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The Effect of α-Cyclodextrin on postprandial lipid and glycemic responses to a fat-containing meal

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ABSTRACT

Objective. α-Cyclodextrin (α-CD), a soluble dietary fiber derived from corn, marketed under the trade name FBCx®, has the potential to help individuals manage their weight and improve their lipid profiles. Initial studies in healthy overweight and/or obese diabetic individuals found that, in those consuming a normal to high fat diet over a 4 or 12 week period, α-CD use was associated with weight loss or maintenance and a reduction in triglyceride (TG) and cholesterol levels in hyperlipidemic individuals. Furthermore, α-CD use was associated with the positive effects of increasing insulin and leptin sensitivities. To date, the immediate postprandial glucose and lipid responses to a fat-containing meal have not been reported.

Materials/Method. This double blinded placebo controlled cross-over trial examined the effect of 2 g of α-CD taken immediately following consumption of a commercially prepared high-fat breakfast meal on the acute postprandial responses in healthy adults.

Results. The coincidental consumption of α-CD with a fat-containing meal was associated with a significant reduction in postprandial TG responses over time when compared to placebo. When incremental area under the curve was calculated, the area under the curve associated with α-CD consumption was significantly smaller than the Placebo area (0.30 ± 1.07 mmol/L/3 h *vs.* 0.98 ± 0.88 mmol/L/3 h, $p < 0.05$). There were no significant changes in glucose or cholesterol levels.

Conclusion. α-Cyclodextrin was shown to significantly lower acute postprandial blood triglyceride levels.

1. Introduction

The prevalence of obesity in the United States remains at remarkably high levels, with 35.5% of adult men and 35.8% of adult women reported to be obese [1]. These high rates of obesity are associated with an increased risk of developing cardiovascular disease and type 2 diabetes as well as an increased risk for metabolic syndrome factors such as increased insulin resistance and increased blood levels of triglyceride (TG) and low density lipoprotein cholesterol (LDL-C) [2]. A reduction in dietary fat intake has the potential to positively influence body weight by reducing energy intake

Abbreviations: α-CD, α-Cyclodextrin; HFD, high fat diet; HDL-C, high density lipoprotein cholesterol; TC, total cholesterol; LDL-C, low density lipoprotein cholesterol; TG, triglyceride; SD, standard deviations.

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and blood lipid levels by reducing the absorption of dietary fat. This, in turn, could lead to a reduction in obesity-related risk factors and disorders.

It is well understood that a reduction in dietary fat intake could result in positive health benefits; however, this requires a change in behavior that is difficult for many individuals to maintain. Tasty high calorie/fat foods are convenient and easily accessible; for many, they are an accepted part of the Western lifestyle. As a reduction in dietary fat intake is difficult for many, reducing calorie intake from fat by blocking its absorption in the gastrointestinal tract would appear to be an appealing alternative. This has led to the search for products that could effectively and safely reduce the absorption of dietary fat. The lipase inhibitor, Orlistat, was the first such pharmaceutical but has fallen into disfavor because of unwanted side effects [3,4].

Dietary fibers have been shown to reduce fat absorption and blood lipid levels, thus reducing the risk of developing cardiovascular disease [5,6]. At the present time, the average fiber intake in the US population is about 15 g, well below the 25–35 g/day recommended by the USDA. Recently a naturally occurring food supplement has shown promise in preventing the absorption of dietary fat. α-Cyclodextrin (α-CD) is a soluble dietary fiber derived from corn and is commercially available in tablet form as FBCx®. It is believed that this particular fiber has the unique ability to form a very stable fat–fiber complex with fat droplets in the stomach. As this fat–fiber complex passes into the duodenum, it is resistant to the lipolytic activity of pancreatic lipases. As it passes into the large bowel the complex appears to remain intact so that the micro flora are unable to ferment either the fat or the fiber, thus avoiding the anal leakage and explosive bowel movements that are associated with lipase inhibitors.

