Community-based Study on Summer-Winter Difference in Insulin Resistance in Kin-Chen, Kinmen, Taiwan

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Background: The aim of this community-based study was to explore the summer-winter difference in insulin resistance in Kin-Chen, Kinmen.

Methods: A total of 2,412 residents aged 40 and over was enrolled in a mass survey in Kin-Chen, Kinmen, by the Yang-Ming Crusade, a volunteer organization of well-trained medical students from National Yang-Ming University. All participants were investigated in winter (first phase, January and February, before Chinese New Year) and summer (secondary phase, July and August) in 2002. Structured questionnaires, demographic and physical data, lifestyle, and blood chemistry parameters were collected.

Results: Higher levels of fasting insulin, HOMA-insulin resistance and triglycerides, but lower levels of high-density lipoprotein cholesterol were found in summer than in winter. The prevalence of metabolic syndrome was higher in summer than in winter, with differences of 7.7% in both genders ($p = 0.0092$ in men, $p = 0.0037$ in women). Body mass index (BMI), age and physical activity were significantly correlated with metabolic syndrome. After controlling for BMI and other risk profiles, summer was independently and positively associated with fasting insulin and insulin resistance regardless of metabolic syndrome.

Conclusion: Fasting insulin, insulin resistance and prevalence of metabolic syndrome were higher in summer than in winter. BMI and season were 2 major determinants of the variation in fasting insulin. The contextual impacts of seasonal variation in shaping metabolic syndrome or insulin resistance in populations need to be reemphasized. [*J Chin Med Assoc* 2008;71(12):619–627]

Key Words: insulin resistance, lipids, metabolic syndrome X, season, triglycerides

Introduction

Several studies have reported increased cardiac events and mortality during the winter months.[1–3] The metabolic syndrome refers to a cluster of metabolic abnormalities, including hypertriglyceridemia, low high-density lipoprotein cholesterol (HDL-C), glucose intolerance, central obesity, and hypertension. These metabolic abnormalities are also known cardiovascular disease risk factors,[4] and the most accepted and unifying hypothesis to describe the pathophysiology of the metabolic syndrome is insulin resistance.[5]

There are few reports[6] regarding the association between seasonal variation and the metabolic syndrome, but there are several studies[7–14] on the effects of season on insulin. Inconsistent findings were noted in these studies. Insulin resistance and triglyceride levels were higher during the summer in some studies,[7,8] while another study showed higher levels in winter than in summer,[9] and several studies showed no significant seasonal variation.[10–14] In previous work,[6] we found that the prevalence of metabolic syndrome in women was higher in the summer than in the winter (30.9% *vs.* 27.6%), but fasting insulin

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was checked only in Kin-Chen, a town in Kinmen county.

Kinmen (Quemoy) lies 2,310 m (< 1.5 miles) away from the southern mainland of China and is located at a latitude of approximately 24°C north.[15] It has a subtropical climate which shows seasonal variations in temperature (winter: 9.8–14.3°C, with a mean of 11.8°C; summer: 25–33°C, with a mean of 27°C). A series of topics including diabetes, hypertension and coronary heart disease has been studied.[16–18] The purpose of this study was to explore the summer-winter difference in insulin sensitivity, insulin resistance and its risk factors in Kin-Chen, Kinmen.

Methods

Study sample

The characteristics of the target population and the methods used in the series of Kinmen studies have been previously reported.[16–18] This study focused on those residents over 40 years old in Kin-Chen, the largest township in Kinmen.[16–18] They were studied in different villages during winter (January and February, before Chinese New Year) and summer (July and August) in 2002. A total of 4,545 residents were eligible for screening and were surveyed by the Yang-Ming Crusade, a volunteer organization of well-trained medical students from National Yang-Ming University.[16–18] Demographic and clinical parameters including body height, body weight, body mass index (BMI, weight/height^2), waist-to-hip ratio, systolic and diastolic blood pressures were acquired with face-to-face interviews and structured questionnaires. Ethics approval was obtained from the Yu-Li Veterans Hospital Ethics Committee, and all subjects gave written informed consent.

