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Controlling energetic intake based on a novel logistic regression model for the metabolic syndrome in a Chinese population

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Abstract

The present study was designed to develop a novel method of energy calculation for controlling energetic intake in patients with the metabolic syndrome. Demographics and dietary data were recorded for 2582 obese subjects. Nutritional education was applied to all the patients. One year later, the data on age, sex, activity intensity coefficient, waistline, environmental temperature and BMI in subjects who lost ≥ 5 % body weight were entered into a multivariate logistic regression analysis model. Energy requirement was calculated from the results of multivariate logistic regression. Four hundred and thirty-four metabolic syndrome patients were then randomly divided into the treated group (216) and the control group (218). The energetic intake in the experimental group was controlled based on the new energy requirement model. The traditional energy exchange method was used in the control group. The independent factors predicting metabolic syndrome prognosis, such as age, sex, activity intensity coefficient, waistline, environmental temperature and BMI, were identified by multivariate logistic regression analysis. The energy requirement model was then constructed by logistic regression analysis. After 6 months of energetic intake control based on the new model, the parameters of the experimental group were significantly different from those of the controls (all $P<0{\cdot}05$): waistline, 89·65 (SD 5·54) *v.* 91·97 (SD 4·78) cm; BMI, 24·67 (SD 3·54) *v.* 25·87 (SD 2·65) kg/m^2; fasting blood glucose, 6·9 (SD 3·6) *v.* 8·7 (SD 4·6) mmol/l; 2 h PG, 8·7 (SD 5·7) *v.* 10·7 (SD 4·5) mmol/l; HbA_1c, 7·7 (SD 1·6) *v.* 8·9 (SD 2·6) %; homoeostasis model insulin resistance index, 3·14 (SD 1·62) *v.* 4·32 (SD 2·25). The new energy requirement model can effectively improve the clinical outcomes of controlling energetic intake in metabolic syndrome patients.

Key words: Metabolic syndrome: Energy requirement model: Energetic intake

The metabolic syndrome is a condition characterised by a cluster of several risk factors, including diabetes and raised fasting plasma glucose, abdominal obesity, dyslipidaemia and high blood pressure (BP)[1,2]. Moreover, epidemiological evidence indicates a link between the metabolic syndrome and several cancers, such as colon and breast cancers[3,4]. Because of its high prevalence, the metabolic syndrome has become one of the major public health challenges worldwide. In China, with a growing obesity population and an increase in unhealthy sedentary lifestyles, the prevalence of the metabolic syndrome is increasing steadily along with associated CVD[5–7]. In the United States, over 40 % of people older than 60 years of age have the metabolic syndrome, predisposing these individuals to type 2 diabetes (T2D) mellitus and CVD[8]. Several prospective epidemiological studies have shown that the metabolic syndrome is associated with increased morbidity or mortality for patients with CVD and stroke[9–11].

Each of the metabolic abnormalities contributing to the metabolic syndrome has a dietary relation, e.g. obesity, hypertension and dyslipidaemia. Foods rich in dietary fibre and/or with a low glycaemic index may help reduce the risk for the metabolic syndrome. The remarkable increase in the metabolic syndrome has increased the demand for novel approaches in diagnosis, prevention and treatment of this condition. It is generally believed that lifestyle changes focusing primarily on weight reduction are the first-line treatment for patients with the metabolic syndrome[12]. Energy requirement in a healthy population is defined as the amount of energy that an average individual would need to ensure stable body weight and composition along with good long-term health. The provision of adequate nutrition support will ensure that patients attain and maintain a desirable body weight and improve nutritional status[13]. The success of nutrition support relies on the provision of adequate energy and nutrients, which in

Abbreviations: BP, blood pressure; T2D, type 2 diabetes.

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turn is based on accurate estimates of energy requirements, avoiding any negative outcomes associated with under- or over-energy intake feeding[14,15].

There are limited reports estimating the energy requirement for metabolic syndrome patients, especially in Chinese populations[16]. In the present study, we developed a method for calculating energetic intake, based on a novel logistic regression model, to estimate the energy requirements of adult Chinese metabolic syndrome patients. We provided evidence supporting that the method was effective in controlling the metabolic syndrome.

