Validating Distinction between Respiratory and Nephropathogenic Infectious Bronchitis Virus Strains via Chicken Tracheal Organ Culture: Insights on Innate Immune Enhancement and the COX-2/PGE2 Pathway

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Background

- Understanding the localized pathogenesis of infectious bronchitis virus (IBV) within the trachea of chicken is crucial for developing effective control strategies against this prevalent poultry pathogen [(1).]
- Current understanding: IBV induces COX-2/PGE2 pathway to enhance its replication in chicken macrophages [(2).]
- Unanswered question: What role does COX-2/PGE2 pathway play in the replication of IBV in trachea

Methods

- IBV genome load and antigen expression were quantified using real-time quantitative PCR and immunohistochemistry. COX-2, interferon (IFN)-α, IFN-β, interleukin (IL)-1β, IL-6, and inducible nitric oxide synthase (iNOS) expressions were measured, along with PGE2 and COX-2 concentrations

Findings

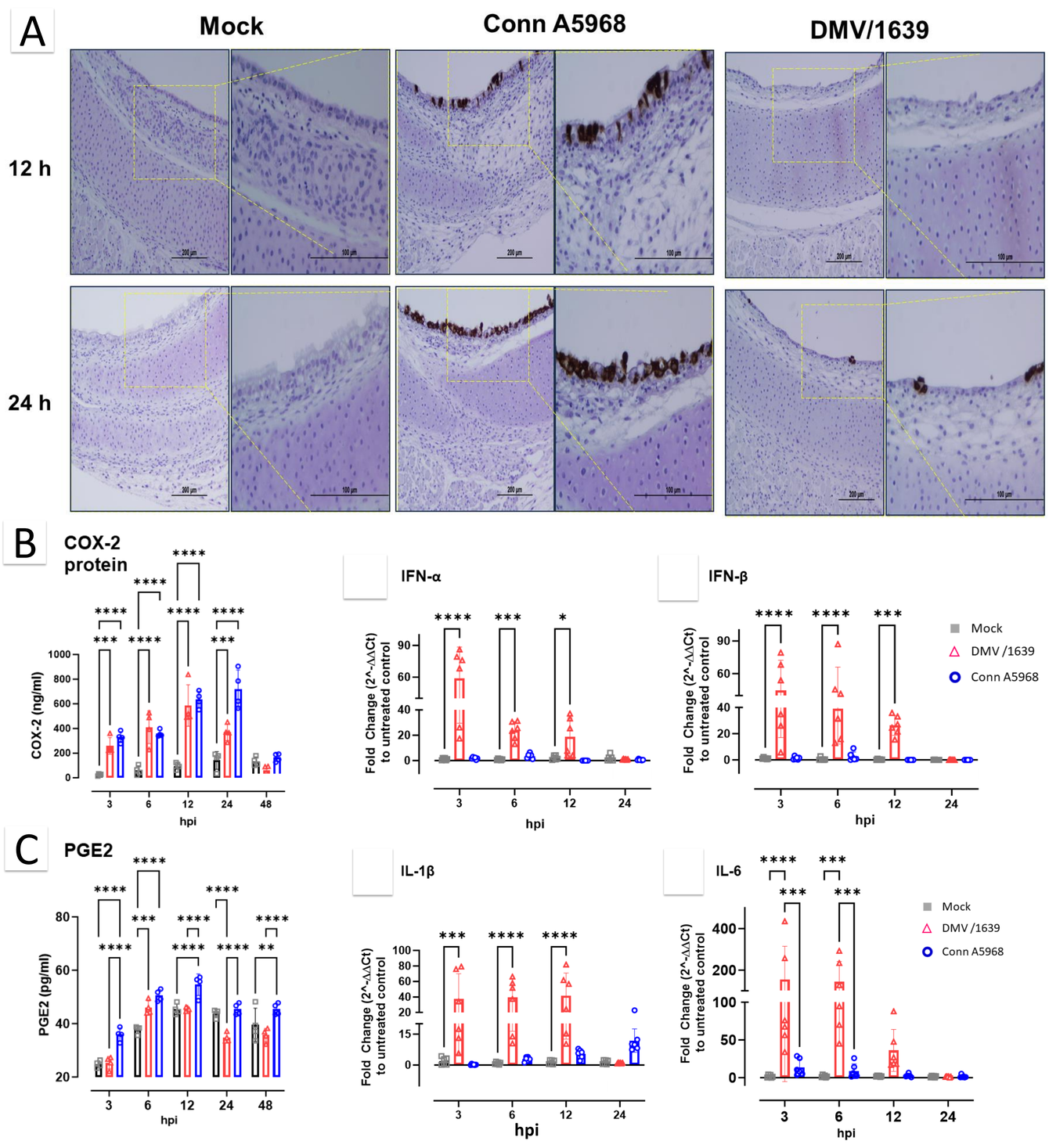
- The findings reveal distinct patterns of COX-2 expression, PGE2 production, and immune responses associated with different IBV strains, highlighting the complexity of host-virus interactions.
- IBV genome load and protein expression peaked at 12 and 24 hpi, respectively.
- Conn A5968-infected TOCs exhibited continuous COX-2 expression for up to 24 hpi, extended PGE2 production up to 48 hpi, and reduced inflammatory cytokine expression.
- In contrast, DMV/1639-infected TOCs displayed heightened inflammatory cytokine expression, brief COX-2 expression, and PGE2 production.
- Treatment with IFN-γ, SC-236, PGE2 receptor inhibitors, or JAK inhibitors reduced IBV infection and lesion scores,
- while exogenous PGE2 or IFN-γ pretreatment with a JAK-2 inhibitor augmented infection.