Definitions of variables

Blood samples were obtained after overnight fasting for determination of plasma glucose, serum uric acid, lipid, and other biochemical measurements, as was previously reported.[16–18] Fasting plasma glucose was determined using the hexokinase-glucose-6-phosphate dehydrogenase method using a glucose (HK) reagent kit (Gilford, Oberlin, OH, USA). Waist girth was measured at the minimum circumference. Serum insulin was measured by radioimmunoassay (Incstar, Stillwater, OH, USA). The detection limit was 2.05 μU/mL. The intra- and interassay coefficients of variation were 7.4% and 9.1%, respectively. "Frequent" physical activity was defined as exercise > 1 time/week or high daily activity (including work); all others were categorized as "infrequent". Three consecutive blood pressure readings, separated by at least 5 minutes, were taken from the right arm from seated subjects. According to the Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III) criteria[4] and the revised definition according to Asia-Pacific criteria for abdominal obesity,[19] the definition of metabolic syndrome is defined as having 3 or more of the following 5 criteria: high blood pressure (systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥ 85 mmHg); high fasting plasma glucose (≥ 110 mg/dL [6.05 mmol/L]); abdominal obesity (waist circumference > 90 cm in men and > 80 cm in women); hypertriglyceridemia (≥ 150 mg/dL [1.65 mmol/L]); low HDL-C (< 40 mg/dL [1.05 mmol/L] in men and < 50 mg/dL [1.30 mmol/L] in women).

We used the homeostasis model assessment (HOMA-IR) model to estimate the levels of insulin resistance in the population.[20] The estimate of insulin resistance obtained by HOMA-IR correlated with estimates obtained by use of the euglycemic clamp ($r = 0.88$, $p < 0.0001$).[20] The formula of the HOMA-IR model is as follows:[21]

$$[\text{Fasting insulin } (\mu U/mL) \times \text{fasting glucose } (mmol/L)]/22.5$$

Statistical analysis

All statistical analyses were performed using the SAS statistical package (SAS Inc., Cary, NC, USA). Data are presented as means and standard deviations for continuous variables and proportions for categorical variables. Two-sample t test and the χ^2 test were used in univariate analysis. To analyze the independent effect of summer-winter difference, multiple logistic regression (for the metabolic syndrome) and multiple linear regression (for log fasting insulin and log HOMA-IR) as a function of covariates, including age, gender, BMI, lifestyle and other risk profiles were assessed. A stepwise procedure was applied to do model selection.

Results

At the beginning of the study in 2000, 4,545 residents (2,296 men and 2,249 women) were eligible for screening, and a total of 2,412 residents participated in the study during 2000 to 2003. The overall response rate was 53.1%. After excluding those with missing data, 2,175 subjects with complete blood data and completed questionnaires were available for analysis.

Table 1 shows the demographic and lifestyle characteristics of the study subjects. Analysis revealed that

Table 1. Characteristics of the study population in winter and summer among residents aged ≥40 years in Kin-Chen, Kinmen, 2002

