

行政院國家科學委員會專題研究計畫 成果報告

不同升糖指數的飲食與運動對脂肪代謝的影響

計畫類別：個別型計畫

計畫編號：NSC94-2413-H-028-003-

執行期間：94年08月01日至95年07月31日

執行單位：國立臺灣體育學院運動健康科學學系

計畫主持人：巫錦霖

計畫參與人員：蔡怡嫻

報告類型：精簡報告

報告附件：出席國際會議研究心得報告及發表論文

處理方式：本計畫可公開查詢

中 華 民 國 95 年 10 月 31 日

行政院國家科學委員會補助專題研究計畫 成果報告

不同升糖指數的飲食與運動對脂肪代謝的影響

The effect of different glycemic index diet and exercise on fat metabolism

計畫類別： 個別型計畫 整合型計畫

計畫編號：NSC 94-2413-H-028-003-

執行期間： 94 年 8 月 1 日至 95 年 7 月 31 日

計畫主持人：巫錦霖

計畫參與人員： 蔡怡姍

成果報告類型(依經費核定清單規定繳交)： 精簡報告 完整報告

本成果報告包括以下應繳交之附件：

- 赴國外出差或研習心得報告一份
- 赴大陸地區出差或研習心得報告一份
- 出席國際學術會議心得報告及發表之論文各一份
- 國際合作研究計畫國外研究報告書一份

處理方式：除產學合作研究計畫、提升產業技術及人才培育研究計畫、列管計畫及下列情形者外，得立即公開查詢

涉及專利或其他智慧財產權， 一年 二年後可公開查詢

執行單位：國立臺灣體育學院運動健康科學系

中 華 民 國 九 十 五 年 十 月 三 十 一 日

不同升糖指數的飲食與運動對脂肪代謝的影響-中文摘要

以升糖指數(glycemic index, GI) 來分類食物中碳水化合物的來源已被廣泛的運用。再過去十年來的流行病學的研究顯示，攝取高GI 碳水化合物的飲食，與心血管疾病，糖尿病，肥胖症，以及一些癌症有密切的關係。上述的這些慢性疾病，與脂肪代謝異常有高度的相關。這有可能是因為攝取高GI 的碳水化合物，導致分泌大量的胰島素，進而促進脂肪的貯存，然而這些流行病學的研究缺乏臨床的實驗來證實。另外，在一些研究指出，低脂高碳水化合物的飲食配合步行運動(brisk walking)，能有較佳的脂肪耐性測驗表現，但是這些飲食的設計，並未考慮其碳水化合物來源的生理效應。本研究探討不同GI 碳水化合物的飲食與運動對於脂肪代謝的影響。六名受測者接受兩次連續三天分別隨機的給予高或低GI 的飲食，飲食控制期結束後第四天實施口服脂肪耐性測驗(oral fat tolerance test)。另外兩次給予受測者相同的飲食控制，並且在飲食控制期，以50%VO₂max 走路運動一小時。結果顯示運動能夠有效的降低餐後血液中的三酸甘油脂濃度，但是在低GI 或者高GI 之間並沒有顯著差異。而低GI 的飲食加上運動能有較佳的胰島素敏感度。關鍵字：升糖指數，運動

The effect of different glycemic index diet and exercise on fat metabolism

Glycemic index (GI) as a classification of carbohydrate has been widely received notice. In the past ten years, many epidemiological studies have been shown strong connection between high GI carbohydrate diet and cardiovascular diseases, diabetes, obesity, and cancers. The above chronic diseases and symptoms are highly related to abnormal fat metabolism, especially on obesity objects. This may be due to high GI carbohydrate induces massive insulin secretion to facilitate fat storage. However, these epidemiological studies are in short of clinical or experimental trials on the effect of GI diet on the fat metabolism. In addition, several studies have been shown the positive effects of low fat diet and exercise on fat tolerance performance. However, these studies did not consider the nature of carbohydrate source. Six healthy male subjects with normal weight was recruited in each study. Subjects consumed either high or low GI diet without exercise (HGI, LGI) or with exercise (HGI+EX, LGI+EX) at 50% VO₂max for three days in a randomized order before undertaking the fat tolerance test (OFTT). The results showed that exercise trials were effectively attenuated postprandial lipemia. Glycemic index carbohydrate effect were no observed. However, LGI+EX may improve insulin sensitivity.

Key words: glycemic index, exercise

Introduction

The glycaemic index (GI) was initially proposed in 1981 (Jenkins et al. 1981). The GI of a food is defined as the incremental area under the blood glucose response curve following the intake of carbohydrate-rich food containing 50g available carbohydrate (CHO), expressed as a percent of the incremental area under the curve (IAUC) of blood glucose response following the intake of 50g CHO from the standard food (glucose or white bread, GI=100) as applied to the same individual.

The GI of a mixed meal can be calculated from the contribution of single food to the whole meal. The mixed meal GI value calculation method was proposed by Wolever and Jenkins (1986) as follows,

TC: Total carbohydrate content of meal (in gram)

GI_a, GI_b, GI_c, ...: GI value of individual food

W_a, W_b, W_c, ...: Carbohydrate content of each food (in gram)

Total GI of the meal = $(W_a/TC) \cdot GI_a + (W_b/TC) \cdot GI_b + (W_c/TC) \cdot GI_c + \dots$.

In a report published in 1998, the FAO and WHO recommended the use of the GI as a method of categorizing CHO foods because the GI provides information on the likely physiological effects of ingested CHO. The GI has been widely used for clinical nutrition, especially for diabetics, and more recently in health promotion, because of the concerns over obesity, cardiovascular disease and specific cancers.

Recently, the GI has been used as one of the key factors for in prevention of obesity, Type II diabetes, hyperlipidaemia, cardiovascular diseases and some specific cancers in the general population (Brand-Miller et al. 2002; Pi-Sunyer, 2002; Leeds, 2002; Jenkins et al. 2002; Liu and Willett, 2002; Willett et al. 2002). It has been suggested that high GI foods are detrimental to health due to postprandial hyperglycaemia and hyperinsulinaemia which can lead to insulin resistance (Brand-Miller 2003).

In addition to epidemiological studies, nutritional intervention studies indicate that a low GI diet might be beneficial in maintaining normal blood lipid profiles in healthy subjects, hyperlipidaemic subjects, diabetes mellitus patients, and patients with CHD (Jenkins et al.

1987; Wolever et al. 1992a; Frost et al. 1996; Frost et al. 1998; Jarvi et al. 1999). Ingestion of low GI foods may be beneficial for weight control in two ways: by promoting satiety and by promoting fat oxidation at the expense of CHO oxidation (Holt et al. 1992; Holt et al. 1996; Wee et al.1999; Ludwig et al. 1999; Brand-Miller et al. 2002; Ludwig, 2000; Febbraio et al. 2000). Some long-term studies in human and animal models have shown the benefit of low GI foods for weight control in obese populations (Wolever et al. 1992b; Slabber et al. 1994; Kabir et al. 1998; Pawlak et al. 2001; Spieth et al. 2000).