Conclsuion

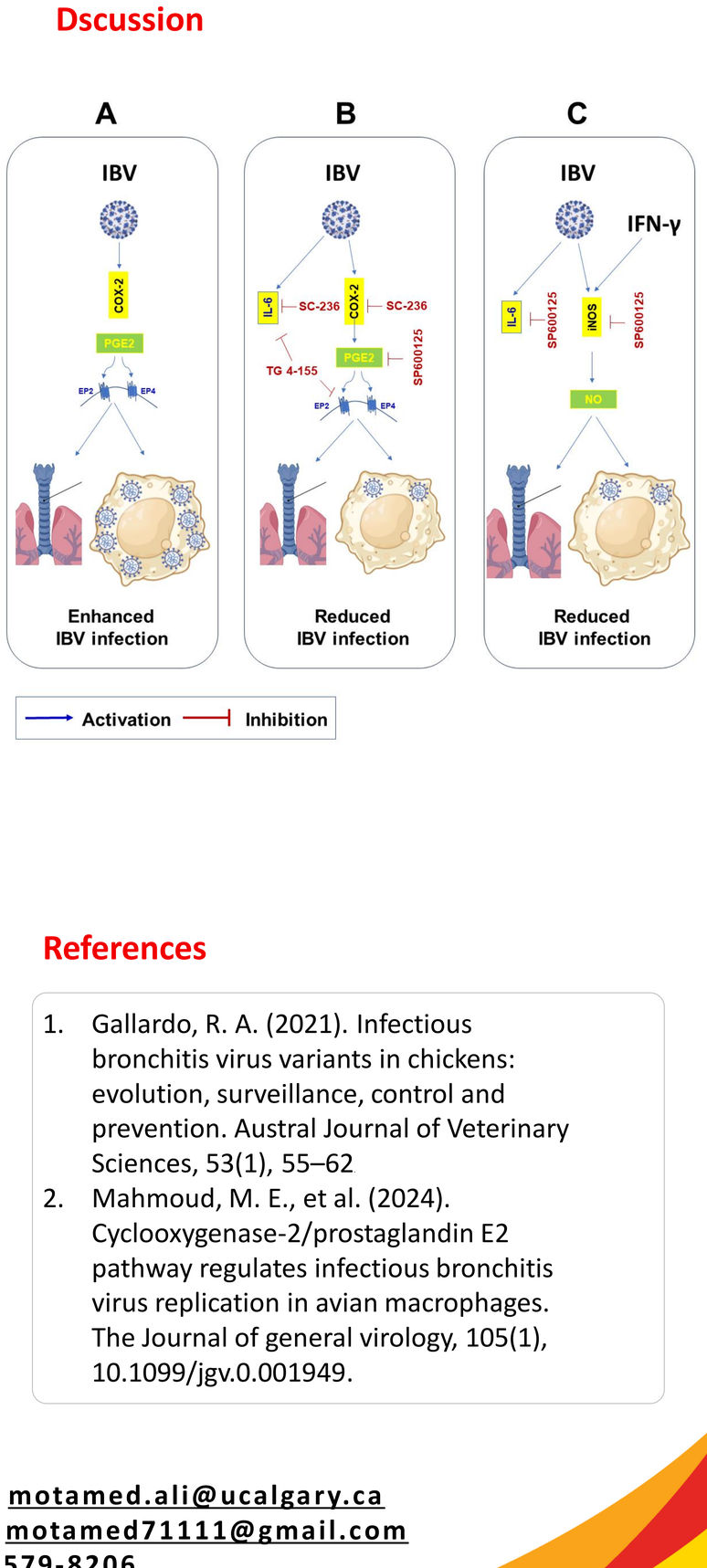
Our study unravels intricate distinctions between the IBV respiratory strain Conn A5968 and the IBV nephropathogenic strain DMV/1639 in IBV-infected tracheal organ explants, shedding light on their divergent pathogenesis.

- The IBV Conn A5968, despite exhibiting higher replication, demonstrated potential immune evasion strategies, marked by lower pro-inflammatory cytokine markers, possibly facilitated by an activated COX-2/PGE2 pathway.
- Conversely, the robust innate immune responses observed in IBV DMV/1639 infection, reflected by heightened pro-inflammatory cytokine expressions, seemed instrumental in restricting its replication and promoting clearance from tracheal explants.

Results



Dscussion



References

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2. Mahmoud, M. E., et al. (2024). Cyclooxygenase-2/prostaglandin E2 pathway regulates infectious bronchitis virus replication in avian macrophages. The Journal of general virology, 105(1), 10.1099/jgv.0.001949.

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