Patients and methods

Patients of energy requirement equation set-up

In order to develop a novel energy calculation method for controlling energetic intake in patients with the metabolic syndrome, a nutritional education and intervention were applied to a total of 2582 patients (1527 men and 1055 women; aged 46·8 (SD 13·2) years) diagnosed as obese in Xi'an Central Hospital from December 1997 to December 2005. Demographic data (sex, age and disease history), height, waist size, body weight and physical activity (type and time) of the patients were recorded. Obesity diagnosis criteria used in the study were as follows: BMI $\geq$ 24 kg/m^2; waist circumference: male $\geq$ 90 cm, female $\geq$ 80 cm; TAG $\geq$ 1·7 mmol/l; fasting blood glucose $\geq$ 6·1 mmol/l. Patients with serious heart, brain, kidney and liver complication were excluded from the present study. The nutrition education and intervention included the following: (1) individual diets were assigned based on the energy intake before joining the experiment and the weight loss plan (loss of 1kg/month). As a reduction of 1 kg fat requires a reduction of intake of 29·29 MJ energy, subjects were counselled to reduce energetic intake by 0·976 MJ/d; (2) the energy composition of the diet was 15–20 % protein, 25–30 % fat and 50–60 % carbohydrate; (3) physical activity: aerobic exercise > 30 min/time, > 3 times/week. Self-reported information on medication, environmental temperature and diet (detailed recipes of three continuous days) was also collected. After a year of follow-up, the patients were divided into two groups: the group with weight loss $\geq$ 5 % (n 1080) and the group with no significant weight changes (< 5 %; n 1502). A balance test was performed in the two groups on medicine use and activity, and no significant differences were found between the two groups. The age, sex, activity intensity coefficient, waist circumference, environmental temperature and BMI of the patients in the weight loss $\geq$ 5 % group were used to set up the energy calculation formula in the present study. The present study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human patients were approved by the Ethics Committee of the Municipal Central Hospital of Xi'an City. Written informed consent was obtained from all the patients.

Patients of energy requirement equation application

Four hundred and thirty-four patients with the metabolic syndrome, diagnosed in Xi'an Central hospital from May 2006 to July 2008, were randomly divided into the treated group (216) and the control group (218). The energetic intake in the treated group was controlled based on the multivariate logistic regression energy requirement model. The traditional energy exchange method was used in the control group[17]. Diagnosis of the metabolic syndrome was based on the International Diabetes Federation definition[1]. The patients had central obesity (waistline; male $\geq$ 90 cm and female $\geq$ 80 cm) plus any two of the following: elevated TAG ($\geq$1·7 mmol/l or under specific medication for this lipid abnormality), reduced HDL-cholesterol (< 400 mg/l or 1·03 mmol/l in males, < 500 mg/l or 1·29 mmol/l in females, or under specific medication for this lipid abnormality), high BP (systolic BP > 130 or diastolic BP > 85 mmHg, or under specific medication for previously diagnosed hypertension) and high plasma glucose (fasting blood glucose $\geq$ 5·6 mmol/l or previously diagnosed T2D). Patients with severe organ dysfunctions, such as heart, brain, lung, liver and kidney dysfunctions, were excluded from the study. Written informed consent was obtained from the parent or legal guardian of each subject, and formal assent was obtained from each subject. The study protocol was reviewed and approved by the Ethics Committee of the Xi'an Central Hospital.

Clinical diagnosis and definitions

All the 434 participants were asked to fill out a structured questionnaire including name, contact information, age, sex, education and disease history. Twenty-four hour dietary recall was used to record the three consecutive days' dietary (type and quantity of food). All the patients received food/lifestyle education during the experimental period. After a 12 h fast, blood samples were obtained for the analyses of plasma glucose, TAG, HDL, glycosylated Hb-A and insulin levels. Plasma glucose concentration was determined by the glucose oxidase method using a Beckman Glucose Analyzer II (Beckman Instruments, Fullerton, CA, USA). Plasma insulin was measured with a commercial RIA kit (Coat-A-Count Insulin Kit, Diagnostic Products, Los Angeles, CA, USA). HbA_1C was measured by the HPLC method described previously[18]. TAG and HDL were measured by the enzymatic method using a Hitachi-7600 analyzer (Hitachi Limited, Tokyo, Japan). The homoeostasis model insulin resistance index was calculated using the following formula: fasting glucose (mmol/l) × fasting insulin (μIU/ml)/22·5[19], where 1 pmol insulin = 6965 μIU. Height, body weight, waist size and BP

were measured. BMI was calculated by dividing body weight (kg) by the square of height (m).