Several studies have shown that consuming low GI carbohydrate pre-exercise meal elicit higher rate of fat oxidation when compared to ingestion of high GI carbohydrate meal during postprandial period and during subsequent exercise (Febbraio et al. , 1996; Spark et al. 1998; Kirwan et al. 1998; Wee et al., 1999; Kirwan et al. 2000; Wu et al., 2003). These evidences might indicate that short term GI diet possibly change metabolic responses especially on fat metabolism. Recently, some studies demonstrated that the short term dietary manipulation could significantly alter fat metabolism (Hardman et a, 1998; Gill et al., 2000, 2001; Koutsari and Hardman, 2001). However, these studies did not consider the GI value of carbohydrate in the diet. Therefore, the study was to investigate the effect of different GI carbohydrate diets and exercise on the fat metabolism. The study may provide a clinical evidence to support those epidemiological studies on the effect of low GI diet on fat metabolism.

An increased plasma concentration of high-density lipoprotein (HDL) cholesterol is the most consistent alteration to the lipid profile evident following a program of regular aerobic exercise (Durstine and Haskell, 1994; Leon and Sanchez, 2001). Kurtsari and Hardman (2001) demonstrated a three-day high-carbohydrate diet (70% energy from carbohydrate) which increased the postprandial triglycerides response to oral fat tolerance test by 35% when compared to an isoenergetic three-day mixed diet (46% energy from carbohydrate). However, 30 min of daily moderate exercise for three days during the high-carbohydrate diet intervention prevented the carbohydrate-induced augmentation in postprandial lipemia. Several studies also demonstrated that the effects of daily exercise (60 min brisk walking) might oppose the postprandial lipemia (Gill and Hardman, 1998; Koutsari et al., 2001; Gill et al., 2000, 2001). However, these studies did not consider the GIs of the high-carbohydrate diet. This study was aimed to examine the effect of daily exercise with different GI diets on the fat metabolism.

METHODS

Subjects

Six healthy, inactive subjects with normal body fat were recruited in this study. The protocol has been approved by the National Taiwan College of Physical Education Human Ethical Advisory Committee and all subjects were given their written informed consent.

Experimental design

Subjects were asked to consume either high GI or low GI diet with or without exercise for three days in a randomized order before undertaking the oral fat tolerance test (OFTT). Each trial was separated by at least one week. On the first day of the dietary control period, subjects reported to the laboratory after 10-12 hours overnight fast. After obtaining subject's height and nude body mass, the dietary control period then began. All the foods were served by the laboratory during the three-day dietary control. Three days later, on the day four, subjects were asked to report to laboratory for the OFTT. Trial 1 and 2 was non-exercised trial (HGI and LGI), and trial 3 and 4 was exercised trial (HGI+EX and LGI+EX) .

Trial 1 and 2	HGI or LGI diet without exercise 3days	OFTT
Trial 3 and 4	HGI or LGI diet with exercise	OFTT
Day 1-3		Day 4

Fig 1. Trial Scheme.

Dietary control

Each subject received an isocaloric and isomacronutrient diet containing either high GI or low GI carbohydrates during three-day dietary control. The energy content depends on subject's body mass which was set at 35 kcal/kg body mass and the macronutrients distribution is 55% carbohydrate, 15% protein, and 30% fat. As the GI values for the mixed diet is calculated by the method proposed by Wolever and Jenkins (1986), the GI values for high GI and low GI diet is 80 and 40 respectively. All the diet was prepared by the laboratory and subjects collected or consumed their meal at the laboratory. All foods used in the diet were purchased in the same supermarket and the commercial products' macronutrient contents were based on the information provided by the manufactures. Subjects were asked to consume the foods served from the laboratory only. Water consumption was *ad libitum* during the dietary control period. During the dietary control period, subjects were asked to avoid any heavy physical activity or exercise during non-exercise or exercise trials.

Oral fat tolerance test meal

Oral fat tolerance test meal comprised whipping cream, cheese, mixed nuts, muesli, white bread and butter. The amount of the macronutrients and energy were given to subjects according to their body mass (1.2g/kg fat, 1.1g/kg carbohydrate, 0.33g/kg protein, and 66 kJ/kg) (Hardman et al. 1998). All foods were purchased at the same supermarket and the macronutrient contents were provided by the manufacturers.

Preliminary exercise tests

After the first two non-exercise-trial, one week before the exercise-trial, subjects were asked to visit laboratory for two preliminary exercise tests: maximal oxygen uptake ($\dot{V}O_{2\max}$) test and submaximal oxygen uptake test.

Maximal Oxygen Uptake Test: Uphill treadmill walking was used to determine the $\dot{V}O_{2\max}$ of each subject. The protocol is modified from Taylor et al (1955). The speed of treadmill was kept constant throughout the test and the inclination will be increased by 2.5 % from an initial 3.5% every 3 min until the subjects reach volitional fatigue. Expired air samples will be measured using a gas analyzer (V_{\max} , USA).

Submaximal Walking Test: A 16 min continuous submaximal walking test, consisting of four stages, was used to determine the relationships between walking intensity and oxygen uptake ($\dot{V}O_2$). Expired air samples was measured for determination of $\dot{V}O_2$. A linear regression equation was then calculated for the relationship between $\dot{V}O_2$ and walking intensity from the 16 min submaximal walking test. The data was to determine the walking intensity corresponding to 50% of each subject's $\dot{V}O_{2\max}$ to be used in the exercise-trials.

On a separate day, one week before the trial, subjects was asked to complete a 30 min walk corresponding to 50% $\dot{V}O_{2\max}$ in order to confirm the relative exercise intensity and to fully familiarize them with treadmill walking and the experimental procedures.

Protocol

After the three-day dietary control (with or without exercise at 50% $VO_{2\max}$), subjects reported to the laboratory after a 12 hour overnight fast in the morning on the fourth day. The subjects were to void before nude body mass was obtained. Body mass of the subjects was

measured using a calibrated balance (Avery 3306 ABV) and height was measured using a stadiometer. After weighing, a cannula was inserted into an antecubital vein while the subject is lying on an examination couch.

Basal blood and gas samples were obtained after the subject has sat for 10 min. The oral fat tolerance test meal was then served, which the subject was asked to consume within 20 min. After finishing the test meal, the six hours postprandial period starts. During the postprandial period, subjects were asked to remain seated, avoiding any unnecessary physical activity. During the postprandial period, water was provided ad libitum in the first trial and then replicated in the next three oral fat tolerance tests.

