

A role for pro-inflammatory cytokines, but not prostaglandin E₂ in avian fever

Manette Marais¹, Shane K. Maloney^{1,2} and David A. Gray¹

1. School of Physiology, University of the Witwatersrand, Johannesburg, South Africa.

2. Physiology, School of Biomedical, Biomolecular, and Chemical Sciences, University of Western Australia, Perth, Australia

Studies with birds have, thus far, failed to find a persuasive correlation between pro-inflammatory cytokines and avian fever. We were the first to investigate a role for pro-inflammatory cytokines in the central component of the avian febrile mechanism using avian cytokines. We injected avian interleukin (IL)-1 β and avian IL-6 into the third cerebral ventricle of Pekin ducks ($n = 8$), and then recorded their core body temperature to determine whether these cytokines are pyrogenic in these birds. We established that both IL-1 β and IL-6 mediate fever in ducks, and we therefore treated birds with antibodies to avian IL-6 (anti-IL-6) and a prostaglandin (PG) synthesis-inhibitor (diclofenac) to ascertain, first of all, if IL-1 β is a precursor for IL-6, and secondly, whether brain-derived PGs are involved in the IL-1 β induced avian febrile mechanism. The febrile responses to IL-1 β were abolished in the anti-IL-6 and diclofenac treated ducks. In addition, we investigated a role for endogenous IL-6 and prostaglandin E₂ (PGE₂) in lipopolysaccharide (LPS)-induced fevers in ducks. We found that the LPS-induced fever was attenuated by anti-IL-6, but it was not associated with an increase in brain derived PGE₂ concentrations.