The effects of prenatal hypoxia on the α-subunits of G proteins in the heart of the Broiler chicken

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Background
• β-adrenoceptors (βARs) are essential for cardiac development and regulation.
• Chronic prenatal hypoxia increases βAR sensitivity to epinephrine in the embryo but decreases it in the juvenile without changing receptor density in either.
• Others suggest that a shift in G-protein subtype expression favouring inhibitory (Gαi) G-proteins could effect βAR sensitivity.

Objective
To evaluate if embryonic and juvenile cardiac Gαs/Gαi expression is altered by prenatal hypoxia.

Hypothesis
Because the β2-AR subtype is known to signal through both stimulatory and inhibitory G-proteins, the hypothesis is that Gαs would increase in the hypoxic embryos, while Gαi would increase in the prenatally hypoxic juveniles.

Methods
• Broiler chicken eggs were incubated in 21% Oxygen (control) and 14% Oxygen (hypoxia).
• Embryonic samples were taken at 19 days of incubation. n=16.
• Juvenile samples were taken at 35 days post hatching. n=20.
• Samples were separated by SDS-PAGE and transferred onto PVDF membranes.
• Membranes were probed with either anti-Gαs or anti-Gαi antibodies.

Results
• Hypoxia increased Gαs, but not Gαi, in the juveniles.

• Hypoxia increased Gαi, but not Gαs, in the embryo.

Conclusions
• Unexpectedly, hypoxia increased Gαi in the embryo, while having no effect on Gαs.
• Chronic prenatal hypoxia increased Gαs in juvenile chickens, with no change in Gαi.
• This suggests the effects of hypoxia are downstream of the G proteins in the signalling cascade.

Perspective
• Others suggest that AC isoforms vary in their susceptibility to inhibition by Gαi, and their sensitivity to Gαs. And changes AC isoforms levels could have an effect on βAR sensitivity.

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