ATYPICAL STRAIN OF TOXOPLASMA GONDII LEADS TO LUNG INFLAMMATION AND WORSENS PULMONARY PHYSIOLOGICAL CHANGES

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Toxoplasma gondii exhibits a genetically diverse population structure, particularly in Latin America, where atypical strains have been associated with more severe disease outcomes. Pulmonary complications have been reported in toxoplasmosis, yet the impact of different strains remains underexplored. This study aimed to evaluate the pulmonary effects of *T. gondii* infection in mice infected with atypical strain TgCkBrRN2 (CK2), isolated in Rio Grande do Norte/Brazil, and the clonal strain ME49. Mice were euthanized in acute (15 dpi) and chronic (60 dpi) phases to assess lung histopathology and pulmonary function via spirometry. During the acute phase, the CK2-infected group showed a more pronounced increase in pulmonary resistance compared to other groups, persisting up to 60 dpi. Both dynamic and static compliance were impaired throughout infection, with strain-specific differences notable in the acute phase, indicating CK2 induces more severe pulmonary physiological alterations. Inspiratory capacity findings were consistent with previous observations, and the Tiffeneau index suggested a restrictive disease pattern in chronic infected animals. Histopathological evaluation aligned with these observations, revealing CK2-infected animals exhibited more severe pathology with higher scores of airway, perivascular, and parenchymal inflammation in both acute and chronic phases compared to ME49 strain. These findings underscore how *T. gondii* infection disrupts pulmonary homeostasis and function, particularly with atypical strains inducing more severe inflammatory responses. Such disruptions may contribute to long-term respiratory impairments, emphasizing the need for further investigation into underlying mechanisms and potential clinical implications.

Cnpq, capes e fapemig.

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