Data analysis

Multivariate logistic regression analysis was performed on the energy requirement (dependent variable) with independent variables. Time comparison has been made to show the difference within the treated and control groups before and after the experiment; and treatment comparison was made for the difference between the treated and control groups, both sorts of comparisons were evaluated by *t* test. Age and course of disease differences between the treated and control groups were also evaluated by *t* test. Sex, education and occupation differences between the treated and control groups were evaluated by χ^2 test. $P<0{\cdot}05$ was considered to be statistically significant.

Results

Energy requirement equation

After 1 year of nutritional education, those with a loss of body weight ≥ 5% were collected in a multivariate logistic regression analysis. Energy requirement (MJ) was used as the dependent factor, and independent variables, such as age, sex (female, 0; male, 1), activity intensity index (0, low physical job; 1, medium physical job), waist circumference (cm), environmental temperature of work place (0, 10–30°C; 1, < 10°C or > 30°C) and BMI (kg/m^2), were entered into the model. It was revealed that age (OR − 0·025, 95% CI −0·026, 0·023), activity intensity index (OR 0·215, 95% CI 0·162, 0·267), waist circumference (OR − 0·006, 95% CI −0·010, 0·003), environmental temperature (OR 0·342, 95% CI 0·241, 0·443), BMI (OR − 0·268, 95% CI −0·284, 0·252) and sex (OR 0·623, 95% CI 0·568, 0·678) were independent risk factors for the energy requirement. There were in total 1080 patients with a weight loss ≥ 5%, and all these patients were included in the equation. Energy requirement (y) was created from the results of multivariate logistic regression as follows:

$$y = 13{\cdot}5 - 0{\cdot}025x_1 + 0{\cdot}215x_2 - 0{\cdot}006x_3 + 0{\cdot}342x_4 - 0{\cdot}268x_5 + 0{\cdot}623x_6,$$

where x_1, x_2, x_3, x_4, x_5 and x_6 represent age, activity intensity index, waist circumference, environmental temperature, BMI and sex, respectively. Energy requirement was limited from 4·18 to 8·79 MJ.

Baseline characteristics

Baseline demographic characteristics of all 434 subjects included in the experiment are shown in Table 1. The baseline parameters, including age, sex, education and disease history, were comparable between the two groups (Table 1, $P>0{\cdot}05$).

Table 1. Demographic data of the patients (Mean values and standard deviations)

	Treated (*n* 216)		Control (*n* 218)	
	Mean	SD	Mean	SD
Age (years)	54	7	66	5
Sex (M/F)	135/81		133/85	
Course of disease (years)	4·4	3·9	4·6	3·5
Education (C/H/P)	33/131/52		39/128/51	
Occupation (W/B)	112/104		124/94	

M, male; F, female; C, college; H, high school; P, primary school; W, white-collar job; B, blue-collar job.

Change in BMI and waist circumference of patients

The BMI of the treated group and the control group before the experiment was 27·16 (SD 3·45) and 26·98 (SD 2·76) kg/m^2, respectively. There was no significant difference in BMI between the two groups ($P>0{\cdot}05$). No significant changes in the BMI of the control group were observed (26·25 (SD 2·83) and 25·87 (SD 2·65) kg/m^2, respectively, $P>0{\cdot}05$) 3 and 6 months after the experiment, compared to that of before the experiment. In contrast, in the same period, the BMI of the treated group was significantly decreased (25·46 (SD 2·35) and 24·67 (SD 3·54) kg/m^2, respectively, $P<0{\cdot}05$). The BMI of the treated group was significantly lower than that of the control group 3 and 6 months after the experiment ($P<0{\cdot}05$).