The favorable gastrointestinal tolerance makes α-CD well accepted by individuals who have difficulty adhering to a restrictive diet. An initial study conducted over a 6 week period in rats demonstrated that, despite comparable energy and food intake, α-CD significantly reduced weight gain and increased fat excretion in growing male Wistar rats that were fed a high fat diet (HFD) containing α-CD relative to rats consuming the same HFD without α-CD. Consumption of a HFD with α-CD also resulted in a 30% reduction in TG levels, a 9% reduction in cholesterol levels and improved insulin sensitivity relative to the control rats consuming the same diet without α-CD. It was estimated from both *in vitro* and *in vivo* data presented in this study that α-CD binds 9 times its own weight in dietary fat [7]. Gallaher et al. [8] reported disproportionately higher levels of fecal saturated fat in their animal study. A study by Wagner et al. [9] examined α-CD use in a mouse dyslipidemia model in which the animals consumed a moderate fat containing diet over a 14 week period. The inclusion of α-CD in the diet was associated with a significant reduction in LDL-C while high density lipoprotein cholesterol (HDL-C) remained unchanged, resulting in an improved lipid profile. The basic research suggests α-CD could be a valuable tool for weight *and* lipid management.

The effect of α-CD on body weight and blood lipid levels has also been examined in healthy overweight but not obese and obese diabetic individuals. In a 2-month, double-blinded crossover study of healthy individuals with BMIs between 25 and 30 kg/m^2, α-CD was associated with a significant reduction in weight, and in hyperlipidemic volunteers, a reduction in total cholesterol (TC) and LDL-C levels during the active month of the study when compared to the placebo month [10]. The second study, which was a 3-month, double-blinded placebo controlled trial, examined the effect of α-CD on body weight and blood lipid levels in obese persons with type 2 diabetes [11]. Participants in the α-CD group, unlike those in the placebo group, maintained their weight (despite increased energy intake) and those with hypertriglyceridemia had a significant reduction in TC levels.

Little is known about the acute postprandial effects of α-CD. It has been proposed that α-CD may reduce carbohydrate digestion, and this in turn may attenuate the postprandial glycemic responses to carbohydrate containing foods. These researchers reported that the addition of α-CD to a meal of boiled rice with 50 g of available carbohydrate reduced postprandial glucose responses [12]. Postprandial increases in blood glucose are associated with increased risk for diabetes and cardiovascular disease [13,14]. Postprandial increases in TG may have health consequences as well, as oxidative stress and increased markers of inflammation have been reported as a consequence of hyperlipidemia following consumption of a high fat meal [14]. Bansal et al. [15] have reported that elevated triglyceride levels 2–4 h postprandial have an independent and strong association with adverse cardiovascular events in women. Nordestgaard et al. [16] have reported similar findings in a large Danish study of both men and women. It has been reported that meals rich in polyunsaturated fats cause lower levels of postprandial lipemia than meals rich in saturated and monounsaturated fatty acids [17]. This is of interest in reference to the mouse study reported by Wagner et al. [9] that has demonstrated that α-CD appears to preferentially lower blood saturated and *trans* fats. The acute glucose and lipid responses to a fat-containing meal with coincidental α-CD consumption, to date, are unknown. After the consumption of a fat-containing meal, we hypothesize that acute glucose and TG levels will be attenuated by the coincidental consumption of α-CD, while TC will remain unchanged [18,19]. This double-blinded placebo controlled crossover study was undertaken to examine the effect of α-CD on the postprandial glycemic and lipid responses immediately following the consumption of a high fat meal by healthy adults.

2. Method and procedures

2.1. *Subjects*

Thirty-four healthy adults (6 males and 28 females) between the ages of 18 and 65 were recruited from a university campus in the Midwest United States. None of the participants were on medications that would lower lipid levels or alter glucose metabolism. Those who were vegetarian, pregnant, did not consume pork, or had a chronic health condition (diabetes, cardiovascular disease and hypertension) were excluded. The mean BMI was 25.04 ± 4.08 kg/m^2, range 19.3–35.9. Participants signed a consent form that had been approved by the university's Institutional Review Board.

2.2. Design and procedure

A double-blind, placebo controlled cross-over study was designed to explore the effects of α-CD in healthy adults immediately following a high fat-containing meal. Upon completion of the screening process the participants signed an informed consent and were then instructed to maintain their usual physical activity and eating pattern on the day before their scheduled arrival at a food laboratory, on 2 separate days, following an overnight fast. Height and weight were self-reported. A wash-out period of 2 days [12] was used. Blood was acquired by finger stick at baseline prior to and at 1, 2, and 3 h following consumption of a commercially available fast-food breakfast meal. Immediately upon completion of the meal each volunteer was asked to take two unmarked tablets, one set of tablets was active (1 g α-CD each); the other placebo (1 g cellulose). Dosage was based upon the manufacturer's recommended dose of two tablets (2 g of α-CD) per fat-containing meal. Active and placebo tablets were identical in appearance (ArtJen Complexus, Bloomfield Hills, MI). The order of tablet presentation was randomized amongst the participants following their first meal. The breakfast meal consisted of a commercially prepared egg sausage biscuit sandwich and 8 oz of bottled water purchased from a popular fast food restaurant. The meal contained a reported 26 g of fat (10 g of saturated fat) (Table 1). Participants were instructed to consume the meal within 10 min. Between blood draws participants were asked to read or engage in light activities.

