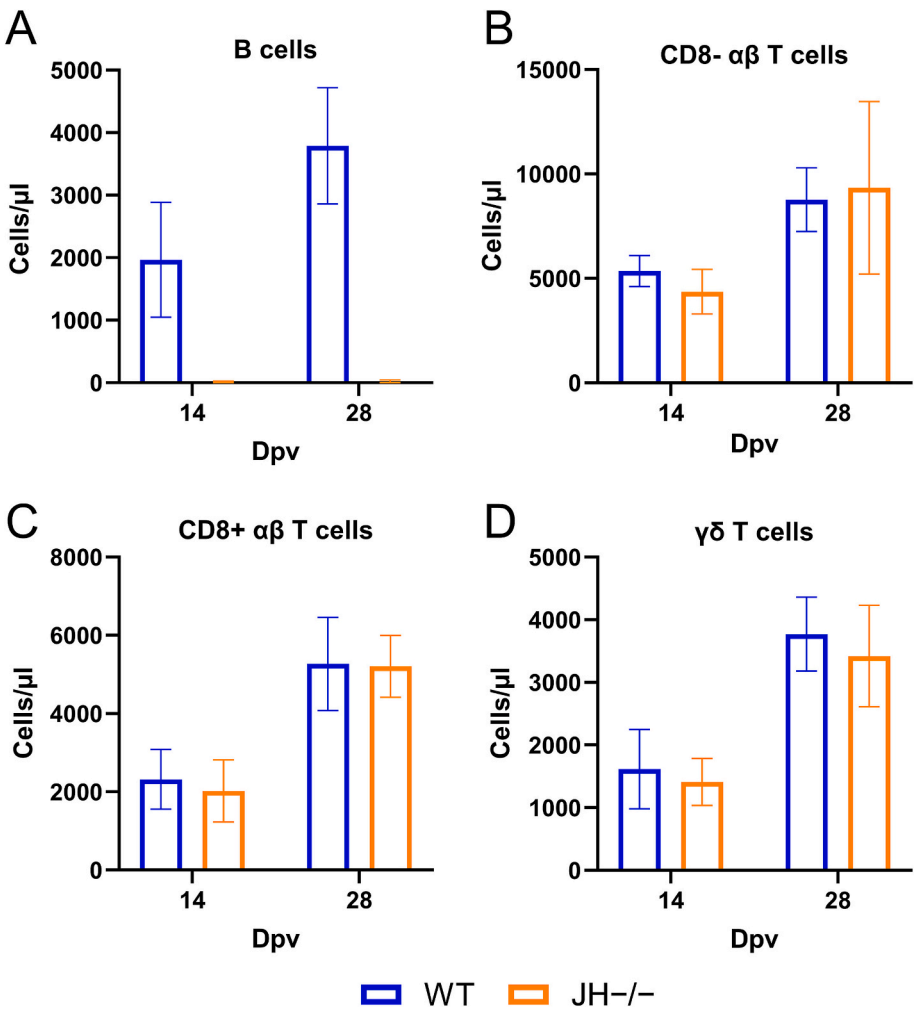


Fig. 2. Immune cell profiles in the blood.
Fluorescence-activated cell sorting analysis of the immune cells of the peripheral blood at 14 and 28 dpv, B cells (A), CD8- αβ T cells (B), CD8+ αβ T cells (C), and γδ T cells (D) count in the blood of $JH^{-/-}$ ($n = 7$) and WT chickens ($n = 8$) ($p > 0.05$, Mann–Whitney U test).

Fabricius and performed vaccine/challenge experiments. In case of CVI vaccination, these bursectomized chickens were still protected against very virulent MDV. This could be either i) due to remaining B cells upon the bursectomy or alternatively ii) indicate that there are differences in the protection provided by CVI988 and HVT. Importantly, CVI988 is very closely related to very virulent MDV strains (in contrast to HVT). Therefore, cellular responses alone are apparently sufficient to provide a complete protection against very virulent MDV.

To further investigate the immune response, we analyzed the absolute number of immune cell in the blood and found no significant changes in the absence of B cells. This indicates that T-cell-mediated immunity remained intact and is not responsible for the increased disease incidence observed in the absence of B cells [22].

MDV-1 load in the blood and spleen were comparable between the B cell knockout chickens and their WT siblings (Fig. 3A, B). These findings are consistent with previous data showing that MDV can efficiently replicate in T cells in the absence of B cells [7,10]. Intriguingly, virus delivery to and/or replication in the FFE was increased in the absence of B cells compared to their WT siblings (Fig. 3C). This increased viral load was also observed in the dust (Fig. 3D). As HVT vaccination has been known to reduce MDV-1 shedding [23], B cells and/or antibodies are potentially involved in reducing viral shedding into the environment. In contrast, Heidari, Zhang [21] reported no or very low MDV-1 genome copies in skin tissues of bursectomized chickens vaccinated with CVI988 upon challenge.

