



## **Abstract Title:** ATP metabolism in RBC as biomarker for post exercise hypotension

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### **Introduction**

The importance of adenosine and adenosine 5'-triphosphate (ATP) in regulating many biological functions has long been recognized, especially for their effects on energy metabolism and cardiovascular homeostasis which may be used for management of hypertension and cardiovascular diseases. In response to ischemia, ATP is broken down to release adenosine. The activity of adenosine is very short lived because it is rapidly taken up by myocardial and endothelial cells, erythrocytes (RBC), and also rapidly metabolized to oxypurine metabolites and other adenine nucleotides. Extracellular and intracellular ATP is broken down rapidly to ADP and AMP and finally to adenosine by 5'-nucleotidase. These metabolic events are known to occur in the myocardium as well as in RBC (Yeung et al., 1997). It has been suggested ATP metabolism in the RBC may be a mechanism for post exercise hypotension (Yeung & Tinkel, 2013) and could be used as a biomarker for cardiovascular protection (Yeung, 2013).

### **Objective**

To investigate the feasibility of exploiting ATP metabolism in the RBC as systemic biomarker for post exercise hypotension and as a target for cardiovascular protection.

### **Methods**

An experimental treadmill exercise rat model was used to probe the relationship between post exercise hypotension and ATP metabolism in the RBC. The cardiovascular protective effect of exercise preconditioning was further investigated in an acute myocardial infarction model using mortality and ATP catabolism in the RBC as endpoints. In addition, an experimental model of endotoxemia in anaesthetized rats was used to validate further the relevance of ATP catabolism in the RBC in the management of sepsis. Data with and without exercise in response to cardiovascular injury or sepsis was compared using t-test and ANOVA and difference considered significant at  $p < 0.05$ .

## Results

We have shown post-exercise hypotension correlated significantly with RBC concentrations of ATP, and that exercise pre-conditioning reduced cardiovascular mortality and breakdown of ATP in the RBC. The post exercise effect was greater in hypertensive than in normotensive rats. Breakdown of ATP in the RBC also occurred in response to endotoxemia. The presentation will also discuss the opportunities, challenges and obstacles of exploiting ATP metabolism as drug targets for cardiovascular protection and prevention.

## Conclusion

ATP catabolism in RBC is a potential biomarker for post exercise hypotension and may be used as a drug target for cardiovascular protection.

## References

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