Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine

Poster presentation

Open Access

The acute effect of a physiological bolus of growth hormone (GH) on insulin signalling pathways in striated muscle in healthy volunteers

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from Danish Society for Emergency Medicine: Research Symposium 2009
Copenhagen, Denmark. 26 April 2009

Published: 20 August 2009

Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine 2009, **17**(Suppl 2):P3 doi:10.1186/1757-7241-17-S2-P3

This abstract is available from: http://www.sjtrem.com/content/17/S2/P3

Introduction

Critical illness impacts GH secretion in a biphasic pattern. The initial stress response consists of activated growth hormone (GH) release, GH secretion is reduced in the chronic phase of critical illness. High levels of GH causes severe insulin resistance and proinflammation, which may contribute to the morbidity and mortality of acute critical illness. The molecular mechanisms subserving these insulin antagonistic effects are unknown, but in vitro studies suggest that insulin and GH share postreceptor signalling pathways. This study was preformed to investigate if there is a crosslink between the two signalling pathways in human striated muscle in vivo.

Methods

8 healthy young men (age: 24,56 year ± 1, 84) participated in a three-armed single-blinded randomised crossover study. Prior to each study day the participants fasted 12 hours. In the morning the participants were randomised to receive either 1) an intravenous GH bolus (0.5 mg). 2) Intravenous GH bolus and oral glucose (75 g.) 3) Intravenous saline and oral glucose (75 g). 4 muscle biopsies were taken each study day at time 0 min, 30 min, 60 min, 120 min. Blood samples were collected regularly throughout each study day.

Results

GH induced an increase in AUCglucose (area under glucose curve) p = 0.05, without any significant changes in insulin levels. GH induced phosphorylation of STAT5 at 30 and 60 min. independently of oral glucose intake. Conversely, oral glucose intake induces phosphorylation of the insulin signalling proteins Akt and AS160 independently of GH exposure.

Conclusion

- GH induces acutely insulin resistance, also during a conventional OGTT (oral glucose tolerance test)

- GH activates STAT5 acutely in striated muscle independently of insulin while oral glucose intake activates insulin signalling pathways in striated muscle independently of GH

- GH-induced insulin resistance does not seem to be mediated by downregulation of insulin signalling pathways in human models.