There was no significant difference in waist circumference between the treated group and the control group before the experiment (93·75 (SD 4·76) and 92·89 (SD 5·76) cm, respectively, $P>0{\cdot}05$). The waist circumference of the control group did not significantly change (92·62 (SD 3·82) and 91·97 (SD 4·78) cm, respectively, $P>0{\cdot}05$) 3 and 6 months after the experiment, compared to that of before the experiment, but that of the treated group significantly decreased (90·24 (SD 5·83) and 89·65 (SD 5·54) cm, respectively, $P<0{\cdot}05$). The waist circumference of the treated group was significantly smaller than that of the control group 3 and 6 months after the experiment (Table 2).

The change in blood glucose, HbA_1C and homoeostasis model insulin resistance index

There was no significant difference in baseline fasting blood glucose between the treated group and the control group before the experiment (10·6 (SD 3·8) and 10·0 (SD 4·8) mmol/l, respectively, $P>0{\cdot}05$). The fasting blood glucose of the treated group was significantly decreased to 8·6 (SD 3·5) mmol/l ($P<0{\cdot}05$) and 6·9 (SD 3·6) mmol/l ($P<0{\cdot}05$), respectively, 3 and 6 months after the experiment, compared to that of before the experiment. The fasting blood glucose of the treated group was significantly

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Table 2. Obesity-related parameters, blood pressure, blood glucose (BG) and homoeostasis model insulin resistance index (HOMA-IR) before and after the education‡

(Mean values and standard deviations)

Group	Time	BMI (kg/m^2) Mean	SD	Waistline (cm) Mean	SD	DBP (mmHg) Mean	SD	SBP (mmHg) Mean	SD	FBG (mmol/l) Mean	SD
Treated	Before experiment	27·16	3·45	93·75	4·76	131	12	83	11	10·6	3·8
	3 Months after treatment	25·46*†	2·35	90·24*†	5·83	129	19	79	08	8·6*†	3·5
	6 Months after treatment	24·67*†	3·54	89·65*†	5·54	128	9	76	14	6·9*†	3·6
Control	Before experiment	26·98	2·76	92·89	5·76	132	16	81	12	10·0	4·8
	3 Months after treatment	26·25	2·83	92·62	3·82	131	13	77	18	9·3	3·4
	6 Months after treatment	25·87	2·65	91·97	4·78	129	17	78	13	8·7†	4·6
		2 h BG (mmol/l)		HbA_1C (%)		HDL-C (mmol/l)		TAG (mmol/l)		HOMA-IR	
Treated	Before experiment	13·6	5·2	10·5	1·4	1·33	0·43	2·75	0·95	4·62	1·89
	3 Months after treatment	9·5*†	5·2	8·8*†	3·5	1·31	0·33	2·89	1·63	3·21*††	1·47
	6 Months after treatment	8·7*†	5·7	7·7*†	1·6	1·34	0·30	2·65	0·78	3·14*††	1·62
Control	Before experiment	12·9	4·8	10·7	3·0	1·28	0·36	2·69	1·42	5·02	2·92
	3 Months after treatment	11·3	5·2	9·6	3·3	1·21	0·63	2·28	1·78	4·72	1·43
	6 Months after treatment	10·7†	4·5	8·9†	2·6	1·30	0·29	2·26	1·53	4·32	2·25

DBP, diastolic blood pressure; SBP, systolic blood pressure; FBG, fasting blood glucose; HDL-C, HDL cholesterol.
Mean values were significantly different between the treated and control groups (treatment comparison): *$P<0·05$.
Mean values were significantly different within the treated and control groups before and after the experiment (time comparison): †$P<0·05$, ††$P<0·01$.
‡ P means statistically significant differences by t test.

lower than that of the control group 3 and 6 months after this experiment ($P<0·05$; Table 2).

There was no significant difference in baseline blood glucose concentration 2 h after food between the treated group and the control group before the experiment (13·6 (SD 5·2) and 12·9 (SD 4·8) mmol/l, respectively, $P>0·05$). The value of the control group did not significantly change (11·3 (SD 5·2) mmol/l, $P>0·05$) 3 months after the experiment, compared to that of before the experiment, but significantly decreased at 6 months (10·7 (SD 4·5) mmol/l, $P<0·05$). At 2 h after food, blood glucose of the treated group was significantly lower than that of the control group 3 and 6 months after the experiment, as 9·5 (SD 5·2) mmol/l ($P<0·05$) and 8·7 (SD 5·7) mmol/l ($P<0·05$), respectively (Table 2).