Ambient temperature and relative humidity was recorded at 30 min intervals during the postprandial period using a hydrometer (Zeal, England). Temperature was maintained between 25-28°C in all trials. All trials were held in the same laboratory under the similar environmental condition.

Blood sample collection and analysis

Blood samples were obtained from a cannula (Venflon 20G, Sweden) in the antecubital vein, which was connected to a 3-way stopcock (Connecta Ltd., Sweden) with a 10cm extension tube. The cannula was frequently flushed with a sterile sodium chloride solution (0.9% w/v) to keep it patent throughout the experiment. Blood samples were obtained with subjects in the sitting position during the postprandial period. In addition to the basal blood sample, further samples were obtained at 30min, 60min, 120min, 180min, 240min, 300min and 360min during the postprandial period.

Ten millilitres of blood was collected at each sampling time. An 8ml blood sample was put into an EDTA tube and centrifuged at 1500g' for 10 min to obtain plasma. The remaining blood (~2ml) was placed into a non-heparinized tube and left to clot for 1 hour and then centrifuged at 1500g' for 10 min for obtaining serum samples. Plasma and serum samples was aliquoted and then stored at -70°C for later analysis. The plasma was analyzed for free fatty acid (FFA) (ACS-ACOD method, Wako NEFA C, Germany), triglycerides (GPO-PAP method, Randox, Ireland), glucose (GOD-PAP method, Randox, Ireland), cholesterol, high density lipoprotein-cholesterol (HDL-C). The serum insulin concentration was analysed by

enzyme-linked immunosorbent assay (ELISA) by an ELISA reader (Ultramark Microplate System, Bio-Rad Ltd. , USA).

Statistical analysis

All results are presented as mean \pm SEM. A two-way ANOVA (time and treatment) for repeated measures was used to analyze differences in the physiological and biochemical responses. The Tukey post-hoc test was applied to locate the differences between means when a significant difference was found. Student's paired t-test was used for non-time dependent variables. Significance was set at the 0.05 level of confidence.

RESULTS AND DISCUSSION

Plasma triacylglycerol (TG)

Plasma triacylglycerol (TG) concentrations were significantly higher during OFTT in the non-exercise trials, LGI and HGI, when compared to the exercised trials, HGI+EX and LGI+EX (Fig 2). The incremental area under the plasma triacylglycerol curve (IAUC) was significantly higher in the LGI and HGI than in the LGI+EX and HGI+EX (Fig 3).

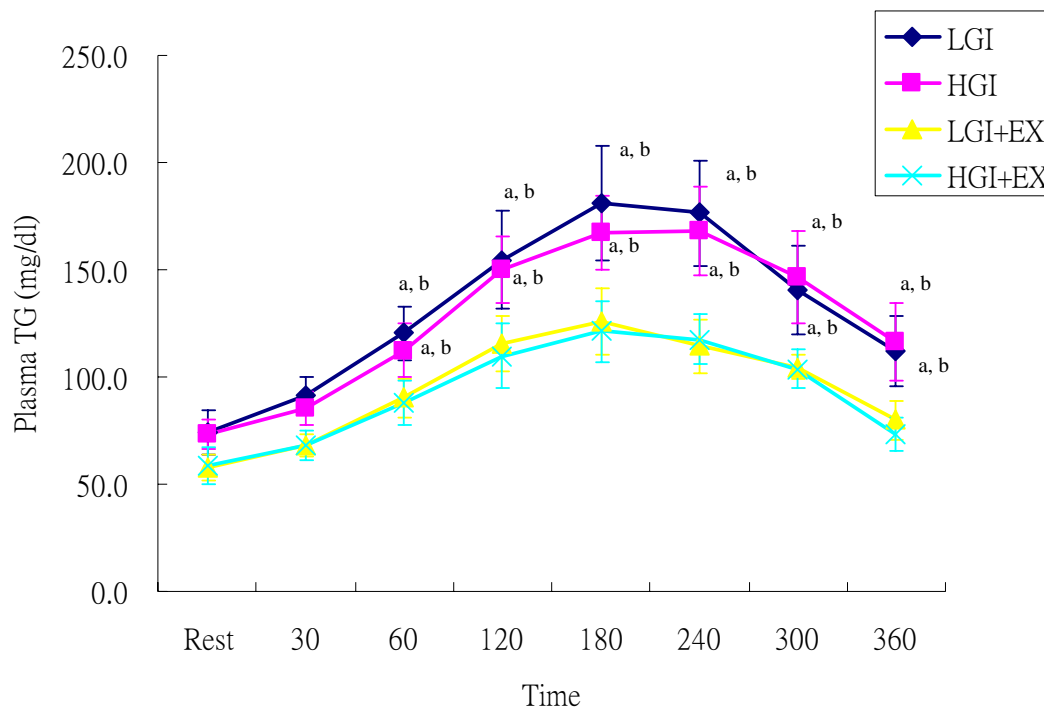


Fig 2. Plasma triacylglycerol (TG) concentrations during oral fat tolerance test (OFTT).

LGI: low glycemic index diet trial. HGI: high glycemic index diet trial. LGI+EX: low glycemic index diet + exercise trial. HGI+EX: high glycemic index diet + exercise trial.

a: Significantly different from LGI+EX

b: Significantly different from HGI+EX

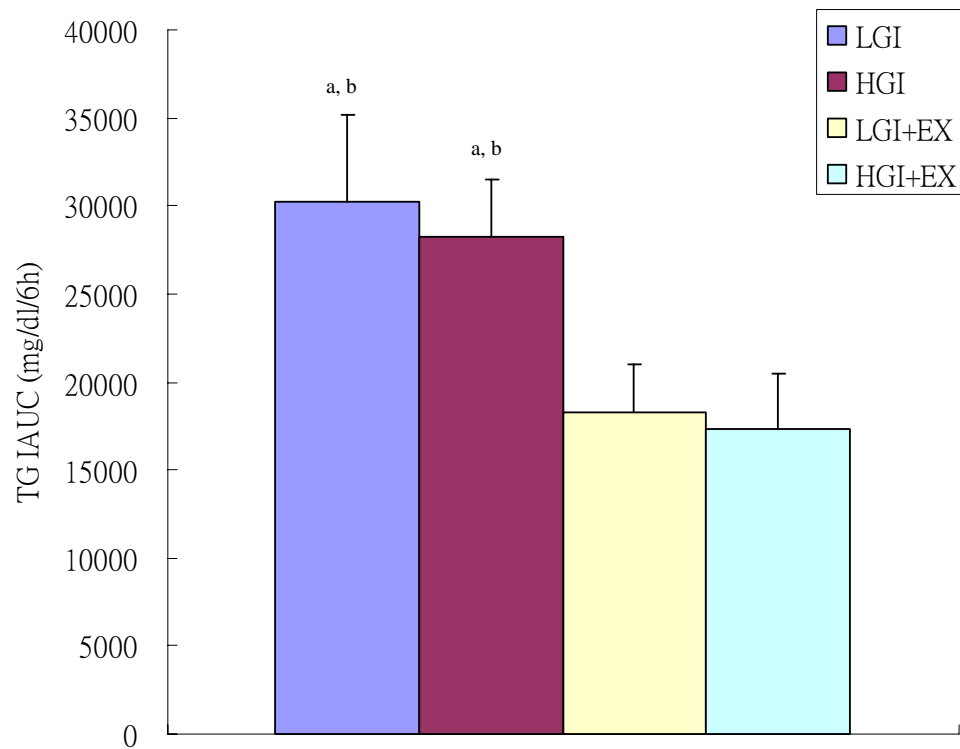


Fig 3. Plasma triacylglycerol (TG) concentrations incremental area under the curve (IAUC) during oral fat tolerance test (OFTT).

