J Glycomics Lipidomics 2015, 5:4
http://dx.doi.org/10.4172/2153-0637.C1.003

OMICS International
conferenceseries.com

International Conference on
Lipid Science & Technology
November 30 - December 02, 2015 San Francisco, USA

Effects of acute intermittent hypoxia on triglyceride levels and adipose tissue lipolytic functions in humans

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Context: Recent animal studies suggest that acute exposure to intermittent hypoxia (IH), a phenomenon observed in obstructive sleep apnea, induces adipose tissue lipolysis and inhibits the activity of adipose tissue lipoprotein lipase (LPL).

Objective: Determine if IH could adversely impact lipid metabolism, namely circulating triglyceride (TG) levels and adipose tissue lipolytic functions in humans.

Design: *In vitro* primary cell culture of human preadipocytes and *in vivo* randomized crossover study design.

Intervention: 10 healthy men were exposed to 6 hours of normoxia (21% oxygen) or IH (pulsed medical nitrogen) following the consumption of a high fat liquid meal.

Results: Using differentiated human preadipocytes, hypoxia significantly inhibits lipoprotein lipase ($P<0.05$) and lipolysis activity. Postprandial TG levels increased in a significant ($P<0.001$) but comparable manner in normoxia and IH sessions. Non-esterified fatty acid (NEFA) levels were significantly reduced over 120 minutes following meal in both sessions. After 120 minutes, NEFA levels significantly rose in both sessions, but to greater extent during IH. Neither meal nor IH affected subcutaneous abdominal adipose tissue LPL activity. Lipolytic responses to catecholamine or isoproterenol (β-adrenergic agonist) and antilipolytic response to α2-agonist assessed in isolated subcutaneous abdominal adipocyte were similar between normoxia and IH sessions.

Conclusions: *In vitro* results suggested that hypoxia significantly inhibits LPL and lipolysis activity. However, *in vivo* study concluded that an acute session of IH is sufficient to increase postprandial NEFA levels, but not circulating TG. Our results do not support impairment in subcutaneous abdominal adipose LPL activity nor intracellular lipolysis in young healthy men.

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