There was no significant difference in HbA_1C between the treated group and the control group before the experiment (10·5 (SD 1·4) and 10·7 (SD 3·0) %, respectively, $P>0·05$). The HbA_1C of the control group did not significantly change (9·6 (SD 3·3) %, $P>0·05$) 3 months after the experiment, compared to that of before the experiment, but significantly decreased at 6 months (8·9 (SD 2·6)%, $P<0·05$). The HbA_1C of the treated group was significantly lower than that of the control group 3 and 6 months after the experiment, as 8·8 (SD 3·5) % ($P<0·05$) and 7·7 (SD 1·6) % ($P<0·05$), respectively (Table 2).

There was no significant difference of homoeostasis model insulin resistance index between the treated group and the control group before the experiment (4·62 (SD 1·89) and 5·02 (SD 2·92), respectively, $P>0·05$). The homoeostasis model insulin resistance index of the control group did not significantly change 3 and 6 months after the experiment, compared to that of before the experiment (4·72 (SD 1·43) and 4·32 (SD 2·25), respectively, $P>0·05$). The homoeostasis model insulin resistance index of the treated group was significantly lower than that of the control group 3 and 6 months after the experiment, as 3·21 (SD 1·47) ($P<0·05$) and 3·14 (SD 1·62) ($P<0·05$), respectively (Table 2).

Change in energetic intake

As shown in Table 3, there was no significant difference in energetic intake between the treated group and the control group before the experiment (7·7 (SD 1·8) and 7·8 (SD 2·0) MJ, respectively, $P>0·05$). The energetic intake of the control group was 7·4 (SD 1·5) and 7·3 (SD 1·6) MJ 3 and 6 months after the experiment, which did not significantly change compared to that of before the experiment ($P>0·05$). The energetic intake of the treated group was significantly decreased, compared to that of before the experiment ($P<0·05$), and was significantly lower than that of the control group 3 and 6 months after the experiment, as 6·5 (SD 1·0) MJ ($P<0·05$) and 7·0 (SD 1·2) MJ ($P<0·05$), respectively.

At 3 months after the experiment, the carbohydrate intake of the treated group consisted of 55 (SD 6) % of the total energy intake, which was significantly higher than that of the control group (51 (SD 6) %, $P<0·05$); the fat intake per day of the treated group was 54 (SD 16) g, which was significantly lower than that of the control group (72 (SD 24) g, $P<0·05$); the fat intake of the treated group consisted of 31 (SD 7) % of the total energy intake, which was significantly lower than that of the control group (37 (SD 10) %, $P<0·05$). There was no significant difference in other parameters.

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Table 3. Energy and nutrient intake in metabolic syndrome patients‡
(Mean values and standard deviations)

Group	Time	Energy (MJ)		Carbohydrate (g)		Carbohydrate (%)			
		Mean	SD	Mean	SD	Mean	SD		
Treated	Before experiment	7·7	1·8	235	68	52	8		
	3 Months after treatment	6·5††**	1·0	212	41	55*	6		
	6 Months after treatment	7·0†	1·2	229	47	55	7		
Control	Before experiment	7·8	2·0	240	66	52	10		
	3 Months after treatment	7·4	1·5	224	57	51	6		
	6 Months after treatment	7·3	1·6	235	64	54	9		
		Protein (g)		Protein (%)		Fat (g)		Fat (%)	
Treated	Before experiment	59	15	13·0	2·0	71	25	35	8
	3 Months after treatment	52	14	13·5	2·5	54††**	16	31†**	7
	6 Months after treatment	57	14	13·5	2·5	59	18	32	7
Control	Before experiment	58	17	12·3	2·3	76	30	36	10
	3 Months after treatment	53	12	12·1	2·5	72	24	37	10
	6 Months after treatment	57	12	13·2	2·4	63	23	33	9

Mean values were significantly different between the treated and control groups (treatment comparison): *$P<0·05$, **$P<0·01$.
Mean values were significantly different within the treated and control groups before and after the experiment (time comparison): †$P<0·05$, ††$P<0·01$.
‡ P means statistically significant differences by t test.