LGI: low glycemic index diet trial. HGI: high glycemic index diet trial. LGI+EX: low glycemic index diet + exercise trial. HGI+EX: high glycemic index diet + exercise trial.

a: Significantly different from LGI+EX

b: Significantly different from HGI+EX

Plasma glucose

Plasma glucose concentrations were elevated and peaked at 30 min and decreased gradually during OFTT. However, there were no significant differences between trials during OFTT (Fig 4)

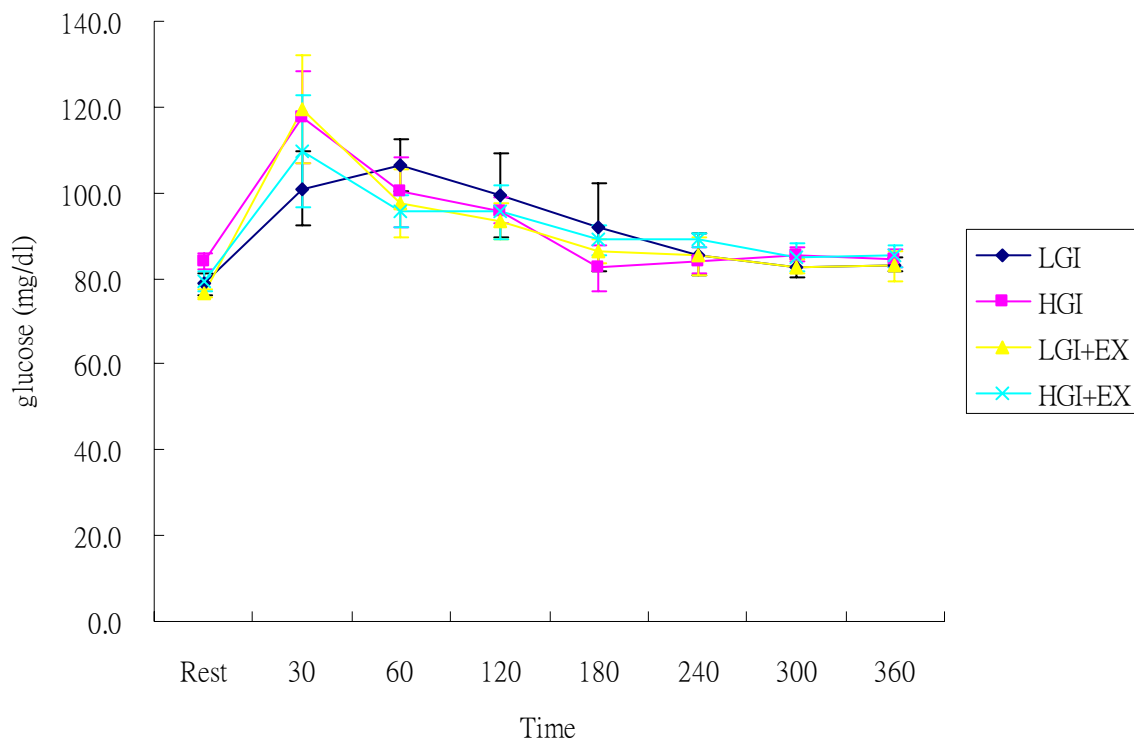


Fig 4. Plasma glucose concentrations during oral fat tolerance test (OFTT).

LGI: low glycemic index diet trial. HGI: high glycemic index diet trial. LGI+EX: low glycemic index diet + exercise trial. HGI+EX: high glycemic index diet + exercise trial.

Serum insulin

Serum insulin concentrations were significantly higher in HGI trial than LGI, LGI+EX and HGI+EX trials at 30 min during OFTT ($p < 0.05$, Fig 5). We calculated the incremental area under the insulin curve (insulin IAUC). The results showed that the LGI+EX were significantly lower than HGI, LGI and HGI+EX ($p < 0.05$, Fig 6)

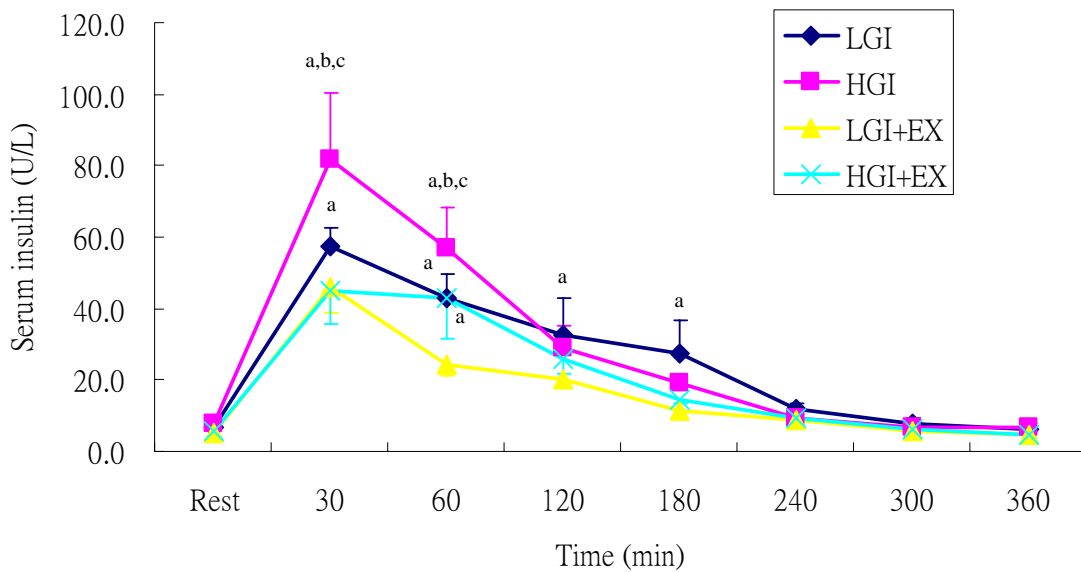


Fig 5. Serum insulin concentrations during oral fat tolerance test (OFTT).

