FISEVIER

Contents lists available at ScienceDirect

Journal of Clinical & Translational Endocrinology

journal homepage: www.elsevier.com/locate/jcte



Brief Report

Effect of intermittent sitting time on acute postprandial lipemia in children



Kara Ross^a, Erica Hinckson^{a,*}, Caryn Zinn^b

^a Auckland University of Technology, National Institute of Public and Mental Health, Centre for Child Health Research, Auckland, New Zealand ^b Auckland University of Technology, Faculty of Health and Environmental Sciences, School of Sport & Recreation, Auckland, New Zealand

ARTICLE INFO

Article history: Received 9 May 2014 Received in revised form 13 March 2015 Accepted 18 March 2015

Keywords: Fat Lipids New Zealand Physical activity Sedentary behavior

ABSTRACT

Objective: To investigate the effect of interrupting sitting time with intermittent moderate exercise on acute postprandial plasma triglyceride (TG) in healthy children following high-fat meal consumption.

Methods: Twelve participants (8 girls; 4 boys), aged 12 ± 2 years (mean \pm SD), completed two trials in the laboratory. On Day 1 (d1), sitting was interrupted with moderate intensity exercise every 30 min, and compared with day 2, (d2), where participants remained sedentary. On each testing day, participants consumed four high fat meals. Blood was sampled in a fasted state and 2-hourly for 6 h with the last sample taken on the 7th hour.

Results: Overall, there were no significant differences in the area under the concentration—time curve between day 1 and day 2, for the 12 participants combined (p=0.98). However, in eight of the 12 participants, triglyceride concentrations remained high on d2 at two, four and 6 h after baseline compared with d1 (p=0.03).

Conclusion: When sitting was interrupted by short bouts of moderate intensity exercise there was a reduction in triglyceride concentrations in eight out of 12 participants. Possible reasons to account for the difference in response may include sexual maturation, gender differences, genetic conditions, or the rate of digestion and intestinal absorption.

© 2015 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Introduction

Animal models have shown that LipoProtein Lipase (LPL) activity is suppressed during prolonged sedentary bouts leading to an increase in chylomicrons (triglyceride-rich lipoproteins) in the plasma [1,2]. LPL is an enzyme that binds to circulating chylomicrons in the blood stream and hydrolises the triglyceride within the lipoprotein [2]. Elevated levels of chylomicrons may play an important role in determining metabolic syndrome [1] and have

been associated with cardiovascular risk factors such as dyslipidemia in overweight children [3]. Postprandial lipemia, the inability to clear chylomicrons quickly following intake of a high fat meal [3], may be related to inactivity, while exercise may decrease postprandial lipemia, through changes to muscle LPL [4]. The purpose of this study was to compare triglyceride (TG) acute response in healthy children following a high fat diet during prolonged and interrupted sitting with intermittent moderate exercise.

Research design and methods

Twelve healthy children (8 girls; 4 boys) aged 12 ± 2 years (mean \pm SD) were recruited from eight randomly selected Auckland (New Zealand) primary schools. Access to schools was granted by school principals. In response to information sheets distributed by teachers to children, parents/caregivers returned consent and assent forms indicating willingness to participate. Participants were

E-mail address: erica.hinckson@aut.ac.nz (E. Hinckson).

Funding: No funding received for this project.

Author contributions: Design: EH, KR; Data collection/analysis: KR; First draft manuscript: KR; Project supervision: EH; Dietary intake: CZ. All authors contributed to the manuscript.

^{*} Corresponding author. Centre for Child Health Research, Faculty of Health and Environmental Sciences, AUT University, Private 92006, Auckland, New Zealand. Tel.: +64 9 9219999x7224; fax: +64 9 9219999.

Table 1Preliminary^a participant characteristics

Measure	Mean	SD
Age (y)	11.5	1.57
Body mass (kg)	48.7	9.72
Height (cm)	152.6	11.10
WHO mean z-score	0.88 ^c	0.59
% Time spent sitting ^b (h)	83.84	6.07
% Time spent standing (h)	10.83	4.04
% Time spent stepping (h)	5.33	2.93
Step count	76.34	21.42
Sit to stand transitions (counts)	7.64	24.4
EE (Met.h)	8.74	15.21

- ^a 14 days before laboratory study.
- ^b Includes sleeping hours.
- c 81st percentile.

excluded if they engaged in high levels of extracurricular physical activity, had a prior medical condition, were taking any medications or had a current condition that would limit activity participation. Ethical approval was received by the Institution's Ethics Committee.