Discussion

In the present study, we developed a new method to calculate and to control the energy intake of metabolic syndrome patients. The modified diet based on the energy calculation was associated with an improvement of the metabolic syndrome. Therefore, the new energy intake calculation method may be an effective strategy for the treatment of the metabolic syndrome.

Insulin resistance is one of the fundamental metabolic defects that underlie the metabolic syndrome[20], and BMI and waist circumference can predict some metabolic disorders[21]. In the model we developed, BMI and waist circumference were included to calculate the energy demand. The modified diet, based on the equation that helps to improve insulin resistance, might have favourable effects on all features of metabolic defects. Ash *et al.*[22] investigated the effects of dietary prescriptions on weight management and glycaemic control in overweight men with T2D. They found that a dietary prescription of 6000–7000 kJ/d was effective in achieving a 6 % weight loss and improving glycaemic control. However, the method of implementation made no difference to the outcomes between groups at 12 weeks or 18 months[22]. Toobert *et al.*[23] tested a comprehensive lifestyle self-management programme (Mediterranean low-saturated fat diet, stress management training, exercise, group support and smoking cessation) in reducing cardiovascular risk factors in postmenopausal women with T2D. The results of the study revealed significantly greater improvements in the treated group, compared with the control group receiving conventional care on HbA_1C, BMI, plasma fatty acids and quality of life at the 6-month follow-up. Patterns favouring intervention were seen in lipids, BP and flexibility, but did not reach statistical significance[23].

The main results of the present study performed in metabolic syndrome patients were similar to the one previously reported. The new energy intake calculation method appeared to improve glucose metabolism. Modified energy intake significantly decreased the levels of fasting blood glucose and HbA_1C. The significantly reduced levels of HbA_1C and fasting blood glucose clearly indicated that the method improved glucose metabolism in those patients[24]. Dyslipidaemia, particularly for high levels of serum NEFA, is a critical factor for insulin resistance and contributes to the metabolic syndrome and the pathogenesis of T2D and obesity[25]. We also evaluated the impact of the energy calculation method on lipid metabolism. The energy intake calculation method did not significantly influence serum TAG and HDL; improvement in both systolic and diastolic BP was not seen, which may be due to a relative short observation period in the present study. The limitation of the present study was the inability to follow-up for a longer period. It was reported that it is important to combine intensive initial intervention with regular participant contact and structured dietary protocol to maximise energy restriction. Without regular follow-up, improvements cannot be maintained[22].

The energy requirement values calculated from the equation of the present study were negatively related to the BMI values. However, almost all the existing energy requirement models, such as those of Harris-Benedict (1919), Henry and Schofield, were established on the base of normal BMR. The BMR values calculated from those equations are positively correlated with body weight[26]. Daly *et al.*[27] confirm that the Harris-Benedict equations overestimate BMR by about 10–15 %. The Italian group reported 3388 BMR data points from a total of 7173 values in the Schofield database, indicating a higher BMR/kg in the studied Italians. BMR studies conducted thus far

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have shown that the predicted values using the FAO/WHO/United Nations University equations overestimate BMR in Asian and Chinese subjects[28]. Henry *et al.*[16] suggest that, when applying the Oxford equation for BMR, a reduction in total energy requirement ranges from 396 kJ (95 kcal) to 841 kJ (201 kcal)/d for males, and 202 kJ (48 kcal) to 863 kJ (206 kcal)/d for females. The patients suffering from the metabolic syndrome always suffered from obesity; therefore, it is plausible to have the value of our equation lower than that of existing models.

We conclude that in metabolic syndrome patients, energy restriction is a critical factor to achieve weight loss and improvements in clinical outcomes. The energy calculation method we developed might be effective in reducing metabolic syndrome risks. Our model also showed that the energy calculated from this model was negatively correlated with the BMI and waistline value. Then, it will be helpful in preventing insulin resistance and metabolic syndrome development among the general population. Further research is required to determine the long-term clinical benefits associated with weight loss.

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