	Total (n=2,175)	Men			Women		
		Winter (n=658)	Summer (n=316)		Winter (n=834)	Summer (n=367)	
Continuous variables				p*			p*
Age (yr)	55.4±11.3	57.0±11.4	55.5±11.1	0.0531	55.3±11.5	52.7±10.5	0.0003
SBP (mmHg)	128.7±17.2	131.9±15.6	130.9±18.1	0.4236	128.3±15.8	122.4±19.6	<0.0001
DBP (mmHg)	80.8±10.6	83.4±10.0	84.2±11.3	0.2865	79.7±10.2	76.1±10.5	<0.0001
BMI (kg/m$^{2)}$	24.5±3.5	24.4±3.4	24.7±3.3	0.282	24.5±3.6	24.3±3.5	0.4673
Waist circumference (cm)	81.6±10.3	85.7±9.6	86.3±8.8	0.379	78.20±10.0	77.9±9.1	0.6024
FPG (mg/dL)	109.3±33.6	114.2±35.8	111.2±42.7	0.2919	107.8±25.5	102.5±35.5	0.0102
Triglycerides (mg/dL)	105.5±72.8	109.6±72.3	126.2±93.2	0.0059	91.6±54.8	111.5±83.0	<0.0001
Cholesterol (mg/dL)	205.4±39.0	211.4±39.0	193.8±33.8	<0.0001	210.6±40.3	193.5±34.9	<0.0001
Uric acid (mg/dL)	6.3±1.7	6.8±1.6	6.8±1.6	0.7219	5.9±1.6	5.7±1.6	0.103
HDL-C (mg/dL)	51.8±14.5	51.9±13.6	42.5±11.0	<0.0001	57.6±14.5	46.6±12.5	<0.0001
Fasting insulin (μU/mL)	13.6±8.8	12.9±9.1	15.8±8.4	<0.0001	12.7±8.9	15.2±8.8	<0.0001
HOMA-IR	3.8±3.6	3.8±3.6	4.6±4.6	0.0086	3.5±3.0	4.0±3.5	0.0134
Categorical variables (%)				$p^{\dagger}$			$p^{\dagger}$
Abdominal obesity‡	33.7	28.0	30.8	0.3678	37.2	38.1	0.7794
High blood pressure‡	51.6	59.7	58.4	0.6982	49.9	35.4	<0.0001
High fasting glucose‡	28.8	37.3	25.4	0.0002	28.3	17.5	<0.0001
Hypertriglyceridemia‡	17.6	20.0	25.3	0.0610	12.0	19.2	0.0011
Low HDL-C‡	33.7	15.5	45.4	<0.0001	31.8	60.4	<0.0001
Metabolic syndrome	24.3	22.1	29.8	0.0092	21.7	29.4	0.0037
Diabetes	11.6	13.7	13.0	0.7753	10.2	9.6	0.7420
Frequent physical activity	41.9	47.6	55.5	0.0267	35.2	35.2	0.9976
Smoking	16.3	34.0	34.7	0.8467	1.8	1.4	0.6269
Alcohol drinking	17.4	33.8	37.2	0.3124	3.4	2.8	0.6032
Menopause					64.3	58.5	0.0688

**t test; †χ^2 test; ‡according to ATP III criteria[4] and revised definition according to Asia-Pacific criteria for abdominal obesity.[19] SBP = systolic blood pressure; DBP = diastolic blood pressure; BMI = body mass index; FPG = fasting plasma glucose; HDL-C = high-density lipoprotein cholesterol; HOMA-IR = homeostasis model assessment of insulin resistance.*

1,492 subjects (68.6%) participated in the winter, and 683 subjects (31.4%) participated in the summer. Their mean age was 55.4 years (range, 40–89 years). The women who participated in winter were of an older age than those who participated in summer. The levels of triglycerides, fasting insulin and HOMA-IR were higher in summer than in winter, while cholesterol level was higher in winter than in summer for both genders. Waist-to-hip ratio, BMI, waist circumference, and uric acid had no significant summer-winter difference in both genders. The levels of systolic blood pressure, diastolic blood pressure, HDL-C and fasting plasma glucose were all higher in winter than in summer, particularly in women. On the other hand, the prevalence of hypertriglyceridemia, low HDL-C and metabolic syndrome were higher in summer than in winter, but high fasting plasma glucose was higher in winter than in summer in both genders. High blood pressure was higher in winter than in summer, particularly in women. There was no significant difference in the prevalence of abdominal obesity between summer and winter in both genders. Men engaged in less frequent physical activity in winter than in summer. The prevalence of smoking and alcohol drinking were not significantly different between summer and winter in both genders. We found no prominent significant association between menopause and season in women.

Table 2 presents the results of multiple logistic regression models for metabolic syndrome. For both genders, the winter season was negatively associated with metabolic syndrome. In contrast, the significantly protective effect of frequent physical activity for metabolic syndrome was only found in men, not in women. Frequent physical activity had no longer protective effect versus metabolic syndrome in the stratified analysis by gender. BMI and age were significantly correlated with metabolic syndrome in both summer and winter. Menopause was a significant risk factor for metabolic syndrome in women.