Preliminary measures

Anthropometric and physical activity measurements were taken two weeks prior to participation. Height (m) and weight (kg) measurements were used to calculate BMI WHO z-scores [5]. Activity measurements were collected through ActivPAL accelerometry. Accelerometers were worn for a total of four days prior to testing (d1 & d2). Recorded activity and inactivity is shown in Table 1.

Experimental design and procedures

All participants were required to attend two trials (08:00–16:15) following a 12-h fast. Each trial was separated by a period of six days. On arrival to the laboratory, participants provided a blood sample and were asked to wear an ActivPAL accelerometer. On d1, participants (experimental trial) engaged in their choice of sedentary activities but completed 4 min of moderate intermittent activity (197 \pm 51 steps) every 30 min starting at 09:25. The

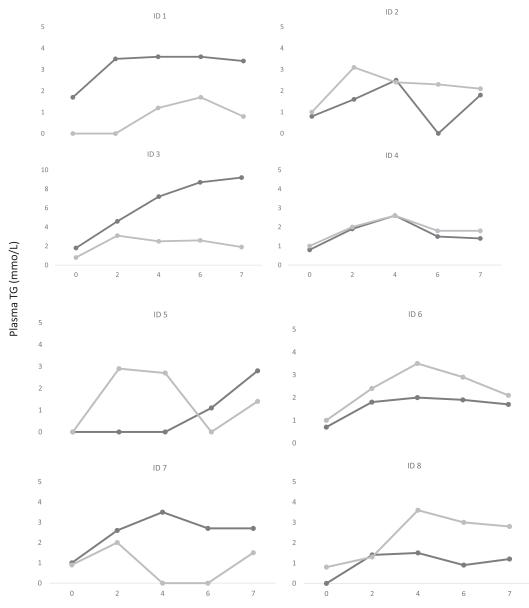


Figure 1. Plasma TG concentrations [mmol/L] in the fasted state and over 7 h after ingestion of high-fat meals for each participant (ID) during sitting interrupted with moderate exercise (
and sitting (
).

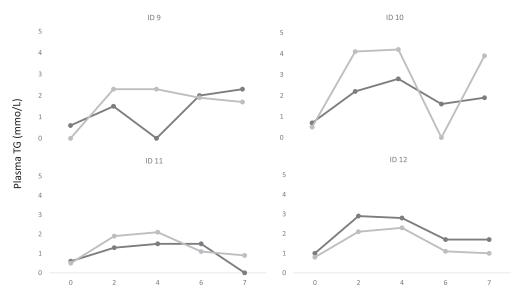


Figure 1 (continued).

number of steps taken and activity intensity were recorded continuously as well as time spent sitting throughout the intermittent activity bout using the ActivPAL accelerometers which then provided cadence levels. Participants engaged in child-friendly activities including soccer, basketball, obstacle courses, stair climbing, dancing, cart wheel races and touch rugby. At 2-h intervals a 500 μl blood sample was taken using pediatric lancets to measure TG. On d2 (control trial) participants followed the same protocol, but remained seated for 6 h.

Dietary intake

On d1 and d2, participants were provided with an identical high fat meal (90 g fat; 140 g carbohydrate; 77 g protein). The first meal was consumed directly following blood collection. Participants consumed three smaller meals throughout each testing day and water was consumed ad libitum. These meals were designed to approximate reported "unhealthy" eating habits of New Zealand children [6]. Participants consumed food according to appetite and the remaining food was weighed. Nutrient intake was analyzed using FoodWorks© Premium.

Blood analysis

Blood samples were collected using OHSA high flow pediatric lancets (BD Microtainer®, BD Australia) and were drawn into sterile heparin tubes (BD Microtainer®, BD Australia). Prior to collection participants were offered the opportunity to use a local anesthetic containing lignocaine and prilocaine (Emla, AstraZeneca Limited, NZ). The finger puncture was performed using a sterile, OSHA approved blood lancet creating a deep puncture (1.5 mm) at the chosen site. The initial drop was discarded and 0.3–0.5 ml of blood was collected for analysis and then centrifuged at a recommended speed of 5000 rpm (revolutions per minute) for 5 min. Plasma was separated from the red blood cells and was immediately frozen. Lipid (TG) analysis was performed in accordance with an accredited laboratory protocol.