LGI: low glycemic index diet trial. HGI: high glycemic index diet trial. LGI+EX: low glycemic index diet + exercise trial. HGI+EX: high glycemic index diet + exercise trial.

a: Significantly different from LGI+EX

b: Significantly different from HGI+EX

c: Significantly different from LGI

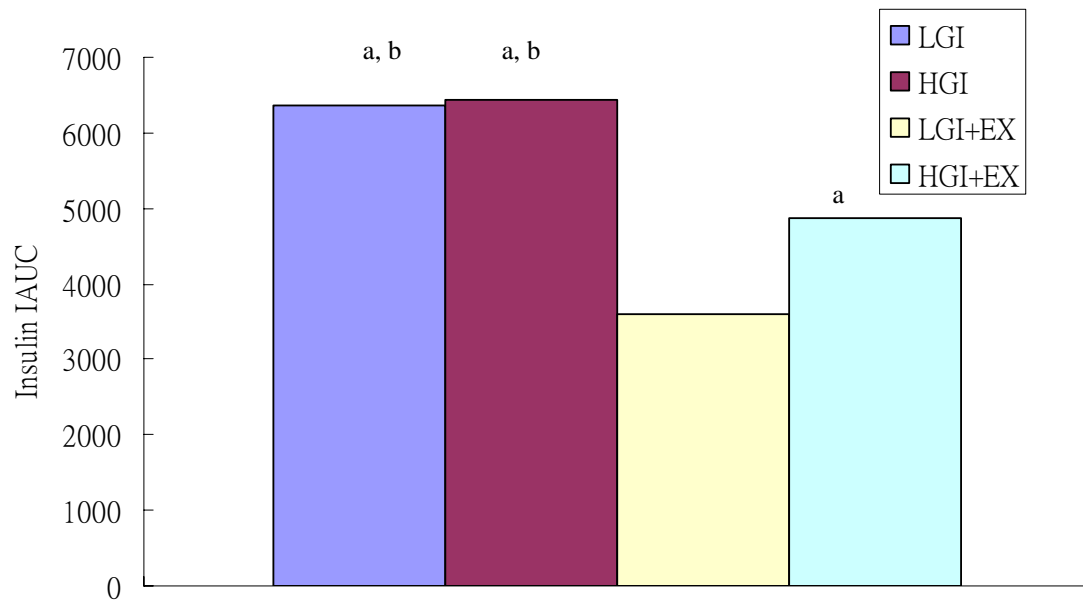


Fig 6. Serum insulin concentrations incremental area under the curve (IAUC) during oral fat tolerance test (OFTT).

LGI: low glyceic index diet trial. HGI: high glyceic index diet trial. LGI+EX: low glyceic index diet + exercise trial. HGI+EX: high glyceic index diet + exercise trial.

a: Significantly different from LGI+EX

b: Significantly different from HGI+EX

Plasma cholesterol (TC), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C)

Plasma TC, HDL-C and LDL-C concentrations were showed in Fig 7. There were no significant differences between trials.

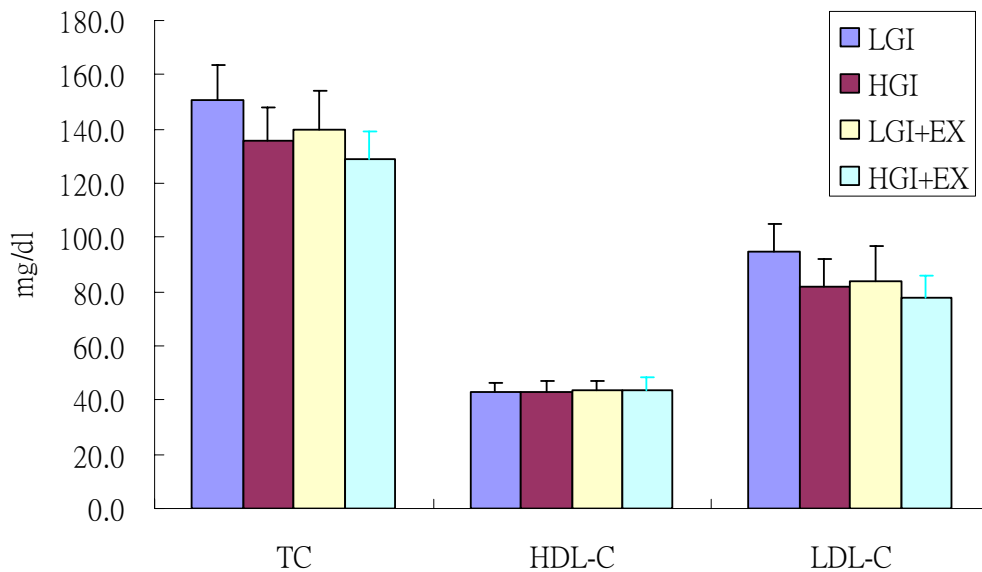


Fig 7. Plasma total cholesterol (TC), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) concentrations at fasting state.

LGI: low glycemic index diet trial. HGI: high glycemic index diet trial. LGI+EX: low glycemic index diet + exercise trial. HGI+EX: high glycemic index diet + exercise trial.

Conclusion

The current data suggested that consecutive 1 hour exercise for 3 days was significantly attenuated postprandial lipemia. The short term low glycemic index diets showed no influence on the postprandial lipid profile. Nevertheless, the low GI diet plus exercise might improve insulin sensitivity.

References:

Barter, P.J., Rye, K.A.. (1996) High density lipoproteins and coronary heart disease. *Atherosclerosis* **121**,1—12.

Brand-Miller, J.C., Holt, S.H., Pawlak, D.B. and McMillan, J. (2002) Glycemic index and obesity. *Am J Clin Nutr* **76**, 281S-5S.

Brand-Miller, J.C. (2003) Glycemic load and chronic disease. *Nutr Rev* **61**, S49-55.

Durstine, J.L., Haskell, W.L.. (1994) Effects of exercise training on plasma lipids and lipoproteins. *Exerc Sports Sci Rev* **22**, 477—521.

FAO/WHO Expert consultation (1998) Carbohydrate in human nutrition. Geneva: Food and Agriculture Organization, World Health Organization. *FAO Food and Nutrition* **66**.

Febbraio, M.A., Keenan, J., Angus, D.J., Campbell, S.E. and Garnham, A.P. (2000) Preexercise carbohydrate ingestion, glucose kinetics, and muscle glycogen use: effect of the glycemic index. *J Appl Physiol* **89**, 1845-51.

Frost, G., Brynes, A. and Leeds, A. (1999) Effect of large bowel fermentation on insulin, glucose, free fatty acids, and glucagon-like peptide 1 (7-36) amide in patients with coronary heart disease. *Nutrition* **15**, 183-8.