To control confounding effects, Table 3 presents the results of multiple linear regression models for log HOMA-IR, log fasting insulin, and stratified by metabolic syndrome, controlling for season, gender, biochemistry and other confounders. In participants without metabolic syndrome, the linear regression model showed that BMI, mean blood pressure, triglycerides and fasting plasma glucose were risk factors for fasting insulin, while winter was significantly negatively associated with fasting insulin. In participants with metabolic syndrome, the linear regression model showed

Table 2. Multiple logistic regression models on metabolic syndrome among residents aged ≥ 40 years stratified by season in Kin-Chen, Kinmen, 2002

Outcome / Variables*	Metabolic syndrome					
	All Participants		Participants in winter		Participants in summer	
	OR	95% CI	OR	95% CI	OR	95% CI
		All				
Season (winter *vs.* summer)	0.51	0.38, 0.67				
Age (yr)	1.05	1.04, 1.06	1.05	1.04, 1.06	1.05	1.03, 1.07
Gender (men *vs.* women)	NS	–	NS	–	NS	–
BMI (kg/m^2)	1.46	1.40, 1.53	1.48	1.39, 1.57	1.43	1.33, 1.55
Physical activity (frequent *vs.* infrequent)	0.73	0.56, 0.95	0.66	0.47, 0.92	NS	–
		Men				
Season (winter *vs.* summer)	0.57	0.38, 0.84				
Age (yr)	1.03	1.02, 1.05	1.03	1.01, 1.06	1.03	1.00, 1.06
BMI (kg/m^2)	1.45	1.35, 1.56	1.46	1.33, 1.59	1.42	1.27, 1.59
Physical activity (frequent *vs.* infrequent)	0.67	0.46, 0.97	NS	–	NS	–
		Women				
Season (winter *vs.* summer)	0.47	0.32, 0.71				
Age (yr)	1.05	1.03, 1.08	1.04	1.02, 1.07	1.08	1.05, 1.12
BMI (kg/m^2)	1.46	1.37, 1.56	1.48	1.36, 1.60	1.44	1.28, 1.61
Physical activity (frequent *vs.* infrequent)	NS	–	0.59	0.36, 0.96	NS	–
Menopause (yes *vs.* no)	2.17	1.23, 3.84	2.77	1.30, 5.88	NS	–

**Other variables of education, alcohol drinking, smoking, fish, seafood, meat and visceral food were not significant. OR = odds ratio; CI = confidence interval; BMI = body mass index; NS = not significant.*

Table 3. Multiple stepwise regression models for correlates of log fasting insulin and log HOMA-IR among the residents stratified by metabolic syndrome in Kin-Chen, Kinmen, 2000–2003 ($n = 2,175$)

Variables	Log HOMA-IR			Log fasting insulin								
	All participants			All participants			Participants without metabolic syndrome			Participants with metabolic syndrome		
	R^2	β*	*p*	R^2	β*	*p*	R^2	β*	*p*	R^2	β	*p*
Intercept*		−1.2363	<0.0001		0.9669	<0.0001		1.0705	<0.0001		1.4576	<0.0001
Season (winter *vs.* summer)	0.0214	−0.1538	<0.0001	0.0460	−0.2052	<0.0001	0.0493	−0.2149	<0.0001	0.0373	−0.1848	0.0003
Age (yr)		NS			NS			NS			NS	
Gender (men *vs.* women)		NS			NS			NS			NS	
BMI (kg/m^2)	0.1070	0.04792	<0.0001	0.1928	0.0474	<0.0001	0.1065	0.0407	<0.0001	0.0922	0.0465	<0.0001
SBP (mmHg)	0.0024	0.0028	0.003	0.0030	0.0027	0.0051	0.0021	0.0023	0.0171		NS	
HDL-C (mg/dL)	0.0036	−0.00232	0.0115	0.0052	−0.0027	0.0007		NS		0.0107	−0.0052	0.0223
Cholesterol (mg/dL)		NS			NS			NS			NS	
Triglycerides (mg/dL)	0.0126	0.0008	<0.0001	0.0213	0.0007	<0.0001	0.0292	0.0017	<0.0001		NS	
FPG (mg/dL)	0.3611	0.0099	<0.0001	0.0339	0.0023	<0.0001	0.0193	0.0024	<0.0001	0.0228	0.0020	<0.0001
Uric acid (mg/dL)	0.0013	0.0164	0.0128	0.0017	0.0131	0.0465		NS		0.0087	0.0284	0.0296
Smoking (yes or no)	0.0027	−0.0758	0.0095	0.0034	−0.0667	0.0154		NS		0.0084	−0.1435	0.0180
Alcohol drinking (yes *vs.* no)	0.0012	−0.06806	0.0018	0.0018	−0.0636	0.0189		NS			NS	
Physical activity (frequent *vs.* infrequent)		NS			NS			NS			NS	