Blood samples were collected with a capillary tube and immediately transferred to a Cholestech LDX System (Cholestech, Hayward, CA) for lipid profile (TC, HDL-C, TG and calculated LDL-C) and glucose analysis. A detailed evaluation of this instrument system has been presented elsewhere [20]. The instrument was calibrated before each use with the calibration cassette provided by the company. The coefficients of variation were about 1.5%. In accordance with normal machine operation, it took about 5 min for each blood sample to be analyzed and results printed out.

2.3. Statistical analysis

Results are reported as means ± standard deviations (SD). For each blood measure, a within subjects repeated measures analysis of variance (RM ANOVA) was applied with 2 within subjects factors, treatment and time. Post hoc comparisons were conducted with paired t-tests. IBM SPSS Statistics 19 (IBM, Armonk, NY) was used for the analysis. Significance level was set at $p < 0.05$.

Table 1 – Reported Nutritional Facts of a Sausage McMuffin® with Egg.

Ingredient	Quantity
Total Calories (cal)	440
Fat (g)	26
Saturated Fat (g)	10
trans Fat (g)	0.4
Cholesterol (mg)	220
Sodium (mg)	910
Carbohydrate (g)	32
Fiber (g)	2
Sugars (g)	2
Protein (g)	20

From: http://www.mcdonalds.ca/ca/en/menu/full_menu/breakfast/sausage_mcmuffin_with_egg.html. Accessed February 4, 2013.

3. Results

Following randomization, 18 participants began the study with Active tablets while 16 with Placebos. There was no difference between gender, age or order of the tablets in response to the meal. No acute adverse gastrointestinal events were noted on either day of testing. Neither glucose nor any of the measured lipid fractions except for triglycerides showed any significant changes between the Active and Placebo meals (Table 2). RM ANOVA results for TG showed a significant effect for time ($p < 0.0001$), and time by treatment interaction ($p < .05$), indicating that α-CD attenuated the rise in TG after the meal. Post hoc paired t-tests indicated a trend for lower TG levels during the Active meals vs. the Placebo meals at 1 h ($p = .07$) and at 3 h the Active meals showed significantly lower levels than observed during the placebo meals ($p < .05$).

Due to the large inter-person and daily variability, all the postprandial responses were normalized as a percent of baseline levels. Differences in glucose, total cholesterol, LDL-cholesterol and HDL-cholesterol responses during the Active phase when compared to the Placebo phase were not significant. When examined as a percentage of baseline

Table 2 – Fasting blood glucose and lipid levels as well as post-prandial levels and the difference between the active and placebo phases (mean ± SD).

	Active	Placebo	Difference	P
Glucose (mmol/L)				
Baseline	4.96 ± 0.52	5.09 ± 0.53	−0.13 ± 0.51	
1 h	5.48 ± 1.26	5.53 ± 1.40	−0.05 ± 0.64	
2 h	5.01 ± 0.61	5.02 ± 0.73	−0.01 ± 0.56	
3 h	4.84 ± 0.32	5.02 ± 0.68	−0.18 ± 0.69	
Cholesterol (mmol/L)				
Baseline	4.63 ± 0.98	4.66 ± 1.06	−0.02 ± 0.39	
1 h	4.47 ± 1.01	4.53 ± 1.03	−0.04 ± 0.35	
2 h	4.47 ± 1.03	4.53 ± 0.98	−0.05 ± 0.38	
3 h	4.50 ± 1.09	4.60 ± 1.01	−0.12 ± 0.42	
LDL Cholesterol (mmol/L)				
Baseline	2.69 ± 0.96	2.74 ± 0.83	−0.03 ± 0.53	
1 h	2.64 ± 0.83	2.61 ± 0.83	0.02 ± 0.43	
2 h	2.48 ± 0.88	2.38 ± 0.75	0.10 ± 0.52	
3 h	2.56 ± 0.91	2.56 ± 0.83	0.01 ± 0.52	
HDL-cholesterol (mmol/L)				
Baseline	1.37 ± 0.44	1.45 ± 0.44	−0.08 ± 0.39	
1 h	1.35 ± 0.41	1.32 ± 0.44	0.02 ± 0.26	
2 h	1.42 ± 0.39	1.42 ± 0.39	0.002 ± 0.17	
3 h	1.32 ± 0.44	1.35 ± 0.44	−0.02 ± 0.31	
Triglycerides (mmol/L)				
Baseline	1.19 ± 0.65	1.07 ± 0.44	0.108 ± 0.45	
1 h	1.26 ± 0.55	1.43 ± 0.71	−0.167 ± 0.51	p = .07
2 h	1.45 ± 0.51	1.49 ± 0.55	−0.053 ± 0.40	
3 h	1.31 ± 0.47	1.51 ± 0.64	−0.200 ± 0.52	p < .05