Gill, J.M.R., Murphy, M.H., Hardman, A.E.. (1998) Postprandial lipemia: effects of intermittent versus continuous exercise. *Med Sci Sports Exerc* **30**,1515—20.

Gill, J.M.R., Hardman A.E.. (2000) Postprandial lipemia: effects of exercise and energy intake restriction compared. *Am J Clin Nutr* **71**,465—71.

Gill, J.M.R., Mees, G.P., Frayn, K.N., Hardman, A.E. (2001) Moderate exercise, postprandial lipaemia and triacylglycerol clearance. *Eur J Clin Invest* **31**, 201—7.

Holt, S., Brand, J., Soveny, C. and Hansky, J. (1992) Relationship of satiety to postprandial

glycaemic, insulin and cholecystokinin responses. *Appetite* **18**, 129-41.

Holt, S.H., Brand Miller, J.C. and Petocz, P. (1996) Interrelationships among postprandial satiety, glucose and insulin responses and changes in subsequent food intake. *Eur J Clin Nutr* **50**,788-97.

Jarvi, A.E., Karlstrom, B.E., Granfeldt, Y.E., Bjorck, I.E., Asp, N.G. and Vessby, B.O. (1999) Improved glycemic control and lipid profile and normalized fibrinolytic activity on a low-glycemic index diet in type 2 diabetic patients. *Diabetes Care* **22**, 10-8.

Jenkins, D.J., Wolever, T.M., Taylor, R.H., Barker, H., Fielden, H., Baldwin, J.M., Bowling, A.C., Newman, H.C., Jenkins, A.L. and Goff, D.V. (1981) Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr* **34**, 362-6.

Jenkins, D.J., Kendall, C.W., Augustin, L.S., Franceschi, S., Hamidi, M., Marchie, A., Jenkins, A.L. and Axelsen, M. (2002) Glycemic index: overview of implications in health and disease. *Am J Clin Nutr* **76**, 266S-73S.

Jenkins, D.J., Wolever, T.M., Kalmusky, J., Guidici, S., Giordano, C., Patten, R., Wong, G.S., Bird, J.N., Hall, M., Buckley, G. and et, a.l. (1987) Low-glycemic index diet in hyperlipidemia: use of traditional starchy foods. *Am J Clin Nutr* **46**, 66-71.

Kabir, M., Rizkalla, S.W., Champ, M., Luo, J., Boillot, J., Bruzzo, F. and Slama, G. (1998) Dietary amylose-amylopectin starch content affects glucose and lipid metabolism in adipocytes of normal and diabetic rats. *J Nutr* **128** , 35-43.

Kirwan, J.P., O'Gorman, D. and Evans, W.J. (1998) A moderate glycemic meal before endurance exercise can enhance performance. *J Appl Physiol* **84**, 53-9.

Kirwan, J.P., O'Gorman, D.J., Cyr-Campbell, D., Campbell, W.W., Yarasheski, K.E. and Evans, W.J (2001) Effects of a moderate glycemic meal on exercise duration and substrate utilization. *Med Sci Sports Exerc* **33**, 1517-23.

Koutsari, C., Hardman, A.E.. (2001) Exercise prevents the augmentation of postprandial

lipaemia attributable to a low-fat high-carbohydrate diet. *Br J Nutr* **86**, 197—205.

Koutsari, C., Karpe, F., Humphreys, S.M., Frayn, K.N., Hardman, A.E.. (2001) Exercise prevents the accumulation of triglyceride-rich lipoproteins and their remnants seen when changing to a high-carbohydrate diet. *Arterioscler Thromb Vasc Biol* **21**,1520—5.

Leeds, A.R. (2002) Glycemic index and heart disease. *Am J Clin Nutr* **76**, 286S-9S.

Leon AS, Sanchez OA. (2001) Response of blood lipids to exercise training alone or combined with dietary intervention. *Med Sci Sports Exerc* **33**, S502—15.

Liu, S. and Willett, W.C. (2002) Dietary glycemic load and atherothrombotic risk. *Curr Atheroscler Rep* **4**, 454-61.

Ludwig, D.S. (2000) Dietary glycemic index and obesity. *J Nutr* **130**, 280S-283S.

Ludwig, D.S., Majzoub, J.A., Al-Zahrani, A., Dallal, G.E., Blanco, I. and Roberts, S.B. (1999) High glycemic index foods, overeating, and obesity. *Pediatrics* **103**, E26

Pawlak, D.B., Bryson, J.M., Denyer, G.S. and Brand-Miller, J.C. (2001) High glycemic index starch promotes hypersecretion of insulin and higher body fat in rats without affecting insulin sensitivity. *J Nutr* **131**, 99-104.

Pi-Sunyer, F.X. (2002) Glycemic index and disease. *Am J Clin Nutr* **76**, 290S-8S.

Sparks, M.J., Selig, S.S. and Febbraio, M.A. (1998) Pre-exercise carbohydrate ingestion: effect of the glycemic index on endurance exercise performance. *Med Sci Sports Exerc* **30**, 844-9.

Slabber, M., Barnard, H.C., Kuyl, J.M., Dannhauser, A. and Schall, R. (1994) Effects of a low-insulin-response, energy-restricted diet on weight loss and plasma insulin concentrations in hyperinsulinemic obese females. *Am J Clin Nutr* **60**, 48-53.

Spieth, L.E., Harnish, J.D., Lenders, C.M., Raezer, L.B., Pereira, M.A., Hangen, S.J. and Ludwig, D.S. (2000) A low-glycemic index diet in the treatment of pediatric obesity. *Arch Pediatr Adolesc Med* **154**, 947-51.

Wee, S.L., Williams, C., Gray, S. and Horabin, J. (1999) Influence of high and low glycemic index meals on endurance running capacity. *Med Sci Sports Exerc* **31**, 393-9.

Willett, W., Manson, J. and Liu, S. (2002) Glycemic index, glycemic load, and risk of type 2 diabetes. *Am J Clin Nutr* **76**, 274S-80S.

Wolever, T.M. and Jenkins, D.J. (1986) The use of the glycemic index in predicting the blood glucose response to mixed meals. *Am J Clin Nutr* **43**, 167-72.

Wolever, T.M., Jenkins, D.J., Vuksan, V., Jenkins, A.L., Buckley, G.C., Wong, G.S. and Josse, R.G. (1992a) Beneficial effect of a low glycaemic index diet in type 2 diabetes. *Diabet Med* **9**, 451-8.

Wolever, T.M., Jenkins, D.J., Vuksan, V., Jenkins, A.L., Wong, G.S. and Josse, R.G. (1992b) Beneficial effect of low-glycemic index diet in overweight NIDDM subjects. *Diabetes Care* **15**, 562-4.