**Intercept in regression model. HOMA-IR = homeostasis model assessment of insulin resistance; β = regression coefficient; R^2 = partial R^2; BMI = body mass index; SBP = systolic blood pressure; HDL-C = high-density lipoprotein cholesterol; FPG = fasting plasma glucose; NS = not significant.*

that BMI, uric acid and fasting plasma glucose were risk factors for fasting insulin, while winter, HDL-C and smoking were significantly negatively associated with fasting insulin. In all participants, BMI was the most important predictor for fasting insulin and explained 10–19% of the variance of fasting insulin. Seasonal effect was the second predictor for fasting insulin and explained 4% of the variance of fasting insulin. Fasting glucose and triglycerides explained 1–3% of the variance of fasting insulin. Mean blood pressure, cholesterol, HDL-C, smoking, alcohol drinking and physical activity explained less than 1% of the variance of fasting insulin. Table 3 also presents the same results of multiple linear regression models in all participants for log HOMA-IR; the most important predictor was fasting plasma glucose, which explained 36.1% of the variance of fasting insulin.

Discussion

The findings from this large-scale, homogeneous Chinese population revealed a higher prevalence of metabolic syndrome in summer than in winter. The results also indicated that summer was positively associated with fasting insulin and insulin resistance. On the other hand, BMI and season were 2 major determinants of the variation in fasting insulin. Fasting glucose, BMI and season were the 3 major determinants of the variation in HOMA-IR.

The seasonal variation in metabolic syndrome and insulin resistance may reflect seasonal patterns in obesity, diet and/or exercise,[5,22,23] and the duration of sunlight[24] or intrinsic biologic rhythms.[22,23] The dietary intake has been linked to individual components of metabolic syndrome[25] and dietary intakes rich in whole-grain foods have been linked to a lower prevalence of metabolic syndrome.[26]

There are no consistent results on the seasonal variation of fasting insulin as shown in Table 4.[7–14] The present study showed higher mean values of fasting insulin and insulin resistance in summer than in winter, and were consistent with a study in Chile.[7] The most plausible explanations for a seasonal variation in insulin sensitivity could be the cyclical changes in body composition that have been previously described[27] or modifications in the dietary intake due to the changing availability of certain food items.

Recent studies have assessed and shown that application of the ATP III metabolic syndrome criteria provides good specificity but low sensitivity in the identification of subjects with insulin resistance.[28–30] The validity of the use of insulin as a surrogate for insulin resistance remains to be established.[31] This drawback may partly explain the controversies about the relationship between insulin resistance/hyperinsulinemia and the metabolic syndrome.[32]

The associations of BMI, age and physical activity with metabolic syndrome and fasting insulin have been established in previous studies.[33–36] Aging is associated with glucose intolerance and insulin resistance,[33] and consequently with an increased level of insulin. In a logistic model, the significant age effect supported previous research evidence.[34]

The importance of obesity as a risk factor for several diseases, including type 2 diabetes and cardiovascular disease, is well documented.[32] It has been reported that body weight increases in winter, which is due to more food intake, less physical activity, and greater use of alcohol.[37–39] Results from the current community-based study were in agreement with those of previous studies.[37–39] A combination of weight loss and enhanced physical activity may reduce the incidence of metabolic syndrome.[5]

The relationship between blood pressure and insulin resistance is well established, and relates to several different mechanisms.[5,40,41] This study showed similar results in all participants.

Season may have an impact on the quantity and type of drinking. The mean number of drinks per week reaches a maximum in late winter and a minimum in summer.[42] In Kinmen, more beer is consumed in summer than in winter, as a Korean study reported.[42] Mild-to-moderate alcohol consumption is associated with a lower prevalence of the metabolic syndrome, with a favorable influence on lipids, waist circumference, and fasting insulin.[43] But in this study, there was a higher prevalence of the metabolic syndrome and insulin resistance in the summer. This inverse result needs further study. Similarly, chronic cigarette smokers have been shown to be insulin-resistant and dyslipidemic.[44] But in our study, we found that smoking was negatively associated with insulin. This issue also needs further study.

The prevalence of the metabolic syndrome increases with menopause and may partially explain the apparent acceleration in cardiovascular disease after menopause.[45] Menopause was found to be a significant risk factor for metabolic syndrome in women in this study. We found no significant association between HOMA-IR/fasting insulin and menopause in women. After adding menopause into the model, the seasonal effect on HOMA-IR and fasting insulin remained more or less the same.

