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

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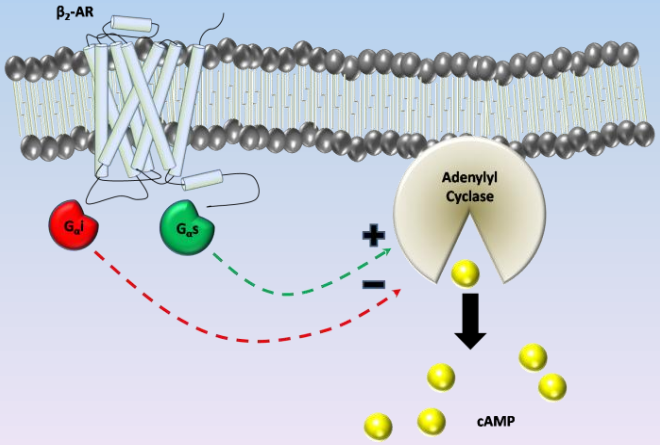
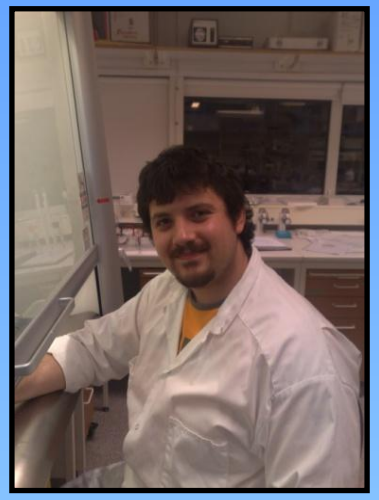
## Objective

To evaluate embryonic and juvenile cardiac $G_{\alpha}s$ and $G_{\alpha}i$ levels in prenatally hypoxic chickens, in order to determine if chronic prenatal hypoxia has an effect on the expression levels of these proteins.

## Hypothesis

Because the $\beta_2$AR subtype is known to signal through both stimulatory and inhibitory G-proteins, the hypothesis is that $G_{\alpha}s$ would increase in the hypoxic embryos, while $G_{\alpha}i$ would increase in the prenatally hypoxic juveniles.



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## Background

• β-adrenergic receptors (βARs) are G-protein coupled receptors that regulate contractility and heart rate through the $G_\alpha s$-Adenylyl cyclase-cAMP cascade. They also play an essential role in the development of the heart.

•Chronic prenatal hypoxia causes an increase in βAR sensitivity to epinephrine in the embryonic heart and, a decrease in sensitivity in the juvenile heart, without changes in receptor density in either age group. (Lindgren and Altimiras, 2009).

•$\beta_2$-adrenergic receptors can signal through both stimulatory and inhibitory G-proteins subtype. And some have suggested that an increase in expression of inhibitory ($G_\alpha i$) G-proteins could decrease βAR sensitivity, while an increase in expression of stimulatory ($G_\alpha s$) G-proteins could increase βAR sensitivity

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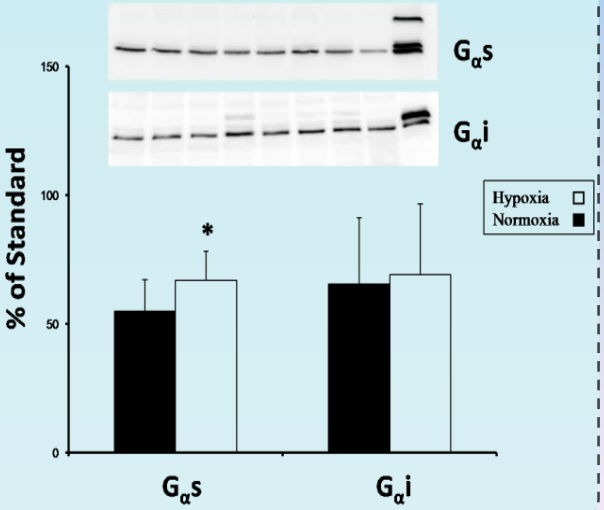
## Methods

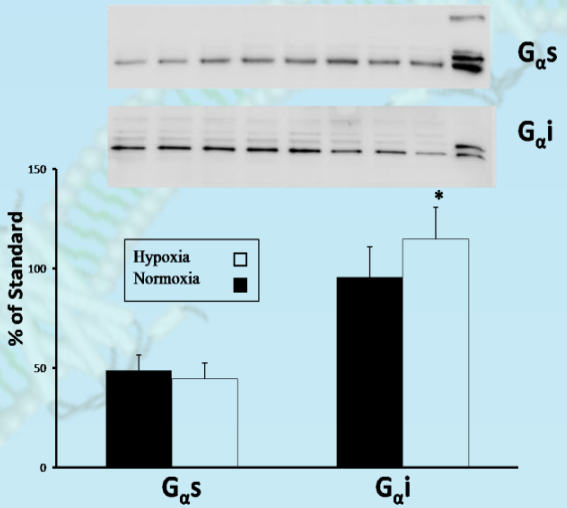
• Broiler chicken eggs were incubated in 21% Oxygen (control) and 14% Oxygen (hypoxia).
• Embryonic samples were taken at 19 days of incubation. n= 16.
• Juvenile samples were taken at 35 days post hatching. n= 20.
• Proteins in the samples were separated by SDS-PAGE and transferred onto PVDF membranes, by electroblotting.
• Membranes were then probed with either anti-$G_\alpha s$ or anti-$G_\alpha s$ antibodies.

## Results

Hypoxia increased $G_\alpha s$, but not $G_\alpha i$ in the juveniles.




Hypoxia increased $G_\alpha i$, but not $G_\alpha s$ in the embryo.




## Conclusions

Unexpectedly, hypoxia increased $G_\alpha i$ in the embryo while having no effect on $G_\alpha s$. Chronic prenatal hypoxia increased $G_\alpha s$ in juvenile chickens with no change in $G_\alpha i$. Demonstrating the long-lasting adverse affect hypoxia has on the chicken heart. The disassociation of G-protein subtype and βAR sensitivity implies that, hypoxia affects the βAR signaling cascade further downstream of the G-proteins. Others have shown that AC isoforms vary in their susceptibility to inhibition by $G_\alpha i$. And changes in AC isoform ratios could affect βAR sensitivity.