Wu, C., Nicholas, C., Williams, C., Took, A. and Hardy, L. (2003) The influence of high-carbohydrate meals with different glycaemic indices on substrate utilisation during subsequent exercise. *Brit J Nutr* **90**: 1049-1056.

計畫成果自評

Although we experienced the difficulty of subject recruitment and shortage of fund on some hormone assays, the project was successfully complete. We have already published part of the data in the 32th Taiwan Nutrition Society Annual meeting at Hungkuang University in Taichung.(不同升糖指數的飲食與運動對於餐後血脂肪濃度之影響，中華民國營養學會第三十二屆年會暨學術研討會，PD-1, pp 161)

The current obtained data is preparing a manuscript to submit to *American Journal Clinical Nutrition* (SCI Journal, 2005 IF=5.853).

出國報告書

參加 2006 年第十一屆歐洲大學運動科學年會(The 11th Annual Congress of European College of Sport Science)出國報告書



服務機關：國立台灣體育學院運動健康科學學系

出 國 人

職 稱：副教授

姓 名：巫錦霖

出國地區：Lausanne, Switzerland

出國期間：7/5-7/8, 2005

目 錄

一、前言：會議解說與參加目的.....	2
二、舉辦城市與地點簡介.....	5
三、會議特色.....	7
四、學習的成果：重點發展領域.....	9
五、結語.....	10
附錄一：發表論文海報.....	11

一、前言：會議解說與參加目的

每年一度的運動科學界的盛事—歐洲大學運動科學學會（ECSS）第十一屆年會（The 11th Annual Congress of European College of Sport Science）—今年假有奧運首都的瑞士洛桑（Lausanne）舉行。

按照傳統慣例，此次會議共計四天，於7月5日展開序幕而於7月8日結束。此次會議規模龐大，共計超過2000名來自世界各地學者與會，超過一千篇運動科學相關論著與研究論文被發表，而參與的人員來自全球的66個國家，堪稱國際上運動科學的盛會。本人有幸代表國立台灣體育學院參與此一學術盛會，以下就本人於此研討會之參與過程，會議內容，課程，與參訪活動做一摘要報告。



圖：大會會場



圖：報到處 (Registration desk)

由於參加的人數眾多，報到處的規模也相當大，是根據姓氏的英文字母來排列，雖然報名參與的人數非常多，但是主辦單位仍然能夠有條不紊的讓與會者迅速，而且順利的完成報到手續。



圖：洛桑教堂



圖：與 Professor Michael Glesson 及英國學者於會場合照



圖：會場一隅

二、舉辦城市與地點簡介

第十一屆歐洲大學運動科學學會(ECSS)年會，今年假夙有奧運之首都—洛桑 (Lausanne) 的舉行。洛桑的位置正於歐洲的心臟，距離日內瓦僅 40 英里，位於日內瓦湖畔，是一個依山傍水的美麗城市，不僅如此，洛桑更是許多運動項目的總會設立的地點，而且 IOC 的博物館正設置在洛桑。



圖：日內瓦湖畔



圖：鳥瞰洛桑



圖：開幕典禮



圖：開幕酒會

三. 會議特色

此次研討會極具規模，整個會議共持續四天，在議程方面，每天都根據不同的專題進行口頭發表(oral presentation)，海報發表(posters)，各領域的主要專題報告(Keynote speakers in different symposia)，以及 workshops 等特別演講。由於發表的論文數量非常多，於同一時間，同時有 4-6 個演講廳同時舉行 Oral Presentation 或者專題演講，並且在海報發表方面，分為四天的發表，發表論文數量超過 1200 篇，因此參加者必須準備充分，並依大會手冊及論文摘要，只能選擇去參加某一個特定的講演，或者觀看海報的發表。

此外大會為了讓更多的年輕的運動科學研究者參與，特別安排了設有青年研究論文獎，由主辦單位挑出本次大會中，優秀的論文，發給優秀論文以及研究獎金獎勵。



圖：會場外觀



圖：會場外一隅

此外大會並且安排了有廠商的展覽，參加展覽的廠商，運動相關產業，運動科學儀器，運動傷害治療，運動營養補充劑，運動相關書籍書商等等，總共有超過 100 個參展廠商，規模之相當龐大。