levels, ingestion of α-CD with a fat-containing meal was associated with significant treatment ($p = 0.01$), time ($p < 0.05$), and time × treatment interaction ($p < 0.05$) effects: TG levels were significantly reduced at all 3 time points when the Active phase was compared to the Placebo phase (Fig. 1). Paired-t test revealed that area under the curve for TG as a percent change from baseline was significantly lower in the Active compared to the Placebo data (362 ± 106%/3 h vs. 417 ± 100%/3 h, $p < 0.001$), while there was no difference between the Active and Placebo when TG absolute values were used. When incremental area under the curve (IAUC) was calculated, again the Active area was significantly smaller than the Placebo area (0.30 ± 1.07 mmol/L/3 h *vs.* 0.98 ± 0.88 mmol/L/3 h, $p < 0.05$).

Participants' data were also grouped according to their baseline TG levels into either hypertriglyceridemic (TG > 1.69 mmol/L, n = 5) or normotriglyceridemic (n = 28) as performed previously [11]. ANOVA with repeated measures, with TG levels as a between subject factor, and IAUC as the dependent variable, shows a significant treatment effect with lower TG IAUC during the Active phase as compared to the Placebo phase ($p < 0.001$). There was a significant treatment by TG level interaction ($p < 0.05$). Participants with hypertriglyceridemia had significantly lower TG IAUC ($p < 0.05$) during the Active phase as compared to the Placebo phase (Fig. 2). For the normolipidemic group, no such difference was observed. There were no differences observed in other blood lipid or glucose levels between hypertriglyceridemic and normotriglyceridemic participants during either Active or Placebo phase.

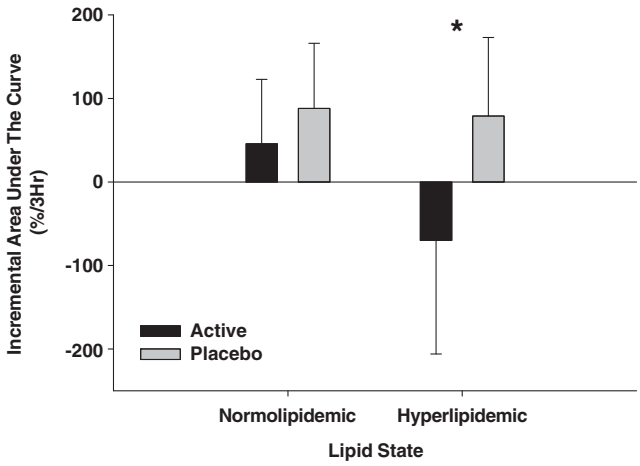

Fig. 2 – Comparisons of blood triglyceride incremental area under the curve according to baseline blood triglyceride levels at Placebo phase and Active phase. Normolipidemic, triglycerides < 1.69 mmol/L and hyperlipidemic, triglycerides >1.69 mmol/L. *: $p < 0.05$.

4. Discussion

This study examined the acute lipid and glucose responses to the consumption of a fat-containing meal with or without α-CD in healthy participants. It was different from previous studies in which these responses were examined after a number of weeks of consuming α-CD. A previous study has demonstrated that 1 g of α-CD binds and eliminates 9 g of dietary fat [21]. With 26 g of fat in the breakfast and 2 tablets (2 g) of α-CD consumed, it was expected that 18 g or about 70% of the fat in the meal would have been eliminated, thus reducing the postprandial blood TG levels. The results showed that the coincidental consumption of α-CD with a fat-containing meal was associated with a significant reduction in postprandial TG responses at 1, 2 and 3 h when compared to placebo. There were also significant differences in the area under the postprandial TG curve. It appears that taking α-CD with a fat-containing meal attenuated the hypertriglyceridemia induced by a high fat meal.