Sample size

We assumed that the overall difference in the mean change of TG will be equal to the within-subject standard deviation. With Type 1

error at 5% and Type 2 error 75%, calculations showed that 12 participants will be needed in the experimental and control groups. In this study the 12 participants served as their own controls [7].

Statistical analysis

All descriptive statistics are presented as means and standard deviations. The areas under the concentration—time curve (AUC) for TG were not normally distributed and therefore were log transformed. Differences between d1 and d2 were then compared using paired *t*-test. Further comparisons were made between d1 and d2 for responders and non-responders. Statistical significance was determined at the 0.05 level. Statistical analyses were performed in SPSS 22.

Results

Descriptive participant characteristics are presented in Table 1. Baseline meal comparisons between d1 and d2 showed a non-significant change of $-4.6\% \pm 5.6\%$ (p=0.2) in the level of fat consumed. Baseline TG levels in all participants also represented a non-significant difference -14% ($\pm 43\%$) (p=0.41).

The raw plasma TG concentrations [mmol/L] in the fasted state and 7 h after ingestion of high-fat meals for each participant during the two experimental conditions are presented in Fig. 1.

TG levels were higher on d2 compared to d1 in eight out of the 12 participants (2,4-6,8-11). The raw combined plasma TG concentrations for all participants (n=12), responders (n=8), and non-responders (n=4), and males (n=4) and females (n=8) are presented in Figs. 2 and 3.

We observed no significant differences in the area under the concentration—time curve between d1 and d2 for the 12 participants combined (p=0.982) (Fig. 2). But significant differences between d1 and d2 were observed when the group was split into responders (-0.21 ± 0.13 , p=0.03) and non-responders (0.42 ± 0.21 , p=0.028).

Discussion

This is one of the first studies to examine the effect of interrupting sitting with moderate exercise on acute postprandial

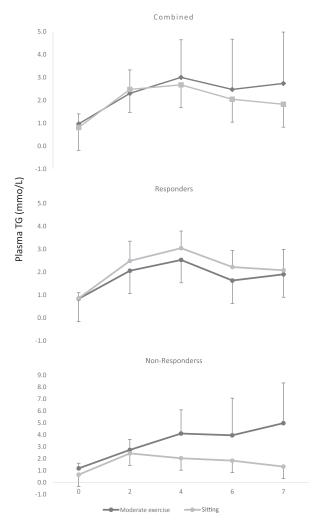


Figure 2. Plasma TG concentrations [mmol/L] in the fasted state and 7 h after ingestion of high-fat meals for all participants (n=12), responders (n=8) and non-responders (n=4) during sitting interrupted with moderate exercise (\bullet) and sitting (\bigcirc) .

lipemia in healthy children with somewhat positive results. The only other study conducted in this area was by Saunders and colleagues [8] who did not find any measurable differences in lipids and other parameters, when 8-h of sitting was compared with interrupted sitting. In our study, when sitting was interrupted by short bouts of moderate intensity exercise, there was a reduction in TG concentrations in eight of the 12 participants. Postprandial clearance was observed as early as 2 h with intermittent sitting compared to continuous sitting. The clearance was maintained for the next 6 h. A possible mechanism for the enhanced clearance with exercise in the eight participants is perhaps through the stimulation of LPL activity. Exercise is known to lead to an increase in muscle LPL activity, and increased skeletal blood flow during exercise can enhance TG clearance [9]. Individual variation in the phenotypic expression of LPL activity may have also been a factor. Additionally, it may be possible that TG response is affected by level of sexual maturation [10], gender differences [11], genetic conditions such as visceral obesity [12], polymorphisms within the genes for apo A-I, A-IV, A-V, E, B, C-I and C-III [13], lipoprotein lipase deficiency [14] or the rate of digestion and intestinal absorption. While it is clear from this study that there are responders and non-responders to our protocol, the nature and scope of this study precluded further investigation. Nevertheless, the results showed a trend towards the expected directions for eight of the 12 participants.