Furthermore, BMI and season were 2 major determinants of the variation in fasting insulin. It remains

Table 4. Characteristics and findings of previous studies with respect to seasonal variation associated with insulin

Reference	Year	Study design	Sample	Fasting glucose*	Triglyceride*	Fasting insulin*	Seasonal variation of insulin
Fahlen et al[10] (Sweden)	1971	Retrospective case-control study	100, 4 groups for 4 seasons	W: 77.2 mg/100 mL (3.28) S: 64.6 mg/100 mL (2.02)	W: 151.4 mg/100 mL (11.39) S: 155.0 mg/100 mL (11.06)	W: 10.3 μU/mL (1.21) S: 14.7 μU/mL (2.2)	No
Gravholt et al[11] (Denmark)	2000	Cohort	10	No seasonal change		Jan: 2,108 pmol/L May: 2,240 pmol/L	No
Walker et al[12] (Scotland)	1997	Cross-section	105, 4 groups for 4 seasons	W: 4.8 mmol/L (0.1) S: 4.9 mmol/L (0.1)		W: 5.4 μU/L (0.6) S: 6.4 μU/L (1.5)	No
Donahoo et al[13] (America)	2000	Cohort	18	W: 82 mg/dL (2) S: 84 mg/dL (2)	W: 99 mg/dL (12) S: 87 mg/dL (8)	W: 7.7 μU/mL (1.0) S: 7.5 μU/mL (0.6)	No
Mavri et al[14] (Slovenia)	2001	Cohort	82, 2 groups	All Mar: 5.5 mmol/L Sep: 5.1 mmol/L	All Dec: 1.79 mmol/L Sep: 1.61 mmol/L	Dec: 11.3 μU/L Jun: 10.7 μU/L	No, but higher sugar and triglycerides in winter
Bunout et al[7] (Chile)	2003	Cohort	84 cases, 65 controls	No seasonal change	Higher in summer	Postprandial insulin increase in summer	Yes, insulin resistance and triacylglycerol levels higher in summer
Campbell et al[8] (England)	1975	Cohort	12	W: 47 mg/100 mL (2) S: 57 mg/100 mL (3)		W: 14 μU/mL (6) S: 15 μU/mL (5)	Yes, higher glucose/plasma insulin ratio in winter (higher insulin level in summer)
Asplund-Carlson et al[9] (Sweden)	1994	Cross-section	1,564	W: 4.55 mmol/L S: 4.47 mmol/L	W: 1.54 mmol/L S: 1.43 mmol/L	W: 8.15 μU/L S: 7.11 μU/L	Yes, serum triglycerides, insulin, and waist-hip ratio were lower in summer. Glucose, cholesterol and BMI did not vary significantly with season.

**Numbers in parentheses are standard deviations. W = winter; S = summer.*

interesting that the causes of metabolic syndrome or insulin resistance thus shifted from the environment as a whole to specific factors within the environment (biological mechanisms) and to the behaviors of individuals.[46,47] Based on the epidemiologic point of view, we therefore suggest that further studies are needed to discuss the roles of macro-level for seasonal effect and incorporate multiple level analysis technology in shaping metabolic syndrome or insulin resistance in populations.

Several possible limitations of this study should be considered when interpreting the results. The cross-sectional nature of the study limits the causal interpretation of its findings. Future longitudinal studies are needed to investigate the role of seasonal variation in the pathogenesis of insulin resistance and metabolic syndrome. Another limitation of this study is its lack of information about patient use of lipid-lowering drugs, which limits the interpretation of the seasonal differences of metabolic syndrome and insulin resistance.

In conclusion, insulin resistance, fasting insulin and the prevalence of metabolic syndrome were higher in summer than in winter. BMI and season were 2 major determinants of the variance in fasting insulin and insulin resistance, and were significantly associated with metabolic syndrome. Based on the epidemiologic point of view, the contextual impacts of seasonal variation in shaping metabolic syndrome or insulin resistance in populations need to be reemphasized. A combination of weight loss and enhanced physical activity in summer may reduce the prevalence of metabolic syndrome and insulin resistance.

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