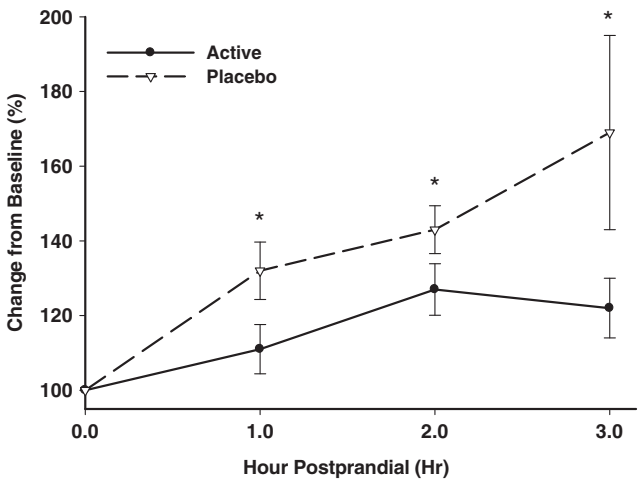

Fig. 1 – Blood triglyceride as percent of baseline value at 1, 2, and 3 h after consuming a fat containing breakfast during active and placebo phases. *: $p < 0.05$.

There were no significant differences in TC, LDL-C, and HDL-C. These were not expected to change since these levels are not known to change following a single meal. There were no significant reductions in glucose levels. Unlike the previous study that examined glycemic responses after a pure carbohydrate meal [12], the meal in the current study was a more typical mixed meal containing fat, protein and carbohydrate. Furthermore, the carbohydrate content in the breakfast was 30 g, much lower than the 50 g used by Buckley et al. [12] and the amount of α-CD consumed was also lower than in their study. These differences in design may explain the different results obtained between the current study and that of Buckley et al.

It appears that α-CD is effective at decreasing the amount of TG that is absorbed and released into the blood during the 3 h following a fat-containing meal. This has the effect of decreasing the amount of energy absorbed as well as decreasing the negative effects of consuming a high fat diet. The reduction in postprandial TG levels was especially significant in individuals with hypertriglyceridemia, as shown in Fig. 2. Considering the fact that both fasting and non-fasting blood TG levels are predictors for cardiovascular disease [22], reducing the postprandial TG levels in hypertriglyceridemic individuals may have clinical significance in

reducing the risk of cardiovascular disease. Using α-CD as a means to reduce postprandial TG levels deserves further attention since α-CD is a Generally Recognized As Safe dietary fiber that does not manifest the unwanted side effects of lipase inhibitors. Elevated postprandial TG is also a risk factor for insulin resistance and metabolic syndrome [22]; reducing postprandial TG levels may reduce these risks.

In summary, α-CD is a naturally occurring dietary fiber that, to date has demonstrated none of the known undesirable side effects of pharmaceutical weight loss products. α-Cyclodextrin use in this study of healthy adults was associated with significantly reduced acute postprandial responses in blood TG levels following consumption of a fat-containing meal. We found no difference in postprandial glucose levels when participants consumed a mixed meal of carbohydrate, fat and protein. The results from the current study and previous studies demonstrate that α-CD exerts both acute and long term benefits in lipid metabolism. These lower TG levels demonstrate reduced energy absorption and have implications for improving health by reducing the postprandial lipemia as well as the pro-inflammatory and pro-oxidative states that are associated with the consumption of high fat diets.

Author contributions

PAJ is responsible for the study design, acquisition of data, analysis of data, drafting of the manuscript and revision of manuscript for final content. K-LCJ and JDA contributed to critical discussion of the study design and analysis and critical revisions of the manuscript. EF and EE assisted with participant recruitment and the collection of data.

Conflict of interest

The study was not funded and there was no conflict of interest. Drs. Artiss and Jen are principals in ArtJen Complexus, USA.

