Brain IL-6- and PG-dependent actions of IL-1β and lipopolysaccharide in avian fever
M. Marais,1 S.K. Maloney 2 and D.A. Gray,1 1School of Physiology, The University of the Witwatersrand, Johannesburg 2193, South Africa and 2School of Biomolecular, Biomedical, and Chemical Science, The University of Western Australia, Crawley, WA 6009, Australia.

We used avian cytokines to investigate a role for proinflammatory cytokines in the central component of avian fever. Inteleukins IL-1 β and IL-6 injected intracerebroventricularly into Pekin ducks (n = 8) initiated robust fevers of equal magnitude and duration, although there was a significant difference in the latency to a febrile response. In addition, the IL-1β -induced fever could be abolished with an intracerebroventricular injection of antibodies to avian IL-6 or an oral administration of a prostaglandin (PG) synthesis inhibitor. Our findings indicate the following sequence of events within the central component of the avian febrile mechanism: IL-1β gives rise to bioactive IL-6, which stimulates an accelerated synthesis of PGs, and these PGs then adjust the sensitivity of warm-sensitive neurons in the avian brain stem to mediate fever. Yet PGE2 was not upregulated in the cerebrospinal fluid of ducks made febrile with lipopolysaccharide. We conclude that IL-1β and IL-6 may well mediate fever by instigating an accelerated synthesis of brain-derived PG, of a class other than PGE2, or that IL-6 serves as one of the terminal mediators of the avian febrile response.