在純學術活動之餘，大會也舉辦許多活動，例如開幕當晚的歡迎酒會，表演之夜，以及划船活動等等。



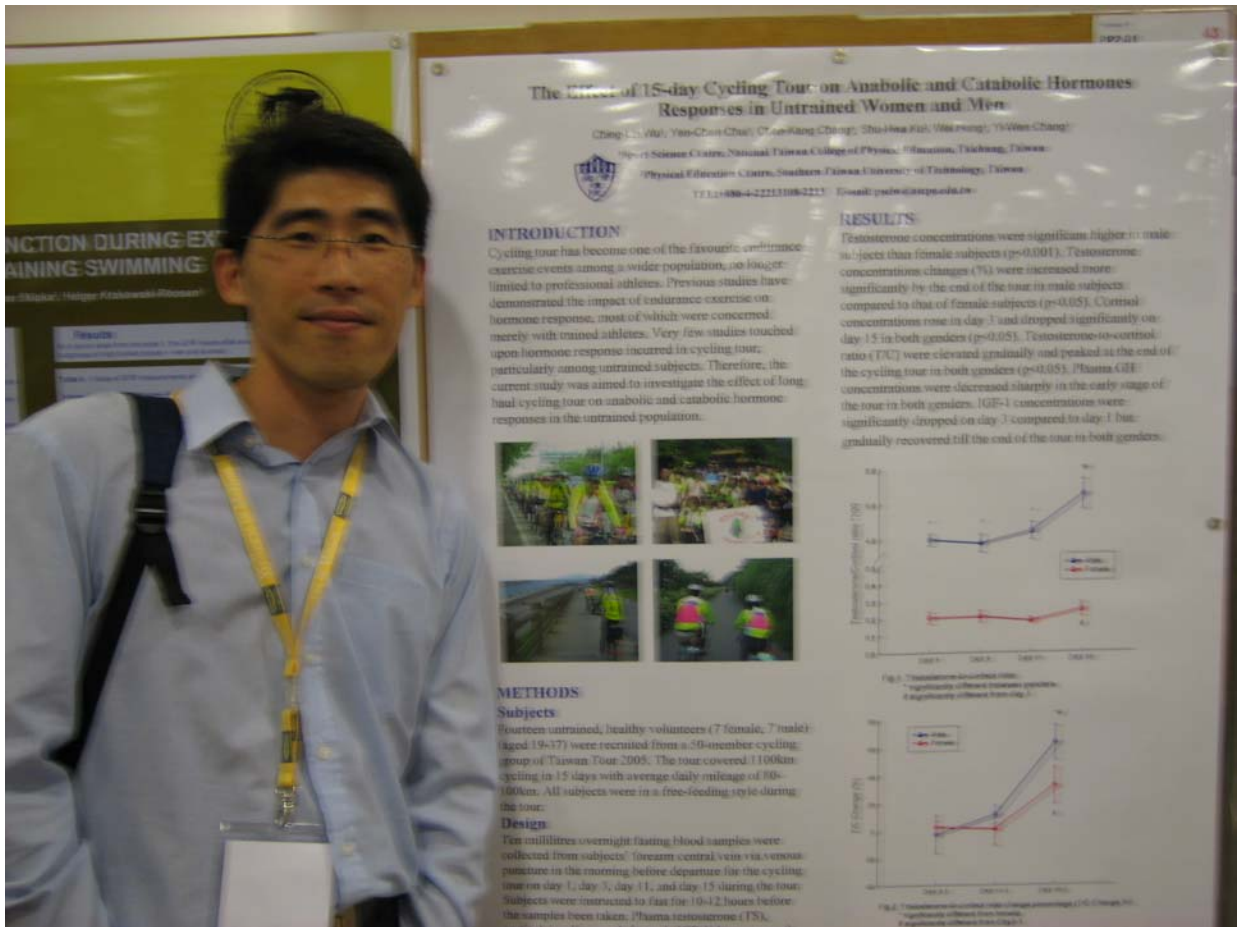
圖：晚宴之夜

四、學習的成果：重點發展領域

經過這幾天密集的聽講還有與其他國家學者的共同討論，發現真是受益良多，很難得有機會參與這麼國際性的會議，與這麼多從事運動科學研究的人才共聚一堂，從這些天的學習與腦力激盪所得的經驗之下，對於運動科學的趨勢發展，有下列幾點發現與感想：

1. 運動科學都不能僅從單一面向來討論，因為不管是運動生理、運動心理、運動生化、或者運動營養等等，每一個環節都需要詳加研究，而每一個研究者，都能夠在所興趣的領域上鑽研，並且藉由此種國際性的會議，來對談與交流，使每個研究者永遠走在尖端上。
2. 從整個會議上，仍不難看出目前仍然重視競技運動，即運動員的表現，因為其所呈現的研究結果能夠對運動員的表現產生直接的貢獻，另外一部分為健康管理，這包運動保健，運動傷害防護與治療，因此基本科目如運動生理學與運動力學都是此次會議發表的大宗。
3. 在運動科學的領域上，已經由巨觀的角度，往微觀方向移動，本次的研討會中已經有一部分的論文探討基因的部分，很多過去無法解答的問題，可能在藉由分子生物

的方法，而有所突破。



圖：本人海報發表

五、結語

此次會議規模龐大，會議本身提供了一個很好的學習，拓展視野與文化交流的機會，學者彼此之間的互動也非常的可貴，我這次參與不但遇到很多台灣過去的老師們，也有幸有機會與在英國的 Professor Michael Gleeson 再次對談，並且與過去在認識的國外學者再次聚首，透過彼此的在學術上交流與研究心得上的交換，獲得了很多寶貴的建議。

The Effect of 15-day Cycling Tour on Anabolic and Catabolic Hormones Responses in Untrained Women and Men

Ching-Lin Wu¹, Yen-Chen Chui¹, Chen-Kang Chang¹, Shu-Hwa Ku², Wei Hung¹, Yi-Wen Chang¹



¹Sport Science Centre, National Taiwan College of Physical Education, Taichung, Taiwan

²Physical Education Centre, Southern Taiwan University of Technology, Taiwan

TEL:+886-4-22213108-2213 E-mail: pschw@ntcp.edu.tw

INTRODUCTION

Cycling tour has become one of the favourite endurance exercise events among a wider population, no longer limited to professional athletes. Previous studies have demonstrated the impact of endurance exercise on hormone response, most of which were concerned merely with trained athletes. Very few studies touched upon hormone response incurred in cycling tour, particularly among untrained subjects. Therefore, the current study was aimed to investigate the effect of long haul cycling tour on anabolic and catabolic hormone responses in the untrained population.



METHODS

Subjects

Fourteen untrained, healthy volunteers (7 female, 7 male) (aged 19-37) were recruited from a 50-member cycling group of Taiwan Tour 2005. The tour covered 1100km cycling in 15 days with average daily mileage of 80-100km. All subjects were in a free-feeding style during the tour.

Design

Ten millilitres overnight fasting blood samples were collected from subjects' forearm central vein via venous puncture in the morning before departure for the cycling tour on day 1, day 3, day 11, and day 15 during the tour. Subjects were instructed to fast for 10-12 hours before the samples been taken. Plasma testosterone (TS), cortisol, insulin growth factor 1 (IGF-1) human growth hormone (GH) were analysed.



RESULTS

Testosterone concentrations were significant higher in male subjects than female subjects ($p < 0.001$). Testosterone concentrations changes (%) were increased more significantly by the end of the tour in male subjects compared to that of female subjects ($p < 0.05$). Cortisol concentrations rose in day 3 and dropped significantly on day 15 in both genders ($p < 0.05$). Testosterone-to-cortisol ratio (T/C) were elevated gradually and peaked at the end of the cycling tour in both genders ($p < 0.05$). Plasma GH concentrations were decreased sharply in the early stage of the tour in both genders. IGF-1 concentrations were significantly dropped on day 3 compared to day 1 but gradually recovered till the end of the tour in both genders.

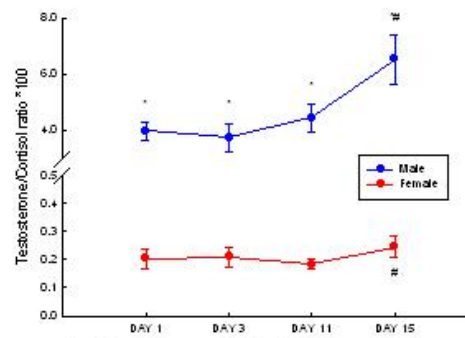


Fig 1. Testosterone-to-cortisol ratio.
* significantly different between genders.
significantly different from day 1.

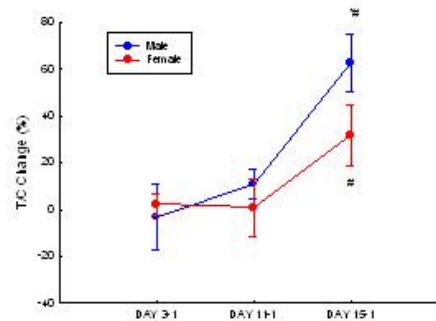


Fig 2. Testosterone-to-cortisol ratio change per stage (T/C Change %).
* significantly different from female.
significantly different from Day 3-1.

CONCLUSIONS

We concluded that the ratio of anabolic hormone to catabolic hormone increased steadily during the long haul cycling tour in both genders. However, higher increase has been observed in males than in females.