In conclusion, our study revealed that the disease incidence was significantly increased in the absence of B cells, while protection against tumor formation remained intact. Even though replication was comparable in the blood and spleen, B cells and/or antibodies can restrict replication in other organs as evident by the increased viral load in the FFE and the dust. Overall, our study provides the first clear evidence that B cells and/or antibodies play an important role in the vaccine protection against MDV-1 provided by the HVT vaccine, which is used to protect billions of chickens worldwide.

CRediT authorship contribution statement

Mohammad A. Sabsabi: Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Ahmed Kheimar:** Writing – review & editing, Methodology, Investigation. **Dominik von La Roche:** Writing – review & editing, Methodology, Investigation. **Sonja Härtle:** Writing – review & editing, Resources, Methodology, Funding acquisition. **Dusan Kunec:** Writing – review & editing, Resources. **Yulin Cong:** Writing – review & editing, Investigation. **Lisa Kossak:** Writing – review & editing, Investigation. **Theresa von Heyl:** Writing – review & editing, Resources. **Benjamin Schusser:** Writing – review & editing, Resources, Funding acquisition, Conceptualization. **Benedikt B. Kaufer:** Writing – review & editing, Writing – original draft, Supervision, Resources, Project administration, Funding acquisition, Conceptualization.

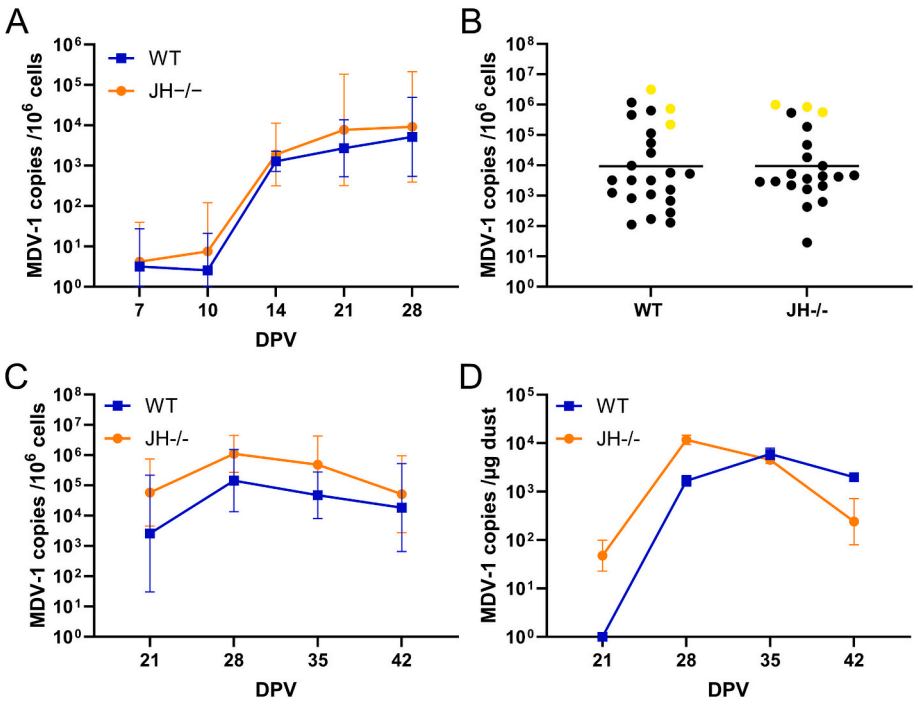


Fig. 3. MDV replication in various tissues in HVT vaccinated B cell knockout chickens.
(A) qPCR analysis of MDV-1 genome copies in the blood of $JH^{-/-}$ (n = 8) and WT chickens (n = 8) at indicated time points. Data is shown as means ± standard deviations ($p > 0.05$, Mann–Whitney U test). (B) MDV-1 load in the spleen of $JH^{-/-}$ and WT chickens measured by qPCR. The chickens with gross tumors (yellow icons) and mean genome copies (line) are indicated ($p > 0.05$, Mann–Whitney U test). Spleen samples were collected upon humane euthanasia upon the onset of MDV-specific clinical symptoms or at termination of the study. Each dot represents one bird. (C) MDV-1 load in FFE at indicated time points ($JH^{-/-}$: n = 8 and WT chickens: n = 8). Data is shown as means ± standard deviations ($p > 0.05$, Mann–Whitney U test). (D) MDV-1 genome copies in 1 µg of dust collected from the $JH^{-/-}$ and WT chicken rooms. Data is shown as means ± standard deviations ($p > 0.05$, Mann–Whitney U test). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Funding

This work was supported by the Deutsche Forschungsgemeinschaft in the framework of the Research Unit ImmunoChick (FOR5130) through the projects HA 8037/2–1, SCHU 2446/6–1 and KA 3492/9–1 awarded to SH, BS and BBK.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Sonja Haertle reports financial support was provided by German Research Foundation. Benjamin Schusser reports financial support was provided by German Research Foundation. Benedikt B. Kaufer reports was provided by German Research Foundation. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

We are grateful to Ann Reum for her technical assistance. We thank the TUM Animal Research Center (ARC) for breeding the required chicken lines for this study.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.vaccine.2025.128048.