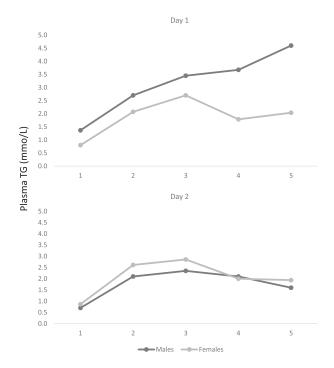


Figure 3. Plasma TG concentrations [mmol/L] in the fasted state and 7 h after ingestion of high-fat meals for all participants (n = 12), males $(n = 4, \bullet)$ and females $(n = 8, \bullet)$ during sitting interrupted with moderate exercise (Day 1) and sitting (Day 2).

The results suggest that postprandial clearance is acutely enhanced in some children after 2 h of interrupting sitting with exercise but not others. Further research is needed on a larger sample of children and with a metabolic dysfunction to test these findings. Additionally, it is important to establish whether longer term intermittent sitting shows similar effects. The results may provide initial evidence into the metabolic effects of sedentary behavior in children.

Conflict of interest

The authors declare they have no conflicts of interest.

Acknowledgments

The authors would like to acknowledge all the participants, the research assistants, placement students of Bath University, our registered nurses, Dr Jun Lu, and the support of the School of Sport and Recreation, Auckland University of Technology.

References

- [1] Zderic TW, Hamilton MT. Physical inactivity amplifies the sensitivity of skeletal muscle to the lipid-induced downregulation of lipoprotein lipase activity. J Appl Physiol 2006;100:249–57.
- [2] Hamilton MT, Hamilton DG, Zderic TW. Exercise physiology versus inactivity physiology: an essential concept for understanding lipoprotein lipase regulation. Exerc Sport Sci Rev 2004;32:161–6.
- [3] Su J, Nzekwu M, Ball G, Jetha M, Proctor S. Postprandial lipemia as an early predictor of cardiovascular complications in childhood obesity. J Clin Lipidol 2009;3:78–84.
- [4] Herd SL, Kiens B, Boobis LH, Hardman AE. Moderate exercise, postprandial lipemia, and skeletal muscle lipoprotein lipase activity. Metabolism 2001;50: 756–62.
- [5] Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ 2000;320: 1240–3.

- [6] Ministry of Health. Obesity in New Zealand. Wellington: Ministry of Health; 2010.
- [7] Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. Med Sci Sports Exerc 2009;41:3.
- [8] Saunders TJ, Chaput J-P, Goldfield GS, Colley RC, Kenny GP, Doucet E, et al. Prolonged sitting and markers of cardiometabolic disease risk in children and youth: a randomized crossover study. Metabolism 2013;62:1423–8.
 [9] Murphy MH, Nevill AM, Hardman AE. Different patterns of brisk walking are
- [9] Murphy MH, Nevill AM, Hardman AE. Different patterns of brisk walking are equally effective in decreasing postprandial lipaemia. Int J Obes 2000;24: 1303–9.
- [10] MacEneaney O, Harrison M, O'Gorman D, Pankratieva E, O'Connor P, Moyna N. Effect of prior exercise on postprandial lipemia and markers of inflammation

- and endothelial activation in normal weight and overweight adolescent boys. Eur J Appl Physiol 2009;106:721–9.
- [11] Couillard C, Bergeron N, Prud'homme D, Bergeron J, Tremblay A, Bouchard C, et al. Gender difference in postprandial lipemia importance of visceral adipose tissue accumulation. Arterioscler Thromb Vasc Biol 1999;19:2448–55.
- [12] Couillard C, Bergeron N, Prud'homme D, Bergeron J, Tremblay A, Bouchard C, et al. Postprandial triglyceride response in visceral obesity in men. Diabetes 1998;47:953–60.
- [13] Lopez-Miranda J, Williams C, Lairon D. Dietary, physiological, genetic and path-
- ological influences on postprandial lipid metabolism. Br J Nutr 2007;98:458–73.

 [14] Gotoda T, Yamada N, Kawamura M, Kozaki K, Mori N, Ishibashi S, et al. Heterogeneous mutations in the human lipoprotein lipase gene in patients with familial lipoprotein lipase deficiency. J Clin Invest 1991;88:1856.