REFERENCES

[1] Flegal KM, Carroll MD, Kit BK, Ogden CL. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999–2010. JAMA 2012;307(5):491–7, http://dx.doi.org/10.1001/jama.2012.39.
[2] Roger VL, Go AS, Lloyd-Jones DM, et al. Heart disease and stroke statistics—2012 update: a report from the American Heart Association. Circulation 2012;125(1):e2-220, http://dx.doi.org/10.1161/CIR.0b013e31823ac046.
[3] Garcia Diaz E, Martin Folgueras T. Systematic review of the clinical efficacy of sibutramine and orlistat in weigth loss, quality of life and its adverse effects in obese adolescents. Nutr Hosp 2011;26(3):451–7, http://dx.doi.org/10.1590/S0212-16112011000300004.
[4] Li MF, Cheung BM. Rise and fall of anti-obesity drugs. World J Diabetes 2011;2(2):19–23, http://dx.doi.org/10.4239/wjd.v2.i2.19.
[5] Brown L, Rosner B, Willett W, Sacks F. Cholesterol-lowering effects of dietary fiber: a meta-analysis. Am J Clin Nutr 1999;69:30–42.
[6] Pereira M, O'Reilly E, Augustsson K, et al. Dietary fiber and risk of coronary heart disease. Arch Intern Med 2004;164:370–6.
[7] Artiss J, Brogan K, Brucal M, Moghaddam M, Jen K-L. The effects of a new soluble dietary fiber on weight gain and selected blood parameters in rats. Metabolism 2006;55:195–202.
[8] Gallaher D, Gallaher C, Plank D. Alpha-cyclodextrin selectively increases fecal excretion of saturated fats. FASEB J 2007;21:A730.
[9] Wagner E, Jen K-L, Artiss J, Remaley A. Dietary alpha-cyclodextrin lowers LDL-C and alters plasma fatty acid profile in LDLr-KO mice on a high-fat diet. Metabolism 2008;57:1046–51.
[10] Comerford KB, Artiss JD, Jen KL, Karakas SE. The beneficial effects of alpha-cyclodextrin on blood lipids and weight loss in healthy humans. Obesity (Silver Spring) 2011;19(6):1200–4, http://dx.doi.org/10.1038/oby.2010.280.
[11] Grunberger G, Artiss J, Jen K-LC. The benefits of early intervention in obese diabetic patients with FBCx™ — a new dietary fibre. Diabetes Metab Res Rev 2007;23:56–62.
[12] Buckley J, Thorp A, Murphy K, Howe P. Dose-dependent inhibition of the post-prandial glycaemic response to a standard carbohydrate meal following incorporation of alpha-cyclodextrin. Ann Nutr Metab 2006;50:108–14.
[13] Esposito K, Giugliano D. Diet and inflammation: a link to metabolic and cardiovascular diseases. Eur Heart J 2006;27(1):15–20, http://dx.doi.org/10.1093/eurheartj/ehi605.
[14] O'Keefe JH, Gheewala NM, O'Keefe JO. Dietary strategies for improving post-prandial glucose, lipids, inflammation, and cardiovascular health. J Am Coll Cardiol 2008;51(3):249–55, http://dx.doi.org/10.1016/j.jacc.2007.10.016.
[15] Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. JAMA 2007;298(3):309–16, http://dx.doi.org/10.1001/jama.298.3.309.
[16] Nordestgaard BG, Benn M, Schnohr P, Tybjaerg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. JAMA 2007;298(3):299–308, http://dx.doi.org/10.1001/jama.298.3.299.
[17] Jackson KG, Poppitt SD, Minihane AM. Postprandial lipemia and cardiovascular disease risk: interrelationships between dietary, physiological and genetic determinants. Atherosclerosis 2012;220(1):22–33, http://dx.doi.org/10.1016/j.atherosclerosis.2011.08.012.
[18] Mora S, Rifai N, Buring JE, Ridker PM. Fasting compared with nonfasting lipids and apolipoproteins for predicting incident cardiovascular events. Circulation 2008;118(10):993–1001, http://dx.doi.org/10.1161/CIRCULATIONAHA.108.777334.
[19] Huff MW. Dietary cholesterol, cholesterol absorption, postprandial lipemia and atherosclerosis. Can J Clin Pharmacol 2003;10(Suppl. A):26A–32A.
[20] Shephard MD, Mazzachi BC, Shephard AK. Comparative performance of two point-of-care analysers for lipid testing. Clin Lab 2007;53(9–12):561–6.
[21] Artiss J, Jen K-L. Dietary fibers and body weight management: a brief review. J Clin Ligand Assay 2005;28:23–6.
[22] Kannel WB, Vasan RS. Triglycerides as vascular risk factors: new epidemiologic insights. Curr Opin Cardiol 2009;24(4):345–50, http://dx.doi.org/10.1097/HCO.0b013e32832c1284.