Data availability

Data will be made available on request.

References

[1] Davison F, Nair V. Marek's disease: An evolving problem. Elsevier; 2004.
[2] Abdul-Careem MF, Hunter BD, Sarson AJ, Parvizi P, Haghighi HR, Read L, et al. Host responses are induced in feathers of chickens infected with Marek's disease virus. Virology 2008;370:323–32.
[3] Schat KA. History of the first-generation Marek's disease vaccines: the science and little-known facts. Avian Dis 2016;60(715–24):10.
[4] Morgan RW, Gelb Jr J, Schreurs CS, Lütticken D, Rosenberger JK, Sondermeijer PJ. Protection of chickens from Newcastle and Marek's diseases with a recombinant herpesvirus of turkeys vaccine expressing the Newcastle disease virus fusion protein. Avian Dis 1992;36:858–70.
[5] Li Y, Reddy K, Reid SM, Cox WJ, Brown IH, Britton P, et al. Recombinant herpesvirus of turkeys as a vector-based vaccine against highly pathogenic H7N1 avian influenza and Marek's disease. Vaccine 2011;29:8257–66.
[6] Wu C, Gan J, Jin Q, Chen C, Liang P, Wu Y, et al. Revaccination with Marek's disease vaccines induces productive infection and superior immunity. Clin Vaccine Immunol 2009;16:184–93.
[7] Yang Y, Dong M, Hao X, Qin A, Shang S. Revisiting cellular immune response to oncogenic Marek's disease virus: the rising of avian T-cell immunity. Cell Mol Life Sci 2020;77:3103–16.
[8] Couteaudier M, Denesvre C. Marek's disease virus and skin interactions. Vet Res 2014;45:36.
[9] Baaten BJ, Staines KA, Smith LP, Skinner H, Davison TF, Butter C. Early replication in pulmonary B cells after infection with Marek's disease herpesvirus by the respiratory route. Viral Immunol 2009;22:431–44.
[10] Bertzbach LD, Laparidou M, Hartle S, Etches RJ, Kaspers B, Schusser B, et al. Unraveling the role of B cells in the pathogenesis of an oncogenic avian herpesvirus. P Natl Acad Sci USA 2018;115:11603–7.
[11] Schusser B, Collarini EJ, Yi H, Izquierdo SM, Fesler J, Pedersen D, et al. Immunoglobulin knockout chickens via efficient homologous recombination in primordial germ cells. P Natl Acad Sci USA 2013;110:20170–5.
[12] Witter RL, Lee LF. Polyvalent Marek's disease vaccines: safety, efficacy and protective synergism in chickens with maternal antibodies. Avian Pathol 1984;13:75–92.

[13] Hernandez R, Brown DT. Growth and maintenance of chick embryo fibroblasts (CEF). Curr Protoc Microbiol 2010;17(1):1–8. Appendix 4:4I.
[14] Schat KA, Calnek BW, Fabricant J. Characterisation of two highly oncogenic strains of Marek's disease virus. Avian Pathol 1982;11:593–605.
[15] Sabsabi MA, Kheimar A, You Y, von La Roche D, Hartle S, Gobel TW, et al. Unraveling the role of gammadelta T cells in the pathogenesis of an oncogenic avian herpesvirus. mBio 2024;15:e0031524.
[16] Seliger C, Schaerer B, Kohn M, Pendl H, Weigend S, Kaspers B, et al. A rapid high-precision flow cytometry based technique for total white blood cell counting in chickens. Vet Immunol Immunopathol 2012;145:86–99.
[17] Djeraba A, Musset E, Lowenthal JW, Boyle DB, Chausse AM, Peloille M, et al. Protective effect of avian myelomonocytic growth factor in infection with Marek's disease virus. J Virol 2002;76:1062–70.
[18] Tahiri-Alaoui A, Smith LP, Kgosana L, Petherbridge LJ, Nair V. Identification of a Neurovirulence factor from Marek's disease virus. Avian Dis 2012;57(387–94):8.
[19] Purchase HG, Sharma JM. Amelioration of Marek's disease and absence of vaccine protection in immunologically deficient chickens. Nature 1974;248:419–21.
[20] Else R. Vaccinal immunity to Marek's disease in bursectomised chickens. Vet Rec 1974;95:182–7.
[21] Heidari M, Zhang H, Hearn C, Sunkara L. B cells do not play a role in vaccine-mediated immunity against Marek's disease. Vaccine X 2022;10:100128.
[22] Omar AR, Schat KA. Syngeneic Marek's disease virus (MDV)-specific cell-mediated immune responses against immediate early, late, and unique MDV proteins. Virology 1996;222:87–99.
[23] Fakhrul Islam AF, Walkden-Brown SW, Groves PJ, Underwood GJ. Kinetics of Marek's disease virus (MDV) infection in broiler chickens 1: effect of varying vaccination to challenge interval on vaccinal protection and load of MDV and herpesvirus of Turkey in the spleen and feather dander over time. Avian Pathol 2008;37:225–35.